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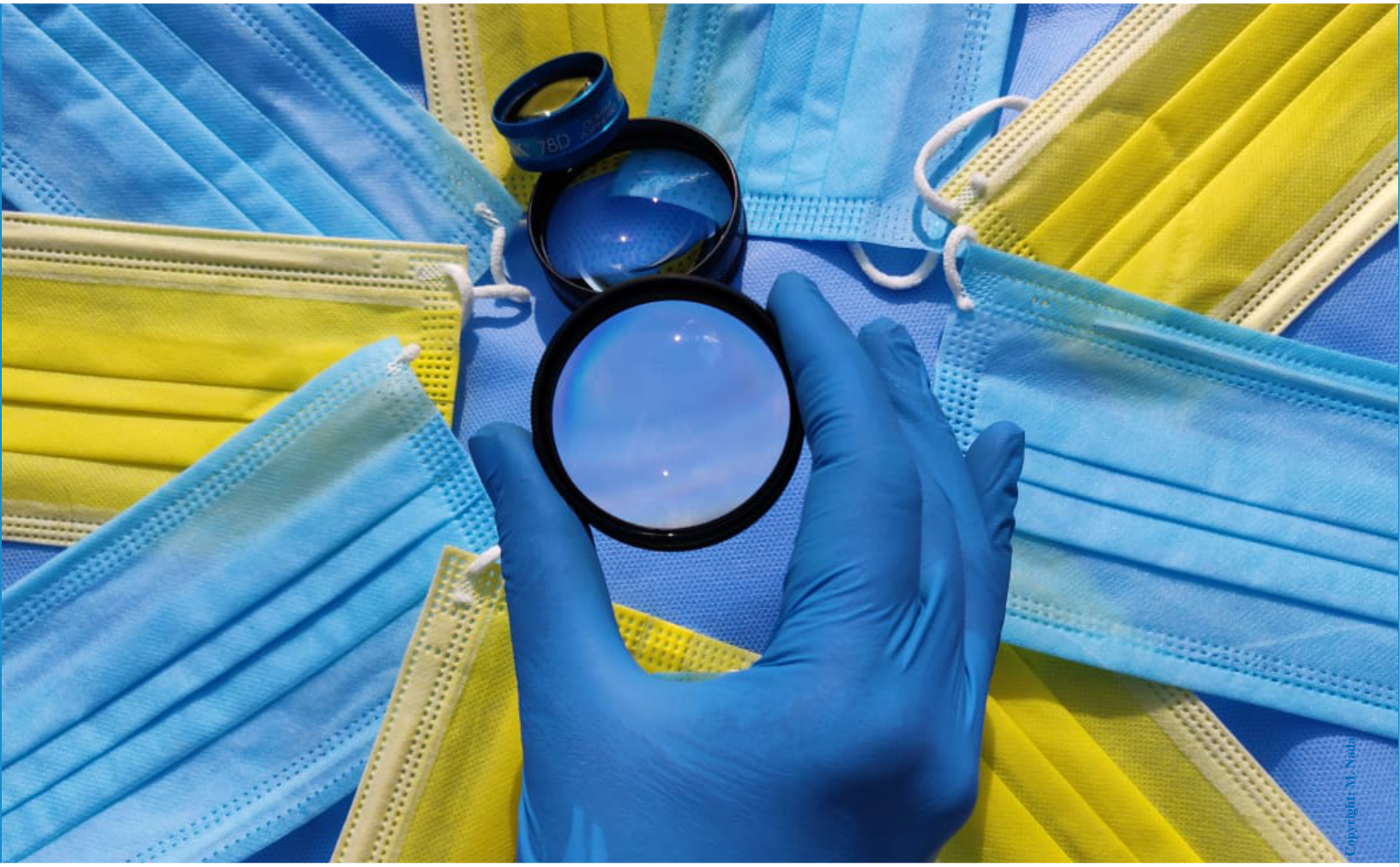

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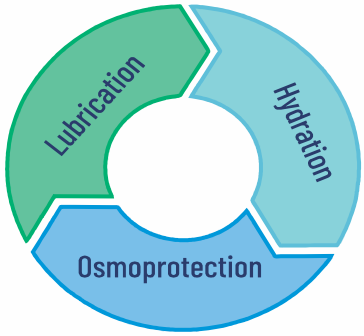

#Levocarnitine

1. Mike Christensen, OD, PhD, and Tressa Larson, OD, Artificial Tears: Looking Beneath the Surface, Review of Cornea and Contact Lenses, February 2016

*Source is Natural Producer of HA (streptococcus equi sub. zoopidemicus)

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Prof. MANISHA NADA
Editor-in-Chief, HJO
RIO, Pt. B.D.Sharma PGIMS, Rohtak
Resi: House No. 22/9J, Medical Campus, Rohtak-124001(Haryana)
Mobile 9896007158, E-mail : manisha_nada@rediffmail.com

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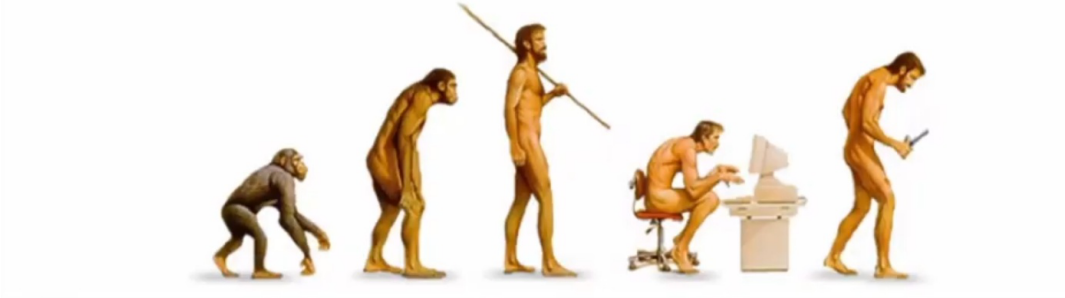

"It is not the strongest of the species that survive nor the most intelligent, but the one most responsive to change".
Charles Darwin

President's Message

Dear Friends,

Warm greetings!!

It is a matter of great happiness that new edition of our journal is being brought out. I congratulate editor Prof. Manisha Nada for her sincere and untiring efforts.

Corona epidemic engulfed the whole world in 2020. India has done well to contain this largely. We have done well at vaccine front with Covaxin and Covishield vaccines. Fifty lakhs health care workers have been vaccinated. Please take care of yourself for next two months and follow covid appropriate behaviour as role model for society.

Please join family benefit scheme of All India Ophthalmological Society and get 20-24 lakhs family benefit. Please join AIOS before you do that.

Our modern system of medicine is in danger because of Mixopathy. Indian Medical Association is leading an agitation. As a doctor of this system, please join Indian Medical Association(IMA), which is leading the agitation as well as fighting case in the Supreme Court.

The membership of Haryana Ophthalmological society has now grown to 959. There are still few, who are not members. I request all of you especially our office bearers to help in enrolling each one in our state.

Please send your proposal for next conference to be held in September-October. Please prepare yourself for taking responsibility as we plan to announce the elections soon.

Few city societies are doing work at local level. Please have your webinar as HOS events. You will get more audience and recognition at state levels. You will be rewarded from HOS.

With best wishes, regards

Jai Hind!

Prof (Dr.) Narinder Kumar Taneja
President

Secretary's Report

Respected Seniors and Dear Colleagues,

Warm greetings from the desk of honorary general secretary !

I congratulate Prof Manisha Nada for continuously carrying out the superb work of the journal of Haryana Ophthalmological Society.

We pay our homage to Dr. Mahesh Minocha from Ambala and Dr. K B Bhargav from Faridabad, whom we lost during these days.

Haryana Ophthalmological Society membership has grown to 959 and for this we thank Dr. Narinder Taneja and Dr. Rajan Gupta for their untiring efforts. We welcome all new members in the society.

We congratulate Dr. Saurabh Kamal, who was amongst the top three from North Zone for Talent Search Series by AIOS.

Tough big physical meetings were a big no due to the covid pandemic, but still webinars played a vital role in educating the recent advances of ophthalmology. Gurugram Ophthalmological Society, Rohtak Ophthalmological Society and Sirsa- Fatehabad Ophthalmological society; our affiliated distt societies need a special mention as they held all their webinars under the aegis of the state society.

Monsoon webinar was done by HOS in August 2020 which was attended by around 500 delegates. HOS did a special webinar for training of paramedical and OT staff all over the state. This webinar was attended by delegates from many states.

Finally, I would like to extend my thanks to all my friends, well wishers, senior colleagues and family members for their love and selfless moral support towards HOS concerned activities.

God bless HOS to live long !

Jai Hind!

Yours,

Prof. Dr Inder Mohan Rustagi
Hon General, Secretary HOS
Triveni Hospitals Pvt Ltd.,
207/13, Subhash Nagar, Old railway road,
Gurugram, HARYANA-122001
Mobile : 9810093892

Editorial

Respected Seniors and Dear Colleagues,

Warm Greetings!

The latest issue of Haryana Journal of Ophthalmology is now online.

The effects of 'Nightmare of 2020-Covid-19' are slowly waning and we have to accept to live with this virus with the confidence that scientific endeavours will bear fruits and we will be able to control this pandemic completely. Alongwith the control of the pandemic, a great concern for the ophthalmologists now is to face the consequences of neglected eyecare of patients. A large number of patients could not get adequate and timely treatment during the lockdown period. A huge number of patients getting intravitreal injections, retina laser treatment for tears and retinal vascular diseases could not turn up due to the risk of corona infection. Owing to the same risk involved, ophthalmologists also shirked seeing patients in the initial phase of lockdown. Due to absence of timely intervention, such patients are showing progression of the diseases, hence the blindness. Urgent changes need to be incorporated in ophthalmic care. This issue needs to be addressed in ophthalmology meetings. We cannot take it lightly as the risk of ensuing blindness is very high. Vision needs to be restored. Digital health innovations should be developed for management of a large number of patients. We have to risk stratify our patients so that patients with stable conditions are put on home-based self monitoring, whereas high risk patients get preferential appointments.

Ophthalmology practice is also coming back to normal and we have to embrace the change and move on. Protection of self and of our patients from corona infection requires that we adopt all set protocols and masks, social distancing and sanitization becomes a habit. Vaccines have been developed against this deadly disease and we all are witness to the world's largest vaccination drive in India. The vaccines have been found to be safe and emergency use authorisation has been given by competent authorities.

The ongoing pandemic has taught us so many things and the horizon is now wider and clearer with 'New Normal' vistas in all the fields. Training of postgraduates is prime function of institutes of ophthalmology. When the lockdown struck and OPDs were closed for elective work our residency training suffered a lot but as the time passed we could improvise and started teaching using online platforms where everyone could attend to the classes safely. Various conferences were initially postponed but digital media took the initiative and conferences could be organised online effectively. Live surgeries were transmitted with very good quality and fruitful discussions could be done. Even postgraduate examination were held with virtual patients and Objective Structured Clinical Examination. Initial inertia and scepticism to adapt to the newer digital solutions has now given way to a new era

full of exciting possibilities. Wet labs and Simulators are a way forward for hands on experience till the time we again come back to actual patient scenarios. There is a need to develop better simulators which are economical too so that this modality can be better utilised.

Medical education in India is all set for a giant leap with major change - National Medical Commission coming into force. Graduate and postgraduate medical education regulations have been changed with emphasis on an outcome driven education and acquisition of competencies.

These dynamic curricula would keep pace with the developments and advances around the world. It stresses on the importance of evidence-based medicine. The curriculum lays down the minimum requirements of infrastructure for ophthalmic services and education. Emphasis is given on research methodology, ethics, and professionalism as well as community ophthalmology. The structured curricula have been developed and their implementation to achieve desired outcomes is definitely going to be a big challenge. We hope it would be a boon to the postgraduates.

Its my proud privilege to have received the guest editorial from Dr. Santosh Honavar, a teacher par excellence, the best ever Editor of our esteemed Indian Journal of Ophthalmology and above all a great human being. I am overwhelmed. I thank him for being kind enough for contributing a very pertinent article on the current times 'Orbital manifestations of COVID-19'.

I am thankful to the President HOS, Prof. Narinder Kumar Taneja and Hon. Secretary, Prof. Inder Mohan Rustagi, for their ever available help.

I thank all the members of the editorial committee for the constant support. I also appreciate the authors who have generously contributed their scientific work.

With the blessings from The Almighty, our respected senior ophthalmologists, teachers and family members,

Let's all flourish together!

Long live HOS!!

Enjoy reading!

Best Regards,

Dr. Manisha Nada
Editor-in-Chief, HJO
Professor,
Vitreoretina Unit,
RIO, Pt. B. D. Sharma PGIMS,
Rohtak-124001 (Haryana)
E-mail : manisha_nada@rediffmail.com
Mobile : 9896007158

Guest Editorial

Orbital Manifestations of COVID-19

Mrittika Sen MD**, Santosh G Honavar** MD, FACS, FRCOphth
Centre for Sight, Road No 2, Banjara Hills, Hyderabad.

Introduction

The pandemic caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has had health implications of unprecedented magnitude. The COVID-19 illness can range from asymptomatic or mild flu like symptoms to severe respiratory distress. It is now known that it can have effects on almost all organs of the body including the cardiovascular, neurological and gastrointestinal systems. Ophthalmic manifestations are varied in terms of presentation, severity and timing. While conjunctivitis is the most common ocular condition seen in COVID-19 patients, other pathologies described in association with the SARS-CoV-2 infection include keratoconjunctivitis, episcleritis, blepharitis, retinal vein or artery occlusions, acute macular neuroretinopathy, paracentral acute middle maculopathy, vitritis, outer retinal abnormalities and reactivation of varicella zoster induced acute retinal necrosis and serpiginous choroiditis. Neuro-ophthalmic complications that have been reported include papillophlebitis, optic neuritis, Adie's tonic pupil, Miller Fisher syndrome, cranial nerve palsies, neurogenic ptosis and acute vision loss following cerebrovascular accident. Orbital manifestations are uncommon and there are only a few isolated case reports. It is important for ophthalmologists to have knowledge about the ophthalmic manifestations of the novel viral infection in order to suspect, diagnose, refer and treat the conditions with skills, machinery and drugs that we already possess. Here we present a brief review of the published literature on orbital pathologies seen in patients with COVID-19 infection.

There are not many orbital manifestations described but it is expected that their incidence will rise considering the interplay of comorbidities and treatment along with the infection itself. The case reports and series published show patients with a mean age of 50.2+/-43 (median 60, 12-76) years. 12/14 patients were males with nine being diabetics and six hypertensive patients. Asthma was notably present in eight patients. Five of these patients presented either with ophthalmic symptoms and were tested for COVID-19 on screening or presented concurrently with systemic symptoms of viral infection. The mean time of presentation from the development of COVID-19 symptoms was 15.8+/-13.8 (median 12, 2-42) days. 10/14 patients had moderate to severe disease.

Dacryoadenitis

Dacryoadenitis is the most common cause of a painful lacrimal gland mass in a healthy young adult and the most common cause of dacryoadenitis is viral infection. In the only reported case, the patient had a four-day history of eyelid swelling and pain. The patient had history of contact with COVID-19 infected patients and his antibody tests for IgM, IgG were positive. Other tests for autoimmune conditions, infectious diseases particularly tuberculosis, mumps, adenovirus, Epstein-Barr virus (EBV), herpes simplex virus (HSV) and herpes zoster virus (HZV) were all negative. A diagnosis of acute dacryoadenitis as a late complication of SARS-CoV-2 virus was made.[1] In the early stages of the disease, the virus can travel to the lacrimal gland via the lacrimal ductules or by direct hematogenous spread. Later, immunological response incited by the virus may affect the lacrimal gland producing inflammation. Acute dacryoadenitis responds well to systemic steroids.

Retro-orbital pain

Bilateral retro-orbital pain may be a prominent and even presenting symptom in patients with COVID-19 and can mimic conditions like dengue.[2] This case highlights the important fact that COVID-19 has features which are highly non-specific and can simulate a lot of other common conditions.

Orbital cellulitis and sinusitis

In the two cases reported by Turbin et al, two adolescent boys developed acute onset unilateral, progressive,

painful orbital swelling.[3] RT-PCR for COVID-19 was done as preoperative investigation. There were no symptoms of chronic sinus disease. Suggested mechanism is that COVID-19 induced upper respiratory congestion can compromise mucociliary clearance with secondary sinus obstruction and bacterial infection. Children have a relatively indolent course of disease with 56% of them being asymptomatic or having mild symptoms.[4] The superior ophthalmic vein thrombosis with facial vein extension may be a thrombotic complication of SARS-CoV-2.

In another case reported by Shires et al, a 76 year old man, diabetic, hypertensive with testicular cancer and COVID-19 developed spontaneously-draining orbital abscess and globe perforation necessitating enucleation with sinus debridement. Cultures grew methicillin resistant staphylococcus aureus (MRSA), Streptococcus constellatus and Peptoniphilus indolicus with negative blood cultures. Intraoperatively, an unusual finding was a highly avascular nasal mucosa. COVID-19 may predispose a patient to infection by bacteria not known to be found in the orbit like Peptoniphilus indolicus which is present in vagina and stomach. Orbital infection with this bacteria has not been reported previously. The avascularity was most likely because of thromboembolic complications of COVID-19.[5]

Mucormycosis

Mucormycosis is a life threatening, opportunistic infection and patients with moderate to severe COVID-19 are more susceptible to it because of compromised immune system with decreased CD4+ and CD8+ lymphocytes, associated comorbidities such as diabetes mellitus which potentiates both the conditions, decompensated pulmonary functions and the use of immunosuppressive therapy (corticosteroids) for the management. Literature shows that rhino-orbital cerebral (ROC) mucormycosis can present concurrently with COVID-19 infection in patients under treatment or diagnosed as a preoperative evaluation.[6-8] Mortality rate is as high as 50% even with treatment. In the series by the authors, all, except one patient presented after recovering from COVID-19.[9] 5/6 cases had received intravenous and/or oral steroids and all were diabetics. Almost 70% of rhino-orbital-cerebral mucormycosis is seen in patients with uncontrolled diabetes and most of them have ketoacidosis at the time of presentation. What is interesting to note in this series is that symptoms of rhino-orbital mucormycosis developed as late as 30-42 days after the diagnosis of COVID-19. High index of suspicion, early diagnosis with histopathological and microbiological evidence, appropriate management with antifungals and aggressive surgical debridement (FESS and orbital exenteration) can improve survival. The signs and symptoms of orbital mucormycosis are not different from those of mucormycosis in non-COVID-19 patients (Figure 1).

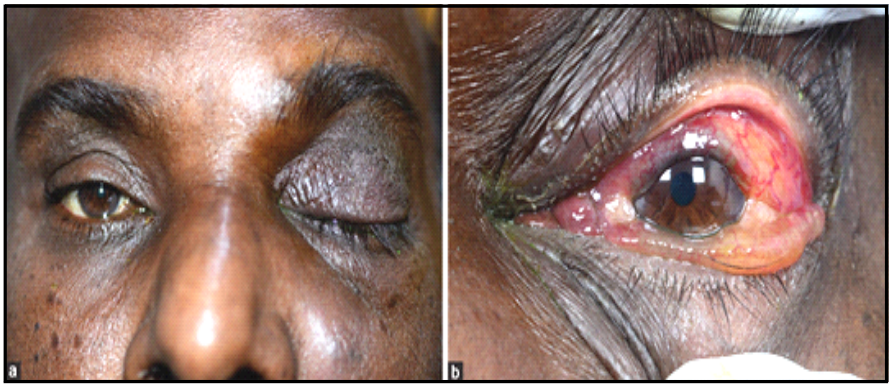

Fig.1 : Clinical picture of a 61-year-old male, who presented with (a) left periocular edema, complete ptosis and (b) proptosis, conjunctival congestion, and severe chemosis 17 days after COVID-19 infection. He had uncontrolled diabetes mellitus and received oral and intravenous steroids as part of COVID-19 management. The patient underwent aggressive paranasal sinus debridement and was initiated on intravenous amphotericin-B, with excellent response to treatment, with eye and life salvage.

Simple tests like vision, pupil, ocularmotility and sinus tenderness can be part of routine physical evaluation of a COVID-19 patient hospitalized with moderate to severe infection or diabetics with COVID-19 or those receiving systemic corticosteroids. A nasal swab for KOH mount and culture is a bedside procedure. Orbital exenteration for life-threatening infection is triaged as an urgent condition requiring surgery within 4-72 hours. Thus, appropriate surgery has to be undertaken with full personal protective equipment. Intravenous liposomal amphotericin B is started based on clinical suspicion or results of deep nasal swab. MRI is very useful to determine the extent of the disease and intracranial extension. Patients should also be made aware about the risks involved with the treatment of COVID-19 and the need for strict glycemic control. Development of unilateral facial or orbital pain, headache, periocular swelling or double vision or diminution of vision should prompt even the COVID-19 recovered patients to seek immediate medical attention. Since majority of the patients developed symptoms of mucormycosis after recovering from COVID-19, follow up of high risk COVID-19 patients for sequelae is imperative.9

Orbital histiocytic lesions

The authors have seen a case (unpublished) of a 77-year-old man with bilateral proptosis, eyelid swelling, enlarged lacrimal gland, orbital mass and cervical, axillary and mediastinal lymphadenopathy and maxillary sinusitis with history of COVID-19 infection six months ago. Incisional biopsy was done and histopathology with immunohistochemistry was suggestive of a benign histiocytic proliferative lesion, possibly Rosai-Dorfman disease. This is very unusual in an elderly individual and the infection with SARS-CoV-2 may have a role in with its influence on the immune system of the body.[10]

The prevalence of ophthalmic manifestations among COVID-19 patients ranges from 2-32%.[11] The causal relation with SARS-CoV-2 is yet to be established with certainty for any of these conditions. While the viral RNA has been identified in different parts of the eye, its replication and infectivity is not established. The transmission of the virus via eye secretions is being actively investigated. The orbital manifestations of COVID-19 can vary from intense retro-orbital pain to life threatening invasive mucormycosis. Orbital emphysema is seen as a complication in intubated patients receiving positive end expiratory pressure ventilation. As with other ophthalmic manifestations, direct effect of the virus, altered immune status, proinflammatory milieu and escalated coagulative profile play variable role in the pathogenesis.

There is an imminent need for establishing evidence-based guidelines for prophylactic use of antifungals in patients with high risk of rhino-orbito-cerebral mucormycosis diagnosed with COVID-19 who require corticosteroids. As we enter the phase of vaccination, a substantial proportion of the population has already been exposed to the SARS-CoV-2 virus, either in the form of overt clinical disease or contact with a patient diagnosed with COVID-19 with subclinical illness. Several countries of the world are experiencing resurgence of cases with mutated strains.

We can expect to see more manifestations of the disease in the eye and even clusters of similar cases. Ophthalmologists should be aware of the different manifestations and keep in mind the importance of asking specific history about COVID-19 infection, contact with infected person or related symptoms. COVID-19 should be included in the lists of causes of common ophthalmic pathologies especially when there is unusual presentation of a disease in an age group or population phenotype where it is not expected like histiocytic lesion in an elderly individual. Knowledge of many of these manifestations being the presenting features can help diagnose the infection early and limit the disease transmission. Tests like nasopharyngeal swab for RT-PCR, antibody titers for previous infection for patients with ophthalmic complaints or CT of the paranasal sinuses to look for sinusitis along with a scan for the chest in high risk patients by physicians treating COVID-19 cases need to be advised conscientiously and logically. Ophthalmologists are also encouraged to report cases seen in association with COVID-19 to add to the pool of knowledge on a global level.

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Address for Correspondence:
Dr Santosh G Honavar
MD, FACS, FRCOphth
Centre for Sight,
Road No 2, Banjara Hills, Hyderabad 500034
E-mail: santosh.honavar@gmail.com
Mobile: 8500452020

Current Topic

New MBBS curriculum- An overview

Jyoti Deswal MS,
Regional Institute of Ophthalmology, PGIMS, Rohtak

The Medical Council of India has revised the MBBS curriculum. The curriculum describes the course content, lays down the minimum requirements of infrastructure and mandates diagnostic and therapeutic procedures required for optimal training. The national ophthalmology undergraduate curriculum for India incorporates the required knowledge and skills for effective and safe practice and takes into account the specific needs of our country.The purpose is to standardize teaching at undergraduate level throughout the country so that there is benefit of achieving uniformity in teaching .This would result in creating competent doctors with appropriate expertise.

This curriculum and examination pattern will be applicable for MBBS course starting from academic year 2019-20 onwards. It has been published in The Gazette of India on 4th November, 2019 as follows:

No. MCI-34(41)/2019-Med./161726.—In exercise of the powers conferred by Section 33 of the Indian Medical Council Act, 1956 (102 of 1956), the Board of Governors in super-session of Medical Council of India with the previous sanction of the Central Government, hereby makes the following Regulations to further amend the "Regulations on Graduate Medical Education , 1997", which will now be called 'Regulations on Graduate Medical Education (Amendment), 2019.

The current undergraduate medical education curriculum focuses on competencies and outcomes and gives emphasis to skill development in all phases. This competency based undergraduate medical education programme is designed with a goal to create an "Indian Medical Graduate" (IMG) possessing requisite knowledge, skills, attitudes, values and responsiveness, so that she or he may function appropriately and effectively as a physician of first contact of the community while being globally relevant. Competency based learning would include designing and implementing medical education curriculum that focuses on the desired and observable ability in real life situations.

Broad Outline of training format

- There shall be a "Foundation Course" to orient medical learners to MBBS programme, and provide them with requisite knowledge, communication (including electronic), technical and language skills.
- The curricular contents shall be vertically and horizontally aligned and integrated to the maximum extent possible in order to enhance learner's interest and eliminate redundancy and overlap.
- Teaching-learning methods shall be learner centric and shall predominantly include small group learning, interactive teaching methods and case based learning.
- Clinical training shall emphasize early clinical exposure, skill acquisition, certification in essential skills; community/primary/ secondary care-based learning experiences and emergencies.
- Training shall primarily focus on preventive and community based approaches to health and disease, with specific emphasis on national health priorities such as family welfare, communicable and non-communicable diseases including cancer, epidemics and disaster management.
- Acquisition and certification of skills shall be through experiences in patient care, diagnostic and skill laboratories.
- The development of ethical values and overall professional growth as integral part of curriculum shall be emphasized through a structured longitudinal and dedicated programme on professional development including attitude, ethics and communication.

- Progress of the medical learner shall be documented through structured periodic assessment that includes formative and summative assessments. Logs of skill-based training shall be also maintained.
- Appropriate Faculty Development Programmes shall be conducted regularly by institutions to facilitate medical teachers at all levels to continuously update their professional and teaching skills, and align their teaching skills to curricular objectives.

Training period and time distribution

- Every learner shall undergo a period of certified study extending over 4 ½ academic years, divided into nine semesters from the date of commencement of course to the date of completion of examination which shall be followed by one year of compulsory rotating internship.
- Each academic year will have at least 240 teaching days with a minimum of eight hours of working on each day including one hour as lunch break.
- Teaching and learning shall be aligned and integrated across specialties both vertically and horizontally for better learner comprehension. Learner centered learning methods should include problem oriented learning, case studies, community oriented learning, self- directed and experiential learning.
- Didactic lectures shall not exceed one third of the schedule; two third of the schedule shall include interactive sessions, practicals, clinical or/and group discussions.
- The period of 4 ½ years is divided as follows:

Table 1: Time distribution of MBBS Programme & Examination Schedule

Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
							Foundation Course	I MBBS			
I MBBS								Exam I MBBS	II MBBS		
II MBBS								Exam II MBBS	III MBBS		
III MBBS Part I									Exam III MBBS Part I	Electives & Skills	
III MBBS Part II											
Exam III MBBS Part II	Internship										
Internship											

- Pre-Clinical Phase [(Phase I) - First Professional phase of 13 months preceded by Foundation Course of one month]: will consist of preclinical subjects – Human Anatomy, Physiology, Biochemistry, Introduction to Community Medicine, Humanities, Professional development including Attitude, Ethics & Communication (AETCOM) module and early clinical exposure, ensuring both horizontal and vertical integration.
- Para-clinical phase [(Phase II) - Second Professional (12 months)]: will consist of Para-clinical subjects namely Pathology, Pharmacology, Microbiology, Community Medicine, Forensic Medicine and Toxicology, Professional development including Attitude, Ethics & Communication (AETCOM) module and introduction to clinical subjects ensuring both horizontal and vertical integration.
- The clinical exposure to learners will be in the form of learner-doctor method of clinical training in all phases. The emphasis will be on primary, preventive and comprehensive health care.
- Clinical Phase – [(Phase III) Third Professional (28 months)]

(a) Part I (13 months) - The clinical subjects include General Medicine, General Surgery, Obstetrics & Gynaecology, Pediatrics, Orthopaedics, Dermatology, Otorhinolaryngology, Ophthalmology, Community Medicine, Forensic Medicine and Toxicology, Psychiatry, Respiratory Medicine, Radiodiagnosis & Radiotherapy and Anaesthesiology & Professional development including AETCOM module.

(b) Electives (2 months) - To provide learners with opportunity for diverse learning experiences, to do research/community projects that will stimulate enquiry, self directed experimental learning and lateral thinking

(c) Part II (13 months) - Clinical subjects include: Medicine and allied specialties (General Medicine, Psychiatry, Dermatology Venereology and Leprosy (DVL), Respiratory Medicine including Tuberculosis), Surgery and allied specialties (General Surgery,

Orthopedics [including trauma]), Dentistry, Physical Medicine and rehabilitation, Anaesthesiology and Radiodiagnosis), Obstetrics and Gynecology (including Family Welfare), Pediatrics and AETCOM module.

New teaching / learning elements

1. Foundation Course

Goal: The goal of the Foundation Course is to prepare a learner to study medicine effectively.

Objectives: The objectives are to:

(a) Orient the learner to:

- the medical profession and the physician's role in society.
- alternate health systems in the country and history of medicine
- medical ethics, attitudes and professionalism
- health care system and its delivery
- national health programmes and policies
- patient safety and biohazard safety
- universal precautions and vaccinations
- principles of primary care (general and community basedcare)
- the academic ambience

(b) Enable the learner to acquire enhanced skills in:

- Language
- Interpersonal relationships
- Communication
- Learning including self-directed learning
- Time management
- Stress management
- Use of information technology

(c) In addition to the above, learners may be enrolled in one of the following programmes which will be run concurrently:

-Local language programme

- English language programme

-First-aid

-Basic life support

-Computer skills

(d) Sports, leisure and extracurricular activities are also integral part of foundation course.

2. Learner-doctor method of clinical training (Clinical Clerkship)

- The Goals of the Learner Doctor program are to provide students experience with

a) Longitudinal patient care

b) Functioning as part of the Health Care team

c) "Hands on" care of patients in the inpatient and outpatient setting

- A designated faculty member will coordinate and facilitate the activity of the student
- The faculty member is (ideally) also responsible for the care of and decisions for the patient.
- Student will be part of the admission team on the admission day.
- He/ she will remain with admission team until 6PM on the admission day EXCEPT during designated class hours.
- Follow and document progress of assigned patient(s) during the course of hospitalization.
- Participate in procedures surgeries delivery etc of the assigned patient (based on the responsibility).
- Document patient encounters and learnings appropriate for level of training in a portfolio or annexure to log book
- Obtain feedback from supervising physician and other members of the health care team.

3. DOAP (Demonstration -Observation - Assistance - Performance) Sessions:

A practical session that allows the student to observe a demonstration, assists the performer, perform in a simulated environment, perform under supervision or perform independently.

Salient points pertaining to Ophthalmology

The emphasis of the curriculum is on skill based learning. The teachers have been instructed to adopt the following methods for teaching the competencies which already have been specified by the MCI:

1. Theory lectures – 30 hours.
2. Clinical postings (DOAP i.e. Demonstrate-Observe-Assist –Perform sessions)- 4 weeks (5 days a week) in second prof and 4 weeks (6 days a week in 3rd prof part-1)
3. Small group discussion (SGD)/ tutorials- 60 hours
4. Self directed learning (SDL)- 10 hours

Eligibility to appear for Professional examinations

(a)Attendance

1. Attendance requirements are 75% in theory and 80% in practical /clinical for eligibility to appear for the examinations in that subject. In subjects that are taught in more than one phase – the learner must have 75% attendance in theory and 80% in practical in each phase of instruction in that subject.
2. If an examination comprises more than one subject (for e.g., General Surgery and allied branches), the candidate must have 75% attendance in each subject and 80% attendance in each clinical posting.
3. Learners who do not have at least 75% attendance in the electives will not be eligible for the Third Professional - Part II examination.

(b) Internal Assessment

1. Regular periodic examinations shall be conducted throughout the course. There shall be no less than three internal assessment examinations in each Preclinical / Para-clinical subject and no less than two examinations in each clinical subject in a professional year.
2. An end of posting clinical assessment shall be conducted for each clinical posting in each professional year.
3. Day to day records and log book (including required skill certifications) should be given importance in internal assessment. Internal assessment should be based on competencies and skills.
4. Learners must secure at least 50% marks of the total marks (combined in theory and practical / clinical; not less than 40 % marks in theory and practical separately) assigned for internal assessment in a particular subject in order to be eligible for appearing at the final University examination of that subject. Internal assessment marks will reflect as separate head of passing at the summative examination.
5. Learners must have completed the required certifiable competencies for that phase of training and completed the log book appropriate for that phase of training to be eligible for appearing at the final university examination of that subject.

University Examinations

- Nature of questions will include different types such as structured essays (Long Answer Questions - LAQ), Short Answers Questions (SAQ) and objective type questions (e.g. Multiple Choice Questions - MCQ). Marks for each part should be indicated separately. MCQs shall be accorded a weightage of not more than 20% of the total theory marks.
- In subjects that have two papers, the learner must secure at least 40% marks in each of the papers with minimum 50% of marks in aggregate (both papers together) to pass.
- A candidate shall obtain 50% marks in University conducted examination separately in Theory and Practical (practical includes: practical/ clinical and viva voce) in order to be declared as passed in that subject.
- There shall be one main examination in an academic year and a supplementary to be held not later than 90 days after the declaration of the results of the main examination.
- A learner shall not be entitled to graduate after 10 years of his/her joining of the first part of the MBBS course.

Appointment of Examiners

(a) Person appointed as an examiner in the particular subject must have at least four years of total teaching experience as assistant professor after obtaining postgraduate degree in the subject in a college affiliated to a recognized/approved/permitted medical college.

(b) For the Practical/ Clinical examinations, there shall be at least four examiners for 100 learners,out of whom not less than 50% must be external examiners. Of the four examiners, the senior-most internal examiner will act as the Chairman and coordinator of the whole examination programme so that uniformity in the matter of assessment of candidates is maintained. Where candidates appearing are more than 100, two additional examiners (one external & one internal) for every additional 50 or part there of candidates appearing, be appointed.

(c) In case of non-availability of medical teachers, approved teachers without a medical degree (engaged in the teaching of MBBS students as whole-time teachers in a recognized medical college), may be appointed examiners in their concerned subjects provided they possess requisite doctorate qualifications and four years teaching experience (as assistant professors) of MBBS students. Provided further that the 50% of the examiners (Internal & External) are from the medical qualification stream.

(d) External examiners may not be from the same University.

(e) The internal examiner in a subject shall not accept external examinership for a college from which external examiner is appointed in his/her subject.

(f) A University having more than one college shall have separate sets of examiners for each college, with internal examiners from the concerned college.

(g) External examiners shall rotate at an interval of 2 years.

(h) There shall be a Chairman of the Board of paper-setters who shall be an internal examiner and shall moderate the questions.

(i) All eligible examiners with requisite qualifications and experience can be appointed internal examiners by rotation in their subjects.

(j) All theory paper assessment should be done as central assessment program (CAP) of concerned university.

(k) Internal examiners should be appointed from same institution for unitary examination in sameinstitution. For pooled examinations at one centre approved internal examiners from same university may be appointed.

(l) The grace marks up to a maximum of five marks may be awarded at the discretion of the University to a learner for clearing the examination as a whole but not for clearing a subject resulting in exemption.

Address for Correspondence:
Dr. Jyoti Deswal
Assistant Professor,
RIO, PGIMS,
Rohtak-124001 (Haryana)
E-mail: jyoti_deswal@yahoo.co.in
Mobile: 9416857905

E-Learning in ophthalmology – Present scenario

A.K. Khurana[1] MS, **Harvinder**[2] MBBS,
Aruj K. Khurana[3] DNB, **Harmanjot Singh**[4] MBBS
Professor and Head[1], Junior Resident[2,4], Assistant Professor[3]
Department of Ophthalmology, SGT Medical College, Hospital and Research Institute

Abstract

Electronic learning is moving from textbooks in electronic format to a truly interactive medium that can be delivered to meet the educational needs of students and postgraduate learners. Computer technology can present reliable, reusable content in a format that is convenient to the learner. It is a valuable tool to add to the medical teacher's toolkit, but like all tools it must be used appropriately. This article endeavors to review the current "state of e-learning and its role in Ophthalmology alongside non-electronic methods—a combination that is currently referred to as "blended" learning. We discuss the various electronic resources and strategies which were used to sustain academics during this pandemic.

Keywords : *Electronic learning, ophthalmology, Covid-19, virtual classrooms.*

Introduction

Ophthalmology, being an image-rich specialty, has been known to adapt the latest and innovative digital technology, much ahead of other medical specialties[1]. E-learning is the use of the internet for the purpose of education. E-learning in medical education is at a nascent stage in our country. E-learning is the use of internet technologies for the purpose of education. It serves to store the instructional material of diverse forms such as print, pictures, animations and videos electronically. The advantages of e-learning include the increased accessibility to information, ease in updating content, personalized instruction, ease of distribution, standardization of content, and accountability. The instructional material is available for access all the time by any number of users at their convenient time.[2]

Academics during COVID-19

On 11th March 2020, the World Health Organization (WHO) declared the COVID-19 a pandemic. Currently, there are more than 3 million cases and one lakh deaths reported, and still counting. This has brought radical changes in all aspects of our lives. Social distancing and restrictive movement policies have markedly deranged traditional educational practices. The time course of these changes is indeterminate. These have affected conventional in-person ophthalmic education and training. There is a pressing need to innovate and implement alternative educational and assessment strategies. The COVID-19 pandemic has provided us with an opportunity to pave the way for introducing digital learning in ophthalmology.[3]

Webinars

Webinars present a great virtual opportunity to engage and stimulate interactions between presenters and participants and can accommodate more participants than a physical conference room setting which could be limited by space and accessibility. Webinars provide participants the convenience of attending an academic presentation from the comfort of their offices or homes while multitasking. A successful webinar session is strongly dependent on the planning activities before the session, aligning the webinar theme to the expectation of the audience, user-friendly webinar platform, and the presenters' expertise and competence. Such webinars have to be announced well in advance and communicated through e-mail to the prospective audience.[4]

Virtual Classrooms

Distance education can be synchronous that happens in real-time, involving online studies, with the aid of chat rooms as well as asynchronous occurring through online channels without real-time interaction. A real classroom has now been replaced by the virtual classroom. Hybrid learning using both can be much more effective and are easier to use.The flipped classroom is a simple strategy which refers to providing learning resources like articles, pre-recorded videos, and YouTube links before the class.

online classroom time is then used to deepen understanding through discussion with faculty and peers. This is a very effective way of encouraging skills such as problem-solving, critical thinking and self-directed learning.E-seminars, case-based discussion, journal clubs, discussion on surgical techniques; clinico- epidemiological presentations can be very effectively conducted using these virtual classrooms.[5]

E-books

Even though paperback and hardbound books have their unquestioned place, the convenience and other advantages of e-books are suited for ophthalmology residents. e-book versions are available for almost all the major textbooks, and they are often stored in every ophthalmology resident's laptop or tablet computer for quick reference. They make it easier to carry around all the necessary books instead of lugging around heavy books. They help in easy searching for particular topics and quick preparation of student lectures. In addition to the standard textbooks which are available for purchase, there are several other books that have been made freely available online by their authors.[6]

Podcasts

A podcast is an online audio resource which is typically published as a series, and this medium is useful in certain scenarios when you want to make maximum use of time. It is particularly useful while driving or working out, when reading or watching videos is not possible, but you can still listen. There are several interesting ophthalmology podcasts, some of which are dedicated to teaching the subject, some catch up on ophthalmic news, while others interview leading ophthalmologists.[7]

Advantages of E-learning

Flexibility and ease of access: E-learning is not fixed or time bound to a particular schedule and is very easily accessible, so learners can choose a place and time of their own liking. Teachers can also update and edit the learning material at their own convenience.

Time-saving: More number of students can be taken up at same time, thereby reducing the time needed for total lectures. The content once made is eternal, and can be revisited any number of times.

Uniformity: Since identical information is posted to all the participants, the curricula uniformity is maintained across the learners. The students posted in off-site campuses also receive identical inputs. This helps in the uniform attainment of learning objectives.[8-11]

Hurdles in Usage of E-learning

Hardware and software issues: The technological infrastructure in the form of hardware and software is compulsory to run e-learning programmes. The issues related to the hardware are cost, shortage of units, and lack of technical knowledge to operate them.

Connectivity: The success of e-learning also depends upon the internet connectivity. Adequate bandwidth would be needed at various levels to ensure proper learning.

Lack of face-to-face interaction: The bulk of the early teaching is with traditional face-to-face interaction; its non-availability in e-learning is a well-recognized challenge.[11]

Conclusion

Ophthalmologists have always been at the forefront in using the latest technology. Even in the e-learning space, there are a lot of valuable online resources that would be very useful for a fresh ophthalmology resident and practicing ophthalmologist as well. E-learning is a significant advance with the potential to change the face of medical education in India in the coming years. For this to happen, Institutions and teachers need to be prepared to accept the change, and put-in the required resources — whether manpower or time or money. For successful incorporation of e-learning in the existing set-up, the following attributes are essential: motivation and self-discipline; ability to study independently or schedule study time; understanding the e–learning process; and adequate equipment and dedicated work support.

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Address for Correspondence:
Dr. A. K. Khurana
Prof & Head, Department of Ophthalmology,
SGT Medical College, Hospital and Research Institute, Budhera, Gurugram
E-mail: drkhurana8@gmail.com
Mobile:9255583523

Simulators in Ophthalmology – A promising tool

Manisha Nada MS DNB, **Manoj P Shettigar** MBBS,
Monika Dahiya MS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Virtual Simulation potentially reduces training costs, increases accessibility, offers objective measurement of training outcomes, and improves patient safety during and after clinician training, all of which can help address the global burden of vision impairment and blindness. This is especially pertinent in unusual situations like the covid pandemic.

Keywords: *Simulators, ophthalmology training, Holo eye.*

Introduction

The development of surgical skills requires long hours of dedicated effort. You need to have a background theory knowledge, a mentor, and patients. Repetitions of the same procedure time and again open up new vistas and slowly you start developing an insight. There is always a definite learning curve. Today's era of speed learning requires that we make this learning curve short and less steep and also ensure safety of our patients. Simulation of the actual scenario by use of 'Simulators' is very useful in quick and structured learning.

Various disciplines that require some motor skill proficiency have developed innovative models to impart practical training to trainees. Airline pilots spend a great deal of time using simulators to acquire robust and mature skills necessary to safely fly whereas, athletes and musicians spend a lot of time in practice and rehearsal before game day or recital. They all learn the finer points to be used for the final job using simulation models. Simulation is an educational activity which is interactive and experiential and is designed to mimic a real-life activity as realistically as possible. Simulation in healthcare is used to develop necessary skills but avoiding exposing patients to undue risks.

Simulators and Ophthalmology

The downstream effects of COVID -19 have influenced the educational experience of both trainees and practicing clinicians around the world. With less number of available patients and social distancing norms use of simulators is much more relevant in COVID era. The first known medical simulation application was developed in 1963 by Entwislel. This application simulated patients with six unique diagnoses and provided medical students feedback on their diagnostic skills.[1]

An eye simulator was developed in 1997 at UC Davis Medical School, California but only slight improvements on this model have been carried out since then. These simulators are designed to demonstrate the effect of disability or malfunctioning of any of the ocular muscles and/or nerves controlling them; however, the representation of the eye and ocular region is very schematic and two dimensional.[2]

Current simulation systems are based on virtual reality and user feedback. Various simulators have been developed in the field of ophthalmology.

Holo Eye

Holo Eye Anatomy application, developed by the L V Prasad Eye Institute, works with the Microsoft HoloLens headset. Based on the concept of augmented reality, the application allows users to interact with a virtual 3D model or hologram of the eye using hand gestures to pan, zoom, rotate, or slice the 3D model into component layers or to the level of individual cells such as the cornea. Learning and visualizing anatomy is key to a deeper understanding of the function and structure, along with pathological process in the visual system. Mixed reality, combining virtual and augmented reality, provides a learning and teaching tool to project 3D eye models in space, allowing engaging and interactive learning from a new perspective.[4](Figure1)

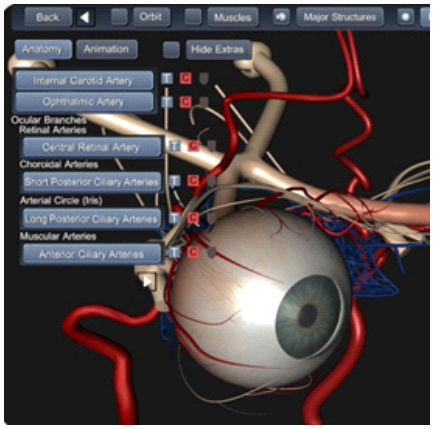

Fig.1: Smart phone app based ocular anatomy simulator

Help me see

Help Me See was founded with the mission of establishing a simulation-based surgical skills training program to teach Manual Small Incision Cataract Surgery (MSICS) HelpMeSee Eye Surgery Simulator is a highly realistic virtual reality trainer for Manual Small Incision Cataract Surgery (Figure 2 and 3). The system provides a scalable approach for training cataract eye surgery specialists and ophthalmologists.

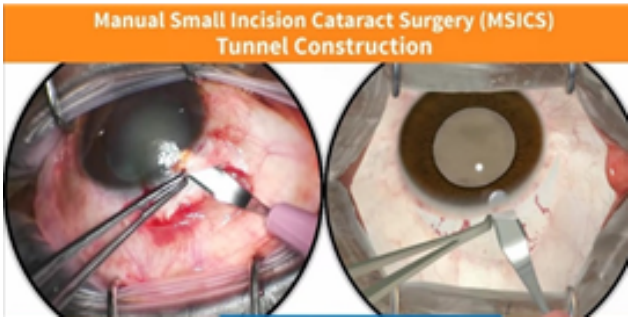

Fig.2: Real eye versus MSICS simulation software

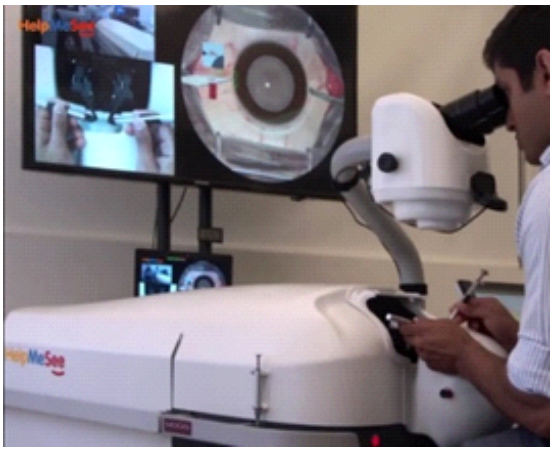

Fig.3: Eye Surgery Simulator

HelpMeSee Eye Surgery Simulator establishes a new standard in virtual reality surgical simulation with quality 3D visuals, an ability to provide haptic feedback for the very low forces experienced in ophthalmic microsurgery, and a feature-rich training system.

The Simulator replicates the mechanics of surgical interaction: angles, forces, friction, flow and resistance with realistic cause and effect. High-fidelity, virtual reality simulation with tactile feedback elucidates the mechanics of ophthalmic surgery. Currently programmed with the tunnel incision step of the MSICS procedure, the Simulator's Simulation-Based Learning System (SBLS) creates an unparalleled learning experience.[3]

Phaco Vision and Eyesi

There are two other commercially available ophthalmic surgical simulators. One is the PhacoVision (Sweden) and the other the Eyesi (Germany). The first system focuses exclusively on the capsulorhexis and phacoemulsification aspects of cataract surgery. The second system was originally designed as a vitreo-retinal surgical training device, though an anterior segment module was subsequently developed. The Eyesi anterior and posterior segment platforms were obtained and implemented at various ophthalmology residency and retina fellowship programs as part of organized surgical training curricula. Recent hardware and software advances have expanded the simulators to include anterior segment training modules also. These include capsulorhexis and phacoemulsification training modules.[5]

The Eyesi simulator consists of a mannequin head with a mechanical eye, various probes which mimic different intraocular instruments, a virtual operating microscope with functioning foot pedal, and a separate instrument foot pedal. The surgeon is shown stereoscopic images of the eye and instruments through the microscope while an observer is able to monitor from a separate viewing screen.[6]

The Eyesi microscope gives a stereoscopic view identical to the real operation theatre microscope. The trainee is required to establish suitable visualization via the microscope's foot pedal's zoom, focus, and X/Y controls. BIOM/ SDI hardware is integrated into

the microscope setup.

Modules are available from Eyesi for vitreoretinal training also (Figure 4). Training sessions include inducing posterior hyaloid detachment and performing peripheral vitrectomies, peeling the internal limiting membrane (ILM) or the removal of epiretinal membranes.

To help trainees in refining manual dexterity skills associated with vitreoretinal surgery Eyesi provides a virtual surgical instrument tray. In the course of performing virtual surgery a trainee will use an illumination probe in their non-dominant hand. As in real retinal surgery Eyesi enables the surgeon's dominant hand to alternate between instruments such as vitrector, endolaser, forceps, scissors etc

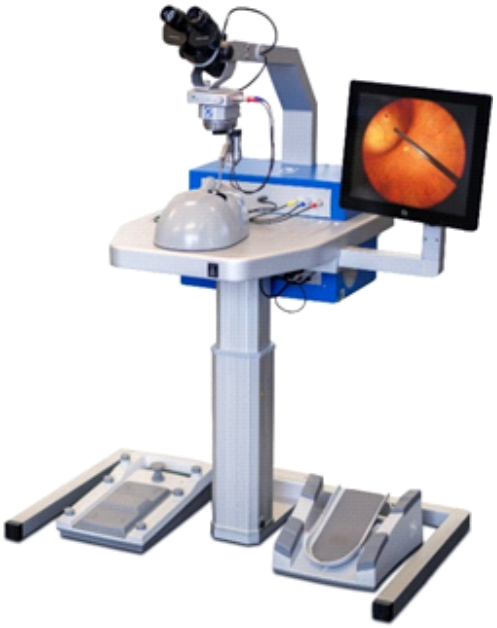

Fig.4: Vitreo retinal surgery simulator

Three-dimensional (3D) technology helps to experience width, depth, and height to simulate real life. This includes VR, printing, and interactive mobile apps. A review study has assessed the recent literature pertaining to 3D simulated surgical training, which provides a rigorous analysis of the quality of assessment of these technologies.[7]

A recent review offers an example of 3D simulation in the printing of rigid gas permeable (RGP) lenses. These lenses are ideal for refractive error correction, treating keratoconus, and corneal transplant. RGP fitting is not easy on irregular corneas and repeated trying on of lenses is not only uncomfortable but also dangerous as it increases risk of corneal shedding or infection. A safer method of fitting RGP lenses uses corneal topography obtained through simulation of a fluorescence evaluation map based on the corneal anterior surface data and RGP lens design parameters. This simulation reduces the number of trial lenses required. Limitations of this technology, however, include the fact that positioning of the lens is influenced by the weight of the lens itself and by eyelid weight, which is even more influential with an irregular cornea.[8]

Conclusion

Simulation in ophthalmology training is an emerging opportunity. Ophthalmologists can learn from those who have been developing robust methods to interpret evidence correctly and to assess validity in comprehensive manner. With the many advantages of simulation for training of clinicians and assessment of patients in a cost- and resource-effective manner, we are poised to embrace the teachings of this powerful emerging field to deliver equitable and timely eye care.

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Address for Correspondence:
Dr. Manisha Nada
Professor,
RIO, PGIMS,
Rohtak-124001 (Haryana)
E-mail : manisha_nada@rediffmail.com
Mobile : 9896007158

Improving low vision through multipronged rehabilitative Techniques among low socio-economic patients

Ashwini Kumar MBBS, DOMS
Mahavir Netralaya, Kankarbagh, Patna

Abstract

Patients with low vision were examined, investigated and treated in the period between May 2020 to July 2020, using multipronged techniques including thorough clinical examination, investigation, counselling, and an assortment of new rehabilitative tools and techniques. Total of 3070 patients were managed and there was improvement in visual acuity as well as improvement in quality of living where patients were able to undertake several household and profession related tasks and activities. Through this, the author concludes that for low vision patients, following proper diagnosis, and treatment, proper rehabilitation tools and techniques are needed to bring improvement in the quality of life.

Keywords: *Low vision aids, low vision treatment, quality-of-life improvement, LVA.*

Introduction

Low vision is the term used to describe significant visual impairment that cannot be corrected fully with glasses, contact lenses, medication or eye surgery[1]. It includes loss of best- corrected visual acuity (BVCA) to worse than 20/70 in the better eye. The terms "partial sight" or "partial blindness" or even "poor vision" were used to describe low vision[1]. However, a proper definition of low vision, related to visual acuity, will help the scientific community to understand low vision better. One accepted definition is "Low vision is a condition caused by eye disease, in which visual acuity is 20/70 or poorer in the better-seeing eye and cannot be corrected or improved with regular eyeglasses. (Scheiman, Scheiman, and Whittaker)[2] Currently, India has around 12 million blind people which makes India home to one-third of the world's blind population[3]. With the increasing life expectancy and thus increasing age related problems, the magnitude of visual impairment is expected to rise in coming years. With this emerging problem, it becomes important to find feasible and effective solutions to improve both visual acuity and quality of life of patients suffering from low vision. While "best possible" correction is the first step towards this, "improving simple life-skills" through a rehabilitative process is also as important.

Material and Method

3070 patients with low vision were examined, investigated and treated at Mahavir Netralaya Kankarbagh in the period between May, 2020 to July, 2020.

Following a systematic screening process including a structured history taking, clinical examination and relevant laboratory investigation, a more specific line of investigation for low-vision patients were undertaken. This included assessing visual acuity through Snellen's chart for distance, near vision chart, assessing colour vision with ishihara charts, examination of ocular movements and Schirmer's test. Newer instruments such as Mono-ocular and Binocular telescopes were used to assess distance vision. Likewise, for near vision- Hand held magnifiers, illuminated and non- illuminated, pocket magnifiers, dome magnifiers, half eye magnifiers, cutaways were all used. According to diagnosis and need, line of treatment included giving different kinds of magnifiers for visual correction and improvement. Special rehabilitation techniques were undertaken; this included M training for the patients with peripheral vision loss, Notex to recognize currency using a variety of new instruments. Also detailed home based rehabilitation exercises and task were given to each patient which were customized according to their situation and needs.

Results

The findings of the study were classified according to socio-economic and clinical aspects.

Socio-economic

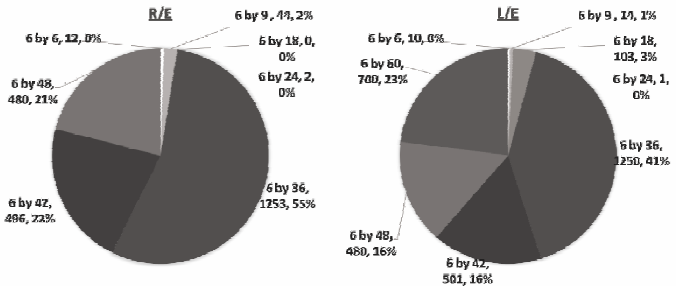
Of the 3070 patients, 84% were males and 16% were

females. The age of the patients ranged from 11 years to 66 years. As the clinic is run by a charitable trust, it caters mostly to people of poorer socio-economic condition and this was reflected in the occupation of the primary earning member, which was 78% unskilled, 8% semi-skilled, 5% skilled and only 9% in service, professional or business.

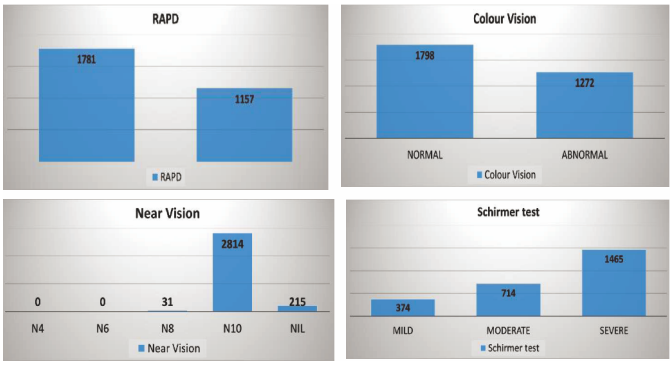
Clinical findings

Visual Acuity

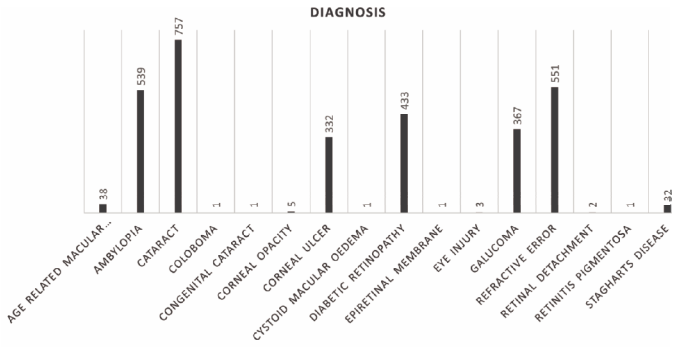
On testing for Visual Acuity, it was observed that a majority of the patients had acuity problems with only 10 persons having 6/6 vision. The majority had visual acuity problems of which over 95% had vision poorer than 6/18 in left eye and 98% with vision poorer than 6/18 in right eye. The worst condition of eye-sight poorer than or equal to 6/42 were 43% in right eye and 55% in left eye.



Other investigations: The findings of other investigations were as follows among the patients investigated; Rapid Afferent Pupillary Defect: 61% no defect, 39% defect. 3029 patients were examined for color vision of which 59% had normal and 41% had abnormal color vision. 3060 patients were examined for near vision of which 91% of patients had near vision of N10. 2553 patients were examined for Schirmer's test of which 57% had severe condition.



The diagnosis of the patients was as per the chart given below:



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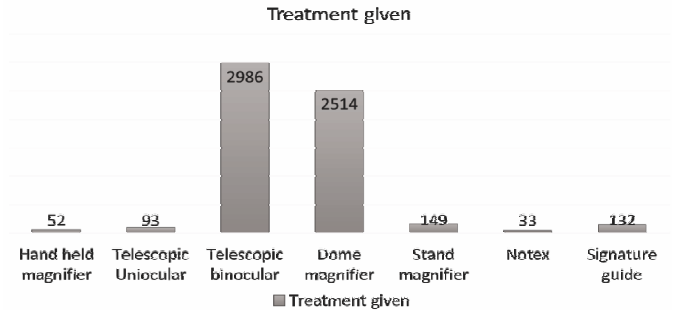
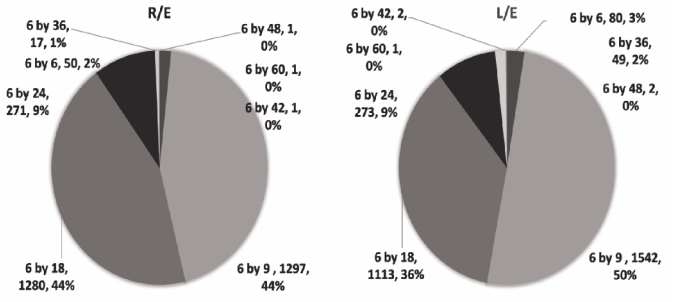


Management

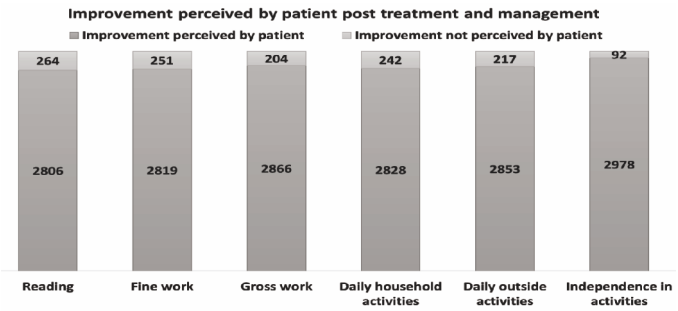
A variety of treatment, management and rehabilitative processes were undertaken to improve the sight of the low vision patients. These included trying out a variety of tools, along with counselling, hand-holding and supporting them to do simple tasks and activities. Each patient was treated differently and rehabilitation plan was customized according to the patient's needs. A few patients were also referred for surgical procedures and tools and therapy given post-surgery.



On testing for visual acuity, it was observed that a majority of the patients had improvement in visual acuity .88% in right eye and 89% in left eye having

visual acuity equal to or better than 6/18. This was in marked contrast to 2% and 4% in right and left eyes prior to rehabilitation and management.

Results in terms of socials aspects, daily tasks and improvement in quality of life included the patients improved ability for the following tasks: reading newspaper, doing fine and gross work, marked improvement in undertaking household and outside activities and a remarkable regaining of overall personal independence and confidence.



Patients completing their rehabilitation were asked to rate their satisfaction with the outcome of the process, and an overwhelming 96% of them stated that they were very much satisfied.

Discussion

The findings of this study have a vital implication because the burden of low vision is gradually increasing; but despite this, there are path breaking and unique tools that can be used in rehabilitation and remarkable improvement of sight. This is not unlike the revolutions in eye-care management brought about by the humble spectacles and the phenomenal cataract surgeries and intraocular lens implantation.

Pal et al did a study on 703 children attending blind schools and ascertained the need for spectacles and Low vision devices (LVD) in children with useful residual vision4. They concluded that there is large potential for these "incurably blind children" to benefit by use of spectacles and Low vision aids and emphasized the need of visual rehabilitation in these children. Khan et al 5. analyzed the perceived barriers to provision of LVDs among ophthalmologists in India and concluded lack of training/knowledge, lack of awareness, and no availability of LVDs as the major barriers. They emphasized the importance of increasing knowledge and awareness of ophthalmologists about the benefits of low vision rehabilitation.

The important finding in this study was that patients were from lower socio economic conditions, and from a wide range of ages making life overall limited and difficult for them, whether it was related to household wok, outside work, schooling or occupation. It was interesting to observe that a significant improvement also occurred in activities indoors and outdoors and a remarkable gain in confidence and satisfaction. This very important inference of the study underscores the need for timely and effective vision rehabilitation to help maximize the quality of life.

Low vision interferes not only with their routine and academic activities but it has also been now well-established that low vision has psychological consequences as well thus making patient feel inconfident and limited. Little things like going outside alone, handling money, reading boosts confidence and enable patients to progress further in life. The tools are also affordable and easy to use, making the case of visual rehabilitation in low vison patients even stronger.

Conclusion

For low vision patients a systematic approach to screening, diagnosis, and treatment is needed, but in addition, nuanced rehabilitation using tools, tasks and techniques both at the clinic and at home can help to bring improvement in the quality of life.

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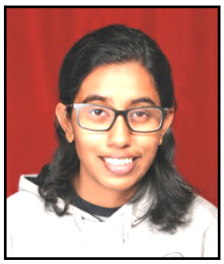
Address for Correspondence:
Dr. Ashwini Kumar
Consultant,
1A4, New Patilputra Colony, Patna.
E-mail : kumarashwini816@gmail.com
Mobile : 9108285969

Correlation of visual acuity with retinal thickness and submacular choroidal thickness in patients of central serous chorioretinopathy



Muralidharan Shruti MBBS, **Sangeeta** B Optom, **Ann Maria Rodrigues** MS, **Reetika Negi** B Optom, **Subina Narang,** MS, *Department of Ophthalmology, Government Medical College and Hospital, Sector-32,Chandigarh, India*

Abstract

Background Central Serous Chorioretinopathy is a self-limiting condition of middle aged males. The course of the disease is varies from spontaneous resolution within 2-3 months to chronic and recurrent forms. Recently increased choroidal thickness has been identified in cases of CSCR using Enhanced depth imaging Optical coherence tomography technology.

Purpose to study correlation of visual acuity at 1 month with baseline retinal and submacular choroidal thickness in patients with CSCR using EDI-OCT.

Method This was a prospective pilot study of 11consecutive cases of CSCR that presented to our retina services over a period of 6 months(March 2019 to August 2019). FFA and Enhanced depth imaging (EDI) OCT was done in all cases and the choroidal thickness was assessed manually using EDI Fast macula scans. All patients were kept under observation and no treatment was given. At 4 weeks follow up repeat OCT scans were done and correlated with the final visual acuity at 1 month follow-up.

Results At initial visit, the mean retinal thickness was 388.7 ± 97.7μm (mean ± SD) and mean height of serous retinal detachment was 173.0 ± 99.6 μm (mean ± SD).The mean choroidal thickness showed Haller's layer thickness of 479±101.4 μm (range 314-580) and Sattler's layer thickness of 121.3 ± 33.6 μm (range 54-170). At 4 weeks follow up, the retinal thickness decreased to 321.7±88.9 μm (Mean±SD) and height of serous retinal detachment to 114.7±106.9 μm (Mean±SD). The thickness of the Haller's layer was 384.5 ± 76.0 μm (Mean±SD) (Range 280-511) and Sattler's layer was 104.36 ± 18.4 μm (Mean±SD) (Range 60-119). At the first visit the logMAR visual acuity was 0.5 ± 0.5 and at the last visit, visual acuity improved to 0.3 ± 0.3 (mean ± SD). The serous retinal detachment (SRD) height and choroidal thickness showed a marked decrease after 1 month in 9 eyes(81.8%), with complete resolution of SRD in 3 eyes(27%). The study shows a positive but weak correlation of retinal thickness, choroidal thickness and SRD height with visual acuity at 1 month.

Conclusion: Acute CSCR undergoes significant and spontaneous improvement in 81% of the cases at 1 month with complete resolution of 27% cases. The height of SRD in CSCR significantly correlates with the final visual outcome. A larger study with more number of patients and longer follow up will give us long term results in this condition.

Keywords: *Visual acuity, retinal thickness, choroidal thickness.*

Introduction

Central serous chorioretinopathy (CSCR) is characterized by serous detachment of the neurosensory retina associated with focal or multifocal areas of leakage at retinal pigment epithelium (RPE). As the name suggests the changes in inner choroid and retina represent the primary and secondary abnormality of this disorder respectively.[1] The increased hydrostatic pressure of choroid, causes detachment of the neurosensory layer from the pigment epithelial layer and accumulation of fluid between the two. Central vision is affected early due to macular involvement and is one of the prognostic factor for CSCR.[2] Although known to be idiopathic, there are several risk factors associated with its causation which include glucocorticoid use, smoking, type A personality, disorders associated with increased endogenous steroids like stress, emotional distress, pregnancy, Cushing's syndrome, systemic hypertension, systemic lupus erythematosus, poor quality of life, psychological problems, and a history of psychiatric illness.

CSCR can occur in both acute and chronic forms; the former is usually self-limiting with simple

observation being the standard management in most cases. The duration in the latter has varied in different studies from persistence of fluid for 3 months , to at least 6 months., However, recurrences within 1 year are commonly seen in approximately 30%–50% of patients.,

The diagnosis of CSCR is confirmed by Fundus Fluorescein Angiography (FFA), Although several studies have described the angiographic characteristics of acute CSCR in the Western population, data in Asians is limited[11]. Recently OCT has emerged as a newer diagnostic tool for evaluating CSCR under the spectrum of Pachychoroid disorders. The altered morphology in the form of abnormally thickened choroid (>395 m) with vascular dilatation (pachyvessels in the Hallers and Sattlers layer), hyperpermeability and alteration of chorio capillaries(inner choroidal layer) can be assessed by the Enhanced Depth Imaging in OCT. OCT thus has become an important tool in assessing the choroidal thickness in CSCR both during diagnosis and subsequent follow up. As the disease is known to affect Type A personality people, OCT prognosticators could help in patient counseling to a great extent.

There have been no studies evaluating the correlation between thickness of choroid and visual outcome in CSCR especially among Indian patients even though CSCR is known to resolve completely with a good visual outcome in the majority. However in some, sequelae are known to occur as a result of which visual gain may not be complete. With this background in mind, we measured the retinal thickness and submacular choroidal thickness with help of OCT and correlated it with visual outcome in patients of CSCR.

Methods

A hospital based, prospective, observational study was carried out among outpatients visiting the retina clinic of a teaching and referral tertiary centre. A total of 11 consecutive cases of CSCR were enrolled between March 2019 to August 2019. A written, informed consent was obtained prior to enrollment.

The diagnosis of CSCR was made if the patient showed a neurosensory detachment of retina on OCT which was further confirmed by FFA. In order to avoid confounding effects of other causes of decreased vision, patients with advanced cataract, macular pathologies, history or evidence of any ocular disease such as uveitis, glaucoma, amblyopia, strabismus or retinopathies of any type were excluded.

The study included 11 eyes of 10 patients (3 females, 7 males) with the age ranging from 27–59 years(Mean 38.6± 11.2SD)). A detailed history was taken from all patients especially for the use of steroids in any form. A trained blinded optometrist recorded logMAR visual acuity using Snellen acuity chart at a distance of 4 meters (S). All patients were subjected to a detailed slit lamp biomicroscopic examination. A thorough dilated fundus examination using slit-lamp biomicroscopy with +90 D lens was done by a trained Ophthalmologist (SN). All patients were subjected to color fundus photography (VisuCam 524), High-Definition (HD) macular cube 512×128 scanning using Cirrus HD-OCT 500(Carl Zeiss Meditec, Inc., 5160 Hacienda Drive, Dublin, CA 94568 USA) (RN) and EDI scan using Zeiss Cirrus HD-OCT 500. FFA was done in all the patients using 10% sodium fluorescein dye. The leakage points and types of leakage pattern seen on FFA were recorded in all. Measurement of SRF height (distance from RPE to photoreceptors) and sub-macular choroidal thickness (distance from inner scleral boundary to RPE) were performed for each patient using the caliper function. Data analysis was done using a two-tailed paired t test to calculate statistical significance of logMAR visual acuity, retinal thickness, choroidal thickness and serous thickness at first visit and 4 weeks. A P value of less than .05 was considered to be statistically significant.The correlation between visual acuity and retinal and choroidal thickness was studied using Pearson correlation coefficient.

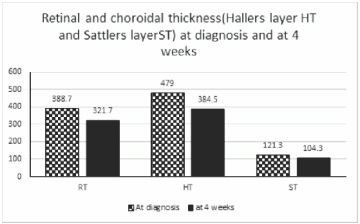
Results

All patients were kept under observation and no treatment was given. The mean± SD age of the study subjects was 38.6±11.2 (Range, 27-59) years. There were 7 males (70%) and 3 females (30%).Of the 11 eyes, at 4 weeks follow up, CSCR persisted in 2 (18.2%), decreased by >10% of SRD in 6 (54.5%) and completely resolved in 3 eyes (27.3%). Bilateral CSCR was present in 1 patient and the rest had

unilateral disease.

The mean ± SD logMAR visual acuity at baseline was 0.5 ± 0.5. The mean ± SD retinal thickness at initial visit was 388.7 ± 97.7 m and the height of serous retinal detachment was 173.0 ± 99.6 m. The mean ± SD choroidal thickness during initial visit showed Haller's layer thickness of 479±101.4 (range 314-580) and Sattler's layer thickness of 121.3 ± 33.6 (range 54-170).

The logMAR VA noted at 4 weeks of follow up 0.0-1.04 (Mean 0.3 ± 0.3SD). It had improved in 8 eyes (72.7%), decreased in 1 eye (9.1%) and remained same in 2 eyes (18.2%). This difference was statistically significant (p=0.05). The mean±SD retinal and SRD height at 4 weeks was 321.7±88.9 and 114.7±106.9 respectively. This too was statistically significant (P= 0.04). Both Haller's layer and Sattler's layer thickness had significantly decreased to 384.5 ± 76.0 (Range 280-511) and 104.36 ± 18.4(Range 60-119) respectively(P=0.05) . Although retinal and choroidal thickness decreased after 1 month, it did not reach normal levels.



We found a positive but weak correlation between final visual acuity and retinal thickness, choroidal thickness and SRD at baseline.

	AT DIAGNOSIS (MEAN ± SD)	AT 4 WEEKS FOLLOW UP (MEAN ± SD)	CORRELATION COEFFICIENT(BASELINE PARAMETERS WITH FINAL VA)
Visual acuity (log MAR) [a]	0.5 ± 0.5	0.3 ± 0.3*	
Total Retinal thickness [b]	388.7± 97.7	321.7 ± 88.9*	a & b 0.17
Serous thickness [c]	173.0 ± 99.6	114.7 ± 106.9*	a & c 0.10
Haller's layer [d]	479± 101.4	384.5± 76.0*	a & d 0.20
Sattler's layer [e]	121.3±33.6	104.3±18.4*	a & e 0.17

* P value < 0.05

OCT at first visit (Figure 1) and 4 weeks (Figure 2) showing resolution of SRF and reduced retinal and choroidal thickness and subretinal detachment height.

The visual acuity of the patient was 0.0 LogMAR.

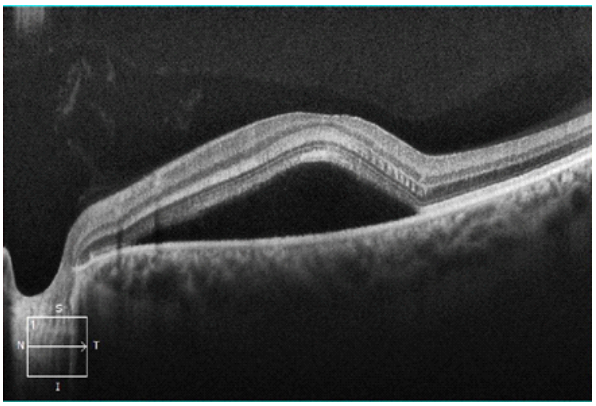


Fig. 1 : SRF at presentation

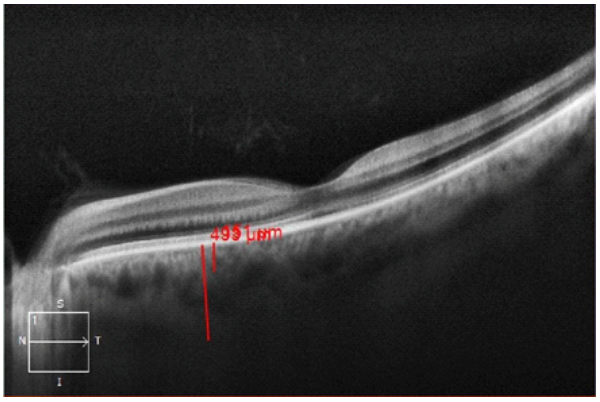


Fig. 2 : Resolution of SRF after 4 weeks

Discussion

CSCR is a relatively common condition and usually affects healthy middle-aged males between 20 and 50 years. Sex ratio (male: female) in our study was 8:3. 9 out of 10 cases had unilateral disease (81.8%) .

In our prospective study we found that CSCR in most eyes was self limiting and resolved at 4 weeks follow up. Our findings also revealed significantly decreased retinal and choroidal thickness in all the eyes from diagnosis to 4 weeks follow up. Furthermore, there was a commensurate and significant improvement in visual acuity in about three fourths of the eyes.

The findings compare favorably with that reported in literature, where a large majority of patients with acute classic CSC resolve spontaneously and experience complete restoration of vision.[16] The visual acuity in our patients had spontaneously improved in 72.7% within 4 weeks and was logMAR 0.0 in 27% patients. This is much higher than previously reported wherein 40% of patients had improved to 6/6, 30% to 6/9 and 10% to 6/12.[17-19]

We observed a positive correlation between visual acuity and the serous detachment height and retinal thickness as measured by OCT. These findings imply that inflammation and fluid accumulation between neurosensory and retinal pigment epithelium layers in CSCR possibly decreases with time.

However some patients may experience permanent visual deficits in the form of decreased visual acuity, contrast sensitivity or color vision or distortion of central vision in the affected eye.[16] Some of the postulated reasons for failure to regain complete vision are photoreceptor damage due to neurosensory detachment, atrophy, RPE pigmentary abnormalities or subretinal fibrosis. CSCR induced retinal serous detachment, central scotoma, metamorphopsia, dyschromatopsia, and decreased visual size and visual contrast sensitivity are well reported visual abnormalities.[20] The follow up period over which these visual abnormalities were reported ranged from 3 months to 14 years. In our study, one patient with bilateral disease showed a deterioration in logMAR visual acuity over 4 weeks due to increase in height of SRD. History of steroid intake or any other medical event was unremarkable.

Similarly Loo et al had shown that serous retinal and RPE detachment correlated with a final visual acuity of 0.5 or less.[21] at a follow up period of 8 years. On the contrary in the current study we found a positive correlation of VA with SRD but not with RPE detachment. This could be explained by the shorter follow up period and fewer patients with RPE detachment in our study as compared to Loo et al[21]

In a retrospective study of 32 eyes; 12 acute non treated and 20 ranibizumab treated chronic CSCR , choroidal thickness(CT) changes as measured by OCT at the following points- subfoveal, nasal and temporal 500 m and 1500 m were compared. At a mean ± SD follow up of 21.6 ± 8 months they found that CT had decreased significantly at nasal 1500 m (N1500) in acute group and all except T500 point in chronic group. Their objective was to study the anatomical and functional difference in CT at diagnosis and after resolution of fluid in both acute and chronic CSCR.[22] However, the authors had not correlated the change in CT with visual acuity as was done by us.

In another study evaluating the role of eplerenone in chronic CSCR and subretinal fluid, 14 eyes were monitored for a period of 3 months wherein visual acuity and choroidal thickness were measured. The findings of this study at 1 month follow up showed concordance with our OCT findings; 10 eyes had decreased subfoveal fluid height as measured on OCT, 2 eyes showed complete resolution of subretinal fluid with mean CT reduced($p=0.07$).[23] However the finding of a corresponding improvement in mean VA was different from ours. More eyes showed improvement at 3 months follow up: 13 eyes had reduced subfoveal fluid height and 9 eyes showed complete resolution with improvement in mean VA suggesting that with longer time the sub-retinal fluid gets further resorbed.

OCT plays an important role in the diagnosis and monitoring the evolution of CSCR. It not only helps in observing the retinal neuroepithelial layer morphology and the changes of RPE, but also in tracking the subretinal fluid changes[24]. Most studies on CSCR have used OCT.

Conclusion

Most eyes with CSCR resolve without treatment at 4 weeks as evident by the significantly decreased retinal and choroidal thickness. The visual acuity had also improved significantly in nearly three fourths of the eyes. There was a positive correlation between visual acuity and the serous detachment height and retinal thickness. The study however failed to show any strong correlation of choroidal thickness with visual acuity and a definite correlation of retinal thickness and visual acuity at 1 month.

The strength of our study is that it is one of its kind from Asian region where there is paucity of literature on CSCR. A short follow up period of 4 weeks seems to be the major limitation of our study. A longer follow up duration would have thrown more light on the evolution of morphological and functional changes and its correlation with visual acuity. This study paves the way for a larger study with more sample size and a longer follow up period to map out the chronic persistent changes in CSCR. This will help in planning definitive targeted therapy for persistent changes.

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Address for Correspondence:

Dr. Shruti Muralidharan

Junior Resident,

Department of Ophthalmology,

Government Medical College, Chandigarh.

E-mail: muralidharanshruti@gmail.com

Mobile: 7508714156

Why is negative pressure operation theatre in ophthalmology not a good idea?

Jatinder Bali[1] MS, **Ojasvini Bali**[2]
Hindu Rao Hospital, New Delhi[1]
Maulana Azad Medical College, New Delhi[2].

Abstract

Human Coronaviruses or HCoVs usually cause common cold. Zoonotic transmission resulting in severe acute respiratory syndrome (SARS) epidemics in 2003 and 2012 occurred. Coronavirus SARS CoV2 has shut down almost one half of human population and devastated countries in less than six months. It affected the ophthalmology practices and surgeries. In many countries the worst of the pandemic appears to be over and they are now contemplating restarting their economies. The ophthalmologists are also planning to go back to doing surgeries and opening clinics. Unique challenges have emerged. Operation theatres are unique environments because of the complexity of construction and criticality of their use. The most important challenge is how to prevent them from becoming hotbeds of disease transmission to operating surgeons, healthcare workers and patients. Phacoemulsification and vitrectomy use vibrating instruments and excimer keratomileusis causes blasts of air to be dissipated. The modifications suggested by some of the ophthalmologists have no backing of evidence and use of negative pressure operating rooms (ORs) is one such idea in which the critical elements involved in ensuring safety have been missed. This article briefly uncovers the gaps and suggests that instead of negative pressure operation rooms the answer may lie in zero pressure or venting mechanisms. It opens the doors for sensitizing the funding agencies to provide resources for research into making the ORs safer.

Keywords: *Severe acute respiratory syndrome, SARS Cov 2 , COVID 19, operation theatres, operating rooms.*

Introduction

Coronaviruses are positive-strand enveloped nucleocapsid RNA viruses. They are divided into 4 genera: alpha, beta, gamma and delta based on genomics and serology. 10% to 30% of all upper respiratory tract infections in adults are linked to coronaviruses. Four human coronaviruses (HCoVs) namely HCoV 229E, NL63, OC43, and HKU1 have become endemic globally.[1] From the turn of the millennium major epidemics by zoonotic HCoVs have been reported which manifested as Severe Acute Respiratory Syndrome(SARS) in 2002-3 and the Middle-East-Respiratory-Syndrome (MERS) in 2012. Humans are competing with wild animals invading their shrinking wild habitats and therefore the zoonotic infections are an inevitable result. The 2020 pandemic due to novel coronavirus 2019 (nCoV 2019) was reported from a cohort of patients with pneumonia traced to Huanan Seafood Market at Wuhan the capital of Hubei Province in China. Novel coronavirus 2019 or Covid-19 infections were mostly mildly symptomatic and self-limiting but acute respiratory distress syndrome (ARDS) requiring ventilation was reported in 5 to 10% of individuals. Mortality and complications were higher in advanced ages. The reports of a bilateral ground glass appearance in lower lungs on computed tomography and a cytokine storm was also reported by some authors. Multiorgan dysfunction syndrome and myocarditis have also been reported in recent studies.[1,2]

The Premise

The current pandemic resulted in over half of the world being locked down. Different countries tried different approaches to battle the virus with varying results. Now the world looks to life after the pandemic. The economies are under threat of recession and even deflation. The healthcare sector is also looking to resume its elective or need based services. The clarion call has come from the Ophthalmology chains which are faced with the threat of liquidity. Heightened risks during surgery were reported in the literature and apprehensions were expressed by the experts in the past.[3] Covid-19 is transmitted through particles or droplets in the air. The anesthetists, ophthalmologists, dentists and otorhinolaryngologists were reported to be at higher risk of contracting the disease especially so when they

carried out procedures that generated aerosols. There were reports of otorhinolaryngologists on ventilators in Britain suspected of contracting the virus through contact with asymptomatic Covid-19 patients.[4]

The patient, surgical team and surgeon wear a sterile clothing along with the usual scrubs and thus would be deemed to be protected. But the problem is something different. The air conditioning in these theatres classically uses 80 percent recirculated air to cut on costs. The temperature and relative humidity are kept at 210 C ± 30C and 20 to 60 percent relative humidity.[5] Aerosols can leave the virus almost everywhere, and they can persist on plastic, metal and cardboard for varying durations. The virus can be present for up to several days on some surfaces. Classically the fumigation procedures would be carried out at the end of the week or end of day in some cases with medical ultraviolet light irradiation. It is not possible to clean the air or surfaces between surgeries.

Discussion

Operating rooms are sterile environments. But that is at the start of the work day. Once aerosols are generated by the drills of the orthopedists or handpieces of the ophthalmologists and the air is affected then we need to consider the air conditioning where the fan that sucks in and circulates the air in the operating theater. The current recommendation of using one hundred percent fresh air thus will have some impact and has some merit. However, Dr Alister Hart, a Briton operating at Britain's largest orthopaedic hospital, contends that if a patient happens to be infected with Covid-19, even if he is asymptomatic then the combination of power tools, high-velocity splatter and ventilation systems can produce a viral wind tunnel. "If the theater has had anyone with the virus in it during the previous 72 hours the airborne aerosol could have landed on some surface," including the ceiling. Even with most stringent cleaning protocols it is impossible to prevent this. According to him the implication is that "very soon all our operating theaters will be covered in Covid-19. And then you switch on the ventilation and you blow it all over the place" and operating rooms will become "viral labs in a wind tunnel" unless we address these concerns.[6] So it appears that soon the "new normal" will have to take this into account.

The air conditioning team responded by recommending 25 air changes per hour and a negative pressure inside the operation theatre. They added some extra ones like using 40-60 percent humidity and setting the temperature at 24-30 degrees centigrade. This response was typical of an industry trying to apply quick fix solutions without actually engaging with the stakeholders. These recommendations found their way into the guidelines of some professional bodies also. What no one realized was that negative pressure operation theatres were prescribed for infected cases and have been there since ages. In ophthalmology, it opened a Pandora's box. Now the negative pressure means that air from the surroundings will find its way into the operation theatre though the air curtains and this is definitely more contaminated than the treated air from the air handling unit over the operating table. The wind tunnel now has another source of adding contagion.

One solution to the problem is using close to zero pressure ventilation systems. This means that the split air conditioners and window units are now useful again if we can ensure disinfection, which we cannot. The second, a little less elegant solution is the use of a venting mechanism with a containment just next to the operation theatre. It is a satisfactory solution if the vented air is treated and vented out at least three feet or more above the inlet. That has still not addressed the question that the paper had raised. One solution is negative pressure hood over the aerosol generating area.

Covid-19 can be transmitted even by those without symptoms which makes this pandemic different from the previous coronavirus infections. Face masks cannot provide sufficient protection. One patient undergoing surgery in Wuhan infected 14 health care workers before a fever was even registered[7]. No unified hospital protocol for managing the coronavirus within surgical settings has been validated and most of the talks are in the realms of conjecture.

The world is trying to grapple with the definition of full PPE in this pandemic. A full-body, light plastic protection with goggles and face shields is the

Chinese version of full PPE. It leaves very little surface area of the body exposed to receive aerosol droplets. This is clearly very uncomfortable over any extended period of time.

Asymptomatic transmission and virus survival on surfaces makes this dangerous for the health professionals. The "viral load" in these settings in surgeons tends to be greater than those who pick the virus up in the community from touching an infected surface and then touching their faces. Whether a higher dose is linked to cytokine storms, in which the body's own defenses overreact, cause inflammation and wreak havoc in the lungs is still unknown.[8]

The mounting evidence seems to suggest that doctors are being exposed to a high-risk environment without proper protection. And no it is not about shortages. They add another dimension to the problem but it has largely been addressed on the scientific forums at least. We are some distance from clarity on what constitutes correct procedure for respirator reuse as the world has acknowledged that we need to find ways to make it last longer or find some more permanent methods.

Universal precautions are the best way forward. All patients, unless proven otherwise, are assumed to have the disease and surgeons take the appropriate precautions. The only problem here is that how do we test the surgeon and his team every time before he enters the operating room.

It does appear that the days of mindless factory style conveyor belt surgery with seventy cases scheduled for a single day may actually not be coming back for several years to come most probably. The results of the drum experiment that demonstrated the persistence for three hours may not be applicable to real life situations, nor would the knowledge of minimum infective dose really be forthcoming for sometime at least. Therefore, it is safe to presume that need based surgery would be a paradigm coming up as even the elective surgery would have to be triaged. Urgently answering the questions like, "are phacoemulsification and vitrectomy surgeries to be considered aerosol generating on the basis of simulation studies" and "is business process redesign to address the air inside the operation theatre relevant to practice of today" is of prime importance with at least some strong evidence. The economic impact of the lockdown is being felt by practices which used debt to grow and the aftershocks of the debt to growth models leveraged very well by the ophthalmic support and logistics microenvironment are being felt by the manufacturers and practitioners alike. However, there is a word of caution as we move towards reopening our practices. The lawsuits can be far more damaging and expensive than the losses incurred by the lockdown.

Conclusion

A cautious and calibrated response based on evidence is the need of the hour. Any clairvoyant guidelines or position statements can at best be deprecated and at worst be detrimental to the interests of the ophthalmologists at large. The discussions should involve the stakeholders and scientific evidence should be evaluated dispassionately before any major changes are envisaged.

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Address for Correspondence:
Dr. Jatinder Bali
55-D, Third Floor, DDA Flats,
Kalidas Road, Gulabi Bagh,
Delhi-110007. INDIA
E-mail: drjatinderbali@gmail.com
Mobile: 9868041025

Keratoconus: Presentation and management

Vandana Sharma MS, **Apoorva Goel** MS,
R S Chauhan MS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Keratoconus is the most common primary ectasia of the cornea usually presenting at puberty. It is a slowly progressive noninflammatory corneal disease characterized by changes in corneal collagen structure and organization. Early management of this disease is important as it can preserve useful vision for the patient and improve the quality of life during the most productive years of life.

Keywords: *Keratoconus, corneal collagen cross linking, corneal topography.*

Introduction

Keratoconus is a clinical term used to describe a condition in which the cornea assumes a conical shape because of thinning and protrusion. The process is non-inflammatory. Cellular infiltration and vascularization do not occur. It is usually bilateral although only one eye may be affected initially. It involves the central two thirds of cornea and is usually centered just below visual axis. The corneal thinning induces irregular astigmatism, myopia and protrusion leading to mild to marked impairment in the quality of vision. Keratoconus, classically has its onset at puberty and progresses until the third to fourth decade of life, when it usually arrests. It may, however, commence later in life and progress or arrest at any age. Rarely, it may be congenital.

Classification

On the basis of Keratometry

- Mild: <48D
- Moderate : 48-54D
- Severe: >54D

On the basis of Morphology

- Nipple cones: 5mm steep curvature may be located apically, centrally or para-centrally
- Oval cones: larger globus, 5-6mm, displaced infero-temporally
- Globus cones: largest>6mm

Presentation of a case of keratoconus

Chief complaints

Symptoms are highly variable and, in part, depend on stage of progression of the disorder. Early in the disease there may be no symptoms and keratoconus may be noted by the ophthalmologist simply because the patient cannot be refracted to a clear 6/6 vision. In advanced disease there is significant distortion of vision accompanied by profound visual loss. Patients with keratoconus fortunately never become totally blind.

Past History

Patient may complain of symptoms suggestive of allergic conjunctivitis and excessive rubbing of eyes. Patient may also give a history of episodes of severe pain, photophobia and watering along with diminution of vision followed by spontaneous recovery indicating previous episode of acute hydrops.

Personal history is usually not significant.

Family history is usually not significant.

Treatment history: Patient may give a history of long-term contact lens usage, rigid gas permeable lens usage or having undergone some corneal procedure.

General physical examination:

GPE may reveal features suggestive of genetic disorders such as Down's syndrome, Ehlers Danlos Syndrome, Osteogenesis Imperfecta, or Lebers Congenital amaurosis.

Features of atopic dermatitis may also be seen.

Ocular examination:

Clinical signs also differ depending on the stage of disease.

External Signs

Munson's Sign- V shaped conformation of the lower lid produced by the ectatic cornea in downgaze. (Figure 1)

Rizzuti's Sign- Sharply focused beam of light near the nasal limbus, produced by lateral illumination of the cornea in patients with advanced keratoconus. (Figure 2)

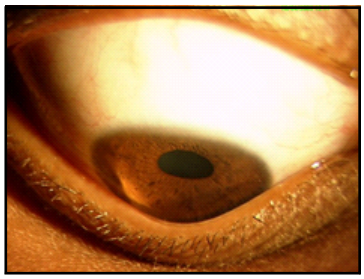


Fig. 1: Munson's sign

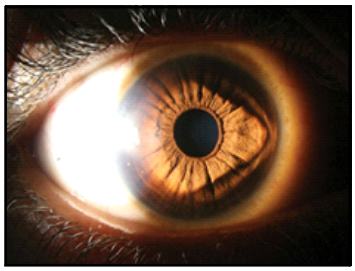


Fig. 2: Rizutti's sign

Slit- Lamp Findings

- Stromal thinning [central or para-centrally, most commonly inferiorly or infero-temporally.
- Conical protrusion of cornea.
- Fleischer ring –An iron line partially or completely surrounding the cone.
- Vogt`s striae –Fine vertical lines in deep stroma and Descemet's membrane that are parallel to the axis of the cone. These lines disappear transiently on gentle digital pressure on the globe.
- Epithelial nebulae.
- Anterior stromal scars.
- Enlarged corneal nerves.
- Subepithelial fibrillary lines.

Retroilumination Signs

- Scissoring reflex on retinoscopy.
- Oil droplet sign CHARLEUX SIGN. (Figure 3)

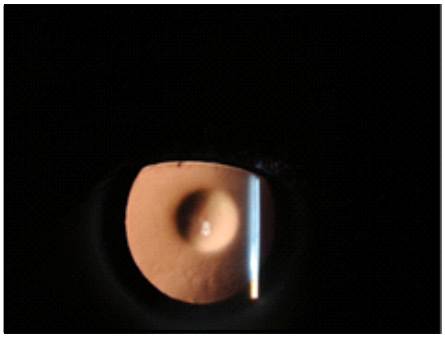


Fig. 3: Oil droplet sign

Photokeratoscopy Sign

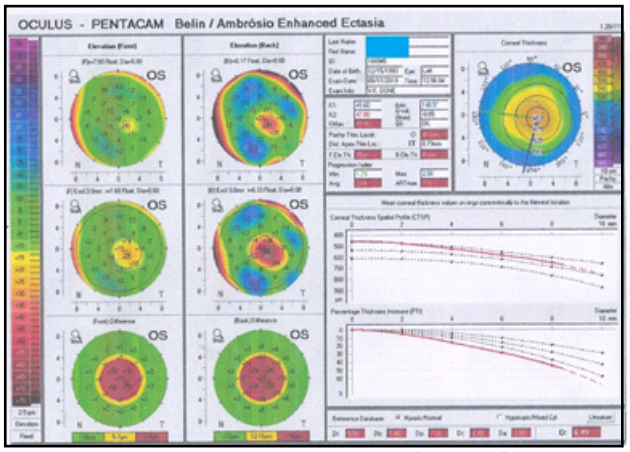
- Compression of mires infero-temporally [egg shaped mires].
- Compression of mires inferiorly or centrally.

Videokeratography Signs

- Localized increased surface power.
- Inferior superior dioptric asymmetry.
- Relative skewing of the steepest radial axes above and below the horizontal meridian.

Corneal Scheimpflug Imaging (pentacam)

- Steepening of cornea mostly in paracentral area.
- Thinning of cornea with apex mostly in infero-nasal quadrant.
- Belin- Ambrosio enhanced ectasia maps reveal minute corneal curvatural anomalies.
- Corneal thickness spatial profile and percentage corneal increase show increased progression of corneal thinning. (Figure 4 and 5)



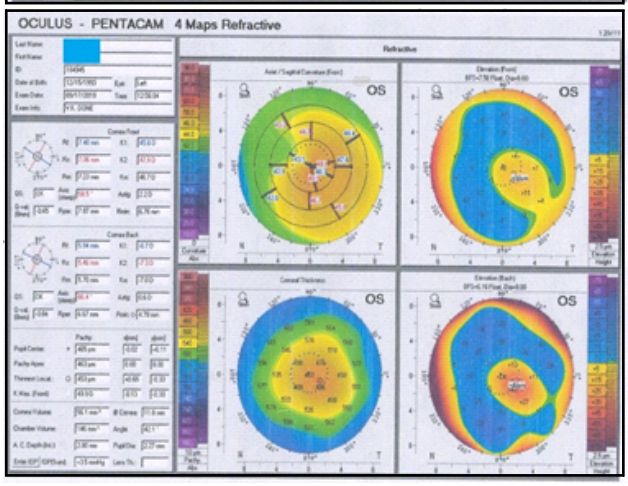


Fig. 4 and 5: PENTACAM of a patient of keratoconus.

Differential Diagnosis

1. Pellucid marginal degeneration
   - Usually detected between the second and fifth decade.
   - Characterized by thinning of inferior cornea from the 4 to 8 'o' clock position.
   - 1-2 mm uninvolved area between the thinning and the limbus.
   - Corneal protrusion is more marked above the area of thinning.
   - Thickness of central cornea is usually normal.
   - Topography has a classical butterfly or crab claw appearance, demonstrating large amounts of against the rule astigmatism.
2. Terrien's Marginal Degeneration
   - Affect both the superior and inferior cornea.
   - Accompanied by lipid deposition and vascular invasion.
3. Keratoglobus
   - Rare disorder in which the entire cornea is thinned most markedly near the corneal limbus.
   - Cornea may be thinned to as little as 20% of normal thickness and it assume a globular shape.
   - Topography reveals simple against the rule astigmatism.
   - The condition is bilateral, but usually is present from birth and tends to be progressive.
   - Cornea in keratoglobus prone to corneal rupture from even minimal trauma, thus hard contact lenses are contraindicated.
   - In advanced keratoconus, the entire cornea can also be thinned and globular, however there may be a small area of uninvolved cornea superiorly that approaches normal corneal thickness.

Management of keratoconus

1. Contact lenses
   - Mainstay of therapy in this disorder and represent the treatment of choice in 90% of patients. The type of contact lens used varies depending on the stage of keratoconus.
   - Four types of contact lenses are used:

   (1) Large diameter rigid gas permeable: useful in early to moderate keratoconus with inferiorly displaced concentration. Belin recommended an initial trial fit with a lens with central posterior curve equal to or 0.5 mm flatter than flat keratometry of the patient.

   (b) Post sphere lens: useful for moderate nipple cone and for inferior displaced cornea.

   (c) Nipple cone lens: better suited for nipple keratoconus.

   (d) Sopar or Meguine lens: used for more ectatic cornea.

   The challenge is to keep the patient contact lens tolerant with good visual acuity in a cornea that may be changing in shape over time.
2. Corneal collagen crosslinking
   - Utilizes riboflavin and ultraviolet-A (UV-A) to increase the biomechanical strength of the cornea by photochemical cross-linking of individual collagen fibers of the anterior stroma.
   - Halts the progression of keratoconus.
   - Early detection and management of keratoconus with C3R ensures a cone with minimal steepening and a better contact lens fitting.
3. Penetrating keratoplasty
   - Indicated when the patient becomes unable to tolerate contact lenses or when the vision obtained is not satisfactory.
   - Progression of cone size towards limbus, necessitating a larger desirable graft.
   - Some studies suggest that patients whose best corrected spectacle visual acuity is 6/12 or worse should be offered cornea transplant.
   - Technically a penetrating keratoplasty is similar to other grafts done for non-vascularized corneal disease .
   - Select the smallest trephine that will encompass the entire cone and still allow an adequate optical zone free from suture, usually between 8.0 mm and 8.5 mm.
   - It is desirable to leave a small amount of with the rule astigmatism, as patients can tolerate this better and it allows for easier contact lens fitting than against-the-rule astigmatism.
4. Lamellar keratoplasty (L.K)
   - Alternative to penetrating keratoplasty for the treatment of advanced keratoconus.

- Criteria for L.K include a healthy host ocular surface, optimal endothelial function, a corneal opacity that spares the Descemet`s membrane or a grossly distorted corneal surface that precludes a contact lens fitting.

5. Epikeratoplasty
   - A form of onlay L.K.
   - Treatment modality in patients with keratoconus who demonstrate good visual acuity with a diagnostic contact lens but who cannot tolerate contact lenses because of severe corneal distortion.
   - Central epithelium is removed from the recipient cornea and a small peripheral keratectomy and keratotomy are created in the anterior stroma.
6. Corneal intacs
   - Two PMMA segments 0.45 mm thickness are implanted in corneal tunnel to achieve maximal flattening of the cornea.

Address for Correspondence:
Dr. Vandana Sharma
B-16/165, Sunder NAgar,
Dhangu Road, Pathankot, Punjab
E-mail: dr_vandana_9@yahoo.in
Mobile: 9463671706

Digital strain due to increased screen exposure during Covid-19 pandemic

Jyoti Deswal MS, **Manisha Nada** MS DNB,
Latika Pandey MBBS, **Surender Kumar** MBBS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Global pandemic has pushed the humanity indoors thus replacing human face to face contact with electronic connection. The digital technology emerged as a boon in this COVID era but the harmful long –term effects of prolonged screen exposure cannot be overlooked. Digital eye syndrome is one of most common ophthalmological complaints these days and rising levels of myopia has set the alarm bells ringing. This article discusses the gravity of these problems related to increased screen exposure and the preventive measures.

Keywords: *Digital strength, screen exposure, digital eye syndrome.*

Introduction

India is a densely populated country with various economic and social disparities and thus, possesses great challenge in this era of the COVID-19 pandemic. In order to confine the spread of this infection and to reduce the pressure on healthcare systems, India went into a lockdown on 25th March 2020. Accordingly, people were confined to their homes with limited access to many services, which are temporarily closed, thus producing huge complications.

The COVID-19 has sent humanity indoors, thus replacing human contact with an electronic connection such as mobile phones, tablets, laptops, television, etc. According to the UNESCO, approximately 1.37 billion students (80% of the world's student population) from >130 countries globally are affected by these lockdown measures, with digital or e-learning approaches replacing face to face, classroom-based learning. One of the major health concerns of the increased use of electronic devices (e-devices) is Digital eye syndrome (DES) or Computer Vision Syndrome. Based on the pathophysiology and symptoms, DES can be categorized into two types: Internal and External DES. Internal DES mainly affects the user's visual system of accommodation, convergence and refraction. Users of e-devices will complain of blurring of distance and/or near vision, difficulty in re-focusing and headaches. External DES manifests with users complaining of tired red eyes, sensitivity to light and general ocular discomfort due to extended screen time. These symptoms are attributable to drying of the anterior surface of the eye, especially the cornea, due to a reduced blink rate.

Another major concern is global rise in the number of children becoming myopic. The combination of more screen time and less outdoor time actually harms children's vision and puts them at a higher risk of developing myopia, that can lead to serious eye problems in the future. The prevalence of myopia among children ages 6-10 years is estimated at round 40% in Europe, North America and even higher in Asia. The Generation R study which involved 5074 children in Rotterdam found an association between increased computer use and myopia at 9 years of age. The combined effect of near work including computer use, reading time and reading distance, increased the odds of myopia. McCrann et al, also found that device recorded smartphone data usage, an objective surrogate for time spent using the smartphone, was independently associated with myopia in a study of 418 students. There is a possibility that a prolonged battle against the COVID-19 virus may lead to an increase in the incidence of myopia by causing long term behavioral changes favorable for the onset and progression of myopia.

Not only eyes are affected by increased exposure to screen but behavioural changes also may arise in children such as lack of sleep, anxiety, stress, anger, loss of social skills, etc. Although school closures may be short-lived, increased access and adoption of such platforms may accelerate the widespread acceptance of digital tools in the longer term. Behavioural

changes that arise from the growing dependence on digital devices may persist even after the COVID-19 pandemic, and it is a possibility that cannot be under-estimated.

Preventive Measures

Though we cannot totally avoid screen exposure as online mode of teaching and working from home has become the 'new normal' in this COVID era, but we can definitely ensure ourselves and our children to adopt some safe screen practices and preventive measures.

- Frequent breaks: Follow the simple 20-20-20 rule which means taking a 20-second screen break every 20 minutes to look at objects 20 feet away from you.
- Frequent blinking: Staring at a screen makes us blink less often, which dries out the eyes. Blinking moistens the eyes, reducing dryness and irritation.
- Increasing the font size: This will help in maintaining a proper distance, and also avoid squinting the eyes while reading long reports.
- Screen etiquette: Learning the basic screen etiquette like adjusting brightness, resolution, and contrast for better clarity and comfort. Avoid using devices in bright sunlight. Also wipe your screen at least once every day to improve visibility.
- Regularly checking device settings: In order to minimize glare as much as possible. Aligning your monitor to the eye level and working in ergonomic settings will also reduce the amount the eyes have to strain while focusing.
- Screen distancing: The distance between the eyes and screen should not be less than 50cm.
- Cutting reflection: Keep monitors away from windows – and never face a window when using a computer or a laptop. Anti-reflection lenses with blue coating are also helpful.
- A healthy diet: To curb the effects of digital eye strain, keep the eyes healthy by eating foods rich in vitamins and minerals, stay well-hydrated and get plenty of sleep each night. Leafy vegetables, carrots, papaya, dates are a great source of vitamin A.
- Hydrating the eyes: Omega 3 oils naturally lubricate the eyes and are found in flaxseed oil and some fish like salmon and sardines. Supplements are also available in the form of capsules. In symptomatic patients, lubricating eye drops may be advised.

Conclusion

This global pandemic has pushed the world to embrace digital technology at an unprecedented pace and scale. While digital technology comes with its benefits in a time like this, it is also important to acknowledge the long-term impact it can have in the form of various health problems. Healthy device habits must be encouraged and more awareness should be created about the effects of prolonged screen exposure among parents and schools. Thus, preventive measures must be followed as the issue of prolonged screen time is here to stay.

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Address for Correspondence:
Dr. Jyoti Deswal
H. No. 25/10A, Jasbir Colony,
Rohtak (Haryana)
E-mail: jyoti_deswal@yahoo.co.in
Mobile: 9416857905

Retinopathy of prematurity and its challenges in recent times

Subina Narang MS, **Parrina** MBBS
Government Medical College Hospital, Chandigarh.

Abstract

India is home to the largest number of preterm babies. About 2,00,000 babies are at risk of developing ROP every year. Of these only 10% require treatment of ROP. Retinopathy of prematurity is the emerging challenge faced by us in the present day. The review article highlights the pathogenesis and the nomenclature commonly used to describe ROP. The referral warranted ROP, treatment requiring ROP and the management of ROP have also been discussed.

Keywords : *Retinopathy of prematurity, screening guidelines, international classification of ROP, laser treatment.*

Introduction

Retinopathy of Prematurity (ROP) is a vision threatening disease of preterm babies which was first described in 1942.[1] It was defined as a complex disease of retina involving abnormal proliferation of retinal blood vessels in premature infants. Once considered to be a virtually untreatable condition, this condition can be tackled at the earliest stage with proper management guidelines. Neonatal care and socioeconomic status of the country play important role in deciding the visual outcome in babies suffering from this disease. As India is seeing a great deal of preterm births and their increased survival rates immense increase in the ROP burden is expected in near future.

Epidemiology

There have been three distinct periods of "ROP epidemics".[2] First epidemic was in the early 1950s. Second epidemic occurred in the late 1960s. The third epidemic involved middle to low-income countries and was first recorded in the 1990s and is still on going. India is presently facing the third epidemic of ROP. The phenotype of ROP in Asian countries affects higher birthweight and higher gestational age premature babies. It is estimated that around 14.5 million children are born premature out of which 1,84,700 children develop any stage of ROP out of which 20,000 suffers blindness or severe visual impairment worldwide.[4] It was reported that around 60-65% of premature babies are born in South Asia and Africa.[4] India accounts for 3.5 million preterm births worldwide. According to the United Nations Children's Fund (UNICEF) survey, it was estimated that around 21 million newborns were having low birth weight (LBW). India is contributing around 1.7 million cases (weight=<2500 g) and 0.4 million (weight=<1500 g), which is third highest incidence rate for LBW.[5]

In high-income countries where most of the babies have an access to neonatal care, ROP-associated blindness is uncommon. But middle-low income countries like India, where there is a lack of infrastructure and capacity ROP related blindness becomes as high as 40% among total cases of childhood blindness, and incidence of ROP may vary between 38%-51.9%. In India, about 2,00,000 are at risk to develop ROP every year considering an incidence of preterm births out of which if about 10% develops treatable ROP, numbers of newborns requiring treatment will be 20,000 every year creating India to be among the worst affected countries with this disease.[6]

Pathogenesis

The ROP pathogenesis is multifactorial where developmental, genetic, and environmental factors come into play. Vascular development in the pathogenesis of ROP is vital. Recently, Flynn et al. divided the retinal vascular development into two phases.[7]

Vasculogenesis The first phase of retinal development begins around gestational age of 14 weeks to 21 weeks. Vascular Precursor Cells (VPCs from mesenchyme) exit the optic nerve and form the four major vessel arcades of the posterior retina

during this phase.

Angiogenesis The second phase involves the endothelial cells proliferation and capillary formation from the existing blood vessels of first phase.

Usually, by 8 months of gestation, the nasal retina gets vascularized and the temporal retina is usually formed by around gestational age of 40 weeks. Thus, at birth preterm babies have a variable extent of incompletely vascularized peripheral retina. After birth, the physiological hypoxia present in-utero is decreased, and after birth the infant is exposed to hyperoxia. The effect of oxygen on the retina on the immature vasculature has been described in two stages-

(1) Primary stage- This is vasoconstrictive stage occuring due to hyperoxia contributing to delayed retinal vascularization and there is reflex vasoconstriction of the capillaries and finally vaso-obliteration. This in turn leads to the suppression of VEGF.

(2) Secondary stage - This is vasoproliferative stage which starts with the shifting of the baby from oxygen to room air. The avascular retina and the developing retinal neurons suffer injury due to hypoxia leading to a sudden surge of VEGF into the vitreous cavity. At this time, there is rise in IGF1 levels which facilitates the effect of VEGF on the retinal angiogenesis resulting in abnormal proliferation of new vessels into the vitreous and tortuosity with dilatation of the retinal vessels with neovascularisation.

Other than VEGF, the role of Cytokines like tumor necrosis factor (TNF-α), interleukin-1β (IL-1β), and IL-6 acting as primary initiators of inflammation following infection or tissue damage were also documented but poorly investigated.[8]

Many house-keeping genes also contribute to the development of the retina and it is suspected that genetic predisposition leads to an increased risk of development of ROP. These genes include angiotensin-converting enzyme (ACE), vascular endothelial growth factor (VEGF), tumor necrosis factor (TNF), endothelial nitric oxide synthase (eNOS), familial exudative vitreoretinopathy (FEVR) causing genes- FZD4, LRP5, TSPAN12, and NDP and variants in complement genes - CFH, CFB, FBLN5, CETP and CXCR4.[9]

Risk Factors

ROP is a multifactorial disease where the prematurity itself is the most consistent and major contributing risk factor for ROP.

One of the largest multicentric trial on ROP, Cryotherapy for Retinopathy of Prematurity Cooperative Group (CRYO-ROP), reported that for every 100 gms increase in birth weight, there is a 27 % decrease in the percentage of threshold ROP and also gave incidence among different gestation period (28-29 weeks= 83%; 30-31weeks=60%; 32-33 weeks=50%).[10] Various other postnatal factors like use of supplemental oxygen, multiplicity, sepsis, apnoea, surfactant therapy, mechanical ventilation, neonatal hyperbilirubinemia, patent ductus arteriosus, intraventricular hemorrhage, double volume exchange transfusion, administration of blood products, thrombocytopenia contribute towards ROP.[1-15] The weight gain in post-natal life could be a predictor for ROP. WINROP (weight, insulin-like growth factor, neonatal ROP) study revealed that weight gain less than 50% of the birth weight by 6 weeks of life predicts the development of ROP with 100% sensitivity and IGF-1 levels at birth had 90% sensitivity for ROP prediction.[16] The use of oxygen, especially in bigger Asian babies has been reported as a risk factor for developing Aggressive Posterior ROP (APROP) in India.

The Supplemental Therapeutic Oxygen for Prethreshold Retinopathy of Prematurity (STOP-ROP) trial 11 has suggested that there was no difference in ROP among two groups who were given different concentration of oxygen (96%-99% SaO2 vs 89%-94% SaO2) while the Surfactant, Positive Airway Pressure, Pulse Oximetry Randomized Trial (SUPPORT) and Benefits of Oxygen Saturation Targeting Study II (BOOST II) study reported less ROP in 85%-89% SaO2 group as compared to 91%-95% SaO2 group but with increased mortality rates and greater survival rates in 91%-95% SaO2 group.[17] In general consensus is to target oxygen levels greater than 90% saturation in order to reduce morbidity rate. Combination of one or more of the above reported factors maybe be important which requires further studies.

Maternal risk factors - Pre-eclampsia, maternal

essential hypertension, diabetes, and antihistaminics drug use. All these factors require further studies.[18]

In a recent meta-analysis any amount of human milk feeding was significantly associated with lower incidence of ROP.[19]

Screening of ROP

ROP screening guidelines has aided in detecting newborns who are at risk of developing ROP and helped in closely monitoring their retinal development. These guidelines have helped to identify the severe form of disease requiring treatment and saving children's eyes from the unfavorable outcome which may lead to childhood blindness due to ROP. The screening guidelines are different in different countries depending upon the ROP phenotype. [26,27]

In India, it has been observed that approximately 13.3 % - 22.6% of ROP babies can be missed using the American and British screening guidelines.[28,29] Hungi et al has reported ROP incidence in India up to 57.6% in older and heavier babies than the American guidelines for ROP screening.[29]

National Neonatology Forum of India gave ROP screening guidelines[30] that are being followed since 2010:

a) Infants born < 34 weeks of gestation and/or weighing <1750 g or

b) Heavier (1750-2000 g birth weight) or older babies (34– 36week gestation) if they have attending risk factors like mechanical ventilation, prolonged oxygen therapy, hemodynamic instability, or adverse respiratory or cardiac disease profile.

Rashtriya Bal Swasthya Karyakram (RBSK) formulated by the Ministry of Health and Family Welfare launched in June 2013 is a government initiative which has included ROP screening. In order to ensure screening of all eligible neonates, emphasis is laid on the first screening of ROP by day 30 of life irrespective of the gestational age or initial 3- 4 weeks after birth. Infants < 28 weeks or < 1200g should be screened earlier than this, at 2–3 weeks of age to enable early identification and treatment of ROP.

Screening of ROP has to be done by a trained ophthalmologist in the presence of peadiatrician under pupillary dilatation. The pupils are dilated using two drops of Phenlepherine 2.5%, Cyclopentolate 0.5% at 20 minutes interval. One must not forget to wipe the access drops as these could be life threatening to small babies due to absorption by the skin. Eye speculum with wire Vectis for globe rotation can be used by a beginner. These are also required for p[roper documentation of the disease. can be used

Telescreening in ROP

RetCam –based screening

Although, in ROP screening indirect ophthalmoscopy remains the gold standard, image-based examination and screening are gaining popularity. The introduction of digital widefield imaging systems has made it possible to perform fundus imaging using RetCam. This technique gives the dynamic image of the retina of the child and facilitates data transfer among ophthalmologists and from technicians to ophthalmologists for early referral. A set of five images of central, extreme nasal, temporal, superior and inferior field to visualize as much retina as possible is sufficient for telescreening. The use of 130 field of view lens usually suffices.

In the Karnataka Internet Assisted Diagnosis of Retinopathy of Prematurity (KIDROP) programme (www.kidrop.org) in India, the Neo camera (developed in India) was used to train doctors, ophthalmic imagers, optometrists, nurses, and paramedics, who were appointed not only to image but also to analyze the images at the first point of contact and it was seen decision making regarding treatment and referral had 93% specificity and 96% sensitivity. Positive predictive value (PPV) was 81.5% as compared to ROP experts. SUNDROP model reported 93.8% PPV.31 Thus, this recent advancement has provided a distinguished gateway for mass-telescreening in remote locations.

Classification of ROP

ROP was first classified in 1987 by The International Classification of Retinopathy Of prematurity (ICROP) to give one common language to the disease. The location of the disease was described on the basis of the three zones (anteroposterior location) all of which were centered around the disc and stages (severity) depending upon the presence of arteriovenous shunt vessels and neovascularisation.[20]

In 2004, The Early Treatment for Retinopathy of Prematurity (ETROP) study grouped ROP into Type 1 and Type 2 ROP.[21] The study concluded that a wait and watch approach with weekly or even twice weekly, follow-up of Type 2 ROP eyes. These presently qualify as referral warranted eyes needing close observation. These eyes should be considered for treatment on progression to Type 1 ROP which at present are also called treatment requiring eyes.[22] In 2005 ICROP study was revised and included the aggressive posterior ROP (APROP) to the earlier classification.[23] ROP is divided into zones depending on the location and stage depending on the clinical appearance of the disease.[24]

Zone I - Circle centered on the disc with a radius twice the distance from disc to the center of macula. Clinically it is recoganized by using +28 Dioptre lens. If the rim of the field of view by +28 D lens touches the nasal rim of the optic disc you are visualizing zone I of the retina.

Zone II- A doughnut-shaped area extending from the nasal edge of zone I to ora serrata nasally and up to the anatomic equator temporally.

Zone III - The outermost residual temporal crescent of retina anterior to zone II. To be sure of zone III, one must visualize the vessels reaching the nasal ora serrata for atleast two clock hours

The stages of ROP define the clinical appearance of the retina at the junction of the vascularized retina and the avascular area. There are 5 stages of ROP –

Stage 1- A demarcation line is seen between the vascular and avascular retina. It is a thin structure that lies in the plane of the retina. The vessels are dilated and tortuous (Figure1a&b).

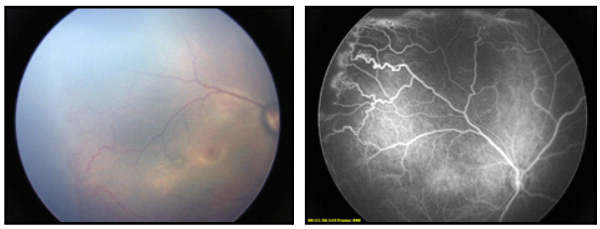

Fig.1 : Stage1 ROP
(a) Fundus picture (b) Fluorescein angiography picture

Stage 2 - The demarcation line grows to occupy a volume and has a height and width to form a ridge above the plane of the retina. Arterio-venous shunts are seen on FFA.

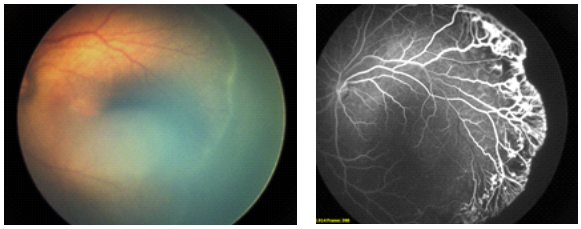

Fig.2 : Stage 2 ROP
(a) Fundus picture (b) Fluorescein angiography picture

Stage 3 - Ridge with extraretinal fibrovascular proliferation into the vitreous. It may be continuous or non-continuous and is posterior to the ridge. Extraretinal proliferation can be seen as fibrovascular tufts on the ridge or as haemorrhagic spots on the ridge (Figure 3)

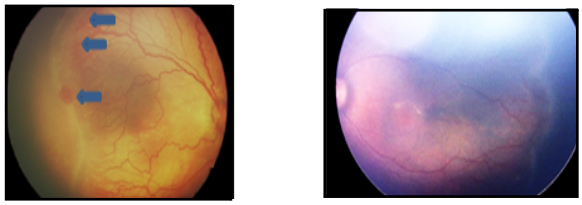

Fig.3 : Stage 3 ROP Fundus picture

Stage 4 – Subtotal retinal detachment (RD)

Subtotal retinal detachment may or may not involve fovea and is classified accordingly as shown in Figure 4 (a) and 4 (b).

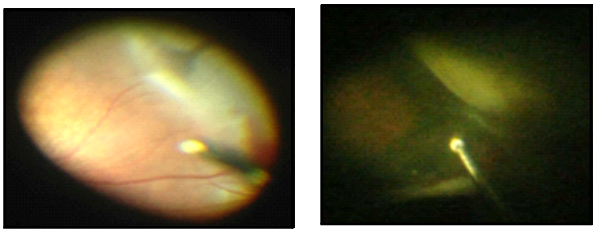

Fig. 4 (a) :RD not involving the fovea
Fig. 4 (b) :RD involving the fovea

Stage 5 - Total retinal detachment which could be of 4 types: open funnel, closed funnel, closed anterior funnel with open posterior funnel or open funnel anteriorly and closed funnel posteriorly.

ETROP[22] study classified ROP into

Type I ROP: Also called high-risk pre-threshold disease (Treatment requiring)

a) Zone I, stage 1, 2, or 3 with plus disease

b) Zone I, stage 3 with or without plus disease

c) Zone II, stage 2 or 3 with plus disease

d) APROP in any Zone

Type II ROP: Known as low-risk pre-threshold disease (Referral warranted)

a) Zone I stage 1 or 2 without plus disease

b) Zone II stage 3 without plus disease

Depending upon the vascularity ROP can be divided into:[27]

Pre Plus disease - Posterior pole vascular dilation and tortuosity which is more than normal but less than plus disease.

Plus disease – Dilatation of posterior veins and tortuosity of arterioles in at least two quadrants. The arteries show totuousity in the posterior pole however in the periphery both arteries and veins show tortuosity. There is growing interest in the quantification of the plus disease using Retcam images and artificial intelligence.

Aggressive posterior ROP (APROP) - Rapidly progressive form of ROP with posterior location, severe-plus disease, and flat intraretinal neovascularization. This may progress directly to stage 5 ROP if not adequately treated in time as shown in Figure 5 (a) & (b).

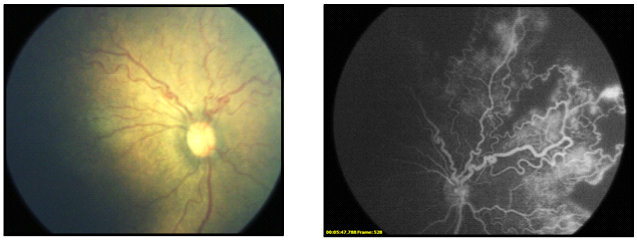

Fig. 5 (a) : Fundus picture of APROP

Fig. 5 (b) : FFA in APROP

Threshold ROP was used for treatment in CRYO-ROP study. It is defined as zone I or II Stage 3 ROP, having five contiguous or eight cumulative clock hours with plus disease.

Pre-threshold ROP includes Type 1 ROP (high-risk pre-threshold) and Type 2 ROP (low-risk pre-threshold)

Spontaneously regressed ROP It is defined as " variable changes seen in spontaneously involuted ROP depending on the severity of initial disease".[24] Changes like abnormally branching or telangiectatic retinal vessels, pigmentary changes, lattice-like degeneration, vitreous membranes, localized tractional detachments, and even retinal breaks may be present. The posterior pole may show tortuous vessels, narrow temporal arcade, disk drag, macular heterotropia, and falciform folds.

Atypical and hybrid forms of ROP: These are not uncommon in the Indian scenario. Eyes with ROP may demonstrate both the flat neovascularization seen in APROP as well as the ridge of staged ROP.[25] The presence of plus disease helps in guiding timely treatment in these eyes. In some cases, only a few vessels arise from the optic disc which does not extend beyond the fovea. This form of severe ROP with poor vascular development is called posterior zone 1 ROP or half zone ROP as shown in Figure 6 (a) & (b).

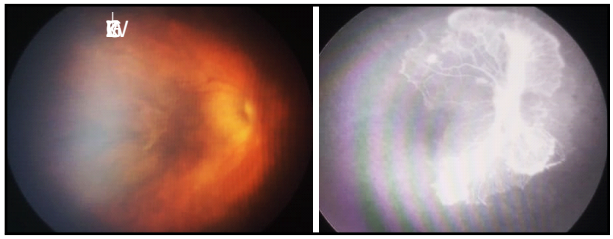

Fig. 6 (a): Fundus picture in half zone ROP

Fig. 6 (b) : FFA in half zone ROP

- Each preterm baby requires repeated visits before reaching the stage warranting treatment. Each avascular retina needs to be followed up till mature retinal vessels are formed till ora serrata, ROP develops and regresses or ROP develops and is lasered. Only 10% of screened babies require treatment

Fundus fluorescein angiography and ROP

The gold standard of treating and diagnosing ROP continues to be indirect ophthalmoscopy. Fundus fluorescein angiography helps us in understanding the pathogenesis and the course of the disease. Klufas et al in his study concluded that the addition of FFA images to color fundus photographs resulted in significant increase in sensitivity for the diagnosis of ROP.[32]

Lepore et al published an atlas of uorescein angiographic ndings in ROP. FFA was seen to clearly de ne the zone I junction between vascularised and non-vascularised retina. The authors also noted the different pattern of vessel branching at the junction between the vascular and avascular retina (AV junction) showing hyperfluorescent cotton wool-like or popcorn-like lesions, focally dilated capillaries, capillary tuft formation, and rosary bead-like lesion

inside vessels of ROP babies.[33]

Azad et al have demonstrated the safety of RetCam assisted FFA in babies with ROP in India. They concluded that in addition to being safe intravenous FFA can help in early diagnosis, prompt management and documentation of complete regression of ROP.[34]

Treatment of ROP

Two large multicentre trials have been the backbone in the treatment of ROP which includes the CRYO-ROP and ETROP study.[22,24]

In CRYO–ROP study, Cryotherapy was used for freezing the full thickness of the avascular retina from the external ocular surface, done in patients with threshold ROP. This trial reported that cryotherapy can decrease the unfavorable outcome to 21.8% compared to 43% in eyes who are left untreated. With the introduction of laser treatment in ROP, both cryotherapy and CRYO-ROP guidelines are no longer used. Cryotherapy was to be done under general anaesthesia and lead to a lot of adnexal reaction.

Laser therapy: Confluent laser spots of the avascular retina in ROP is gold standard treatment for ROP. The National Eye Institute in 1999 funded the ETROP trial where the Laser treatment was given to babies with ROP. This proved to be a major help in the treatment of zone 1 ROP. According to the ETROP study, ROP babies falling into High-risk ROP as described following were given treatment:

1) Zone 1, Stage I to III ROP with plus disease
2) Zone 1, Stage III ROP without plus disease
3) Zone II, Stage II to III ROP with plus disease
4) Aggressive posterior ROP

Follow up visits till spontaneous regression of ROP required falling under low risk :

1) Zone 1, Stage I to II ROP without plus disease
2) Zone 1I, Stage I to III ROP without plus disease
3) Peripheral avascular retina without any stage of ROP

In India, laser therapy of the avascular retina is usually done under topical anaesthesia and a sugar pellet may be used to facilitate the laser treatment. Follow ups are done at least once weekly for zone 1 ROP and one to two weeks for Zone II ROP till ROP regresses or progresses to treatable ROP.

The study concluded that laser therapy in Type 1 ROP could reduce the unfavorable from 15.6% to 9.1%. Various studies have reported overall favorable outcome in 86-93% for threshold disease and 100% in pre-threshold disease treated with diode laser

Anti–VEGF therapy - Introduction of anti-VEGF treatment has shifted the trend of treatment for ROP towards pharmacotherapy. Giving Intravitreal bevacizumab as an initial monotherapy can cause regression in 88% cases of type 1 ROP with only 9% requiring additional laser treatment and 1% requiring additional injection.[35] Another randomized clinical trial, Bevacizumab Eliminates the Angiogenic Threat of ROP (BEAT-ROP) results were compared between the anti-VEGF (bevacizumab- 0.625 mg in 0.025 ml used) group and conventional laser group for treating stage 3 + ROP having zone I or II posterior disease and has shown the superiority of anti-VEGF treatment over conventional laser therapy for infants for zone I but not in zone II disease. The rate of recurrence was 26% vs 6% between laser and anti-VEGF groups.[36] The role of more and more anti-VEGF agents are being investigated as an adjunctive or alternate therapy. Ranibizumab 0.2mg was found to be superior than laser therapy with fewer unfavorable ocular outcomes in multicentric RAINBOW study. [37] In one comparative study, the efficacy of intravitreal pegaptanib and laser photocoagulation was investigated and it was seen that 89.7 % of injection treated eyes had favorable anatomic outcomes with stable regression of ROP as compared to only 60.8 % of laser-treated eyes[38]. Recently, one-year outcomes of intravitreal aflibercept injection were evaluated in one of prospective nonrandomized interventional case series study in 26 eyes with type 1 pre threshold ROP and it found that favorable anatomical and visual outcome were present in 96% and 80% of eyes respectively.[39] Ranibizumab and bevacizumab have been compared and similar efficacy was found in causing regression of ROP.

But numerous studies in the literature have reported late recurrence even after pharmacotherapy as anti-VEGF effects remain for 6 weeks post intravitreal injection. Reactivation has been reported even after 3 years of treatment. It has been now used as primary therapy for APROP, Agressive anterior ROP, or poor media clarity due to posterior disease to improve visualization for laser treatment or persistent

neovascularization, tractional elements, and tractional retinal detachment before surgery which occurs following failed laser treatment.

Surgery - Surgical modalities including scleral buckling, lens-sparing vitrectomy, and lensectomy with vitrectomy are indicated for Stage 4A, Stage 4 B, and Stage 5 ROP depending upon stage, extent, and location of the traction. Lens sparing vitrectomy (LSV) is the most commonly performed and preferred surgery for Stage 4 ROP giving gratifying results. Lensectomy with vitrectomy is the surgery for stage 5 ROP. Best anatomical and functional outcome seen in stage 5 ROP are in open funnel configuration. The visual outcome for stage 5 is very poor and can lead to permanent visual impairment. Various studies in the literature have reported anatomical success ranging from 84 – 100% after LSV for Stage 4A ROP and 14.3% - 45.5% in Stage 5 ROP.[40]

Follow-up - The therapy for ROP goes much beyond the management in infancy. Once the baby develops ROP, a long term follow-up is required in these babies to give them useful vision. These babies have high incidence of sequelae like refractive error, strabismus, anisometropia, amblyopia, glaucoma, cataract, disc and macular drag, retinal breaks, retinal detachment etc.

Conclusion

Retinopathy of prematurity has become a modern epidemic that recently emerged as a worldwide health problem. Since improvements in neonatal care in recent decades, the rise in ROP has been noted recently. New operational guidelines have provided a more comprehensive approach to managing the disease. But still, there is more research is required in the field of ROP to decrease the overall financial burden on the country.

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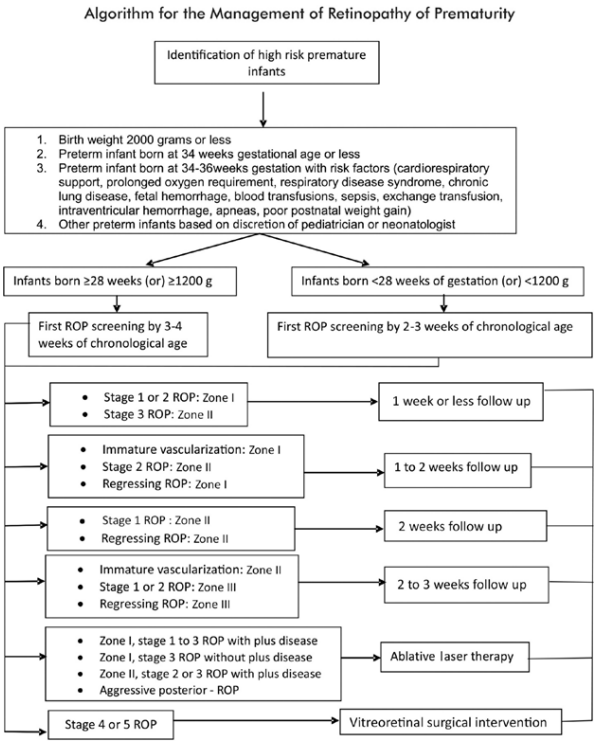

Figure 1: Algorithm for the follow-up and management of retinopathy of prematurity. (Reproduced with permission from "Project operational guidelines. Prevention of Blindness from Retinopathy of Prematurity in Neonatal Care Units, https://phfi.org/wp-content/uploads/2019/05/2018-ROP-operational-guidelines.pdf")

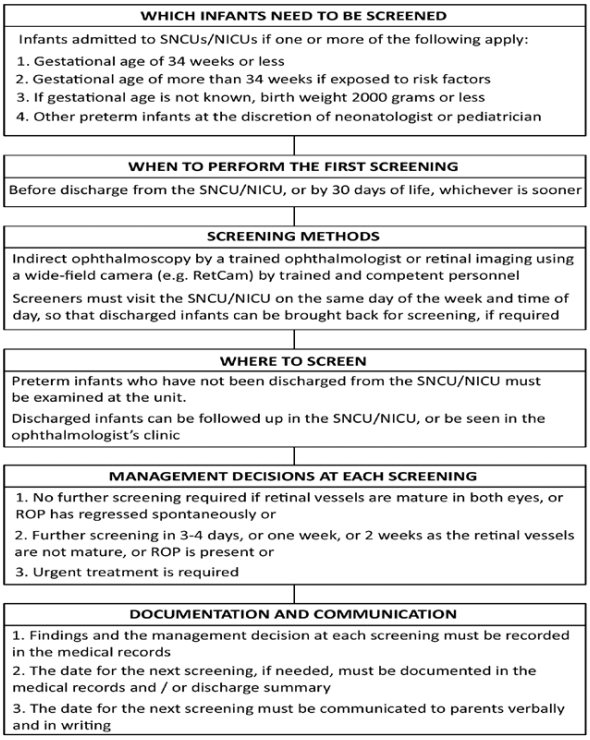

Figure 2: Retinopathy of prematurity screening guidelines. (Reproduced with permission from "Project operational guidelines. Prevention of Blindness from Retinopathy of Prematurity in Neonatal Care Units, https://phfi.org/wp-content/uploads/2019/05/2018-ROP-operational-guidelines.pdf")

WHICH INFANTS NEED TO BE TREATED
Infants developing signs of sight threatening ROP in one or both eyes
WHEN SHOULD TREATMENT BE GIVEN
Within 48 hours as the disease can progress very fast to retinal detachment
WHO SHOULD TREAT
An ophthalmologist trained and competent in treating sight threatening ROP
METHOD OF TREATMENT
Laser treatment delivered by indirect ophthalmoscope to the avascular peripheral retina Pain control and /or sedation are required. Staff trained in neonatal resuscitation must be present Note: Once retinal detachment has occured, complex vitreoretinal surgery by an expert can prevent blindness in some cases
WHERE TO TREAT
In the SNCU/NICU if an inpatient. If discharged from the SNCU/NICU: in an eye department if infrastructure and personnel are available
FOLLOW UP AFTER TREATMENT
Ophthalmologist should follow up at one week. Further laser treatment may be required
LONG TERM FOLLOW UP
Infants treated for sight threatening ROP are at high risk of refractive errors and other eye conditions. They should be follow up until at least 5 years of age

Figure 3: Retinopathy of prematurity treatment guidelines. (Reproduced with permission from "Project operational guidelines. Prevention of Blindness from Retinopathy of Prematurity in Neonatal Care Units, https://phfi.org/wp-content/uploads/2019/05/2018-ROP-operational-guidelines.pdf**)**

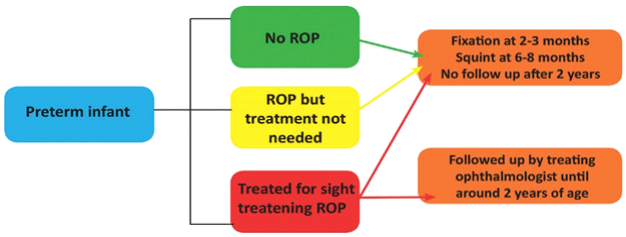

Figure 4: Ophthalmic evaluation guidelines for preterm infants. (Reproduced with permission from "Project operational guidelines. Prevention of Blindness from Retinopathy of Prematurity in Neonatal Care Units, https://phfi.org/wp-content/uploads/2019/05/2018-ROP-operational-guidelines.pdf**)**

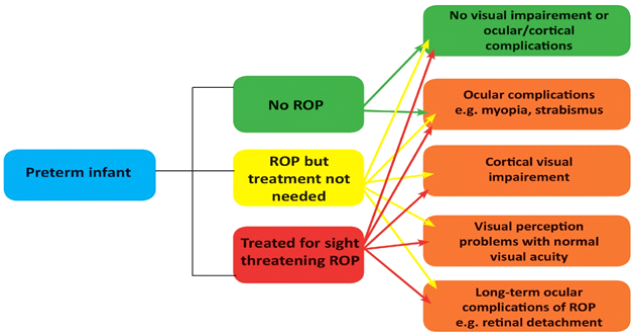

Figure 5: Ocular and cortical morbidity in preterm infants – follow-up evaluation guidelines. (Reproduced with permission from "Project operational guidelines. Prevention of Blindness from Retinopathy of Prematurity in Neonatal Care Units, https://phfi.org/wp-content/uploads/2019/05/2018-ROP-operational-guidelines.pdf**)**

Address for Correspondence:
Dr. Subina Narang
Professor Govt. Medical Hospital,
Sector-32, Chandigarh
E-mail : subina.navya.sn@gmail.com
Mobile : 9646121587

Exfoliation syndrome

Sahebaan Sethi MS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Exfoliation syndrome , previously known as pseudoexfoliation syndrome, is characterized by the production and progressive accumulation of a fibrillar extracellular material in lens, ciliary body, zonules, iris, trabecular meshwork, corneal endothelium, conjunctiva and orbital structures. Ocular hypertension or glaucoma develops within 10 years in approximately 40 percent, a risk approximately a ten times higher than that found in the general population.Annual checkups for early detection of glaucoma should be done so that medical management,laser trabeculoplasty and glaucoma surgery can be planned.This would help preserve the visual acuity for a longtime.

Keywords: *Artificial intelligence, ophthalmology, diabetic retinopathy, digital screening, machine learning.*

Introduction

Exfoliation syndrome (XFS), previously known as pseudoexfoliation syndrome, is an age-related disease characterized by the production and progressive accumulation of a fibrillar extracellular material in ocular tissues throughout the anterior segment like lens, ciliary body, zonules, iris, trabecular meshwork, corneal endothelium, conjunctiva and orbital structures. It is now known to be a systemic disease [1,2] related in part to genetic and environmental factors with close associations with systemic pathology, including cardiovascular and cerebrovascular diseases[3].

Pathophysiology

Exfoliation syndrome an ocular manifestation of a systemic disease. Defects in elastin metabolism has been proposed to be responsible for the synthesis of exfoliative material since association with specific mutations of the lysyl oxidase-like protein 1(LOXL1) gene, which is important in elastin metabolism, is strongly associated with XFS and exfoliation glaucoma(XFG). Histochemically, exfoliative material is made of glycoconjugates surrounding a protein core. The origin of exfoliative material is unclear; however, evidence suggests emergence from intraocular cells (trabecular and corneal endothelium, ciliary and lens epithelium and iris) and extraocular cells (fibrocytes, vascular and muscle)[4].

Ocular pathology associated with XFS includes peripupillary iris depigmentation, trabecular meshwork hyperpigmentation (an early feature), secondary open-angle and/or angle-closure glaucoma, cataract, lens subluxation, corneal endothelial compromise and central retinal vein occlusion[5].

Clinical Features

XFS is three times more common in females than males[6]. This condition rarely occurs before 50 years of age and the incidence increases steadily with age. XFS typically presents unilaterally. The fellow eye develops signs of exfoliation in more than 40% of cases, but exfoliation material can almost always be demonstrated in fellow eyes on electron microscopy and conjunctival biopsy[7].

Lens The most commonly recognized feature is the 3-ring sign on the anterior lens capsule, formed by a central disc, a peripheral ring, and a clear zone, in between(Figure 1).

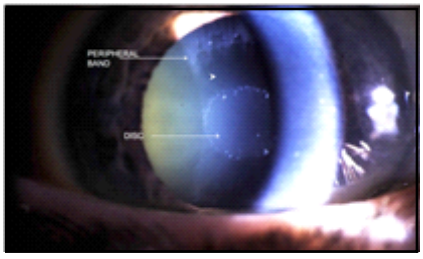

Fig. 1 : Image of exfoliation material on the lens capsule, demonstrating classic findings of central disc, lucid interval (arrowhead) and peripheral band.

The clear zone varies in diameter and may exhibit curled edges. The central disc measures 1-2.5 mm in diameter and has well-demarcated borders. The

peripheral ring typically is seen after pupillary dilation. Its size is variable, and its inner border has many radial striations. The translucent zone most likely is created by the physiologic rubbing of the posterior surface of the iris against the lens.

Iris Fine flaky white material can be seen on the pupillary border of the iris without dilation(Figure 2).

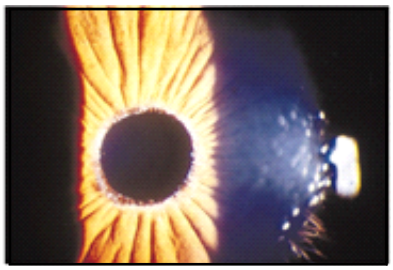

Fig. 2 : XFM on pupillary border. Photograph courtesy of Dr Robert Ritch, MD. XFS, exfoliation syndrome.

Physiological rubbing of iris against the lens scrapes the pseudo exfoliative material from the surface of the lens. This scraping results in a secondary pigmentary dispersion syndrome, with a loss of melanin from the iris pigment epithelium at the pupillary margin adopting a sawtooth-like morphology. Peripupillary iris atrophy is a common and distinctive finding. It is best visualized using infrared transillum-ination (Figure 3).

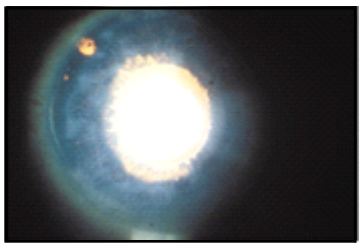

Fig. 3 : Typical diffuse iris sphincter region transillumination. Photograph courtesy of Dr Robert Ritch, MD. XFS, exfoliation syndrome.

Pupil Eyes with XFS often dilate poorly[8]. Eyes with XFS may also constrict less well to topical 4% pilocarpine[9]. Even without mydriatics, the pupil in the involved eye may be smaller[10]. Pigment dispersion in the anterior chamber is common after pupillary dilation and may be profuse causing marked IOP elevation[11].

Cornea Scattered flakes of pseudoexfoliative material may be observed on the endothelial surface of the cornea. A greater than normal frequency of cornea guttata in eyes with XFS has been suggested[12].

Zonules and Ciliary Body Exfoliation material(XFM) may be detected earliest on the ciliary processes and zonules and this creates a tendency to spontaneous subluxation or dislocation of the crystalline lens in advanced cases.

Anterior Chamber Angle Gonioscopy shows a discontinuous pigmentation of the trabecular meshwork, pigment characteristically is deposited on the Schwalbe line or anterior to the Schwalbe line (the Sampaolesi line)(Figure 4).

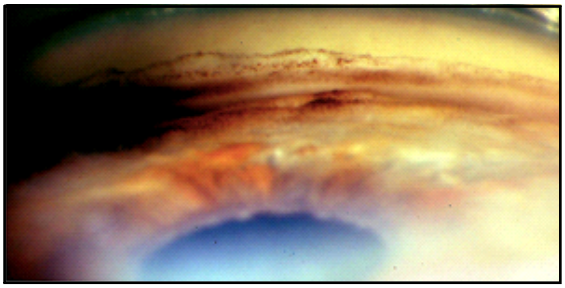

Fig. 4 : Goniophotograph of an angle of an eye with XFS demonstrating variegated (irregular) pigment. There is also a meandering line anterior to the meshwork in the peripheral cornea termed 'Sampaolesi's line'. This feature is also commonly seen in XFS, particularly in the inferior angle; however, it is non-specific for the condition. Photograph courtesy of Dr Robert Ritch, MD. XFS, exfoliation syndrome.

A high incidence of narrow or occludable angles in eyes with pseudoexfoliation has been reported, sometimes with peripheral anterior synechiae.

Vitreous After cataract extraction, XFM may be found on the vitreous face or on vitreous strands when the face is ruptured, on the posterior capsule and on IOLs indicating that the presence of the lens is unnecessary for its continued formation.

Glaucoma in Exfoliation Syndrome

Glaucoma is a secondary event. Exfoliation syndrome itself does not cause optic nerve damage. Glaucoma occurs more commonly in eyes with XFS than in those without it, about six times more[13]. Glaucoma in XFS has a more serious clinical course and worse prognosis than POAG. There is a significantly higher frequency and severity of optic nerve damage at the time of diagnosis, worse visual field damage, higher

baseline IOP, greater diurnal fluctuation of IOP, poorer response to medications, more severe clinical course, more rapid progression, and more frequent necessity for surgical intervention[7]. Exfoliative glaucoma undergoes periods of exacerbations and remissions and appropriate management.

Mechanism of Open Angle Glaucoma

Blockage of the trabecular spaces by XFM promotes accumulation of pigment and cellular debris in the juxtacanalicular tissue, which causes obstruction of the aqueous channels and limits access to the Schlemm canal that leads to narrowing of the canal lumen, collapse of its walls, disruption of its endothelium, and partial obliteration.

Mechanism of Angle Closure Glaucoma

Zonular laxity allows forward movement of the lens, causing decreased anterior chamber depth and pupillary or angle closure glaucoma.

Nuclear cataract is often more frequently found in eyes with XFS than in eyes without it[14]. Patients with XFS are much more prone to having complications at the time of cataract extraction[7]. Pupillary diameter and zonular fragility (and/or phacodonesis) have been suggested as the most important risk factors for capsular rupture, zonular dehiscence and vitreous loss(5-10 times more common)[15,16] and should serve as a warning sign to the surgeon.

Posterior capsular opacification is more in eyes with XFS compared to those without XFS[17]. Late postoperative decentration of IOLs and capsular bags was reported to be significantly higher in eyes with XFS and was also related to zonular weakness[18]. Capsule contraction syndrome is particularly common in eyes with XFS, particularly if the capsulorrhexis is small, and can lead to IOL displacement[19].

Work Up

History- Patients may be asymptomatic, or they may complain of decreased visual acuity secondary to cataract or glaucomatous visual field changes.

Slit lamp examination- Pseudo exfoliation syndrome is diagnosed clinically by slit lamp examination with an 85% sensitivity rate and a 100% specificity rate[20]. In an undilated eye, pupillary border may show XFM with peripupillary iris transillumination defects. On dilated examination, characteristic 3 ring sign on the anterior lens capsule may be seen. Presence of glaucomatous disc damage will indicate XFL glaucoma.

Gonioscopy shows a discontinuous pigmentation characteristically on the schwalbe's line or anterior to it (sampaolesi line).

Investigations- Preoperative assessment prior to cataract extraction includes Ultrasound Biomicroscopy to diagnose any zonular laxity or lens subluxation. Specular microscopy can be done to ascertain reduced endothelial cell density or morphologic changes in size and shape of the endothelial cells[21].

In pseudoexfoliative glaucoma, Visual Fields are confirmatory of any glaucomatous disc damage. The glaucomatous disc changes and visual fields defect pattern is the same as in POAG. Various imaging technologies like Optical coherence tomography (OCT) and confocal scanning laser ophthalmoscope (Heidelberg retina tomograph [HRT]) are also being used to document and monitor changes due to glaucomatous damage in the optic disc and the retinal nerve fiber layer. These imaging techniques are similar to that in POAG. Anterior Segment OCT can suggest presence of any synecial angle closure or reduced depth of the anterior chamber.

Differential Diagnosis

Pigmentary glaucoma: characteristic signs are Krukenberg spindle, mid peripheral iris transillumination defects and homogenously darkly pigmented trabecular meshwork/sampolesi line. It is common in young males with myopia.

Uveitis: Photophobia is a common symptom. In both exfoliative and uveitic glaucomas, corneal endothelial deposits are common. The raggered peripheral anterior synechiae of some inflammatory glaucomas in not seen in exfoliative glaucoma however angle closure due to narrow angles in not very uncommon in exfoliative glaucoma.

Capsular delamination/True exfoliation: Trauma, exposure to intense heat(glass blowers), or severe uveitis can cause peeling off of a thin membrane of anterior lens capsule.

Primary amyloidosis

- Primary open angle glaucoma
- Fuchs heterochromic uveitis

Treatment of Exfoliative Glaucoma

Medical Management

Glaucoma associated with XFS tends to respond less well to medical therapy than does POAG[22].

Prostaglandins are effective in XFL glaucoma because they facilitate trabecular outflow directly and also by having an inhibitory action on matrix metalloproteinases and reducing the formation of exfoliative fibrils[23]. Latanoprost is associated with a narrower range of diurnal IOP fluctuation[27].

XFG was reported to respond less favorably to timolol therapy than eyes with COAG[24], but some studies suggest an equal[25] or greater[26] ocular hypotensive response.

Dorzolamide is almost as effective as timolol and also is additive with it[28].

Cholinergic agents are effective and probably have a greater additive effect with beta-blockers in XFS than COAG[29].

Aqueous suppressants by decreasing aqueous secretion, result in decreased aqueous flow through the trabecular meshwork. Reduced perfusion results in failure of the meshwork to survive[30]. Continued administration of oral acetazolamide produces a reduction in outflow facility and an elevation of IOP to greater than baseline after discontinuation[31].

Theoretically, miotics should be the first line of treatment. However, frequent presence of nuclear sclerosis in such patients, chance of development of posterior synechiae and tendency to cause pigment release with miotics make them less popular[32].

Lasers

Laser trabeculoplasty is particularly effective in XFL glaucoma owing to the relatively pigmented angles however the amount of IOP reduction is modest and long-term success drops to approximately 35–55% at 3–6 years[33-36]. Also, eyes with XFS may have a greater post-laser inflammatory reaction than eyes without[37].

Selective laser trabeculoplasty (SLT) has been shown to be equivalent to Argon laser trabeculoplasty in terms of lowering IOP[38,39]. The theoretical advantage of SLT is that SLT is a repeatable procedure because it does not produce thermal damage to the trabecular meshwork.

Laser iridotomy is the procedure of choice for angle-closure glaucoma. Angle-closure glaucoma caused by anterior lens movement or subluxation may also require argon laser peripheral iridoplasty to mechanically pull the iris away from the trabecular meshwork[40].

Surgery

If IOP remains uncontrolled following medical and/or laser treatment, surgical management is warranted. Trabeculectomy has similar efficacy and safety outcomes in XFG as in POAG[41].

Glaucoma drainage device implantation is also an option, especially in eyes with previous conjunctival manipulation[42,43].

Trabeculotomy as well as trabecular aspiration (TA) have been shown to be effective in the management of XFG[44,45]. TA aims to improve trabecular outflow by removing pigment and exfoliative material[46]. Trabecular aspiration combined with phacoemulsification is more effective than cataract surgery alone in reducing postoperative IOP and the necessity for antiglaucoma medication[47] but not as effective as phaco-trabeculectomy[48].

There is some support for the use of Trabectome as well as the use of iStent, but prospective studies with longer follow-up are needed before one can recommend widespread use of these and possibly other microinvasive glaucoma surgical approaches[49,50].

Deep sclerectomy has been proposed in XFG, and one study found that patients with XFG had significantly higher success following deep sclerectomy with an implant compared with patients with POAG[51].

The role of endoscopic cyclophotocoagulation (ECP) in XFG is minimal as exfoliation material accumulates on the ciliary body and zonules, and high laser energy can result in rupture of ciliary processes with significant inadvertent haemorrhage[52].

Taken together, non-medical management of XFG is guided by mechanism, stage of glaucoma, degree of IOP elevation, ocular and systemic factors and patient and care provider preferences.

Cataract extraction in Exfoliation Syndrome

It is recommended not to delay cataract surgery. Moreover, a significant IOP-lowering effect has been found following cataract surgery in patients with PXF[53]. Plausible theories for this include: washing out of fibrillar material from the angle, structural

alterations such as deepening of anterior chamber angle, decrease in irido-lenticular contact and inflammation leading to better aqueous outflow (trabeculoplasty-like effect).

Conclusion

Key recommendations to prevent complications during cataract extraction are summarised in the box below.

Pearls for Safer Cataract Surgery [54-57]
Capsulorhexis ? Large enough to enable nucleus to prolapse into the anterior chamber as needed in order to reduce strain on zonules.
Hydrodissection ? Occasionally tap on centre of nucleus to decompress fluid pressure on weak posterior capsule.
Phacoemulsification ? Prevent anterior chamber collapse and protect corneal endothelium using cohesive as well as dispersive ophthalmic viscosurgical devices (OVD).
Cortical removal ? Use tangential vectors to carefully separate cortical material from capsular bag, particularly if a capsule tension ring (CTR) has been inserted.
Small pupil ? Consider cohesive OVD, mechanical and pharmacological dilation.
Zonular weakness? Consider CTR or capsule tension segments. If diffuse weakness, could consider intracapsular or pars plana removal.
PCIOL? Posterior chamber intraocular lens is generally well tolerated.
Postop ? Potential for fibrinoid reaction postoperatively due to weak blood–aqueous barrier. Postop management should include aggressive anti-inflammatory treatment with steroids and NSAIDs.

XFS patients have approximately a 40% chance of developing ocular hypertension or glaucoma within 10 years, a risk approximately a tenfold higher than that found in the general population58 hence they should have annual checkups for early detection of glaucoma. Exfoliative glaucoma patients should also be more frequently followed up than POAG patients as progression can occur more rapidly.

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Address for Correspondence:
Dr. Sahebaan Sethi
Arunodaya Deseret Eye Hospital,
Sector 55, Gurgaon.
E-mail: sahiba401@gmail.com
Mobile: 9600175053

Low vision aids

Suman Chahar MS, **Manisha Nada** MS DNB, **J P Chugh** MS,
Ashok Rathi MS, **Rohan Madan** MBBS, **Dixit Soni** MBBS
Regional Institute of Ophthalmology, PGIMS,Rohtak

Abstract

Visual impairment is a challenging problem worldwide. Low vision impairs the social life of patients. Low vision aids can improve patient s social life.Lack of awareness about low vision aids in practitioners as well as patients remain a barrier to their use.The article highlights the use of different kinds of low vision aids with their advantages and disadvantages.

Keywords: *Low vision, low vision aids, vision loss.*

Introduction

As per the International Statistical Classification of Diseases and Related Health problems (ICD-10) published by World Health Organization (WHO), visual disturbance and blindness is classified as H53-54.9. Low Vision (Visual impairment Categories 1 & 2) is defined as "A person with low vision is one who has impairment of visual functioning even after treatment, and/ or standard refractive correction, and has a visual acuity of less than 6/18 to light perception or a visual field of less than 10 degrees from the point of fixation, but who uses, or is potentially able to use, vision for the planning and/or execution of a task for which vision is essential".[1] Blindness (Visual impairment Categories 3, 4 & 5) is defined as visual acuity of less than 3/60 or a corresponding visual field loss of less than 10 degrees in the better eye with best possible correction. The term visual impairment includes both blindness as well as low vision.[2]

Visual impairment is a pressing public health challenge, with blindness being one of the most common disabilities world wide.[3] Globally, the number of people of all ages visually impaired is estimated to be 285 million, of whom 19 million are children.[4] The burden of childhood blindness may not seem to be large in number, but it is the second largest cause of blind-person years worldwide (following cataract

Lack of awareness among eye care practitioners about low vision management remains a barrier in the use of low vision.[7-11] Thus, in the wake of the changing trends in the management for low vision, we discuss about the current management options for low vision in this review. Quality of life is reduced in low vision persons. Thus, the purpose of low-vision rehabilitation is to improve quality of life with increasing social interaction by optical/non-optical devices.

Low Vision Aids

An optical/non-optical device that improves or enhances residual vision by magnifying the image of the object at the retinal level. Rehabilitation depends to the type of visual loss and also on individual's choice or expectations. Reading has been identified as the most common problem in patients with low vision.[12] Improvement in reading for distance as well as for near has been reported using optical aids in several studies.[13]

Indications for low vision aids

- Children : Albinism, ROP, Congenital malformation ,Optic neuropathy.
- Young adult : Keratoconus, Ocular injuries, Late manifestation of congenital malformation.
- Old age : Glaucoma, ARMD , Diabetic maculopathy, Macular degeneration ,Retinal degeneration, Chorioretinitis, Optic atrophy, Myopic degeneration.

Types of Devices

1.Optical 2.Non-optical

Basic principle of LVA -Optical LVAs are based on the fact that with sufficient magnification, the normal retina surrounding the damaged central retina can be used for central vision.

Optical LVA :

- Magnifying spectacles
- Hand magnifiers
- Stand magnifiers

- Telescopes
- Other optical devices

Magnifying Spectacles

Magnification by a convex lens is obtained by bringing the object within it•s focal length. An erect, virtual and magnified image is produced. High plus lens is used to magnify the images (Figure 1).

- Magnification is 1/4th the power of the lens.
- Suited for near and intermediate distance.
- Mono-ocular or binocular

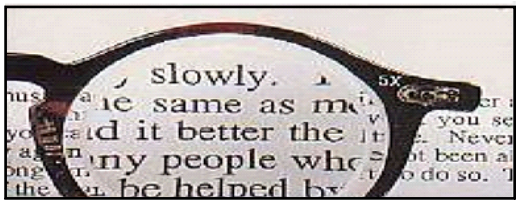

Fig. 1: Magnifying spectacles

Instructions for use - Patient should be instructed to hold the material very close and then move it out and scan the lines one by one. Light must be properly adjusted.

Advantages

- Cosmetically acceptable.
- More comfortable and easy to use.
- Both hands are free to hold the reading material.
- Field of vision is large.
- Simultaneous both near and distance vision.

Disadvantages

- Spherical aberration.
- Higher the power, closer the reading distance.
- Close reading distance causes fatigue and unacceptable posture.
- Patients with eccentric fixation are unable to fix through these.
- Illumination problem.

Hand magnifiers

- Indicated for spot or short-time tasks in patient with field of vision reduced to 10 degrees or more.
- Used for near vision (Figure2).
- Available from + 4.0 to + 40 D

Fig .2: Handheld Magnifiers

Instructions for use - The patient should be shown how to put the magnifier flat on the reading surface to begin with and raise it until the image is clear and distorted.

Advantages

- Working distance is more.
- Accommodation is not required.
- Easy to manipulate for viewing eccentrically.
- Some have light source which further enhances vision.

Disadvantages

- It occupies both hands.
- Not useful in absence of manual dexterity.
- Field of vision is limited as compared to spectacles.
- Need to be held at the correct distance.

Stand magnifiers

It forms a virtual image a short distance behind the lens. The patient needs to place the stand magnifier on the reading material and move across
the page to read. It has a fixed focus (Figure 3).

Fig.3: Stand Magnifiers

Instructions for use - Patient should be taught to place the stand magnifier flat on the reading material, and look at the image through reading glasses or bifocals to converge the divergent rays coming from the magnifier lens. Because of the reduced aperture of these magnifiers the eye must be closer to lens surface to obtain the full width of the reading field.

Advantages

- Technically simple.
- They are a choice for patients with tremors, arthritis and constricted visual fields.

Disadvantage

- Small field of vision.
- Too close reading posture is uncomfortable for the patient.
- Difficult to use if the surface is not flat .[14]

Bar magnifiers

- Contains plano - cylindrical lens.
- Magnifies the height of the letter which

becomes readable. Lies flat on the page, elongates the letter but don't separate them, magnifies in the vertical meridian only(Figure 4).

- Person with small central field who needs minimum magnification are benefitted.
- Available in low magnification power only.
- Power range 2 to 3.5 D.

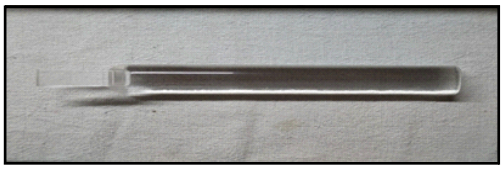

Fig. 4: Bar Magnifiers

Telescope

They magnify the apparent size of distant objects, making them appear closer to the patient. The magnification ranges from 2x to 10x. The patient has to spot the object he wishes to see and then brings the telescope in front of the eye. The optics of the telescopic systems is based on two basic principles- Galilean or Keplerian. It could be hand-held, Clip-on/spectacle-mounted or abioptic design.

Uses

- Reading from a blackboard (distance > 2m).
- Finding an entrance to a building.
- Watching games, television.
- Reading traffic signals, street signs, bus numbers.

Advantages

- One of the most popular device to enhance distance vision.
- Can be used in classroom for blackboard reading or outdoors.

Disadvantages

- Major drawback is the restriction in the field of view.
- Focusing requires good hand-eye coordination.

Bioptic telescope

It magnifying up to six times, which can be embedded into the spectacle glass. It allows the wearers to switch their sight between their regular vision and the magnified vision of the device by just a slight tilt of the head, similar to how one uses bifocal spectacles. Several recent studies have highlighted the effectiveness of bioptics for driving among the visually impaired patients. However, the evidence regarding the safety and efficacy of bioptic driving is still unclear, and laws surrounding it are ambiguous. Bioptic telescope use is legalised conditionally in some provinces in United States, Canada and The Netherlands.[14]

Other optical devices

Absorptive lenses - reduce glare and dark adaptation time(Figure5).

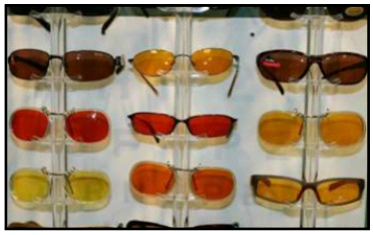

Fig. 5: Absorptive Lenses

Tinted lenses-Low absorptive high transmission are best for constant use Photochromatic lenses- Use for light sensitive person.[15]

Filters – Contrast can be enhanced by using spectacles with yellow and amber filters.

Polarization- Reduces glare.

Visual field enhancement devices

- Fresnel prism - retinitis pigmentosa. Hemianopic mirrors.
- Central scotomas and eccentric viewing - prism incorporated into reading glasses by diverting the light.

Non-optical devices

- Approach magnification
- Lighting
- Contrast enhancement
- Increased size object
- Electronic magnifiers (CCTV, LVIS, V-max)
- Writing and communication devices
- Orientation and mobility LVAs

Approach magnification

Partially sighted patients should be encouraged to move as close as possible to the object(Figure 6).

Fig. 6: Approach Magnification

Illumination

Positioning

- To the side of better eye.
- Moving light closer.

Higher levels of illumination is required : macular degeneration Glaucoma Diabetic retinopathy Retinitis pigmentosa Chorioretinitis.

Reduced illumination required

- Albinism.
- Aniridia.

Contrast Enhancement

- Using a typoscope
- Contrast modification of visual environment

Fig. 7: Contrast Enhancement

Relative size devices

Large print material , Enlarged clocks, telephones, Calendars, computer keyboards Large type playing cards (Figure 8)

Fig 8: Relative size devices

- Electronic magnifiers
- Close-circuit television
- Desktop CCTV with enhanced features (autofocus, speech commands, flat screens, text manipulations).
- Handheld cameras/portable devices.
- Head mounted systems where camera and LCD displays are combined in a single unit.

Advantages

- Higher magnification up to 70x.
- Binocularity.
- Patient can sit at comfortable reading distance.
- Hands are free for writing, etc.

Disadvantages

- Expensive.
- Need more training and practice.

Computer Education Software

- JAWS screen Reading software: It converts a normal personal computer into a talking computer so that one can learn to operate the computer independently.
- Connect out load internet and e-mail software: Access to internet through speech and braille output.
- MAGic 8.0 screen magnification system with Speech: It has a magnification range of 2x to 16x and it also reads the information aloud.

Portable Electronic Low-Vision Aids

Interest is increasing among the patients and the physicians in portable electronic low-vision aids. Some of these devices are Optelec Compact+, Optelec Compact 4HD, Schweizere Mag43, and Eschenbach Mobilux Digital, Aumed –EYE-C.

One portable artificial vision device (OrCam) is an optical character recognition device, capable of recognizing text, monetary denominations, faces, and can be programmed to recognize other objects. It consists of a miniature camera and an earpiece that can be mounted on the spectacle frame. When activated OrCam can click pictures and even read aloud any text found on the pictures that can be heard by the user via the earpiece.(16) The Or Cam was recently made commercially available in the United States and its usefulness has been elucidated in a recent study.[17]

Smart Phone/ Tablet/ Electronic Readers

With a widely available internet access, internet-ready devices like smart phones and tablets are being commonly used worldwide and off late are being increasingly used as visual aids. These devices are incorporated with features of image enlargement, high contrast screens, invert colors and bespoke apps. The textural characteristics like font size, format, word spacing, line spacing, color can also be manipulated. Several recent studies have shown that with proper training these devices can be a valuable tool for low vision patients especially as a reading aid. Another exciting ongoing development is the Google glass technology and research is on way for its use in cases of visually impaired.

Mobile friendly low vision apps

Claria Zoom- Easy to see interface for the visually

impaired. Such as - big characters and color themes
Low Vision Clear Sight - Multi-featured application for low vision having options to view enlarged contacts, camera, calculator, clock and GPS with bold color themes
Eye – D- For visually impaired it is intelligence based smart phone assistant app. It helps user to be location aware, explore and navigate to nearby places of interest.
Zoom Plus Video magnifier - It enlarges text and allows changing colors and contrast of the text and background like video magnifier.
Macular Society, Low Vision keypad free, blind and senior music player etc. are few more which can be used.

Mobility assisting devices

Patients with low vision suffer a major problem of mobility and following devices can be useful

- Long canes
- Strong portable lights

Life skill devices-

- Pre-set insulin syringe: the patient feels the pre-set level notes and knows how much to inject even if he is not able to see the markings.
- Notex: scientifically accepted device for currency identification
- Needle threader: it helps in easy threading.
- Talking clock and watches: these are readily available in the market at low cost. They have raised buttons with speech output option

Why to prescribe

The prescription of low vision devices gives the person-

- Independence.
- Increase adaptation to the daily activities material.
- Exposure to enriching experinces.
- It constitutes an important factor for socioeconomic and cultural integration.

How to prescribe

A careful clinical history is important to setting goals based on real expectations. A person who participates in selecting the aid is more likely to learn how to handle it.

- Determine the best corrected VA for near/ distance.
- Determine the visual acuity that the patient requires.
- Calculate the magnification needed to achieve the goals

Select the aid according to the characteristics of the device : the needs , goals, and clinical aspects of the patient.
To achieve better acceptance of any aid , consider the person needs, goals and ability to handle the aid, as well as the weight, cost, and timing of prescription. It is important for the person to be comfortable with the aid proposed.
For reading activities, besides achieving vision for certain size of optotype, the child should be evaluated for reading. In the presence of eccentric fixation or difficulties with proposed aid, training should precede prescription.
Each category of visual device has its advantages and disadvantages. In low vision daily practice, it is common for a child to have more than one aid.
The patient should be monitored frequently to evaluate the effective use of the aid prescribed and difficulties presented in daily activities and to set up more advanced goals.

When to prescribe

Optical aids for near vision are introduced when the reduction of the distance between the object and the eye does not allow the necessary range or when the accommodative effort is too large.
At school age, with VA up to 20/ 200, reducing the distance between the object and the eye is recommended until the second grade. From this stage, a stand magnifier or a hand magnifier can be used for reading small – print books such as dictionaries.
For VA less than 20/200 (0.1 log Mar), optical aids should be prescribed earlier if the VA is less than 20/400 and central scotoma greater than 30 degree, a video magnifier is indicated.
For VA equal to or less than 20/800 aids such as Braille and computer sound system should be included, with or without other resources. Orientation and mobility technique should be encouraged at all low vision level.
When a telescope is first prescribed, a manual, monocular telescope with lower magnification is recommended. The telescope can be prescribed for reading blackboards, and later to use constantly at far. It is important to let the child experiment with the telescope in different situations.

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Address for Correspondence:
Dr. Suman Chahar
Phogat House, 1447, Sector-2,
Rohtak- 124001 (Haryana)
E-mail: chahar.suman88@gmail.com
Mobile: 8947906258

Collagen corneal cross linking with riboflavin (C3R) Technology and Treatment

Sumpi Chinging MBBS, **Apoorva Goel** MBBS,
Chetan Chhikara MS, **R S Chauhan** MS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Corneal collagen cross-linking (CXL) is a new modality of treatment, based on collagen cross linking in the corneal stroma with the help of UVA and the photosensitizer riboflavin to increase the formation of intra and interfibrillar covalent bonds by photosensitized oxidation. In vitro studies have shown that the cornea absorbs approximately 30% of UVA light with an additional 50% of UVA absorption occurring in the lens.CXL is proven to halt the progression of corneal ectasias such as keratoconus, pellucid marginal degeneration and iatrogenic post Lasik ectasia by strengthening the corneal stroma. CXL may also be effective in the treatment of infectious keratitis due to elimination of infectious agent by UVA irradiation. Combination of CXL with refractive vision improving interventions, such as intracorneal ring segment implantation, limited topography-guided photoablation and conductive keratoplasty have shown success.

Keywords: *Corneal collagen cross-linking , riboflavin, corneal ectasia, bullous keratopathy.*

Introduction

The term crosslinking (CXL) is used to express the formation of chemical bridges. It can be formed by chemical reactions initiated by heat, pressure, or radiation, resulting in change of the physical properties of the cross-linked material. Corneal cross linking is also known as C3R or CXL or CCL.C3R. It is a new approach to increase the mechanical and chemical stability of corneal tissue. It utilizes riboflavin as a photosensitizer and UVA to increase the formation of intra and interfibrillar covalent bonds by photosensitized oxidation.[1]

In vitro studies have shown that the cornea absorbs approximately 30% of UVA light with an additional 50% of UVA absorption occurring in the lens.1 UVA absorption can be considerably increased using Riboflavin. With 0.1% riboflavin and an irradiance of 3 mW/cm2 of UVA, almost 95% of UVA light will be absorbed within the cornea reducing irradiance to 0.15 mW/cm2 (at the endothelial level), which is well below 0.36 mW/cm2, the threshold considered cytotoxic for the endothelium.[2-4]

The irradiation levels may still exceed the threshold leading to keratocyte apoptosis in the anterior stromal layer and a demarcation line between the treated and untreated cornea.2[-5]

Caporossi et al6 performed confocal microscopy analysis in humans after X-linking in vivo. They detected the effective depth of treatment by identifying distinct vertical and lateral transition areas at a depth of 270 to 330 m. The anterior stroma showed oedema with only a few keratocyte nuclei and poor reflectivity and the posterior stroma showed regular keratocyte population with normal reflectivity.

CXL changes the intrinsic biomechanical properties of cornea, increasing its strength by almost 300%.CXL is a non-surgical procedure and it is only known procedure that is proven to halt the progression of the keratoconus.

Biochemistry of C3R

The UVA 365nm irradiates the riboflavin molecules causing them to lose their internal chemical balance and produce oxygen free radicals. The riboflavin molecule is unstable, it can be stabilised by linking it to two collagen fibrils leading to formation of crossed bridge between the collagen fibrils, producing a general strengthening of the cornea.

Technique

Photo polymerization using UV-A is activated by a photosensitizer (riboflavin) and a wavelength (U V-A) which is deeply absorbed enough to protect deeper layers of the eye.

Riboflavin has two important functions in this technique; absorption of UV-A radiation and acts as a photo-sensitizer, leading to generation of reactive oxygen species.

Riboflavin and UV-A Light

Riboflavin, a naturally occurring photosensitizer is also known as Vitamin B2, a precursor of Flavin mononucleotide (FMN). UV-A has deleterious effect on the ocular structures but with the use of riboflavin as photosensitising agent, transmission rate of UV-A is only 7% across the cornea, limiting the UV-A irradiance of the lens and retina.

Physiology of collagen corneal cross linking

In this procedure, riboflavin eye drops are applied to cornea which is then activated by UV light. Riboflavin is exited into its triplet state generating reactive oxygen species (ROS) by using UVA at 365nm. Reactive oxygen species is mainly singlet oxygen and to a much less degree superoxide anion radicals; which further react with various molecule including chemical covalent bonds bridging amino group collagen fibrils.[7] Because of the absorption maximum of riboflavin at 365 nm, this wavelength was specially chosen for the treatment with UV light. This achieves 90% absorption of the UV light in a 400 m thick de-epithelialized cornea without endangering the lens or the cornea. It is caused by an increase in collagen fibre diameter due to interfibrillar and intrafibrillar covalent bonds by photosensitised oxidation cross-linking. The cornea becomes more compact due to cross linking and are more resistant to biochemical deformation or ectasia.

Exclusion criteria

- Corneal thickness less than 400 m at the thinnest position.
- Active ocular disease.
- Herpes keratitis.
- Diabetes.
- Pregnancy.
- Previous ocular surgery other than laser refractive surgery.
- Immunocompromised patients.
- Patients with known sensitivity.

Clinical indications for CL-UVR treatment

1. Progressive keratoconus

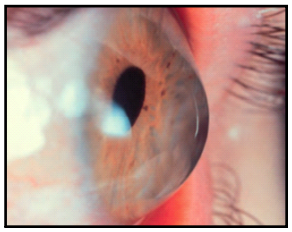

Fig.1 : Picture showing conical protrusion

Keratoconus is a corneal non-inflammatory degeneration. It is characterized by bilateral conical protrusion and thinning (Figure1). It commonly occurs in the second decade of life. Differences between normal and keratoconic corneas is seen in biochemical and immunohistochemical studies of the proteoglycans in the matrix. With CXL, covalent binding occurs between collagen molecules leads to stabilization of the collagen scaffolds and changing of several tissue properties.[8]

2. Iatrogenic post refractive keractasia (post Lasik ectasia)

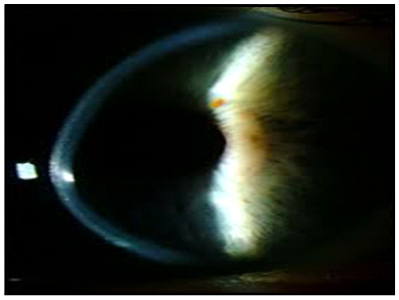

Fig.2 : Picture showing corneal ectasia

Keractasia characterized by a progressive corneal steepening, can occur centrally or inferiorly and can be associated with severe refractive changes, loss of BCVA, with stromal thinning of the cornea (Figure2). CXL should be considered for patients with post LASIK progressing ectasia to strengthen artificially weakened corneal stroma and stabilize the cornea.[9]

3. Pellucid marginal degeneration

Fig.3 : Picture showing corneal thinning in pellucid marginal degeneration

It is a rare ectatic disorder that typically affects the inferior peripheral cornea in a crescentic fashion, mostly in males, between the second and fifth decades of life (Figure3). It has been observed that patients with PMD have large amounts of against-the-rule astigmatism, making it difficult to fit in contact lenses than in patients with keratoconus.[10,11]

4. Infectious keratitis and the melting process

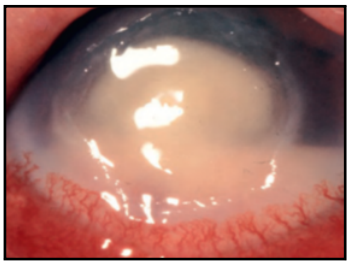

Fig.4 : Picture showing corneal melting

CXL treats corneal melts (Figure4) or infectious keratitis by strengthening of collagenolytic cornea because of cross linking and elimination of the infectious agent by UVA irradiation.[12]

5. Bullous keratopathy

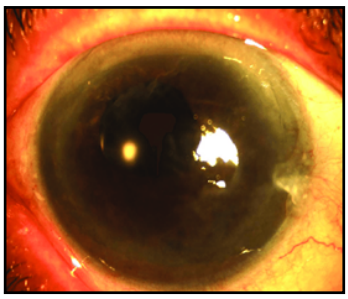

Fig.5 : Picture showing bullous keratopathy

Wollensak et al. has shown the anti oedematous effect of CXL on the cornea (Figure5). The bullous changes markedly improve and the patients do not report of any pain or discomfort.[13]

Pre-operative work up for CXL-UVR treatment

A detailed ophthalmological examination is required which includes the following:

- Visual acuity assessment (UCVA, BCVA, Contrast sensitivity)
- Intraocular pressure recording
- Slit lamp examination specially for Vogt's striae, Fleischer's ring and corneal scarring
- Slit lamp photographs
- Pentacam
- Evaluation of central corneal thickness and thinnest pachymetry
- Corneal topography
- OCT examination

Pre-requisites

- Clear cornea.
- Contact lens tolerance or good spectacle corrected visual acuity.
- Disorders should be progressive in nature.
- Thinnest corneal pachymetry higher than 400 m.
- No central corneal scarring.
- Maximum corneal curvature should not exceed 60 D.

Machine-Technical details

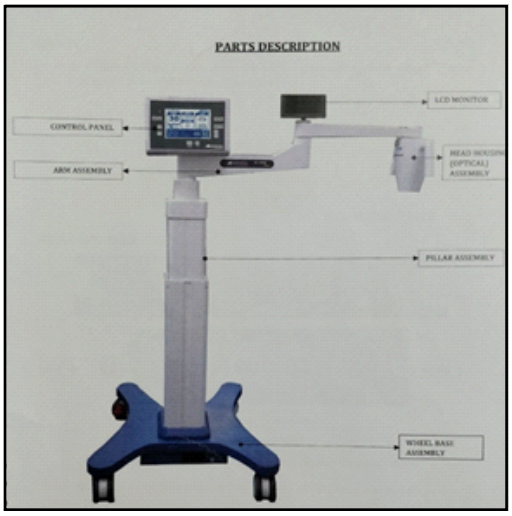

Fig.6 : Parts of machine

- UV-A light is harmful to normal eye.It is necessary to use specified goggle (shown in below Figure 7), while treating the patient.

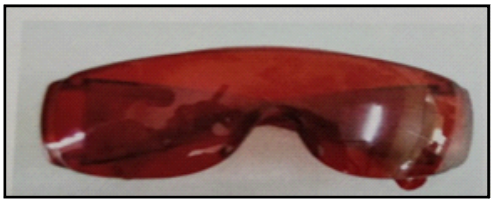

Fig.7 : Goggle

Rear panel description

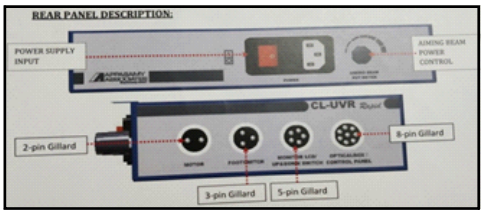

Fig.8 : Rear panel

- The input power supply (110/230V AC) is given through the power chord.
- The power switch is meant to turn ON and turn OFF the power supply to the device.
- Three Pin Footswitch connector used to connect the footswitch.
- Aiming Beam Power control Knob.

Head housing (optical) assembly

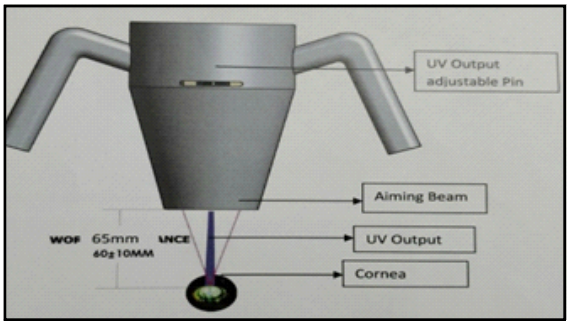

Fig.9 : Optical assembly

- Important section of machine, from where UV-A light is projected to the eye.
- Adjust two aiming beams (Red LED) to become one point (focus point) from the Head housing assembly using footswitch and it should be placed 65mm distance from the patient's eye (cornea).

Solar meter

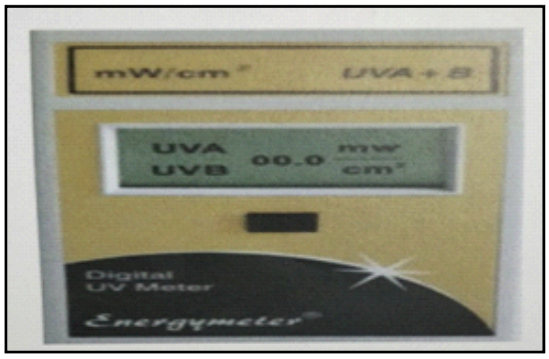

Fig.10 : Solar meter

- Used to measure UV-A light component.
  The output is expressed in terms of Mw/cm.2

Footswitch

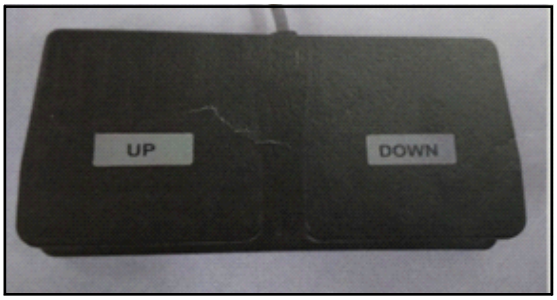

Fig.11: Solar meter

- Allows the user to move pillar up/downs.

CL-UVR rapid control panel menu description

Fig.12 : CL-UVR rapid control panel menu description

- Power control section control the power of the UV-A light source.
- Timer control section is used to preset the timer.
- Start/pause buttons are used to start and terminate the process.
- Video Asstt. is used to monitor the patient's eye position in LCD display while doing procedure.
- Save can be done in two different settings as U1 and U2 for quick access.

Surgical procedure

CXL with removal of epithelium (epithelium off)

The treatment is performed in an operation theatre under sterile conditions. Removal of the epithelium is required for efficient penetration of riboflavin.

Procedure is done under topical anesthesia after cleaning and draping of the eye. Scraping of the corneal epithelium out to 7 mm is required for better diffusion of riboflavin. Ultrasound pachymetry should be performed at the thinnest point of the deepithelialized cornea, to ensure a minimal corneal thickness of 400 m. Riboflavin solution, 0.1% in 20% dextran is instilled every 3 min for 30 min to saturate the cornea with riboflavin and for corneal hydration.

Prior to treatment, calibration of the intended irradiance of 3 mW/cm2 surface irradiance (5.4 J/cm2 surface dose) is done using a UVA meter at a working distance of 6 cm. Irradiance is performed.

After the treatment, bandage contact lens is placed after a drop of topical antibiotics is instilled. Instillation of topical antibiotics four times daily until contact lens removal is advised to the patient. The contact lens is removed on the third day and the patients are instructed to instil topical steroids four

times daily followed by a tapering schedule over 2 months.

CXL without removal of epithelium (Epithelium on or transepithelial cross linking)

The corneal epithelium is left intact, which requires a longer riboflavin loading time. It is likely less painful and would be ideal if keractasia is efficiently stabilised. Several substances have been used to increase the permeability of riboflavin by loosening the tight junctions of the epithelial layer. Various clinical studies have been conducted to evaluate the clinical effects of transepithelial CXL on keratoconic eyes pre-treated with substances enhancing epithelial permeability. Enhanced riboflavin permeability with trometamol, BAC, EDTA, and gentamicin have been tried. The results of clinical observations varied from "less effective than standard CXL" to "moderately effective" to "appearing to halt keratoconus progression, with a statistically significant improvement in visual and topographic parameters."[14-16] Alcohol 20% has also been applied to devitalize corneal epithelium.[17] Samaras et al. in their study concluded that complete removal of the corneal epithelium appears to be necessary to allow sufficient riboflavin absorption into the stroma to alter the normal light transmission properties of the porcine cornea.[18]

Complications after CXL

1. Infectious disease

Kymionis et al. published a case report which showed that CXL can induce herpetic keratitis with iritis even in patients with no history of herpetic disease.[19] Australian authors reported a case of polymicrobial keratitis caused by Streptococcus salivarius, Streptococcus oralis and coagulase-negative Staphylococcus sp. in a patient.[20]

2. Corneal haze

Greenstein et al. conducted a study to determine the natural history of CXL associated corneal haze measured by Scheimpflug imagery (densitometry) and slit-lamp bio microscopy in patients with keratoconus or iatrogenic keractasia. Changes in haze did not correlate with postoperative clinical outcomes.[21]

Herrmann et al described a temporary subepithelial haze in a patient who underwent CXL for keratoconus and treated with topical steroids. The haze disappeared several months after the procedure.[22]

3. Corneal scarring

On retrospective evaluation of haze development after riboflavin-ultraviolet (UV) A-induced corneal collagen cross-linking (CXL),8.6% of the eyes developed significant stromal scarring at 1 year after CXL. Based on these results, advanced keratoconus should be considered at higher risk of corneal scarring after CXL due to low corneal thickness and high corneal curvature.[23]

4. Cell density loss

Kymionis et al reported the outcomes after CXL in patients with thin corneas(<400 m)[24]. In 1 year of follow up time, they detected a significant decrease in endothelial cell density from 2733 ± 180 cells/mm2 to 2441 ± 400 cells/mm2.

5. Corneal melt

Gokhale and Vemuganti reported a case of acute corneal melt with perforation in a patient with keratoconus after uncontrolled use of topical diclofenac and proparacaine eyedrops[25] who had corneal melting following CXL.[26]

Combination of CXL with refractive surgeries

Collagen CXL stops, slow, or, in many cases, partially reverses the ectatic process in eyes with keratoconus and ectasia after LASIK, as measured by visual acuity and corneal curvature. Corneal curvature irregularity must be significantly reduced and regularized. Some of the means to regularize the ectatic cornea in combination with CXL are described below.

1. CXL combined with intracorneal rings

A prospective comparative randomized consecutive study was conducted in Dunya Eye Hospital, Istanbul, Turkey. It compared 2 sequences of combined intrastromal corneal ring segment (ICRS) implantation and ultraviolet/riboflavin-mediated corneal collagen crosslinking (CXL) in progressive keratoconus. They concluded that Implantation of ICRS followed by CXL resulted in greater improvement of keratoconus.[27] Miranda et al. reported a reduction of spherical equivalent and max K of more than 2.50 D and 6.00 D, respectively, using Ferrara rings.[28]

2. CXL combined with topoguided photorefractive keratectomy

Kymionis et al. conducted a prospective study to present the results after simultaneous photorefractive keratectomy (PRK) followed by corneal collagen cross-linking (CXL) for progressive keratoconus and showed favorable results. PRK followed by CXL seems to be a promising treatment capable of offering functional vision in patients with keratoconus.

4.CXL combined with conductive keratoplasty

Kymionis et al. evaluated the combined effect of conductive keratoplasty (CK) followed by corneal collagen cross-linking (CXL) in 2 patients with keratoconus and showed that corneal remodeling with CK in patients with keratoconus seems to have a temporary effect despite the subsequent application of CXL.

Conclusion

Corneal CXL halts the progression of corneal ectasia, with very low incidence of complications or side effects. It is an efficacious procedure in the treatment of keratoconus and iatrogenic ectasia. CXL has shown to reduce the corneal curvature, spherical equivalent refraction and refractive cylinder in eyes with corneal instability and progressive irregular astigmatism seen in patients with keratoconus and ectasia. This has been used as an adjuvant treatment to conventional therapeutic modalities in infectious keratitis, corneal melting processes and bullous keratopathy. A sequential or simultaneous combination of limited topography-guided PRK and CXL seems to be a promising treatment capable of offering functional vision in patients with keratoconus.

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Address of Correspondence:
Dr. Sumpi Chinging
Junior Resident,
RIO, PGIMS,
Rohtak-124001 (Haryana)
E-mail : sumpi.chinging79@gmail.com
Mobile: 9717625459

Electroretinography

Chetan Chhikara MS, **Manisha Nada** MS, DNB
Aakash Sharma MBBS, **R S Chauhan** MS, **Manoj P Shettigar** MBBS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Electroretinography (ERG) is a functional test of the outer retina. During an examination, the retina is selectively stimulated. The stimulation of the retina produces a response of the individual retinal cells and reveals information about its function. The ERG examination requires very specific conditions in order to avoid undesirable factors which may adversely affect the recordings. The electroretinography examination may be performed for a short period ("rapid protocol"), commonly used to access retinal activity. The "long protocol" is used for the differential diagnosis of retinal disorders. It is mainly used in diagnosing and evaluating retinal dysfunction when there are no ophthalmic lesions present. The main indications for electroretinography are the pre-operative examination of cataract patient and the early diagnosis of inherited retinal diseases.

Keywords: *Electroretinography, inherited retinal disease, photoreceptors, mfERG.*

Introduction

Electroretinography (ERG) is the mainstay of clinical ophthalmic diagnostic testing. The ERG provides an objective, quantitative measure of retinal function and allows the clinician to monitor the function of rod cells, cone cells, and ganglion cells in each eye. It uses electrodes placed on the cornea or adjacent to the orbit to monitor changes in the electrical potential of the eye in response to specific stimuli[1]. Careful manipulation of the stimulus and testing conditions allows the clinician to investigate different cell types and layers of the retina. The ability to distinguish between different cell layers and cell types means that ERG's can be used to discern between myriad inherited retinal disorders and dystrophies that may otherwise prove clinically indistinguishable.Several different types of ERG test provide specific information about the patient's visual function. The full-field erg, or fferg, is the most common form ofelectroretin-ographic testing. It provides an assessment of general retinal function and can distinguish between the various cell types, revealing the function of photoreceptors, bipolar cells, ganglion cells and amacrine cells, but cannot provide specific information about individual sectors of the retina. The most recent advance in ERG technology is the multifocal electroretinogram (mfERG) which provides a detailed assessment of the health of the central retina and measures the response in each of a large number of small sectors, typically either 61 or 103, of the retina.It thus provides a map that allows the clinician to locate specific areas of malfunction. The pattern ERG, or pERG, measures the response to a temporally changing pattern of contrast at a constant level of luminance, providing information about ganglion cells and generalized macular function.

Basic Principle

Photoreceptors and downstream neurons in the retina maintain a non-neutral electrical

"resting potential" by manipulating the intracellular and extracellular concentrations of

positive sodium, potassium, and calcium ions and negative chloride ions, as well as larger electronegative molecules. Human rod cells present a model system of phototransduction. The chromophore, or light sensing pigment, in rods is 11-cis-retinal, which is bound to an apoprotein called opsin, forming rhodopsin. When a photon strikes 11-cis-retinal, causes it to

isomerize into all-trans-retinal[2-5]. This conformational change causes rhodopsin to activate

transducin, a heterotrimeric G protein[6] Activated transducin binds to the inhibitory subunits of phosphodiesterase 6 (PDE6),thereby de-inhibiting it. The newly active PDE6 hydrolyses cyclic Guanosinemonophosphate (cGMP), reducing intracellular cGMP levels and closing cGMP-gated cationic channels (CNG) in the rod cellular

membrane.[7] This reduces the influx of NA+ and Ca2+ into the cell, thereby hyperpolarizing it.The hyperpolarization of the cell causes it to cease transmitting glutamate across synapses to bipolar cells, inducing changes in their polarization. Bipolar cells transmit this signal either directly to ganglion cells, each of which has an axon proceeding out of the orbit along theoptic nerve, or to amacrine cells, which then activate ganglion cells or alter the output of other bipolar cells. Photoreceptors, bipolar cells, and amacrine cells operate via graded potentials, but ganglion cells generate action potentials in response to incoming signals from bipolar and amacrine cells; these action potentials help to propagate the information along the optic nerve. The function of each of these cell types can be measured precisely using various electroretinographic techniques.

Components of ERG

a-wave: Initial corneal-negative deflection, derived from the cones and rods of the outer photoreceptor layers

This wave reflects the hyperpolarization of the photoreceptors due to closure of sodium ion channels in the outer-segment membrane. Absorption of light triggers the rhodopsin to activate transducin, a G-protein. This leads to the activation of cyclic guanosine monophosphate phosphodiesterase (cGMP PDE) eventually leading to a reduction in the level of cGMP within the photoreceptor. This leads to closure of the sodium ion channels resulting in a decrease of inwardly directed sodium ions, or a hyperpolarization of the cell. The a-wave amplitude is measured from baseline to the trough of the a-wave[8]

b-wave: Corneal-positive deflection; derived from the inner retina, predominantly Muller and ON-bipolar cells

The hyperpolarization of the photoreceptor cells results in a decrease in the amount of neurotransmitter released, which subsequently leads to a depolarization of the post-synaptic bipolar cells. The bipolar-cell depolarization increases the level of extracellular potassium, subsequently generating a transretinal current. It is this transretinal current that depolarizes the radially oriented Muller cells and generates the corneal-positive deflection. The b-wave amplitude is generally measured from the trough of the a-wave to the peak of the b-wave. This wave is the most common component of the ERG used in clinical and experimental analysis of human retinal function.[9-10]

c-wave: It is derived from the retinal pigment epithelium and photoreceptors

The c-wave is a reflection of the resulting change in the transepithelial potential due to the hyperpolarization at the apical membrane of the RPE cells and the hyperpolarization of the distal portion of the Muller cells. The c-wave generally peaks within 2 to 10 seconds following a light stimulus, depending on flash intensity and duration. Due to the c-wave response developing over several seconds, it is susceptible to influences from electrode drift, eye movements, and blinks.

d-wave: This is produced from the off bipolar cells.

The a-wave, sometimes called the "late receptor potential," reflects the general physiological health of the photoreceptors in the outer retina. In contrast, the b-wave reflects the health of the inner layers of the retina, including the ON bipolar cells and the Muller cells.[11]

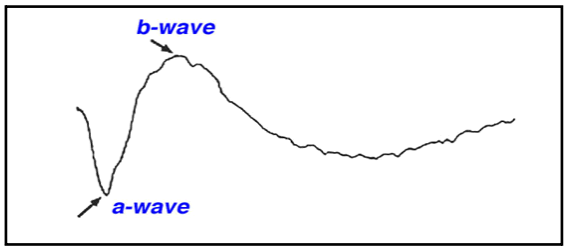

Fig. 1: Basic waveform of ERG

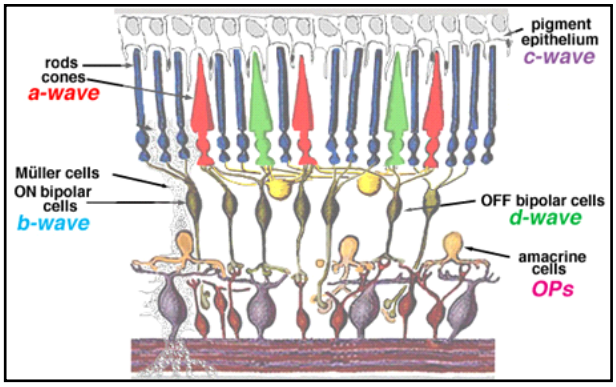

Fig.2 : Showing from where the major components of ERG originate.

Two principal measures of ERG waveform are-

Amplitude

- a- wave- from the baseline to the negative trough of the a-wave.
- b-wave measured from the trough of the a-wave to the following peak of the b-wave.

Time

- (t)a from flash onset to the trough of the a-wave.
- (t)b from flash onset to the peak of the b-wave.

These times, reflecting peak latency, are referred to as "implicit times" (Figure3) .

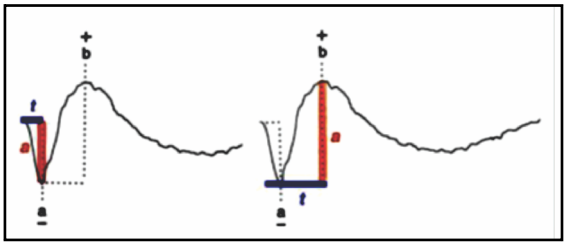

Fig. 3: Showing amplitude and implicit time of a & b wave.

The ERG of a normal full-term infant looks similar to a mature ERG. A normal ERG in a new born infant can be small amplitude the first couple of months. The ERG attains peak amplitude in adolescence and slowly declines in amplitude throughout life.[12] After age 55-60 years the amplitude of the ERG declines even more. Implicit times slow gradually from adolescence through old age as well.

ERG recording electrodes

1. Recording electrodes/Active Electrode
   - Over cornea, bulbar conjunctiva or skin of lower eyelid (Figure 4).
   - Protect corneal surface with non-irritating ionic conductive solution (artificial tears or contact lens solutions containing sodium chloride and no more viscous than 0.5% methyl cellulose) & topical anesthesia for contact lens electrodes.
   - Conjunctival sac – used in pattern ERG
   - Cornea (contact lens electrode) in flash ERG
2. Reference electrodes
   - These electrodes connect to the negative input of the system.
   - Bipolar electrode- incorporated within the contact lens-speculum assembly- may produce lower amplitude than the monopolar electrode at a separate area.
   - Monopolar electrodes may be attached to skin near each temporal orbital rim, avoid placing over muscle masses.
3. Common electrodes/ Ground electrode
   - Connected to common input of the system.
   - Placed over earlobe/mastoid/forehead.

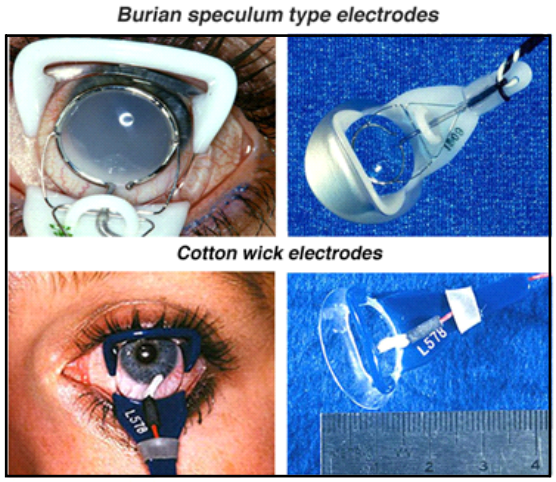

Fig.4: Burian & Cotton Wick electrodes.

There are yet other simpler ERG recording devices using gold Mylar tape that can be inserted between the lower lid and sclera/cornea. Most electrodes are monopolar, i.e., are referred to another electrode site most commonly on the forehead. Some are bipolar with the reference electrodes built into a metal surface on a speculum(Figure 5).

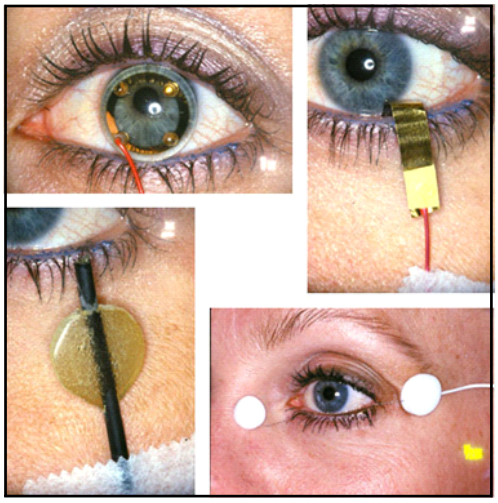

Fig. 5: Corneal electrodes.

If electrodes are to be reused, they should be sterilized with a solution that neutralizes prion-transmitted diseases such as Creutzfeldt-Jakob disease (CJD).

Types of ERG

The focal ERG (fERG; also known as the foveal ERG) is used primarily to measure the functional integrity of the fovea and is therefore useful in providing information in diseases limited to the macula. A variety of techniques have been described in the literature for recording fERGs. Differing field sizes varying from 3 degrees to 18 degrees and light stimulus frequencies have been used in the various methods, however each technique deals with the challenge of limiting amount of light scattered outside the focal test area. Focal ERG is useful for assessing macular function in conditions such as age-related macular degeneration, however requires good fixation from the subject. The full-field ERG (Ganzfeld; ffERG) measures the stimulation of the entire retina with a flashlight source under dark-adapted (scotopic) and light-adapted (photopic) types of retinal adaptation. This is useful in detecting disease with widespread generalized retinal dysfunction i.e. cancer associated retinopathy, toxic retinopathies, and cone-rod dysfunction. Due to the massed retinal electrical response, small retinal lesions may not be revealed in ffERG recordings.[13]

The multifocal ERG (mfERG) simultaneously measures local retinal responses from upto 250 retinal locations within the central 30 degrees mapped topographically. This new technology was developed by Erich Sutter in the early 1990s and involves powerful computers and high –intensity display monitors. The light stimuli are presented on a video monitor in one of a large number of arrays consisting of hexagonal elements. The hexagonal elements in the array are distributed so that the focal retinal responses have an approximately equal signal-to-noise ratio. The central hexagons are smaller than those in the periphery. The elements are stimulated in a pseudo-random sequence of light and dark, called a maximum length sequence (or m-sequence). The resulting waveforms are similar to those of the ffERG: initial negative deflection (N1 or a-wave), followed by a positive deflection (P1 or b-wave), and a second negative deflection (N2 or c-wave). MfERGs are useful in detecting localized abnormalities within the retina in conditions such as retinitis pigmentosa, branch retinal artery occlusion, fundus flavimaculatus, and Stargardt's disease. Degree of retinal toxicity related to certain drugs such as hydroxychloroquine or ethambutol is better detected using mfERG compared to ffERG. Early visual field defects due to glaucoma may also be detected sooner using mfERG compared to automated perimetry.[14]

The pattern ERG (PERG) uses pattern-reversal stimuli similar to VEP testing and captures retinal ganglion cell activity predominantly in the N95 waveform component. Itis used to detect subtle optic neuropathies. In demyelinating optic neuropathy, the PERG is relatively normal, while it may be abnormal in ischemic optic neuropathies. P50 evaluates the macular function.[15]

Light stimulation for ERGs.

There are also several methods of stimulating the eye. Some laboratories use a strobe lamp that is mobile and can be easily placed in front of a person whether sitting or reclining. The mobility of a strobe lamp(Figure 6) or an array of LEDs is a necessity in some situations such as at the hospital bedside or in the operating room.

Fig.6: Strobe lamp light source.

For patients over 5 years of age most laboratories use a Ganzfeld (globe) with a chin rest and fixation points(Figure7). The Ganzfeld allows the best control of background illumination and stimulus flash intensity. Either strobe lamp or Ganzfeld methods of flash presentation can be used to record the ERG following a single flash or to average responses to several flashes with the aid of a computer.

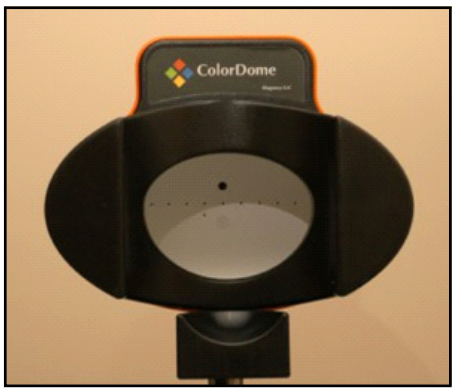

Fig. 7: Ganzfield Stimulation Globe

ERG recording guidelines according to ISCEV 2015 guidelines-

- Maximally dilate the pupils.
- Before dark adapted protocols- 20 min of dark adaptation.
- Before light adapted protocols- 10 min of light adaptation.
- Present low strength flashes before stronger flashes- so that the partial light adaptation due to bright light does not occur.
- Insert corneal contact electrodes (when these are used) under dim red light after dark adaptation period. Avoid strong red light. Allow 5 min of extradark adaptation after insertion of contact lens electrode.
- Allow at least 30 min recovery time in ordinary room illumination after use of strong light for retinal imaging (fundus photography, fluorescein angiography and others).
- Request the patient to fix and not move eyes. Ocular movements can change the positions of electrodes, can cause blockage of light by eyelids or electrode and may induce electrical artifacts.

Separating rod and cone ERGs

Most disorders of the retina are detected by an attenuation of amplitude. Implicit times, of both a- and b-waves are also affected in some conditions. Implicit times and amplitudes vary depending upon whether the eye is dark adapted or not, background illumination, brightness, rate of stimulation and colour of the light stimulus. These parameters allow separation of rod and cone activity in any duplex retina. Rods and cones differ in number, peak color sensitivity, threshold and recovery. There are about 120 million rods in each retina and about 6-7 million cones. Because of sheer numbers, the ERG following a white flash is dominated by the mass response of the rods. Peak wavelength sensitivity for rods is around 510 nm and the peak sensitivity of cones as a group is about 560 nm[16-17]

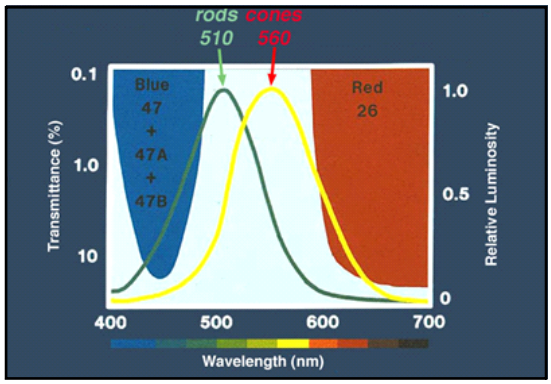

Fig.8: Filter conditions used to isolate rod and cone components of the ERG using dim scotopic flashes.

Using different rates (flicker) of stimulus presentation also allows rod and cone contributions to the ERG to be separated. Even under ideal conditions rods cannot follow a flickering light up to 20 per second whereas cones can easily follow a 30 Hz flicker, which is the rate routinely used to test if a retina has good cone physiology.[18]

ERG recording methods

This includes 6 protocols named according to the strength of the stimulus in candela. second/square meter (time integrated luminance).[19-20]

1. Dark-adapted 0.01 ERG (a rod-driven response of on bipolar cells).
2. Dark-adapted 3 ERG (combined responses arising from photoreceptors and bipolar cells of both the rod and cone systems; rod dominated).
3. Dark-adapted 10 ERG (combined response with enhanced a-waves reflecting photoreceptor function).
4. Dark-adapted oscillatory potentials (responses primarily from amacrine cells).

5. Light-adapted 3 ERG (responses of the cone system; a-waves arise from cone photoreceptors and cone Off- bipolar cells; the b-wave comes from On- and Off-cone bipolar cells).
6. Light-adapted 30 Hz flicker ERG (a sensitive cone-pathway-driven response).

Factors affecting ERG responses

- Duration of stimulus
- Size of retinal area illuminated
- Interval between stimuli
- Size of pupil
- Development of Retina
- Clarity of Ocular Media
- Age, Sex, and Refractive Error- Adult ERG by the age of 2 years, Women > Men.

Abnormal ERG responses

There are 4 types of abnormal responses.[21-22]

- Supernormal Response – Amplitude of both a & b wave is 2 standard deviation above the normal .Eg.Albinism, Early Siderosis Bulbi.
- Subnormal Response- Both a & b wave have amplitude less than 2 standard deviation beneath the mean normal. Eg. Early RP, HCQ Retinopathy, Retinal Detachment, Vitamin A deficiency etc.
- Extinguished Response- Complete absence of response. Eg. Advanced RP, Complete RD, Advanced Siderosis Bulbi, Choroideremia, Leber's Congenital Amaurosis etc.
- Negative Response- Large a wave with small or no b wave (b/a ratio <1) Gross disturbance in retinal circulation. Eg. Arteriosclerosis, Giant cell arteritis, CRAO, CRVO etc.

Clinical applications of ERG

1. ERG in Retinitis Pigmentosa- like disease- Retinitis pigmentosa shows minimal or sub-normal a- and b-wave amplitudes (response primarily from cone system) and can even result in completely extinguished ERG waves in severe retinitis pigmentosa. There are a number of central nervous system syndromes with RP-like ocular involvement. Prominent among these are the mucopolysaccharidoses such as the Hurler, Scheie and Hunter syndromes & neuronal ceroid lipofuscinoses such as Batten's disease which have abnormal ERGs.

   Other syndromes that may include retinitis pigmentosa are Bassen-Kornzweig syndrome (a-beta-lipoproteinemia), Alagille syndrome, Cystinosis, Kearn's-Sayres syndrome, Laurence-Moon-Bardet-Biedl syndrome, Myotonic dystrophy, Refsum's disease, Usher's syndrome etc. [23]
2. Congenital stationary night blindness (CSNB) - Schubert-Bornschein type can vary in ERG appearance but the classic form has reduced b-wave amplitudes & Riggs type CSNB the a- and b-wave ERG amplitudes attenuate proportional to degree of expression.
3. Enhanced S-cone syndrome, sometimes called Goldman-Favre syndrome - ERGs show a poor rod photoreceptor response and increased ERG responses to blue flashes.[24]
4. X-linked juvenile retinoschisis - ERG has a specific abnormality showing a normal a-wave but no b-wave that is a negative ERG.
5. Cone Dystrophies - Markedly depressed photopic response and less affected scotopic response.
6. ERGs in retinal vascular disease - Vascular occlusions such as central retinal artery thrombosis produce a characteristic avascular appearance to select areas of the fundus and an ERG with no b-wave. Ophthalmic artery occlusions usually result in unrecordable ERGs. Central retinal vein occlusion shows attenuation of b-wave amplitude and delay in 30 Hz flicker implicit time to beyond 35 milliseconds. In general, focal disease including due to vascular insufficiency, detachment, trauma, or focal toxicity reduces the full-field ERG amplitude proportional to amount of area affected.
7. Foreign bodies and Trauma -In Siderosis a transient supernormal response then negative pattern followed by non-detectable response in severe cases (rod function more affected than cones; reduction of b-wave amplitude more than a-wave) however a small piece of stainless steel or plastic outside the macula may have a minor effect on the retina. In general, if b-wave amplitudes are

reduced 50% or greater compared to the fellow eye, it is unlikely that the retinal physiology will recover unless the foreign body is removed. In traumatic cases ERG changes depends upon the extent of retina involved.

8. Drug toxicities - Several drugs taken in high doses or for long periods of time can cause retinal degeneration with pigmentary changes like thioridazine, chlorpromazine, Vigabatrin, and chloroquine or hydroxychloroquine. The American Academy of Ophthalmology guidelines recommend a baseline examination for patients starting these drugs to serve as a reference point; and to rule out maculopathy an annual screening after 5 years of use unless there is suspicion of toxicity or presence of unusual risk factors[25]
9. Systemic disorders and the ERG - Systemic metabolic disorders are reflected in retinal physiology. Liver and kidney disease and drugs that affect those organ systems, usually reduce ERG b-wave amplitudes, particularly in scotopic dim flash ERGs. For example, deferoxamine, an iron chelating drug used to reduce iron overload, can be toxic to the retina. Vitamin A deficiency shows reduced ERG amplitudes particularly under scotopic conditions. [26]

Conclusion

Electrophysiological recording is a valuable asset for the clinician. Because ERG tests can measure the function of different cell types and cell layers, they can aid the clinician in distinguishing between symptomatically similar diseases. Furthermore, because they provide an objective measure of retinal function, they can help clinicians evaluate very young patients, very old patients, and others that may otherwise be difficult to diagnose. The quantitative results that ERG tests provide make them useful as tools for both prognosis and disease monitoring.

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Address for Correspondence:
Dr Chetan Chhikara
Senior Resident,
RIO, PGIMS,
Rohtak- 124001 (Haryana)
E-mail: chetanchhikara@gmail.com
Mobile: 7206004050

Acute onset isolated sixth nerve palsy due to neurocysticercosis – An unusual presentation.

Neha Chauhan *MBBS,* **Nitin Vichare** *MS, DNB*
Command Hospital, Chandimandir

Abstract

We report a case of nine years old girl, presented to a tertiary care centre with complaint of esotropia right eye associated with frontal headache of one week duration. Ocular evaluation revealed right lateral rectus palsy and fundus examination showed bilateral papilloedema. On investigations, peripheral blood smear showed eosinophilia and MRI Brain and orbit showed multiple ring enhancing lesions in bilateral cerebral hemispheres suggestive of extensive neurocysticercosis with right medial rectus muscle involvement. The patient was managed with Intravenous mannitol and dexamethasone followed by oral steroids with oral albendazole for a month in consultation with the paediatrician. The patient responded well to treatment, with resolution of right lateral rectus palsy and papillodema after two months.

Keywords : *Esotropia, papilloedema, neurocysticercosis.*

Introduction

Human cysticercosis, results from infestation with the larvae of the pork tapeworm, Taenia solium. It is a major public health problem in the country and the developing world.[1]

Cysticercosis occurs in human host by faecal-oral contamination with T solium eggs by eating under cooked pork or by contaminated water, food or vegetables. T solium has a complex two-host life cycle. Human beings are the only definitive host and harbour the adult tapeworm (taeniasis), whereas human can also become accidental intermediate host.Infection with T.solium results in the formation of cysticerci in the human body.[2,3]

The central nervous system, subcutaneous tissue, skeletal muscle, heart muscle and eye are the sites for predilection for the development of cysticerci in human host.

Ocular cysticercosis can be extraoular or intraocular and may have varied clinical presentations. However, the association between orbital cysticercosis and systemic cysticercosis is considered rare.[4]

Here we present a case of acute onset sixth nerve palsy due to raised intracranial pressure in a case of neurocysticercosis with medial rectus involvement.

Case Report

A previously healthy 9 years old girl, non-vegetarian by diet, presented to a tertiary care centre with a history of sudden onset, painless, inward deviation of right eye associated with frontal headache of one week duration. No history of head or ocular trauma, fever, diplopia, vomiting or seizures. There was no history of any contact with pets or passage of worms in stools. No past history of spectacle use or refractive error. On evaluation, distant visual acuity was 6/6 both eyes on Snellen's chart with no refractive error on cycloplegic refraction. There was obvious right face turn. Extra ocular movements showed restricted abduction(Figure1) in right eye. On torch light examination, Hirschberg test showed 30 degree of esotropia right eye in primary position(Figure 1).

Cover test confirmed the deviation. On cover uncover test, right eye could not take fixation.

Maddox rod test showed 50 degree prism dioptre base out deviation in right eye. No palpebral fissure changes, diplopia in primary gaze or nystagmus was found. Force duction test ruled out medial rectus contracture and force generation test showed lateral rectus paresis. Anterior segment examination was normal in both eyes. Pupils were bilaterally round and briskly reacting to light. Dilated fundus examination revealed optic disc raised, blurred margins with loss of physiological cup suggestive of bilateral papilloedema (Figure 2). Intraocular pressure was 15 mm of Hg in right eye and 16 mm of Hg in left eye .Systemic examination was unremarkable and examination of other cranial nerves was essentially normal. A provisional diagnosis of isolated sixth nerve palsy with papilloedema was made.

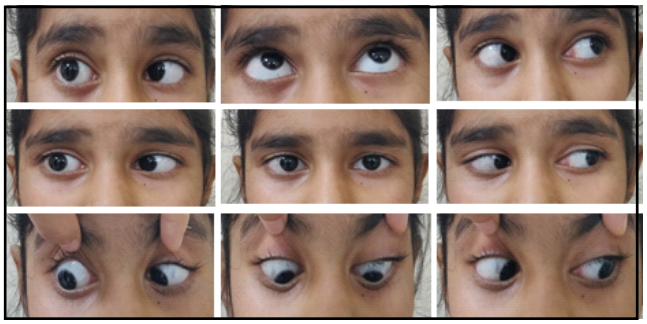

Fig.1: Abduction deficient in right eye

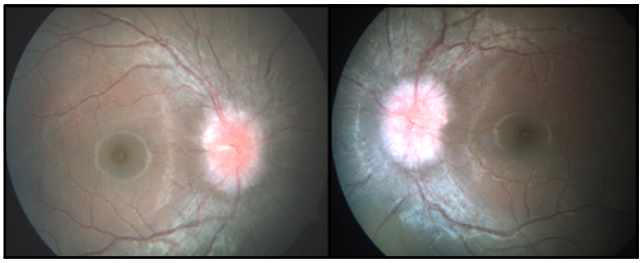

Fig. 2: Papillodema both eyes

Investigations

Blood investigations revealed raised eosinophil count on peripheral blood smear. Serum Cysticercosis antibody Ig G was positive. Stool examination was normal . Patient underwent MRI brain and orbits which showed multiple discerete randomly distributed ring enhancing lesions in bilateral cerebral hemispheres and left cerebellar hemisphere along with bulky medical rectus with well -defined cystic lesion suggestive of neurocysticercosis with medial rectus muscle involvement (Figure 3). Cerebrospinal fluid analysis excluded any infectious etiology or malignancy

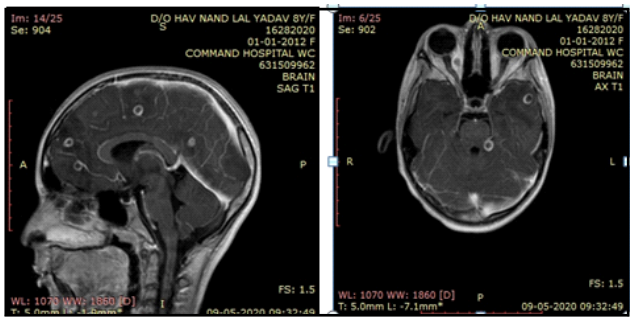

Fig.3: Multiple discrete randomly distributed ring enhancing lesions in bilateral cerebral hemispheres and left cerebellar hemisphere along with bulky medical rectus with well defined cystic lesion on MRI brain and orbits

Treatment

The patient was started on Intra venous mannitol (0.2g/kg/day) and dexamethasone (0.1mg/kg/day) for 3 days followed by with oral albendazole (15mg/kg/day) and oral prednisolone (1mg/kg/day) for 4 weeks in consultation with the paediatrician. Oral prednislone was tapered over next four weeks

The patient responded well to treatment with convalescence of headache, clinically improvement in ocular movements and resolution of papilloedema (Figure 4 (a) and 4 (b)

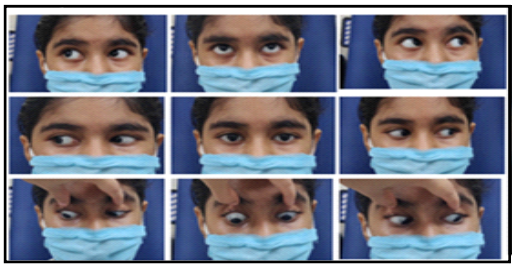

Fig. 4 (a): Extraocular movements – post therapy,

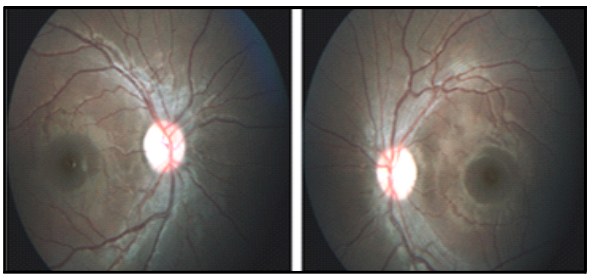

Fig. 4 (b): Resolution of papillodema

Discussion

Taenia solium inhabits the small intestine of man and has world-wide distribution. Scholl and Soemmerring discovered a live Cysticercus cyst in the anterior chamber in 1889.[5] Ocular infestation by its larva, Cysticercus cellulosae, is commonly found in the developing countries. In Indian subcontinent, 78% of the cases of ocular cysticercosis have been reported from Andhra Pradesh and Pondicherry.[6]

It has myriad clinical presentations and depends on the location, size, relation to the adjacent structures and the stage of evolution of the cyst. Neurocysticercosis is the most common form of systemic involvement. Ocular cysticercosis can be extraocular (in the subconjunctival or orbital tissues) or intraocular (in the vitreous, subretinal space, or anterior chamber). The extraocular muscle form has been reported to be the most common form of orbital and adnexal cysticercosis.[7] Disturbance in ocular motility is the most common presentation in extraocular form. Amongst the extra ocular muscles,

superiour rectus, lateral rectus, medial rectus and the superior oblique muscles are most commonly affected.[8]

Almost a third of cases with neurocysticercosis presents with headache and vomiting. With extraocular muscle involvement, simultaneous brain involvement is reported in 16% of cases. Papilloedema has been reported in 2.39 to 6.6% of paediatric cases.[9] Changes in intracranial pressure either increased or decreased may result in downward displacement of the brainstem causing stretching of the abducens nerve which is tethered as its exits the pons inside the Dorello's canal.[10] Patients with sixth nerve palsy present with binocular horizontal diplopia, worse in the distance, and esotropia in primary gaze. Examination for sixth nerve palsy involves documenting the presence or absence of papilledema, examining the ocular motility, evaluating the eyelids and pupils, and excluding involvement of other cranial nerves (eg, V, VII, VIII). In this patient, we found unilateral esodeviation with limitation in abduction corresponding to abducens nerve palsy and bilateral papilloedema.The rest of cranial nerves were normally functioned. CT/MRI are imaging modalities of choice for diagnosing myocysticercosis involving extraocular muscles.[11] As our patient was having headache, we went in for MRI with showed neurocyticercosis with medial rectus muscle involvement.However, the child presented with unilateral abduction deficit due to false localising sign with raised intra cranial pressure. Oral albendazole acts by blocking glucose uptake of the parasite and depleting its glycogen stores. This leads to death of the larva with release of toxins which causes severe inflammation. Concurrent usage of oral steroids suppress this inflammation and its sequelae. Steroids have also been reported to increase the plasma levels of albendazole.Pandey et al. started oral steroids 3 days prior to therapy with albendazole. This was noted to suppress the inflammatory reaction that peaks on the third day following beginning of therapy.[12] In this case, initially mannitol with dexamethasone was given to manage raised intracranical pressure followed by oral steroids with albendazole and complete recovery was achieved after 2 months.

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Address for Correspondence:
Dr. Neha Chauhan
Junior Resident,
Command Hospital (Western Command)
Panchkula, Haryana
E-mail: cneha91@yahoo.com
Mobile: 9564540666

Ocular myasthenia gravis

Yuvika Bansal[1] MS , **Gaurav Goyal**[2] MS
Sanjivani Multispeciality Hospital, Sirsa[1]
Eye Q Hospitals, Hisar[2]

Abstract

Myasthenia gravis is primarily an autoimmune disorder of neuromuscular junction at the skeletal muscles. The hallmark is fatigability and variability in muscle weakness. It has a diverse presentation because of the variation in the extent of muscles involved. Most often ophthalmologists are the first to diagnose it, as systemic myasthenia may have ocular involvement either at the onset or later in the course of the disease. This review article highlights the presentation, differential diagnosis, investigations, and treatment of ocular myasthenia.

Keywords : *Myasthenia, acetylcholine, neuromuscular junction, antibody.*

Introduction

Ocular Myasthenia gravis (OMG)is a localized form of generalised myasthenia in which antibodies directed against acetylcholine receptors destroy or bind the acetylcholine receptors at neuromuscular junction.

Ocular muscles are involved in around 95 % of patients during the course of the disease, whereas 40 – 90 % of generalised myasthenia may initially present with droop and diplopia and around 50 -80 % of ocular myasthenia may eventually progress to general myasthenia.[1] Ocular Myasthenia Gravis (OMG) may affect any age and sex, age distribution is bimodal, women are afflicted more in their mid 20s whereas men show their peak at around 40s.

Pathophysiology

Neuromuscular junction is the site of connection between a nerve terminal and a muscle. When action potential generates, there is release of acetylcholine (a neurotransmitter) from the nerve terminal which then binds to the Ach(nicotinic) receptors at the post synaptic membrane of striated muscle which then initiates the muscle contraction. In myasthenia antibodies are directed against acetylcholine receptors and decrease the available receptors by blocking or complement mediated membrane damage thereby decreasing the signal transduction. Acetylcholine receptor antibodies are present in around 90 % generalized myasthenia and 50-70 % of ocular myasthenia[2].

Extraocular muscles are the most susceptible to it as they have a high frequency of synaptic firing with fewer number of acetylcholine receptors. Moreover they are at constant tension in maintaining gaze in a particular direction. Hallmark of OMG is variability in muscle weakness and fatigability. Most common presentation is diplopia and ptosis .The pupillary fibres are devoid of nicotinic acetylcholine receptors, and thus pupils are unaffected in myasthenia gravis[3].

Presentation

Majority of patients present with pupil sparing ocular motility disorder with ptosis either at presentation or during course of disease. Around 20 % may remain purely ocular, rest may progress to generalized form and chances of progression are high in 2 years from the onset.

Ptosis is one the most common presentations of OMG (Figure 1 a). It is due to the involvement of levator palpebrae superioris. Ptosis worsens as the day progresses, or after repeated or sustained use of muscle throughout the day. Patient typically feels better after a period of rest. One should always rule out OMG in a case of acquired ptosis.

Ptosis can be unilateral or asymmetrical bilateral. Important clinical signs in favour of myasthenia are "Cogans lid twitch sign" in which there is overshooting of lid as patient is instructed to look downwards for at least 15 seconds and then gaze upwards to primary gaze. Test is considered positive when there is over shoot followed by downward drift

of the lid. This is not specific for myasthenia, as it may be seen in thyroid eye disease [4].

Other clinical sign due to the lid involvement is "see saw ptosis or enhanced ptosis" in case of bilateral ptosis which is elicited by first asking patient to look upwards , ptosis will worsen after prolonged upgaze and now one of the lid is elevated manually , there will be worsening of ptosis or enhancement of ptosis in contralateral eye . This can be explained by Herring's law of equal innervations. This test helps to differentiate from other causes of bilateral ptosis like nuclear third nerve palsy , myotonic dystrophy[5].

There is decreased tone in orbicularis oculi muscle which can be elicited by an attempt to open the eyes manually by the examiner after patient is being asked to forcefully close the eyes. There is lack of inhibition due to decreased orbicularis tone. The lids may fall apart even without forceful opening and there is peeking of underlying sclera. This sign is called "peek sign".

Extraocular involvement can vary from single muscle to multiple muscles. Ocular myasthenia is a big masquerader and may mimic supranuclear, internuclear , or infranuclear cranial nerve palsies [6] . It may mimic any cranial nerve palsy which is pupil sparing. Thus OMG should be kept as a differential of unilateral/ bilateral / painless ophthalmoplegia sparing pupil with or without ptosis.

Diagnosis and tests

Myaesthenia is a clinical diagnosis which is complemented by clinical, serological and electrophysiological tests.

Clinical Tests

Common bed side tests are fatigue test, ice test and sleep test.

- Fatigue Test

There is worsening of ptosis or extraocular muscle impairment after a period of sustained upward gaze and on voluntary contraction of antagonist orbicularis muscle there is temporary recovery of ptosis.[7]

- Ice and Sleep Test

Improvement in symptoms especially ptosis with cooling is the basis of ice test. Lowering the temperature decreases the activity of acetylcholinesterase at neuromuscular junction thereby increasing the available acetylcholine molecules for muscle contractions. There is improvement of around 2mm droop after keeping ice pack over lids for around 3 to 5 minutes (Figure 1 b). Ice test is a simple bed side test with sensitivity and specifity of 80-100 % in OMG. Golkin etal concluded that the sensitivity of the Ice test is less in complete ptosis but its reliability can be compared with edorphonium test.8 Positive sleep test is improvement in ptosis after 30 mins of rest.

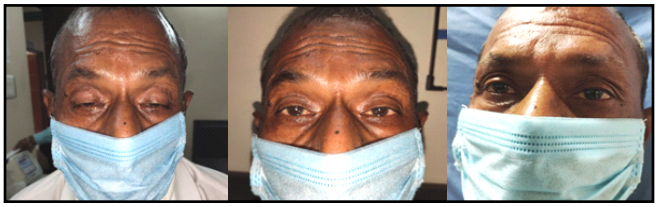

Fig. 1 : (a) At presentation 1(b) After ice test 1 (c) Tensilon test

- Edrophonium Test

Edrophonium is a short acting, quickly hydrolyzed anticholinesterase. It competitively inhibits the enzyme acetylcholinesterase thereby increasing the acetylcholine. It has a rapid onset of action (30 seconds) with short duration (< 5 minutes). It is of particular use in cases where we have evident ptosis.

The dose of edrophonium is 0.15mg/kg in children and total dose is no more than 10 mg in adults . After noticing and documenting the position of eyelids , test dose 2mg of tensilon is injected intravenously. Patient is observed for any improvement in ptosis, also watch out for idiosyncratic cholinergic side effects. If there is no side effect with the test dose, incremental dose of 3-4 mg is injected after 2 minutes. If there is no positive response after 1 minute rest of the dose is given in incremental dose.

Tensilon test should be done under proper observation and monitoring of vitals. Atropine sulphate (0.4-0.6mg) should be available immediately, some even premedicate by injecting 0.4mg atropine subcutaneously. Positive tensilon test is suggestive of myasthenia but not pathognomic, some patients with intracranial lesions may show positive response. The sensitivity of tensilon test is 95 % in generalized myasthenia gravis (GMG) and 86 % in OMG. (Figure 1 c)

- Prostigmine and Neostigmine Test

Neostigmine is a longer acting anticholinesterase ,

permitting the proper evaluation of improvement in ocular motility. It is of special use in patients with diplopia without ptosis, in children who are not too cooperative for evaluation. Mix of 0.6mg of atropine with 1.5mg of prostigmine is injected in one of the deltoid muscles. The onset of action is 15 mins and effect is most obvious 30 minutes after injection.

Electrophysiological Tests

- Rapid nerve stimulation test (RNS)

Supramaximal electric stimuli are delivered to proximal and facial muscles repetitively (6-8 times) at lower frequencies (2-3 hertz) and amplitude of the compound action potential is noted. In myasthenia there is characteristic decremental response (usually more than than 10 %) after 3rd or 4th stimuli .Sensitivity of RNS is 70- 80% in GMG and 50 % in OMG . The response is also subjective to the type of muscle excited. Moreover, it is not specific for myasthenia as we may get decremental response in other Neuromuscular disorders.

- Single fibre electromyography (SFEMG)

It is the most sensitive test for neuromuscular disorders. Two muscle fibres innervated by single axon are stimulated with a special concentric needle electrode with a recording surface. SFEMG studies the adjacent action potentials from same motor unit. The latency between the potentials is noted. The variation in the time interval between two action potentials is called jitter. SFEMG records this jitter. This jitter is also not specific of myasthenia and can be seen in other neuromuscular disorders.

Serological Tests

- Acetylcholine receptor antibody test

It measures the IgM and IgG antibodies to Ach receptors. It is highly specific of myasthenia with increased sensitivity for generalized myasthenia (around 80%) as against 50 % for OMG. The absolute titres may also help in prognosticating the progression from ocular to generalized myasthenia.[10] Changes in antibody titres correlate with disease severity. Patients with negative acetylcholine receptor antibody test are seronegative patients .Of these patients, 30 % have antibodies against muscle specific kinase (anti MuSK Ab). Anti Musk antibodies are particularly helpful in cases with strong clinical suspicion who are seronegative along with negative tensilon test[11].

Imaging Tests

Patients with positive ice test and positive response to tensilon test may not require neuroimaging. Atypical cases should undergo neuroimaging to rule any intracranial pathology. Confirmed cases of myasthenia should undergo CT mediastinum to rule out thymoma. About 15% of myasthenia patients have thymoma and one half of the thymoma patients develop myasthenia gravis.[12]

Treatment

Goal of therapy in myasthenia is to relieve patient symptomatically, reduce the acetylcholine receptor antibodies and induce remission, prevent progression to generalized myasthenia, and avoid the long term side effects of treatment.[1]

Modulation of neuromuscular transmission

Acetylcholine esterase inhibitors: They remain the first line treatment to relieve patient symptomatically. The aim is to increase the duration of neurotransmission for muscle contraction.

Pyridostigmine bromide :

Pyridostigmine(mestinone) is initially given at 30mg twice daily to four times daily and may be increased to a maximum of 1500mg daily. Studies have shown that pyridostigmine given alone does not help in remission of disease[13]. It has good effect on ptosis but diplopia and ocular motility does not improve much with pyridostigmine.

Immunosuppressive therapy

- Long term immunosuppression

Oral corticosteroids in low or moderate dose relieve patients of diplopia. The response can start within days and maximum benefit can be achieved in weeks. There are two common approaches of steroid therapy- high dose / rapid induction regime or low dose / slow titration regimen. High dose therapy carries the risk of exacerbation of myasthenia crisis. After remission,dose is gradually tapered after 4- 6weeks and shifted to alternate day regime. In low dose regimen, 10mg is given and gradually increased by 10mg every week till maximum dose of 1mg/ kg which is maintained for 6-12 weeks and then tapered gradually.[14] Steroids induce remission in 85% of OMG and decrease the progression from OMG to

GMG. Common side effects of long term steroid use are obesity, hirusitism, osteoporosis, hypertension and increased risk of opportunistic infections.

Steroid sparing agents

- Azathioprine- It is a purine antagonist and interferes with T and B cell proliferstion. It has been used either as an adjunct to steroid or as monotherapy. Randomized controlled trial of the adjunct therapy has shown increased remission, lower relapse, decrease in antibody titre and low steroid maintenance dose at the end of 3 years of follow up. [15] Initial dose is 50mg/day which is increased by 50mg every week till maximum of 2-3mg/kg/day in two to three divided doses. Initial response is delayed (more than 6 months) which peaks by 2 to 3 years.

Potential side effect are hepatotoxicity and bone marrow suppression. Blood counts and liver function tests should be done bi-weekly for the first 2 months after initiating treatment and monthly thereafter. Dose is reduced if count falls below 4000/mm 3 and therapy should be discontinued if it is below 3000/mm3,[16]

- Mycophenolate mofetil- It blocks purine synthesis and has been used widely in organ transplant patients. Initial dose is 500-1000mg / day which is titrated over weeks to maximum dose of 1.5gm twice daily. Effects are seen earlier than AZA, it has favourable side effect profile. Randomized trials have shown no added benefit of combination of mycophenolate with steroids over steroids alone.[17]

Other immunosuppressive agents which have been studied and tried are Methotrexate, Cyclosporine A, Tacrolimus, Cyclophosphamide . Although results are encouraging with these steroid sparing agents but one has to be cautious about the risk benefit ratio of the treatment.

- Short term immunomodulation
- IVIG and plasmapheresis

High doses of immunoglobulin (IVIG) and plasma exchange represent short-term treatments, are indicated in severe cases of GMG, myasthenia crisis , preoperatively to prepare patients for thymectomy. These therapies are usually not indicated for patients with pure ocular myasthenia gravis.[18]

Thymectomy

Thymectomy is indicated in thymomatous myasthenia gravis, in patients early in the course of their disease and those younger than 60 years of age. Studies have even shown remission and improvement in generalized myasthenia without documented thymic enlargement.It is not indicated in ocular myasthenia.[19]

Supportive therapy

It includes measures to elevate the lid using crutch glasses, and lid surgery if ptosis has been stable for long. Small ocular deviations and diplopia can be managed with prisms, strabismus surgery can be done in ocular alignment which is stable for 6 months.

Paediatric Myasthenia

It includes neonatal, congenital and juvenile myasthenia. The etiopathogenesis of all three types is different. Neonatal myasthenia is a temporary myasthenia due to transplacental transmission of antibodies from a MG mother. Congenital myasthenia presents at birth and persists life long, it is due to genetic defect at neuromuscular junction which is not responsive to anticholineesterases.[21]Juvenile myasthenia gravis is a rare condition of childhood and prepuberty with antibodies against Ach receptors. It is usually ocular and has good remission rates[20,21]. (figure 2 a and b)

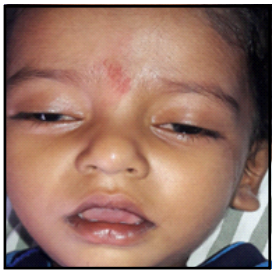

Fig. 2:(a) At presentation

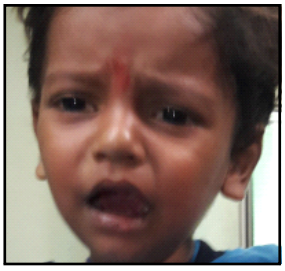

Fig. 2(b): After tensilon test

Conclusion

Ocular myasthenia is a masquerader and can present as variable, unilateral or asymmetrical bilateral, pupil sparing painless ophthalmoplegia with or without ptosis. The diagnosis is supported by clinical bed side tests, serological, and various electrophysiological tests. Anticholine esterases give symptomatic relief but do not help in remission or preventing progression of OMG to GMG. Oral corticosteroids alone or along with other immunosuppressive agents help in remission of disease and conversion to generalized myasthenia. Surgery for ptosis and diplopia can be

considered if there is no response with medical therapy and the measurements are stable for at least 6 months.

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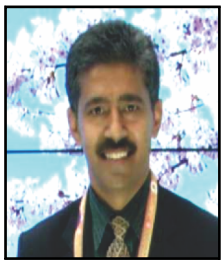
Address for Correspondence:
Dr. Yuvika Bansal
Consultant,
Sanjivini Multi-speciality Hospital,
Sirsa (Haryana)
E-mail : bansal.yuvi@gmail.com
Mobile : 9992800232

Acute onset curvularia- An unusual presentation



RL Sharma MS, **Kusum Bhanoo** MS,
Lalit Gupta MS, **Kamlesh Sharma** MBBS
Department of Ophthalmology, IGMC, Shimla

Abstract

Pigmented fungi (Dematiaceous fungi) have emerged recently as important opportunists and seen commonly in debilitated and immuno-deficient hosts. They produce characteristic corneal lesions which can be diagnosed by examination of 10% KOH preparations provided an index of suspicion is kept.

Keywords: *Keratitis, dematiaceous fungi, immunodeficient, curvularia.*

Case Report

A 58 years old male farmer came with history of trauma to right eye with wheat husk 35 days back and painful gradual diminution of vision, redness, watering of right eye for last one week. Past history revealed inability to close right eye & sagging of right angle of mouth occurring spontaneously during childhood but not congenital. His visual acuity was 20/200 in the right eye & 20/60 in the left eye. There was lagophthalmos in the right eye & Bell's phenomenon was present due to right facial palsy. On slit lamp examination there was a blackish pigmented plaque in the center of right cornea along with 2mm thick immobile
Hypopyon (Figure 1). There was pigment deposition over the lens, pupil was constricted and left eye was normal.

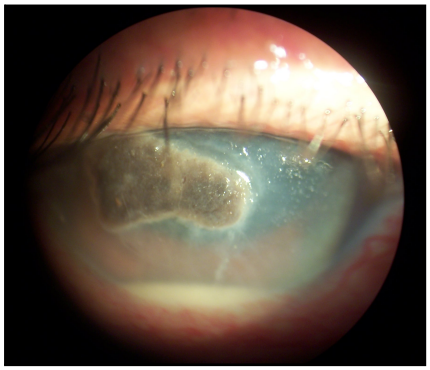


Fig.1: Right eye showing blackish plaque on cornea and hypopyon at presentation

Systemic evaluation of the patient was done, which on chest x-ray showed fibrotic lesion bilateral upper zone & mass in left upper zone with foci of calcifications (Old healed pulmonary tuberculosis). Under topical anesthesia pigmented plaque could be lifted up as a whole leaving underlying tissue clean (Figure 2). This was sent for 10% KOH wet mount and fungal culture on Saboraud's dextrose agar medium.

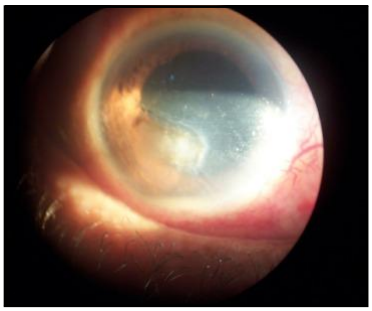


Fig.2 : Right eye after the black plaque removal

KOH wet mount revealed pigmented, septate, branching hyphal structures along with terminal & intercalary chlamydospores (Figure 3). The characteristic brown pigmentation of the Curvularia nata was confirmed on histological staining (Fontana Masson) and growth on SDA. Patient was treated with 2 hourly Natamycin 5 % eye drop,6 hourly Moxifloxacin eye drops and 12 hourly Cyclopentolate . Patient improved symptomatically within 3 days of therapy (Figure 4). The vision was 6/36 at 6 weeks of follow up with central macular corneal opacity.

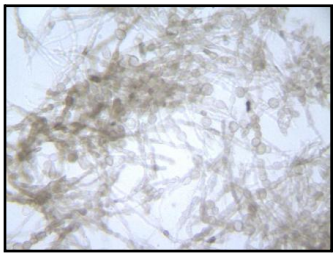


Fig.3: KOH smear showing pigmented hyphae of curvularia

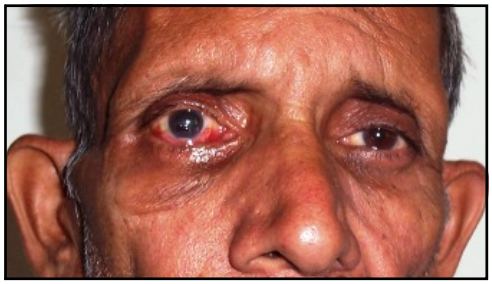


Fig.4: Patient after 3 weeks of natamycin therapy

Discussion

Fusarium and Aspergillus are recognized as ocular pathogens for a long time, but the dematiaceous fungi have emerged recently as important opportunists and seen commonly in debilitated and immuno-deficient hosts as our patient was debilitated and had old treated tuberculosis. Among the pigmented fungi (Curvularia, Cladosporium, Bipolaris, Alternaria and Exophilia) Curvularia is the most prevalent one[1] whose filamentous fungi colonizes soil, vegetation & spreads by airborne spores. Out of 40 known species of Curvularia some are phytopathogens & others are zoopathogenic. Corneal infection caused by Curvularia was reported in 1959 as the first human disease.[2] The histopathological recognition of Dematiaceous hyphomycetes is based on seeing tissue invasion by pigmented hyphae. Curvularia is one of the several genera of the "black fungi". Dematiaceous moulds live and linger in the soil, & on the plants in warm climates[3]. Curvularia accounts for 29% of all fungi isolated from patients with mycotic keratitis in North India.1 Trauma is most common cause in patients with Curvularia keratitisapart from post-keratorefractive surgery, corneal exposure, climatic droplet keratopathy & rarely seen contact lens user. In this patient in addition to trauma he had Bell's palsy of the involved eye that predisposed him for trauma and lack of blinking probably lead to the formation of this plaque on corneal surface. Curvularia keratitis has a slower course & less inflammation than other fungal corneal infections. Melanin in the cell wall of Dematiaceous hyphae & conidia resists killing & could be involved in pathogenicity. Although melanin production by Dematiaceous fungi is down regulated at body temperature[4] but our patient had visible superficial corneal pigmentation which is rare in Curvularia[3]. The main feature of the keratitis in this case was the characteristic pigmented plaque visible on the cornea, which was peeled off from the surface as such leaving the underlying tissue clean as excision is the best treatment in these fungi.

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Address for Correspondence:

Dr. Ram Lal Sharma

Professor & Head,

Department of Ophthalmology,

IGMC, Shimla (Himachal Pardesh)

E-mail : rls_10@rediffmail.com

Mobile : 9418200098

Corneal blood staining after traumatic hyphema- a case report

Manpreet Kaur MS, **Sabhia Jan** MS,
Akram Khan MS, **Yogesh Kumar** MS
SHKM Government Medical College, Nalhar

Abstract

A 7 years old boy presented with the complaints of pain,redness,watering and loss of vision in RE following trauma with a wooden stick 5 days back. On examination, he had full chamber hyphema with blood staining of 2/3rd of cornea with raised IOP. Anterior chamber paracentesis was done after controlling the IOP.The patient developed lenticular changes and is on follow up.

Keywords: *Traumatic, hyphema, intraocular pressure, anterior chamber, blood staining.*

Introduction

Corneal blood staining has been described as a rare complication of contusion injury induced hyphema of relatively long duration with a raised intraocular pressure (IOP) and results from impregnation of the corneal stroma with hemoglobin and hemosiderin . A pressure >25 mm Hg for >5 days may cause corneal staining[1]. It is infrequently seen after penetrating injuries, in which the tension is usually low. The reported incidence is 2–11% of traumatic hyphema cases and even higher for total hyphemas[2-4]. Other corneal blood staining etiologies are hyphema induced by intraocular surgery[5] or any other bleeding in the anterior chamber (AC). In case of hyphema, maximal medical and surgical efforts should be applied to prevent cornea staining, since this condition may end up with an irreversible corneal opacity, hence permament blurring of vision. Irreversible corneal staining may require corneal transplantation. We herein report a case of traumatic hyphema resulting in corneal blood staining.

Case Report

A 7 years old boy presented with the complaints of pain, redness, watering and decreased vision in the right eye following trauma with a wooden stick 5 days back. On examination, the visual acuity was recorded as PL present and PRaccurate in RE and 6/6 in LE. RE examination further revealed mild lid edema and conjunctival congestion on gross examination. On Slit lamp examination, there was ciliary congestion, corneal staining of lower 2/3rd of cornea with full chamber hyphema. The IOP was 50.6 mmHg by Schiotz tonometer in RE and 14.6 mmHg in LE.B-scan showed clear vitreous and intact retina. CT scan of orbit revealed no globe, bony orbit or optic nerve injury. The patient was kept on intravenous mannitol, oral acetazolamide, antibiotics and vitamin C along with topical b-blockers, antibiotic steroid eye drops and atropine ointment. The IOP in RE was reduced to 20.6mmHg. So the patient was taken for anterior chamber paracentesis under GA after PAC fitness and negative RTPCR report for COVID 19. Clotted blood was aspirated out and AC was formed with BSS (balanced salt solution) and air. Next day, the anterior chamber had no hyphema and superior clear cornea was easily visualized with blood staining of lower 2/3rd of cornea (Figure 1 a and b).

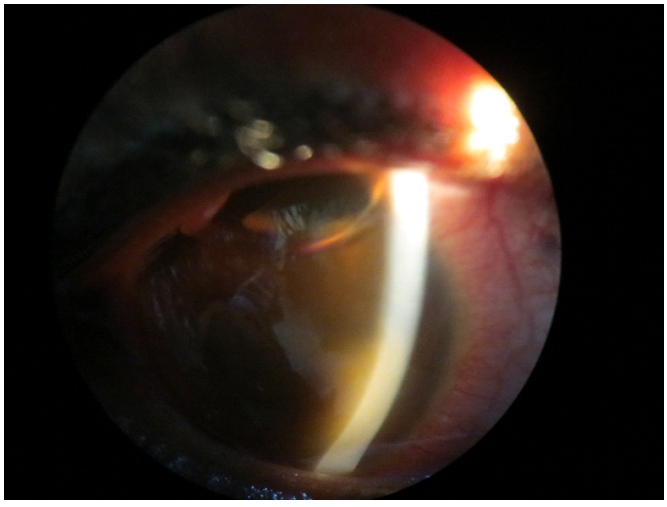

Fig. 1 a : corneal blood staining (1st postoperative day)

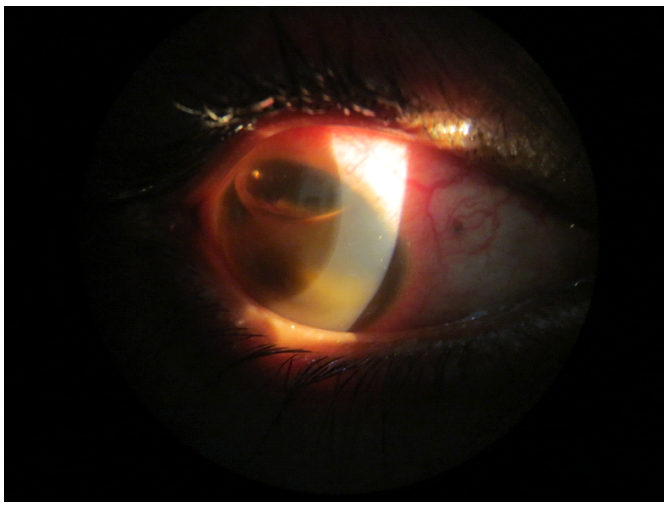

Fig. 1 b : Air bubble(arrow) with superior clear cornea

The visual acuity improved to 6/12p (through superior clear cornea). The IOP became normal, so systemic anti-glaucoma drugs were discontinued and the patient was discharged on topical treatment as prescribed earlier with oral antibiotics, analgesics, vitamin C and prednisolone 20mg tablets after 2 days. At first follow up after 1week, there was no fresh hyphema, IOP was 14mmHg with persistent corneal staining.The oral steroids were tapered over 1week and topical medications were continued. At one month follow up,the VA decreased to 6/60 as the patient had developed lenticular changes(seen through superior cornea) and persistent corneal staining. The patient is under constant follow up.

Discussion

Hyphema is one of the most challenging clinical problems encountered by the ophthalmologists. Traumatic hyphema is encountered in children and adults. Hyphema is usually the result of blunt trauma that hits the exposed portion of the eye despite the protection of the bony orbital rim. Various missiles and objects have been incriminated, including balls, rocks, projectile toys, air guns pellets and the human fist [6].

Most hyphemas (71-94%) result from tears in the anterior face of the ciliary body, with disruption of the major arterial circle and its branches, recurrent choroidal arteries, or ciliary body veins, the remaining percentage arises from ruptured iris vessels, cyclodialysis, or iridodialysis [7]. Predisposing factors for corneal blood staining include prolonged duration of hyphema, large or total hyphema, sustained increased IOP and dysfunction of the corneal endothelium[8]. The reported case exhibited a total hyphema with markedly elevated IOP for 5 days prior to consultation.

The following pathogenesis of corneal blood staining has been postulated. Initially, endothelial degeneration occurs because of mechanical disruption, toxicity from erythrocytic debris and possibly porphyrin-induced phototoxicity. Subsequently, haemoglobin and breakdown products diffuse into the posterior stroma. Haemoglobin is phagocytosed by posterior keratocytes and degraded to haemosiderin, which is subsequently phagocytosed by anterior stromal keratocytes. Excess intracellular haemoglobin and haemosiderin causes keratocyte death. The cornea is repopulated with keratocytes as a result of ingrowth of fibrocytes from the limbus; corneal clearance therefore begins peripherally[9-11].

The natural history of corneal blood staining has been documented by slit-lamp microscopy. Initially, corneal oedema occurs as a result of endothelial dysfunction indicating that blood staining is imminent. The earliest sign is the presence of fine yellow granules in the posterior stroma. A straw yellow discoloration of the stroma subsequently develops, progressing to a reddish-brown discoloration over several days. Over time, the discoloration changes through various shades of greenish-black to grey. Corneal blood staining extending to Bowman's layer and the epithelium has been reported in severe cases , and a greyish discoid opacity deep in the stroma can simulate an anterior dislocation of the lens[9-10].Clearance of the blood staining begins peripherally and progresses centrally, and can take up to 3 years12. Occasionally clearance is incomplete leading to permanent opacification 13.

Therapeutic efforts should be made at the earliest to prevent corneal blood staining in cases of hyphemas presenting early. Corneal blood staining is most likely with a total hyphema and an IOP greater than 25 mmHg for longer than 6 days9. Surgical evacuation of the hyphema is indicated at this stage or at the first microscopic sign of blood staining14. Thereafter, management options include awaiting spontaneous

clearance or penetrating keratoplasty11,15. In this case, corneal staining was present at the first consultation only. Long time follow up will be done to determine the outcome in this case.

Conclusion

Corneal bloodstaining is undoubtedly a vision-threatening complication of total hyphema after ocular trauma. Timely control of IOP is important to prevent corneal blood staining and optic nerve damage. Furthermore, removing the whole hyphema as soon as possible may decrease the severity of corneal blood staining and shorten the course of clearing up spontaneously.

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Address for Correspondence:
Dr. Manpreet Kaur
Professor and Head,
Department of Ophthalmology,
SHKM Government Medical College, Nalhar
E-mail : dr_manpreetkaur@yahoo.co.in
Mobile: 9992223729

Primary localised conjunctival amyloidosis presenting as unilateral ptosis

Neebha Anand MS, **Urmil Chawla** MS, **Reena Gupta** MS,
Jyoti Deswal MS, **Suman Chahar** MS, **Preetam Kurrey** MBBS
Regional Institute of Ophthalmology , PGIMS, Rohtak

Abstract

A 23 years old female presented with watery discharge from right eye and mass in the medial aspect of eye with drooping of right upper lid. On examination, the patient had moderate ptosis of right upper lid with a pinkish red multi-lobulated, friable mass, bleeding to touch involving the medial canthus. CECT showed ill-defined mild enhancing soft tissue attenuating lesion involving and encircling the anterior aspect of the right globe. Patient was taken up for excision biopsy which revealed it to be amyloidosis. Systemic investigations turned out to be normal. This case is being reported here for its rarity.

Keywords : *Amyloidosis,conjunctiva,ptosis.*

Introduction

Amyloidosis is an accumulation of heterogenous, amorphous, proteinaceous material in extra ocular space or any tissue.[1,2] Amyloidosis has mainly two forms, systemic and localized.Systemic amyloidosis is a serious and life-threatening condition because of destruction of tissue and their function due to accumulation of amyloid material while localised amyloidosis has very good prognosis which frequently include head and neck area without systemic manifestation. Primary localized conjunctival amyloidosis is a rare subtype in which abnormal amyloid material accumulates in substantia propria and around conjunctival vessels, it presents as painless,well- vascularised subconjunctival lesion which can be solitary or multiple and usually bilateral. Sometimes it may present with subconjunctival haemorrhage,yellow subconjunctival mass, orbital mass, lid thickening and blepharoptosis.Several factors which attribute to ptosis are frequent episodes of haemorrhage causing recurrent eyelid swelling resulting in levator aponeurosis dehiscence from tarsal plate. Other causes may be a large amyloid mass causing mechanical ptosis or muscle infiltration with abnormal amyloid causing mechanical ptosis. Continuous rubbing of conjunctival mass may cause ocular surface irritation.[3-8]

Here, we present a rare case of primary localised amyloidosis presenting as unilateral ptosis with histopathological finding suggestive of extracellular amorphous and eosinophilic hyaline deposits underneath the conjunctival epithelium in substantia propria.The tissue stained pink with Congo red staining and showed apple green bi-refringences under polarised light, thus confirming the diagnosis of amyloidosis

Case Report

A 23 years old female presented to our eye OPD with watery discharge from right eye and mass in the medial aspect of upper lid, medial canthus and inferior fornix. On examination, the patient had moderate ptosis of right upper lid with a pinkish –red multi-lobulated, friable mass, bleeding to touch involving the medial canthus. On everting the upper lid the mass was seen to be involving medial half of the right upper lid till the superior tarsal edge. Ptosis was moderate with good LPS action (Figure 1 and 2).

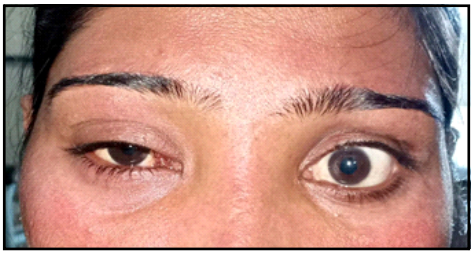

Fig.1: Right eye showing ptosis

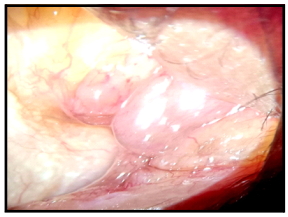

Fig.2: Pinkish lobulated mass in medial canthus

Ocular movements were full in all directions. A probable diagnosis of conjunctival papilloma/ conjunctival squamous carcinoma was kept and CECT orbit was advised. CECT showed ill- defined mild enhancing, iso to hypodense soft tissue attenuating lesion involving and encircling the anterior aspect of the right globe with mild oedematous changes in the preseptal segment of right upper lid(Figure 3). Tiny calcific foci were seen in right upper lid with a probable diagnosis of neoplasm. Patient was taken up for excision biopsy. The lesion was removed carefully from the bulbar, palpebral and forniceal conjunctiva(Figure 4).

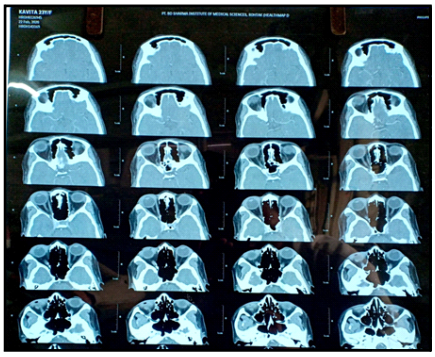

Fig.3: CT scan showing soft tissue hypodense mass medially in right eye.

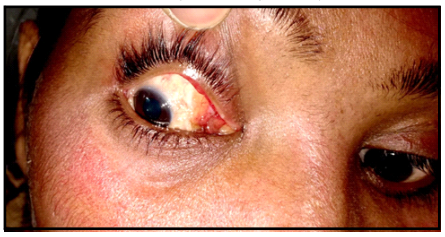

Fig .4: Right eye after excision of the mass

It was found not to be involving the deeper tissues (deeper to conjunctiva) of the upper lid. Histopathological examination showed extracellular amorphous and eosinophilic hyaline deposits underneath the conjunctival epithelium in substantia propria (Figure 5).

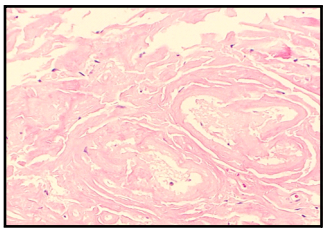

Fig.5(a): Histopathology of conjunctival tissue showin eosinophilic hyaline deposits and amorphous material

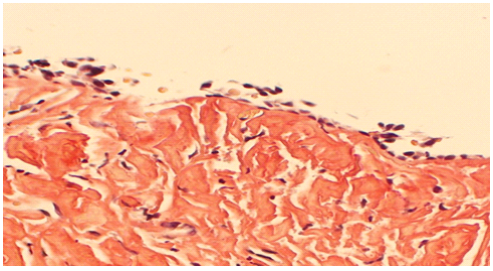

Fig.5(b): Higher magnification of biopsy specimen

Lesion was composed of a few foci of calcifications. The tissue stained pink with congo red staining and showed characteristic apple green bi-refringences under polarised light suggestive of amyloid deposition. To rule out systemic amyloidosis, certain specific tests were done like urine routine analysis, CBC, abdomen and pelvis ultrasound scan and ECG , which all were normal.

Discussion

Localized amyloidosis is a rare disorder with orbit involvement seen in 4% of all cases involving head and neck region. Primary amyloidosis can involve all ocular structures[9] Usually focal orbital amyloidosis has deposition in eyelid or conjunctiva and in superior portion of the orbit. Typically, patient presents with unilateral or bilateral solitary or multiple firm, rubbery, painless bleeding mass. It is usually found in young and middle age group and rarely in old age. Most common non familial ophthalmic manifestation of amyloidosis is the localised form in conjunctiva. Condition is usually unilateral as in our case. Our patient had ptosis which does not seem to be due to neurological cause because there is no restriction of movement in any gaze. The amyloid deposition in levator palpabrae superiosis probably attributes to the ptosis in the patient.Amyloidosis must be considered as a differential diagnosis of conjunctival neoplasm.[10-12]

Various treatment modalities are available for amyloidosis including conservative local excision,debulking,cryotherapy, and superficial cobalt therapy. Surgical complications have been minimized due to careful debulking of deposits with a spooned curette, preservation of anatomic planes, avoidance of normal lid tissue sacrifice and careful dissection with diathermy needle.Mass excision is the

gold standard but it should be as conservative as possible. Total excision of the lesion is usually impossible and surgery should be performed to excise main part of the lesion with preservation of the palpebral lobe of the lacrimal gland, the levator palpabrae superioris and the extraocular muscles.[13-15] Congo red staining is the best method in histopathological examination with findings of a green birefringence under a polarizing light. Histopathological findings in the case were consistent with amyloidosis. Calcification was probably due to chronicity.Systemic examinations and laboratory tests were normal which excludes systemic amyloidosis.

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Address for Correspondence:
Dr Neebha Anand
Professor, Squint and Oculoplasty Unit,
RIO, PGIMS,
Rohtak-124001, Haryana
E-mail : drneebha@yahoo.co.in
Mobile : 9215557072

Management of limbal dermoid

Neha Yadav MBBS, **Sumit Sachdeva** MS,
Manisha Rathi MS, **Priyamvada Yadav** MS, **Shreay Vashisth** MBBS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Epibulbar dermoids are benign, congenital choristomas that may vary in size, location and depth causing variable symptoms like astigmatism, amblyopia or total loss of vision. Their management presents with various challenges because of varied presentation and cosmetic reasons. This article summarizes the various surgical and medical management options for different grades of limbal dermoids along with a case report of a young female who presented with a limbal dermoid with mild astigmatism, which was corrected with glasses.

Keywords: *Dermoid, limbal, choristoma.*

Introduction

Choristoma is a benign congenital tumor that consists of histologically normal tissue that is derived from germ layers foreign to that anatomic location. Limbal dermoid is a form of epibulbar choristoma constituting collagenous connective tissue with a sebaceous component. They can be unilateral or bilateral, single or multiple, and mostly located on bulbar conjunctiva, cornea or limbus.[1] Amongst limbal dermoids, the most common location is inferotemporal[2]. It is an embryological anomaly occurring at 5-10 weeks gestation in which there is metaplastic transformation of mesoblast between surface ectoderm and rim of the optic nerve[3]. Epibulbar dermoids are congenital lesions with connective tissue hyperproliferation with sebaceous component. Histopathological examination shows sebaceous gland acini and hair follicles inside a well defined nodule of fibrous connective tissue.[4] Limbal dermoid can be isolated finding or may be associated with other ocular findings like aniridia, aphakia, microphthalmia, scleral staphyloma or corneal staphyloma. It can also be associated with some syndromes like Nager acrofacial dysostosis or Goldenhar syndrome which is now named Goldenhar-Gorlin syndrome after including vertebral anomalies[5]. Limbal dermoids are classified into three grades anatomically as described below:[6]

- Grade 1- superficial lesions measuring less than 5 mm and localized to limbus
- Grade 2- lesion covering most of cornea (more than 5 mm) and extending deep into stroma but not involving Descemet's membrane
- Grade 3- lesion covering whole cornea and extending through histological structures between anterior surface of eyeball and pigmented epithelium of iris.

The clinical features may be cosmetic disfigurement or diminution of vision due to astigmatism depending upon the size of dermoid. Management includes medical management and surgical correction.

Case Report

A 17year old female presented to our OPD with an asymptomatic lesion in her right eye which was present since birth. She was referred from Medicine department for eye opinion regarding the lesion. She had normal facial features and no skeletal anomalies were noted. Audiometry was done to rule out any hearing deficit, and was found out to be within normal limits. Detailed examination of eye showed nasal limbal dermoid in right eye (Figure 1). Pupil was eccentric and reacting to light.

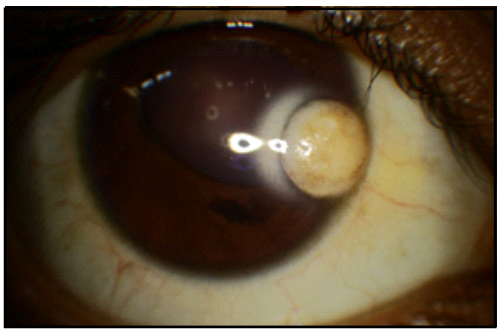

Fig. 1 : Right eye showing limbal dermoid

Vision(unaided) in right eye was 6/9 and left eye was

6/6. Refraction corrected the vision in right eye to 6/6 with addition of cylindrical lens. Slit lamp examination showed the dimensions of dermoid to be 3mm × 3mm and depth extending to stroma classifying it as grade 2 dermoid. The lesion also showed 2 hair strands projecting out from it. Anterior chamber was of normal depth and other structures also showed no deviation from normal anatomy. (Figure 2) Fundus examination showed no abnormality.

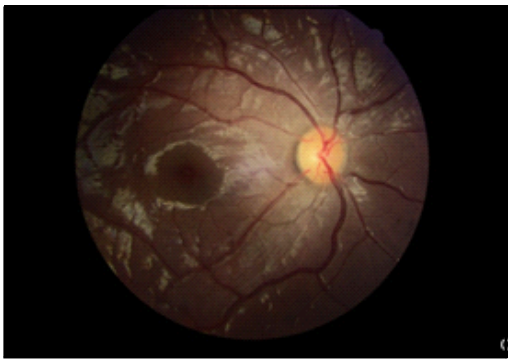

Fig. 2 : Right eye showing normal fundus

Detailed examination of the other eye turned out to be completely normal. Patient was advised removal of lesion under local anaesthesia for cosmetic reasons but she refused for any intervention and didn't come for follow up.

Medical Management.

Management in literature mostly says to "leave these lesions alone". Medical management is recommended in grade1dermoids with upto 1Dioptre astigmatism. Patients who are managed conservatively with spectacles should be kept on regular follow-up and compliance should be ensured. Regular visits should be planned every 2-3 months. Visual acuity, refraction, size of lesion using digital photography should be monitored at every visit. Possibility of amblyopia should also be kept in mind in pediatric age group. On any visit, if any of above parameters are affected or patient is not compliant, surgery should be considered.

Surgical Management

Dermoids are benign lesions, but they might require surgical excision in many cases. Enlarging lesions can cause astigmatism, disturbance of tear film leading to irritation and frequent rubbing of eyes.

Surgical correction should be considered in conditions described below

Table 1

1	Chronic eye rubbing due to irritation and recurrent conjunctivitis
2	Amblyopia unresponsive to medical management
3	Progressive dellen, with corneal surface decompensation
4	Growth encroaching into pupillary area or optical zone
5	Irregular astigmatism
6	Inadequate lid closure
7	Cosmetic indication

For dermoids greater than grade 2, surgical management is indicated. Surgeries vary from simple excision to keratoplasty. Simple keratectomy is advised for small and superficial lesions, with major disadvantage being the residual astigmatism.[8]

Keratoplasty can be lamellar or penetrating, with only central or full curvature graft. The advantage of these procedures include correction of astigmatism[9,10] as well as improved cosmesis while the disadvantages being re-epithelization, interface neovascularisation, steroid-induced glaucoma and graft rejection.[11]

Newer techniques include corneal- limbal scleral donor graft transplantation and surgical resection followed by amniotic membrane transplantation.[10,12,13] Multilayered amniotic membrane graft is considered better for volumetric filling of the defect created by excision.[14] Some studies have proven autologous limbal stem cell transplantation as a good option to compensate for the defect created due to excision of large dermoid[15] Recent advances state that use of fibrin glue to transplant amniotic membrane has reduced chances of graft rejection.[16] Ongoing studies suggest the future management of limbal dermoid with pericardial patch graft as reconstructing alternative along with amniotic membrane multilayered graft.

The best surgical option for every case can vary according to various factors and it is summarized in table 2.

Table 2 : Recommendations for surgical removal of ocular dermoids[7]

Grade of ocular dermoid	Recommended techniques
Grade 1 (<50µm thickness and <1mm diameter)	Simple excision
Grade 1 (<100µm thickness and <1 mm diameter)	Keratectomy +AMT +ALSCA
Grade 2 and deeper grade 1	Keratectomy +AMT +ALSCA +PPG versus anterior or deep anterior lamellar keratoplasty ±AMT
Grade 3	Total anterior segment reconstruction

Abbreviations: AMT- amniotic membrane transplant, ALSCA- autologous limbal stem cell allograft, PPG- pericardial patch graft

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Address for Correspondence:

Dr. Neha Yadav

Junior Resident,

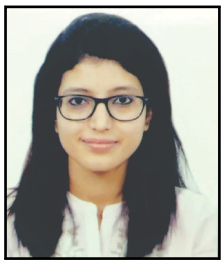
RIO, PGIMS,

Rohtak-124001 (Haryana)

E-mail: dryadavneha93@gmail.com

Mobile: 8398061082

Pituitary macroadenomas presenting as isolated sixth nerve palsy



Preeti Yadav MBBS, **Manisha Nada** MS DNB,
Monika Dahiya MS, **Jitender Phogat** MS, **S V Singh** MS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Abstract

Pituitary adenoma is the most common cause of sellar masses, after the third decade of life, which may present with various neuro-ophthalmic manifestations. Common visual manifestations of adenoma are visual impairment and third nerve palsy. Here, we are reporting a case of ACTH positive pituitary macroadenoma presenting as isolated sixth nerve palsy without apoplexy in a middle-aged male.

Keywords: *Diplopia, pituitary adenoma, sixth nerve palsy.*

Introduction

Pituitary adenoma is an eminent pathology comprising a heterogenous group of tumors with an overall incidence of 20-25% in general population.[1] Pituitary adenoma is the most common cause of sellar masses, after the third decade of life, which may present with various neuro-ophthalmic manifestations due to intimate anatomical proximity of pituitary gland with optic chiasma and cranial nerves in the cavernous sinus.[2] Visual manifestations of pituitary adenoma range from being asymptomatic to advanced deterioration of visual acuity up to blindness. The classical presentation is bitemporal hemianopia on perimetry secondary to compression of optic chiasma.[3] Ocular motility disorders in pituitary adenoma are rare manifestations, only few case reports are available in literature. Lateral extension of tumor into cavernous sinus can cause compression of third cranial nerve, resulting into diplopia and restriction of ocular motility.[4] However, isolated sixth nerve palsy is a rare presenting manifestation of pituitary adenoma. These tumors typically grow slowly unless pituitary apoplexy occurs, which is defined as an acute haemorrhagic infarction of the pituitary adenoma resulting into increased intrasellar pressure often resulting in acute onset of headache, nausea, vomiting, ophthalmoplegia and visual loss.[5] We report a case of an isolated sixth cranial nerve palsy as the presenting clinical feature of a rapidly expanding ACTH positive silent tumor without any apoplexy.

Case Report

A 44 year old male with no significant past medical history presented to our OPD with a chief complaint of vague headaches and diplopia since 5 days. His ocular examination showed BCVA of 6/6 in both eyes with +3 DS correction with normal anterior segment. Neuro-ophthalmologic exam revealed an isolated, incomplete, right sixth nerve palsy resulting into Right Convergent Squint (RCS) with restriction of right lateral rectus (Figure 1).

Fig.1(a): RLR restriction Fig.1(b): RCS in primary gaze

Humphrey visual field testing with 24-2 testing strategy was normal and dilated fundus examination revealed the absence of papilledema and a normal retinal periphery (Figures 2&3). There was no clinical evidence of either myasthenia gravis or thyroid ophthalmopathy.

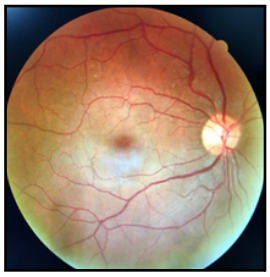


Fig 2: Right eye fundus

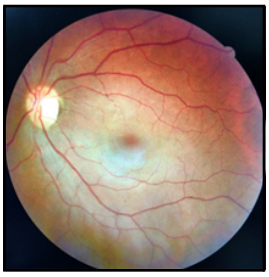


Fig.3: Left eye fundus

An MRI of the orbits and brain revealed a large T2W/FLAIR heterogenous hyperintense and T1W hypointense lesion of size 26x27x20 mm with tiny cystic areas, located in pituitary fossa, extending into right cavernous sinus and inferiorly bulging into sphenoid sinus suggestive of pituitary macroadenoma.

The patient was immediately admitted to the neurosurgery department for preoperative studies and scheduled for urgent transsphenoidal hypophysectomy. Preoperative and systemic work up was initiated, inclusive of complete blood count, metabolic profile, coagulation studies, sedimentation rate, thyroid function tests, acetylcholine receptor antibodies,(both binding and blocking), angiotensin converting enzyme and pituitary hormones level. All tests were within normal limits aside from an elevated ACE and ACTH.

The patient underwent urgent transsphenoidal hypophysectomy for this rapidly progressive tumor. Intraoperatively, the tumor was found to have clinical extension into the cavernous sinus. Pathologic examination of the mass demonstrated a pituitary adenoma positive for ACTH immunostain. No frank haemorrhage or necrosis was noted. Post-operatively the patient was placed on intravenous decadron, which was tapered over 10 days. His VIth nerve palsy and horizontal diplopia resolved over the subsequent three months. Now, 6 months after his surgery, he remains free of symptoms with complete ocular motility, 6/6 BCVA and full visual fields.

Discussion

The cavernous sinus contains the carotid artery as well as the oculomotor, trochlear, ophthalmic and maxillary divisions of the trigeminal and abducens nerves. Extraocular palsy generally indicates compression of the caver- nous sinus wall or direct extension of the pituitary adenoma into the cavernous sinus.[6] The incidence of ocular palsy occurring with pituitary tumors has been reported to be between 4.6 and 32%.[7-10] Most commonly oculomotor nerve is affected and rarely abducens nerve get involved.[11] The sixth cranial nerve runs lateral to the internal carotid artery, but medial to the third, fourth, and first and second divisions of the fifth cranial nerves which run superior to inferior within the lateral dural border of the cavernous sinus. It is therefore more often spared because of its more sheltered position within the sinus.[10]

Several published studies had suggested various mechanisms for the pathophysiology of cranial nerve palsies in pituitary adenoma; including either indirect compression on the ocular cranial nerves by compressing the cavernous sinus or direct compression through cavernous sinus invasion.[12] Adenomas that usually invade the cavernous sinus grow through fragile medial sinus wall with mediolateral expansion resulting in lateral displacement of the internal carotid artery, with third nerve compression in the oculomotor trigone. Thus the most commonly involved by the compression is the third nerve. At the oculomotor trigone there is a whole cistern of cerebrospinal fluid which represents a possible route for tumor invasion.[13,14] Vascular occlusion also has been reported as a mechanism for third nerve palsy, due to compression of its blood supply originating from the internal carotid artery.[14] Other proposed mechanism for ocular palsy was the occurrence of pituitary apoplexy with rapid deterioration of symptoms. Isolated sixth nerve palsy as a presentation of pituitary adenoma is very rare. The sixth nerve passes within isolated fourth nerve palsy is very rare except in case of massive compression of the cavernous sinus, involving all the ocular motor nerves.[15]

Conclusion

Pituitary macroadenoma presents as isolated sixth nerve palsy rarely, but we should consider it in cases of acute onset of ophthalmoplegia and diplopia. The most common ocular cranial nerve involved is the isolated third nerve palsy followed by sixth nerve palsy. Early diagnosis and timely tumor excision with cranial nerves decompression has a favourable outcome regarding the improvement of ocular motility.

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Address for Correspondence:
Dr. Preeti Yadav
Junior Resident,
RIO, PGIMS,
Rohtak-124001(Haryana)
E-mail : preetiyadav31@yahoo.com
Mobile: 8930688130

Approach to a case of diplopia

Gunjan Chadha MS, **Akanksha Rani** MBBS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Introduction

Diplopia chart is the record of separation of the diplopic or double images in thenine positions of gaze. It can be plotted or charted in patients who cooperate and can appreciate the double vision with inconcomitant or concomitant deviation.

Methods

The patient should be made to sit comfortably with head erect throughout the procedure. The test is carried out in dark room. Patient is asked to wear Red - Green glasses. RED in front of Right eye and Green in front of Left eye. It is desirable to use Armstrong goggles since these are shaped to fit the orbital margin and therefore patient would be looking only through the colored medium. The examiner holds vertical source of light at least 1/2 m or 1 m (distance should be mentioned on the chart).

The light is directly held in front of the patient. If the patient complains of double image in primary position, the direction in which double image appear and the maximum separation of the images should be noted. If the patient notes a double image, the relative position of these images is noted. The light is now carried to the right and then to the other 8 positions of gaze. In each gaze position the patient must be asked whether the images are parallel or tilted; if the torsion is present colored pencils can be given to an observant patient to show the separation in torsion. Also, in each gaze patient should be asked the amount of separation subjectively and its increase in a particular gaze.

Interpretation

To interpret the diplopia chart the most important questions are-

1. The position in which diplopia appears
2. The position in which the separation of the image is the greatest In the direction of the action of the paralyzed muscle, the double vision or the separation would be greatest because of the under action of the muscle and overaction of the antagonist muscle and yoke muscle.

Interpreting Diplopia chart

If two images are joined together – then there is no no diplopia. If images are separated it confirms diplopia. Maximum separation is in the quadrant in which (the muscle moves the eye) the muscle is restricted. The image is displaced towards the field of action of the paralysed muscle. If horizontal separation with uncrossed images is noted then esodeviation is present. If horizontal separation with crossed images arepresent ,then make the diagnosis of exodeviation. If vertical separation with uncrossed images are noted by you then think that the oblique muscles are involved. If vertical separation with crossed images are present , vertical recti muscle are involved.

Fig. 1 : Diplopia in right gaze - where L denotes patients left and R denotes patients right and red googles are worn in front of right eye and green in front of left eye.

In the above figure diplopia is maximum in horizontal gaze in patient's right gaze i,e dextro version . Muscle involved in dextro version are Right lateral rectus and Left medial rectus. The red image is distal it shows Right lateral rectus is paralysed and the deviation of eye in right lateral palsy would be in esodeviation and the image formed in eso deviated eye leads to uncrossed diplopia.

Diplopia chart (precautions)

1. Head must be kept straight during the examination
2. The goggles must be well-fitting
3. The light should be kept upright (or horizontal in case of vertical or torsional diplopia) and held at a consistent distance.
4. The patient should be asked about the tilting of images
5. The light should be visible to both the eyes
6. Never interpret the diplopia chart in isolation, clinical examination and Hess chart should be used in conjunction to come to a conclusion

Conditions where the Hess chating cannot be done-

1. Torsional diplopia
2. Patient who is bed ridden
3. Unable to plot hess chart
4. Unable to plot Binocular field of fixation
5. Diplopia chart should be interpretated with clinical findings and other investigation like the binocular field of fixation, Hess chart.

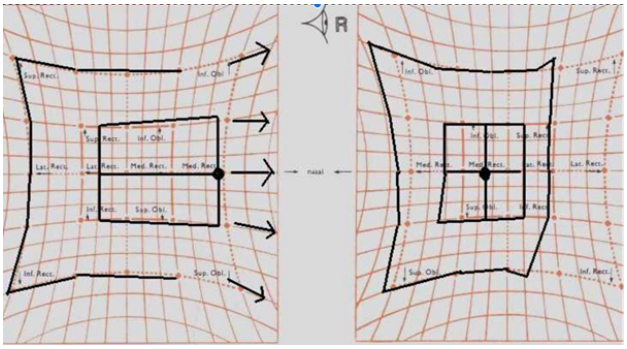

Fig.2: shows right eye is the abnormal eye as it has smaller fields and fixation is towards the nasal side which means eye is in esotropia and right eye shows the restricted field in th direction of lateral rectus this is right lateral rectus palsy

As seen in the left eye the point of fixation is shifted nasally and there is overreaction of medial rectus muscle due to herring's law.

Address for Correspondence:
Dr. Gunjan Chadha
Senior Resident,
RIO, PGIMS,
Rohtak-124001 (Haryana)
E-Mail: gunjanchadha64@gmail.com
Mobile : 8527311237

Approach to a patient with proptosis

Priyamvada Yadav MS, **Urmil Chawla** MS DNB
Regional Institute of Ophthalmology, PGIMS, Rohtak

Introduction

Proptosis is abnormal protrusion of eyeballs and is the hallmark of orbital disease. While developing an approach for evaluation of a patient with proptosis is very important to reach a specific diagnosis with good clinical skills, intelligent use of imaging studies & plan a management algorithm.We should be able to develop some differential diagnosis on the basis of history and physical examination. Imaging studies are used to refine the differential diagnosis. Biopsy (excisional/incisional) may be required to reach the final diagnosis. To reach the differentials ,we have to think of all the different tissues that are present in the orbit viz. glandular tissue, nerves, vessels, muscles, connective tissue, the blood cells within the blood vessels etc. Any abnormality arising from any of the structures present in orbit may lead to proptosis.

Surgical spaces of orbit

Intraconal space contains the optic nerve and orbital fat Extraconal space contains lacrimal gland, superior oblique muscle and trochlear nerve, blood vessels in the extraconal orbital fat.

Subperiosteal space is a potential space between the bones and periorbita.

Subtenon space lies between the globe and the tenon's capsule .

Extra-orbital space surrounds the orbit and is comprised by bone, sinuses, brain ,nose and skin Proptosis True or pseudo.

First objective is to differentiate proptosis from pseudo proptosis.There can be racial differences in globe placement in orbit . So ,asymmetry between the two eyes gives a better idea of presence of proptosis , unless it is bilateral.We should Rule out unilateral enlargement of globe as in high myopia or unilateral lid retraction or presence of contralateral enophthalmos , ptosis etc.

History

History should be elaborate with particular emphasis on certain points.

Age and gender : Most of the orbital pathologies are age specific and tend to occur occur at certain ages. Many of them occur with variable frequency in males and females.

Common causes of proptosis in adults

1. Thyroid orbitopathy
2. Cavernous hemangioma
3. Optic nerve tumors e.g. Meningiomas
4. Lymphoid lesions of orbit
5. Lacrimal gland tumours
6. Metastatic and secondary orbital tumours

Common causes of proptosis in children

1. Orbital cellulitis
2. Dermoid cyst
3. Capillary hemangioma
4. Lymphangioma
5. Optic nerve glioma
6. Rhabdomyosarcoma

Onset and Progression

Whether it has been acute (within hours to days), sub-acute (within weeks), or chronic (several months or years).

Acute onset proptosis is noted in infective causes like orbital cellulitis or with trauma like orbital hematoma. Sub-acute onset is noted in neoplasms, inflammatory causes like thyroid orbitopathy or orbital inflammatory diseases. It should be noted whether the disease is static, progressive or waxing - waning. Patients usually remember the onset & progression with acute illnesses but in case of chronic ones , they may not be able to identify the exact onset or progression ,giving rise to a situation of 'disease first noted when'.FAT (Family Album Tomography) i.e. review of old photographs of patient comes to help in these cases.

Associated features

Deterioration in vision, double vision, watering, swelling around the eye or any other associations have to be noted. Pain is one very important symptom that can act as a clue to the diagnosis .Pain suggests

inflammation ,infection , sudden changes in pressure ,some tumor growing into bone or nerves.

Medical and Systemic History

Any history of weight loss ,smoking, presence of any other systemic disease along with its control status have to be noted .Diagnosis of neoplasm anywhere else in body must be enquired.History of trauma to the face in the past is very important .

Ocular examination

Visual acuity : may be reduced because of exposure corneal exposure or optic nerve compression

Refraction : high myopia may give appearance of proptosis. Any mass indenting posterior pole of globe may give rise to acquired hyperopia .

Intraocular pressure : may be elevated due to restriction of eye movements or in cases of arteriovenous fistula.

Eyelids : look for lid retraction, S shaped deformity of lid, lagophthalmos

Conjunctiva : look for chemosis or prolapse .Dilated episcleral vessels are seen in carotid cavernous fistula. Salmon coloured patch in lymphoma.

Cornea : look for exposure keratopathy

Iris : look for Lisch nodules

Pupil : look for the size and reactions particularly for relative afferent pupillary defect

Ocular motility : restriction may occur due to to compression of nerve ,direct muscle involvement, mechanical limitation, cavernous sinus thrombosis

Fundus examination : look for disc edema ,disc pallor , globe indentation, choroidal or internal limiting membrane folds

Assessment of mass

Inspection

A worm's eye view gives good idea of presence of proptosis. Note should be made for any features of temporal fossa fullness that may be seen in sphenoid ridge meningioma. Also look for any features of craniosynostosis.

Direction of proptosis

Whether it is axial like that seen in cases of thyroid orbitopathy or intraconal disorders like optic nerve tumors , cavernous hemangioma , or non -axial displacement should be noted. If non-axial, look for the direction of displacement .If the eye is pushed downward ,think of problems in the area of lacrimal gland or defects in orbital roof that may be due to trauma in or frontal sinus mucocele formation. In case of lateral displacement, the problem usually lies in ethmoidal sinus that may be a subperiosteal abscess arising in sinus, extending into subperiosteal space, sinus carcinomas or mucoceles .Upward displacement is seen in maxillary carcinomas.If the globe is medially displaced, that is usually due to lacrimal gland pathologies.

Globe Displacement	Common causes
1) Axial proptosis	Thyroid orbitopathy
	Cavernous hemangioma
	Optic nerve gioma
	Optic nerve meningioma
2) Non-axial proptosis	
Lateral	Ethmoid sinus mucocele/abscess
Medial	Lacrimal gland masses
Superior	Maxillary sinus carcinoma Orbital fat tumours
Inferior	Frontal sinus mucocele Sphenoid wing meningioma
3) Enophthalmos	Scirrhous carcinoma breast

Orbital masses usually push the eye out , an exception is infiltrative sclerosing tumor (scirrhous carcinoma of the breast)that leads to enophthalmos.

Hertel's exophthalmometer can be used to measure the degree of proptosis .

Asymmetry between left and right sides is more important than actual measurement.

(To diagnose proptosis on imaging ,a line is drawn between the anterior most part of zygomatic processes. This line should meet the globe at or behind the equator. Another line is drawn from the cornea perpendicular to the above said line .This distance should not be more than 21 mm .If it is so, it is labelled as proptosis. Asymmetry between the two eyeballs is also noted and difference should not be more than 2 millimeter normally.)

Palpation

Start with palpation of orbital rims and then move towards the eye. Feel the superior and inferior fornix for presence of any mass .

If a mass is palpable , it should be evaluated for its size ,consistency ,extent ,tenderness, whether a finger can be insinuated between the mass and the bony orbital rim .Whether the mass is separate from the adjacent tissues or infiltrating ,whether is it is fixed to a bone or nearby structure along with status of overlying skin should also be noted.Infectious or inflammatory disorders cause the skin to be erythematous and

warm.

Any mass which is big enough to cross the midline is usually malignant. Resistance to retro displacement of globe may indicate a tumor or thyroid orbitopathy .

Regional lymph nodes should also be palpated.

Pulsation is best detected on applanation tonometry or on lateral view. This may be noted in arteriovenous fistula or it may be a pulsation transmitted through bony orbital wall defect like in sphenoid wing dysplasia seen in neurofibromatosis .

Feeling for the radial pulse at same time will reveal synchronisation.

Auscultation

A bruit may be heard by stethoscope's bell .

Bruit is heard or thrill felt if it's a high flow lesion. Venous lesions show enlargement with Valsalva maneuver and with dependent head position.

Investigations

Imaging studies are usually required to reach a definite diagnosis. Urgency of the investigation is decided by careful history and examination.

A patient presenting with proptosis ,with history of progression over months to years is not in that much urgency of imaging as is a patient with progression over days to weeks. Other factors like presence of relative afferent pupillary defect also suggest the need for urgent imaging due to optic nerve involvement and endangered vision while a patient with stable thyroid orbitopathy may wait for same.

CT scan is the modality of choice for imaging studies in proptosis. MRI is indicated only in specific cases like lesions at orbitocranial junction .

Biopsy (incisional or excisional) is done depending upon the need.

On imaging we get an idea of what is causing the proptosis - either it is arising from some orbital structure or an orbital mass not arising from a specific structure. It may be well-circumscribed(benign) or infiltrative(malignant). Localise it to a particular orbital compartment and note its characteristics, its relationship to adjacent soft tissues and bone ,whether it gives rise to fossa formation(benign)or causes bony erosion (malignant). Look for the shape, size, internal characteristics (homogeneous/ heterogeneous) of the mass and contrast enhancement.

Localisation of the mass to an orbital compartment is also critical for biopsy and planning a surgical approach.

The patient should be looked at as a whole instead of just the eye. Taking a good and relevant history, meticulous clinical examination with imaging tailored to need and appropriateness for the case, help come to differentials diagnosis, while also providing for planning of surgical approach if needed, supplemented by excisional/incisional biopsy.

A systematic approach with knowledge of anatomy is a must for the same.

Address for Correspondence:
Dr Priyamvada Yadav
Senior Resident,
RIO, PGIMS,
Rohtak-124001 (Haryana)
E-mail:dr.priyamvada29@gmail.com
Mobile : 8979565652

Approach to a case of diabetic retinopathy

Manisha Nada MS DNB, **Monika Dahiya** MS
Aakash Sharma MBBS, **Sakshi Lochab** MBBS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Introduction

Diabetes Mellitus (DM) is one of the most important emerging public health challenges of 21st century and is a global epidemic now. It is a metabolic disorder characterized by hyperglycaemia due to either insulin deficiency or insulin resistance. Diabetic retinopathy is a well known complication of long standing, poorly controlled diabetes mellitus and a leading cause of blindness worldwide. Broadly DR is classified into Nonproliferative Diabetic Retinopathy (NPDR), Proliferative Diabetic Retinopathy (PDR) and diabetic maculopathy.

History

Chief complaints- It primarily depends upon whether associated diabetic maculopathy or PDR is present or not. Patient may present with gradual or acute loss of vision, floaters or paracentral scotoma.

History of present illness - Patient should be asked for onset of loss of vision whether it was insidious onset or sudden onset, gradually progressive or not, painless/painful, corrected by prescription of glasses or not and was it associated with any other ocular complaints like pain, redness, photophobia, watering, discharge, headache/ browache, coloured halos, glare, frequent change of glasses, flashes of light and night blindness.

Past history- Any history of intraocular surgery, ocular trauma, retinal laser for DR and prior treatment with intravitreal injections should always be asked.

Medical history- History of DM must be taken including type of DM, duration, control of blood sugar and whether on regular treatment or not. Patient should be thoroughly asked whether he/she is on OHA or insulin. Comorbid conditions must be ruled out like hypertension, hyperlipidaemia, anaemia and pregnancy as they can worsen diabetic retinopathy.

Personal history- Smoking and alcohol intake must be asked in every patient.

Family history- Ask if there is any history of diabetes mellitus and hypertension.

Examination

General Physical Examination: A detailed general physical examination must be done in every patient to rule out hypertension, pallor, xanthelasma, any associated neuropathy/nephropathy and diabetic foot.

Ocular Examination

Visual acuity- Best Corrected Visual Acuity (BCVA) should be noted both for distance and near.

Eyeball- Look for cranial nerve palsies. Classically pupil sparing 3rd nerve palsy and 6th nerve palsy are signs of reversible ischemic mononeuropathy.

Lid- Xanthelasma (suggestive of hyperlipidaemia) can be seen. Recurrent hordeola and blepharitis can be seen in uncontrolled diabetes.

Conjunctiva- Increased risk of developing bacterial infections.

Cornea- Look for corneal hypoesthesia secondary to associated neuropathy and tear film abnormalities as a manifestation of autonomic neuropathy.

Iris- Look for Neovascularisation of Iris (NVI) as a manifestation of PDR.

Pupil

- Ectropion uvea due to eversion of posterior pigmented layer at pupillary margin secondary to contraction of fibrous tissue accompanying neovascularisation.
- Difficulty in dilating pupil as a manifestation of diabetic neuropathy resulting in reduced functional innervation to dilator muscle.
- Argyll Robertson Pupil- Bilateral small pupils which accommodate but do not constrict when exposed to bright light.
- Sluggish pupil may be present in diabetic papillopathy.
- IOP-DR can be associated with POAG and NVG.

Lens

- Reduction in accommodative ability may be noted.
- Typically nuclear and cortical cataract formation in chronic and progressive cases.
- Acute cortical cataract formation with profound elevations in blood glucose
- Snow flake cataract : white subcapsular opacification may be seen in young patients with type 1 DM.
- Rapid progression of senile cataract.

Vitreous- Precocious liquefication and Posterior Vitreous Detachment (PVD) due to abnormal collagen cross linking and nonenzymatic glycation. Asteroid hyalosis can also occur in diabetics.

Gonioscopy to rule out Neovascularisation of Angle (NVA)

Fundus- Grading of DR should be done based on fundus findings and make coloured fundus diagram.

Table 1: Fundus changes in Diabetic Retinopathy

Clinical Sign	Pathogenesis	Layer of retina affected	Effect of treatment
Cotton wool spot	NFL infarcts	Nerve fibre layer	Persists
Microaneurysms	Secondary to capillary wall outpouching due to pericyte loss.	Superficial retinal layers	Resolves if controlled
Dot and blot haemorrhages	Microaneurysm rupture in deeper layer of retina	OP&IN layer	Resolves gradually
Flame shaped haemorrhages	Splinter haemorrhages in NFL	NFL	Resolves gradually
Hard exudates and Retinal edema	Breakdown of blood-retina barrier, allowing leakage of serum protein and lipids	OP&IN layer	Complete resolution is unlikely

ETDRS Classification of DR

DR is divided into NPDR and PDR which is further subdivided-

Non Proliferative Diabetic Retinopathy (NPDR)

1. Mild NPDR: microaneurysms only
2. Moderate NPDR: Microaneurysm, retinal hemorrhages, hard exudates, cotton wool spots, venous looping/beading, IRMA.
3. Severe NPDR: Any one of 4:2:1 rule
   - Blot hemorrhages in all 4 quadrants
   - Venous beading in 2 quadrants
   - IRMA in 1 quadrant
4. Very Severe NPDR: Any 2 or above of mentioned 4:2:1 rule.

Proliferative Diabetic Retinopathy (PDR)

1. Early PDR
2. High Risk PDR: NVD>1/4-1/3 DD
   - NVD with preretinal bleed or vitreous hemorrhage
   - NVE>1/2 DD with preretinal bleed or vitreous hemorrhage

Clinically Significant Macular Edema (CSME): Most common cause of moderate vision loss in DR patients.

1. Thickening of retina at or within 500 microns of the center of the macula
2. Hard exudates at or within 500 microns of the center of the macula
3. Retinal thickening of at least 1 DD size, with any part within 1 disc diameter of the macula center.

CSME can be focal or diffuse and with or without hard exudates. In focal CSME, microaneurysms are the main leaking lesions. It clinically appears as focal areas of macular thickening surrounded by ring of hard exudates. While in diffuse CSME, FFA shows leakage from widespread capillary abnormalities, dilated capillary bed and associated with areas of capillary nonperfusion (CNP). Clinically it may not be associated with hard exudates as diffuse breakdown of inner blood retinal barrier (BRB) allows passage of smaller molecules, not lipoproteins.

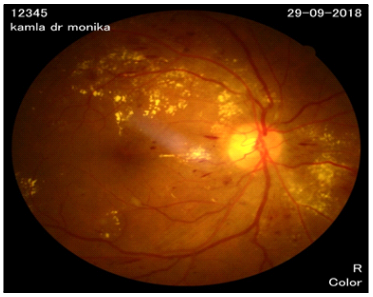

Fig. 1: Right eye showing severe NPDR with CSME

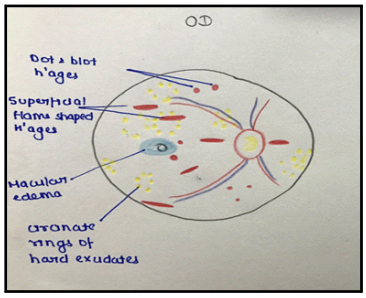

Fig. 2: Colour fundus diagram of right eye

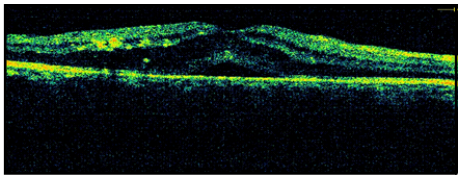

Fig. 3: OCT of right eye showing spongiform macular edema with hard exudates and with subfoveal neurosensory detachment

Investigations

1. HbA1C to see glycemic control over past 3 months
2. Lipid Profile
3. Complete hemogram
4. OCT
   Macular thickening can be quantified on OCT and it helps in deciding treatment protocol of diabetic macular edema, whether medical management or surgical management.

OCT based classification of DR -

- Spongiform macular edema
- Cystoid macular edema- cystic spaces separated by hyperreflective septas
- Serous Retinal Detachment- hyporeflective space under neurosensory retina
- Tractional macular edema- Secondary to Vitreofoveal traction (VFT)/Vitreomacular traction (VMT) and needs surgical management.
- Taut Posterior Hyaloid Membrane- Leads to recalcitrant diabetic macular edema and seen as hyperreflective membrane on retinal surface and needs surgical management.

5. FFA- To guide focal and grid laser
   To assess amount of ischemia in severe NPDR
   To look for subtle NVE

Management of DR

Aim Target HbA1C <7.0
Target Systolic BP<130 mmHg
Target LDL cholesterol < 2.5mmol/L
Target triglycerides < 2mmol/L

• NPDR

Focal laser -100 micron laser burns applied to areas of leaking microaneurysms sparing central 500 micron.
Grid Laser- 100 micron threshold burns applied in grid pattern for diffuse leakage, ideally guided by FFA. It is associated with minor loss in visual field, transient increase in macular edema, subretinal fibrosis, inadvertent foveolar burns and rarely CNVM.

Intravitreal injections- For diabetic macular edema anti-VEGFs (Bevacizumab, Ranibizumab, Aflibercept) and intravitreal steroids (Triamcinolone Acetate, Ozurdex) are given.

• PDR

Pan Retinal Photocoagulation (PRP) should be done as soon as possible avoiding areas showing traction.
Anti-VEGFs can be given to prevent rebleed.

Vitrectomy

Type 1 DM with severe vitreous haemorrhage (VH), severe PDR and VFT/VMT

Follow Up Schedule

Mild NPDR- 1 year
Moderate NPDR- 6-12 month
Severe NPDR- 4 months
Very Severe NPDR- 3 months
PDR- 2 months
If CSME is present in any stage of DR then follow up should be done based on OCT.

Address for Correspondence:
Dr. Manisha Nada
Professor,
RIO, PGIMS,
Rohtak-124001 (Haryana)
E-mail : manisha_nada@rediffmail.com
Mobile : 9896007158

Approach to a case of retinal detachment

Monika Dahiya MS, **Manisha Nada** MS DNB,
Jitender Phogat MS, **Manoj P** MBBS, **Preeti Yadav** MBBS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Introduction

Retinal detachment refers to separation of the neurosensory retina from the underlying retinal pigment epithelium. It is classified mainly into 3 types:

1. Rhegmatogenous Retinal Detachment (RRD)- it is most common type of retinal detachment. It occurs secondary to a hole, tear or break in the neuronal layer which allows fluid from vitreous to seep in between the neurosensory retina and underlying RPE resulting into Rhegmatogenous Retinal Detachment.
2. Tractional Retinal Detachment (TRD)- It results from adhesions between the vitreous gel/ fibrovascular proliferation and the retina.
3. Exudative/Serous Retinal Detachment- it results from exudation of material into the subretinal space from retinal vessels as in HTN, CRVO, vasculitis, uveitis, tumors et

History

Chief complaints- a case of RRD usually presents with flashing lights, floaters, loss of vision and visual field defects.

History of Present Illness (HOPI)

- Photopsia- it includes sensation of a flashing light related to retinal traction. It is typically described as sensation of falling stars, even when eyes are closed or in a dark room. A shower of floaters and vision loss usually accompany it.
- Floaters- It is very common visual symptom and to distinguish its etiology, always elicit a detailed history. Sudden onset of large floaters in the centre of visual axis may indicate posterior vitreous detachment (PVD) and patient observes a circular floater. Description of hundreds of tiny black spots or insects floating in front of eye is usually suggestive of vitreous hemorrhage resulting from retinal blood vessel tear caused by a retinal tear or mechanical traction of a vitreoretinal adhesions.
- Field defects- Patient may report a black curtain or shadow in peripheral visual field, which over a period of few days may spread to involve the entire visual field. Bullous RD produces dense visual field defects while flat RD produces a relative visual field defects. Visual field defects can be helpful in guessing the probable quadrant of detachment.
- Vision Loss- If RD involves macula, patient presents with sudden painless loss of vision.

Past Ocular History- For RRD, we should enquire regarding family history of retinal disorders or degenerations, myopia or prior retinal therapies. History of any ocular trauma or prior intraocular surgery must be taken. For tractional RD and exudative RD, systemic history of Diabetes mellitus, hypertension, tuberculosis, tumors and infections is very important.

Past Surgical History

- History of vitreous loss during cataract surgery
- Previous laser capsulotomy
- IOFB removal

Family History- It is helpful in certain cases of familial RD where retinal degenerations are hereditary. Example- Familial exudative vitreoretinopathy (FEVR), Familial retinal dialysis, Stickler syndrome, Knobloch syndrome, Marfan syndrome, Homocystinuria etc.

Ocular Examination

Visual Acuity- Check VA for near and distance and correction for refractive error must be noted. Never forget to look for myopia in fellow eye.

External examination- for any signs of trauma.

Anterior segment- Look for signs of trauma: stability of lens barrier and media clarity. Uveitis and NVI may be seen in tractional and exudative RD.

Pupil- a fixed dilated pupil may indicate previous trauma; a positive Marcus-Gunn pupil can occur with

any disturbance of the afferent pupillomotor pathway, including retinal detachment. RAPD is more likely to be seen clinically if RD is bullous, involving >2 quadrants and especially if the macula is off.

IOP- a relative hypotony of >4-5mmHg than fellow eye is quite common. If IOP is extremely low, choroidal detachment may be present. IOP may be raised in Schwartz-Matsuo syndrome in which RRD is associated with a mild anterior uveitis and blockage of angles by parts of photoreceptors.

Vitreous- Look for Shaffer sign i.e pigment or tobacco dusting which is suggestive for retinal tears in 70% of cases with no previous eye disease or surgery. Look for Vitreous haemorrhage in TRD cases, retrolental cells in exudative RD and vitreous degeneration in rhegmatogenous RD.

Fundus Examination- Indirect ophthalmoscopy is the definitive means of diagnosing RD. Direct ophthalmoscopy can diagnose vitreous hemorrhage and large detachment of posterior pole, but is inadequate for complete examination because of lower illumination, lack of stereopsis and limited view of peripheral retina. All findings must be recorded in a modified Amsler-Dubois chart. RD is seen as marked elevation of retina, which appears gray due to loss of transparency with dark blood vessels that may lie in folds. The detached retina may undulate and appear out of focus. Shallow detachments are more difficult to detect; thus suspected area should be compared with an adjacent normal quadrant to detect any change in transparency. A pigmented/non-pigmented line may demarcate the line of detachment. Never forget to examine fellow eye for for the risk factors of RD.

	Rhegmatogenous RD	Tractional RD	Exudative RD
History	Photopsia Visual field defects	DM, penetrating trauma, sickle cell disease	Malignant HTN, eclampsia, renal failure, uveitis, trauma
Retinal break	Present	No primary break, may develop secondary break	No break or coincidental
Extension	To ora serrata	Does not extend to ora	Gravity dependent
Retinal Shape	Convex	Concave	Extremely high convex
Motility	Undulating	Taut, peaks to traction point	Smoothly elevated bullae
Subretinal fluid	Clear	Clear	May be turbid, shift rapidly to dependent location
Choroidal mass	None	None	May be present
PVR	+	-	-

Differential Diagnosis

- Posterior uveitis/scleritis
- Posterior Vitreous Detachment
- Vitreous hemorrhage
- Vitreous syneresis
- Thick hyaloid
- Vitreous membranes
- Retinoschisis
- Retinal cyst
- Subretinal exudates
- Retinal mass
- Choroidal detachment

Investigations

USG B-Scan: In the presence of hazy media, it helps in differentiating type of RD, rules out IOFB, subretinal mass, choroidal detachment etc.

Table 2- Different type of RD on USG B Scan

Rhegmatogenous RD	Tractional RD	Exudative RD
Convex elevation	Concave	Extremely high convex
Undulating folds	Bridging tractional bands	Shifting fluid +

Table 3- Differentiating features of RD, PVD and CD on B Scan

PVD	RD	Choroidal detachment
Smooth, with or without disc insertion	Smooth or folded with disc insertion	Smooth, convex shaped with no disc insertion
<100% spikes	100% spikes	Double 100% spikes
Marked after movements	Moderate	None

Management

Rhegmatogenous RD - Usually surgical including vitrectomy, scleral buckling and pneumatic retinopexy.Laser treatment is done wherever needed.

Tractional RD - Observation or vitrectomy depending on the macular status with or without lasers.

Exudative RD - Observation or steroids.

	Scleral buckling	VR surgery	Pneumatic retinopexy
Indications	RRD Inferior retinal breaks Retinal dialysis Pediatric RD	RD with PVR changes RD with GRT RD with VH RD with IOFB	Single break, in superior 8 clock hours Break should not be more than 1 clock hours Multiple breaks within 1-2 clock hours
Contraindications	Posterior breaks Opaque media PVR	Bleeding disorders Suspected tumors like RB, Melanoma	Break > 1 clock hour Break in inferior 4 clock hours Uncontrolled IOP Hazy media
Complications	Perforation Raised IOP Extrusion Diplopia Anterior segment ischemia ERM Recurrent RD	Iatrogenic breaks Lens trauma Secondary glaucoma PVR Cataract progression	Vitreous incarceration Subconjunctival gas Missed breaks PVR Persistent SRF Vitreous haze Sudden rise in IOP
Benefits	Excellent anatomic results Good visual outcomes	Visualization of all tear/breaks Anatomic success in complicated detachments	Minimally invasive Reduced recovery time Better postoperative visual acuity

Address for Correspondence :
Dr. Monika
Senior Resident,
RIO, PGIMS,
Rohtak-124001(Haryana)
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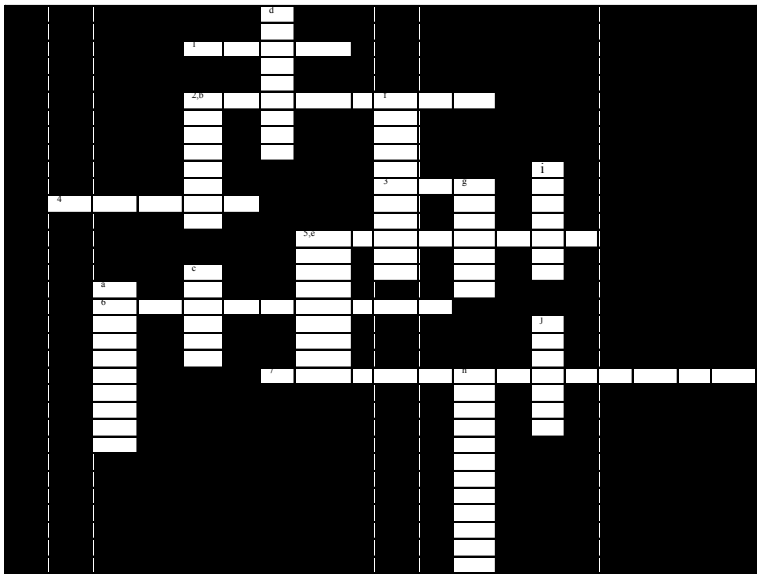
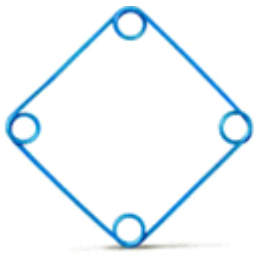
Mobile : 8813023464

HOS QUIZ- JANUARY 2021
OPHTHALMIC CROSSWORD



HORIZONTAL ROWS:

1. A prospective randomized controlled trial of intravitreal bevacizumab or laser therapy in the management of diabetic macular edema.
2. The brown iron line in the cornea anterior to the head of the pterygium.
3. Fourth layer of cornea.
4. A partial thickness corneal transplant procedure.
5. Identify this ring…



6. Identify this instrument…

7. Measurement of corneal sensation.

VERTICAL COLUMNS

a. Principle on which applanation tonometry is based.

b. Most common inherited cause of rhegmatogenous retinal detachment.

c. A clinical test to determine the reflux of fluid through the puncta.

d. He invented direct ophthalmoscope.

e. A term used for loss of eyelashes and eyebrows.

f. An inherited connective tissue disorder which may present as blue sclera, microcornea or subluxation of lens.

g. Identify this needle holder…

h. Identify this instrument…

i. A circular deposit of pigment over anterior lens capsule resulted from blunt trauma.

j. A visual field analyser.

Quiz compiled by-
Dr. Jyoti Deswal, Dr. Manisha Nada

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Editor-in-Chief, HJO
Regional Institute of Ophthalmology
Pt. B.D. Sharma PGIMS,
Rohtak-124001 (Haryana)
Mobile : 9896007158
E-mail : manisha_nada@rediffmail.com

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celebrates
World Diabetes Day
Let's be aware of Diabetic Blindness
(14th November, 2020)
at
Pt. B.D. Sharma PGIMS, Rohtak

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Retina Symposium

On

Diabetic Retinopathy-Diagnostic and Treatment Novelties

29th November 2020 Sunday

7.00PM to 8.30PM

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Dr. S. V. Singh, HOD, RIO Rohtak

Organising Secretary

Dr. Manisha Nada

Welcome address

Speakers

7:10 – 7:15 PM
Diabetic Retinopathy and OCT - A Prologue
Dr. Manisha Nada

7:15- 7:25 PM
OCT as your guide in managing Diabetic Retinopathy
Dr. Chekitaan Singh

7:25 -7:40 PM
Predictive biomarkers for Anti VEGFs and Steroids in Diabetic Macular Edema
Dr. Rajeev Raman

7:40 PM– 7:50 PM
Target, aim and shoot the leaky vessels
Dr. Salil Gupta

7:50 – 8:00 PM
Individualising treatment of Diabetic Macular Edema
Dr. Sumeet Khanduja

Discussion: 8 pm onwards


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MONSOON WEBINAR
August 21, 2020
07:00 - 08:30 PM
PANELISTS
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Dr. Rajan Gupta
Dr. Kapil Vohra
Dr. Manisha Nada
Dr. Urmil Chawla
Dr. Parveen Arora
Dr. Chekitaan Singh
Family Benefit Scheme - Decoded
Dr. Inder Mohan Rustagi | 8 min
ICL mishits! - Tips to avoid and tackle
Dr. Ajay Sharma | 10 min
Anterior segment injuries - 10 Surgical Pearls
Dr. Rajender Singh Chauhan | 10 min
Patient as Partners - New norms
Dr. Neeraj Sandhuja | 15 min
Question Answer
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THURSDAY
10TH September
2020
TIME:
7:00 pm to 8:00 pm
AGENDA
Time
Topic
Speaker
7:00 PM—7:15 PM
Convenience in Post Cataract Inflammation Management
Dr Rajan Gupta
7:15 PM—7:30 PM.
Biometry in Post Lasik Cataract.
Dr Gaurav Goyal
7:30 PM—7:45 PM.
Management of Chemical Injuries.
Dr Vikas Mittal
7:45 PM—8:00 PM.
Q&A
All Delegates


LIVE
WEBINAR
Under the aegis of
Haryana Ophthalmological Society,
Sirsa Fatehabad Ophthalmological Society
enovation technology
Dr. Praveen Arora
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Dr. Mahip Bansal
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Topic
• 'ENOVA' an Innovation In Itself : Dr. Nikhil Gandhi
• Diabetic Retinopathy – An Overview : Dr. Nikita Arora
Speakers
Moderator
Dr. Nikhil Gandhi
Anupam Eye Hospital & Laser Centre, Akluj
Dr. Nikita Arora
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DATE : 1st October 2020 | 7:00 – 7:45 PM IST
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Posterior Segment Complications of Phacoemulsification
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Dr. Ashish Lal
Dr. Vaibhav Sethi
Dr. Deependra V Singh
PCR Management : Tips and Tricks
Dr. Harbans Lal
10 min
PCR Management using Fluid Dynamics
Dr. Ashish lal
8 min
Tackling lens Fragments & IOL in Vitreous
Dr Vaibhav Sethi
8 min
Suprachoroidal H'ge: Managing the Extreme
Dr. Deependra V Singh
8 min
BEYOND ACADEMICS
Medicolegal Aspects in Ophthalmology
Dr. A. A. Deshpande
15 min
Disclaimer: The order of talks can change on the day of the program.

Optic Disc Edema -Features of its common manifestations

Sakshi Lochab MBBS
Regional Institute of Ophthalmology, PGIMS, Rohtak

Disc edema is an ophthalmoscopic finding defined by unilateral or bilateral swelling of the optic disc. Axonal distension and elevation of the optic disc leads to a swollen disc. Disc edema indicates swelling of axons present at the disc along with increased fluid around the axons. This axonal swelling can occur because of obstruction in axonal transport at lamina cribrosa. There are several synonyms used to describe this finding including papillitis, papilledema, swollen or choked discs, and the most commonly used term – optic disc edema (ODE). Careful history along with thorough fundus examination helps to identify the cause of disc edema.

Presentation: Patients with ODE are often asymptomatic but can present with a range of symptoms based on the underlying cause. Symptoms vary from transient obscuration of vision, visual loss, headache, diplopia, vertigo, giddiness, vomiting, etc. For example, a patient who has disc edema due to increased intracranial pressure (ICP) may present with positional headache, visual obscurations, or pulsatile tinnitus. If the cause in infectious, systemic symptoms may be present. An ipsilateral relative afferent pupillary defect may be present.

Differential Diagnosis: It is useful to separate differential diagnoses associated with ODE into unilateral disc edema or bilateral disc edema.

Table 1: Causes of unilateral optic disc edema

Cause	Symptoms	Fundus findings
Anterior Ischemic optic neuropathy (AION)	Arteritic AION: scalp tenderness, jaw claudication, fever, joint pains – associated with Giant cell arteritis.	Arteritis AION: Unilateral optic disc edema with a white, chalky appearance.
	Non-arteritic AION: history of vascular risk factors (Diabetes, Hypertension, Hyperlipidemia)	Non-arteritic AION: Unilateral optic disc edema, cup-less "disc at risk" in the fellow eye
Neuroretinitis	Viral URI symptoms, fever, lymphadenopathy, rash	Optic disc edema with a macular star
Papillitis/Optic Neuritis	Orbital pain with visual changes (blurring, field defect, or decreased color vision) and an afferent pupillary defect	Usually only optic disc edema
Papillophlebitis	Young, healthy patient with blurred vision, with or without a field defect	Optic disc edema, venous dilation, and disc hemorrhages
Retinal Vein Occlusion	History of vascular risk factors (Diabetes, Hypertension, Hyperlipidemia), sudden painless vision loss, afferent pupillary defect	Optic disc edema with dilated and tortuous vessels, extensive retinal hemorrhages, cotton wool spots

Table 2: Causes of bilateral optic disc edema.

Cause	Symptoms	Fundus Findings
Increased intracranial pressure (Papilledema)	Headache, pulsatile tinnitus, visual obscurations, nausea and vomiting	Bilateral optic disc edema
Diabetic Papillopathy	History of diabetes, visual changes (blurring or field defects)	Bilateral optic disc edema, microaneurysms, cotton wool spots, hard exudates, retinal hemorrhages, neovascularization
Hypertensive Papillopathy	History of hypertension, visual changes (blurring or field defects), headache	Bilateral optic disc edema, cotton wool spots, hard exudates, retinal hemorrhages, arteriosclerosis
Pseudopapilledema (Optic Disc Drusen)	Generally asymptomatic	Round, white/yellow bodies on and buried in the optic nerve head

Diagnosis: For diagnosing disc edema fundus examination plays a key roel. In general, on fundoscopic or slit lamp exam one can find enlarged retinal venules, blurred disc margins, elevation of the optic disc, obscuration of blood vessels traveling through the optic disc, and in severe cases, hemorrhage overlying the optic disc. Blurred disc margins is earliest sign of disc edema. Dilatation of venules leads to hyperemic appearance of the disc. In advanced cases disc elevation is seen which begins in periphery and progresses towards the centre. Retinal or choroidal folds also known as Paton lines are also found in these cases.These findings together with patient symptoms and history are key to arriving at the proper diagnosis.

Unexplained optic disc edema necessitates further imaging studies. An MRI and MRV of the brain will evaluate for the presence of increased intracranial pressure including structural lesions and venous outflow obstruction, which can both lead to papilledema. A lumbar puncture may also be needed to evaluate for infection and elevated ICP. The presence of elevated ICP in the absence of a cause found on imaging may lead to the diagnosis of idiopathic intracranial hypertension, also known as pseudotumor cerebri.

Address for Correspondence:
Dr. Sakshi Lochab, Junior Resident, RIO, PGIMS, Rohtak-124001 (Haryana)
E-mail ; sakshi94lochab@gmail.com Mobile ; 8295444178

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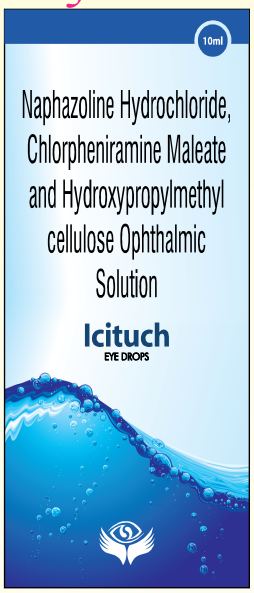

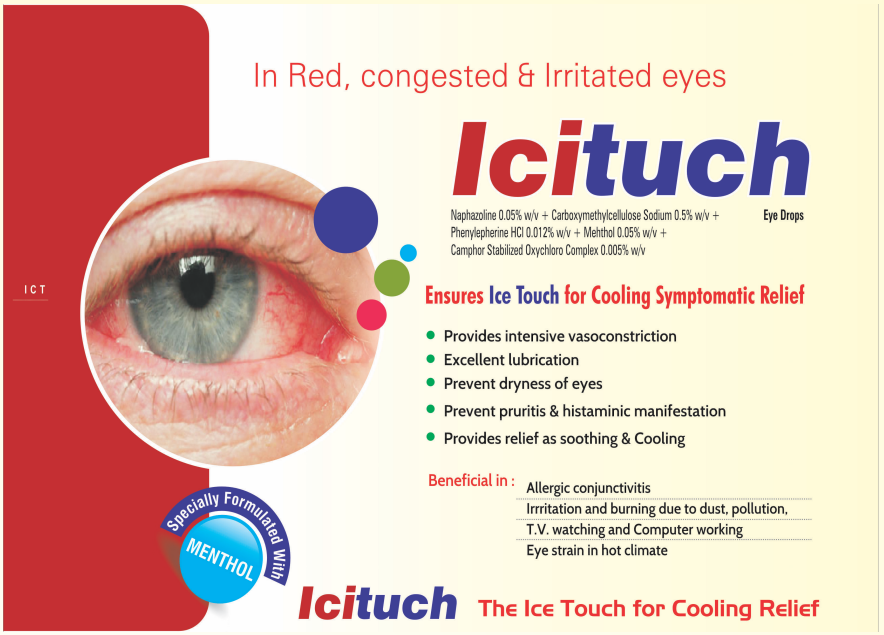

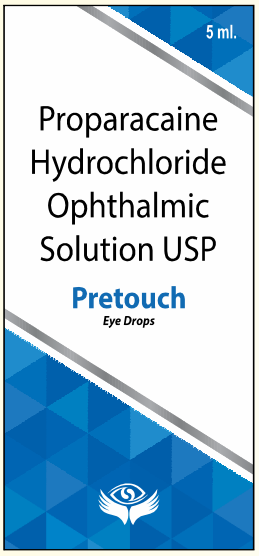

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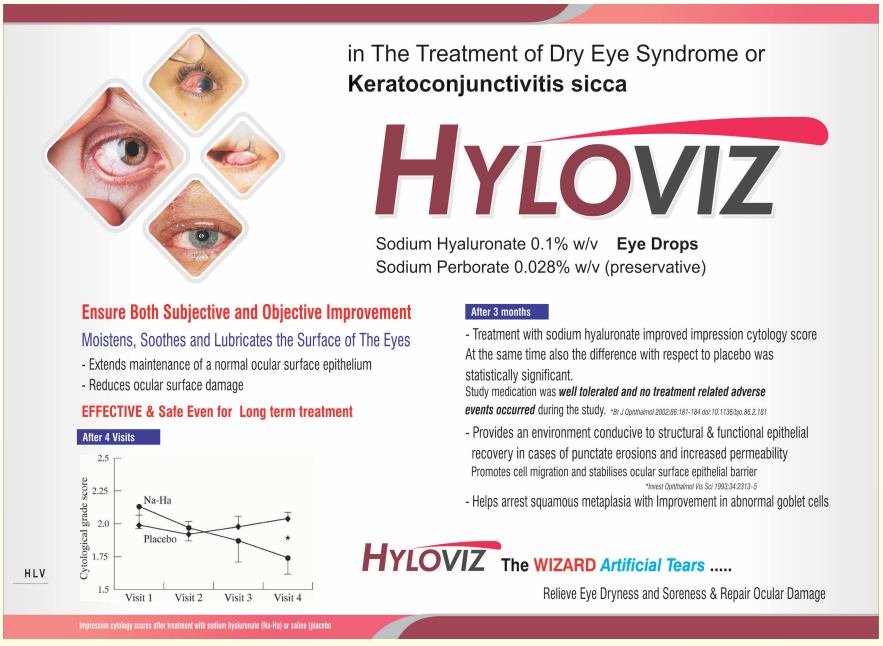

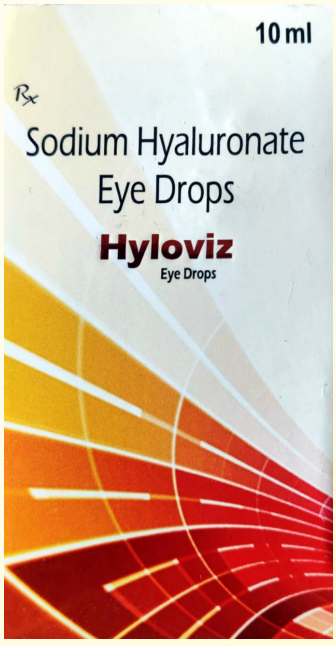

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