TOS TODAY

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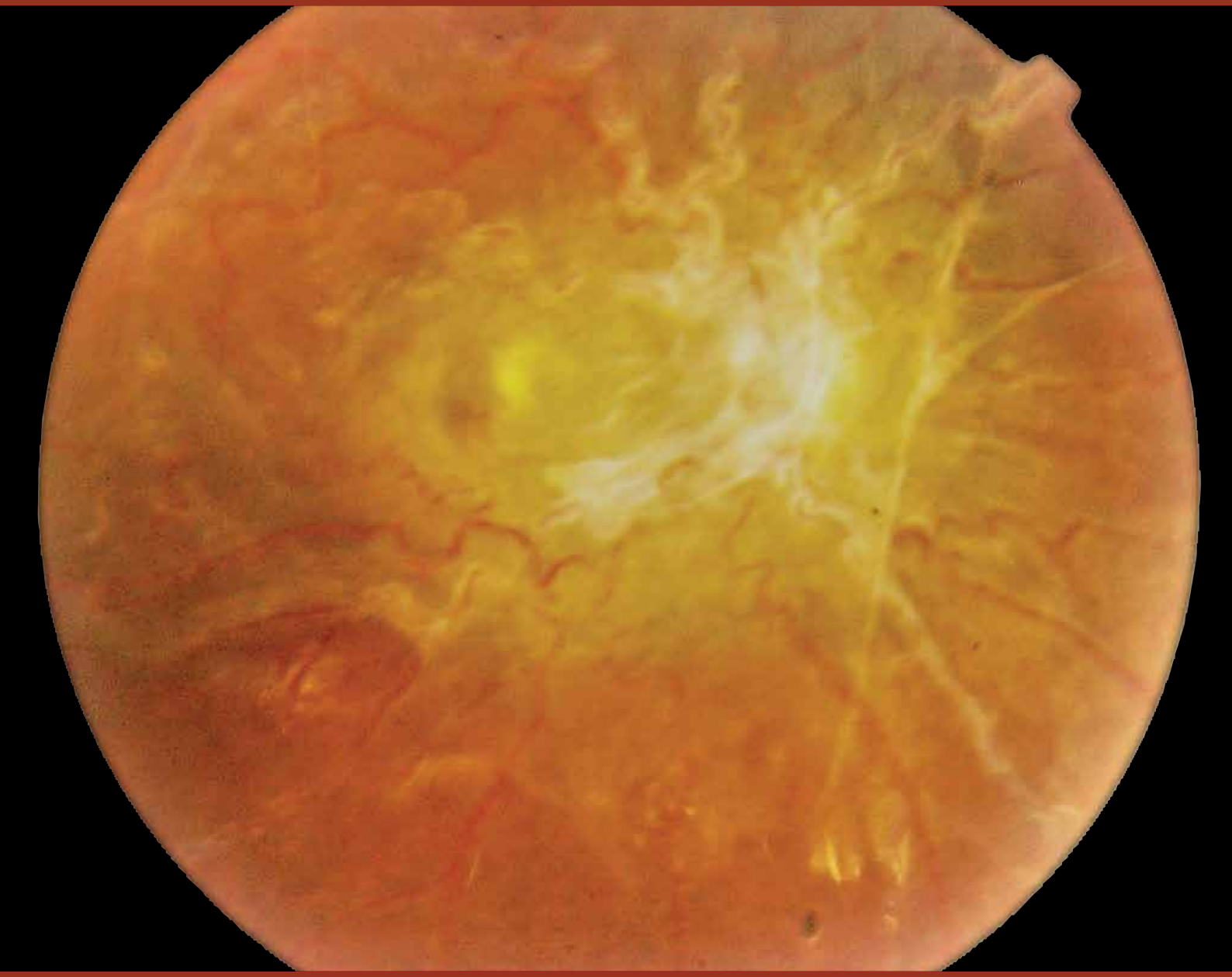

Submacular deposit with epimacular membrane and vascular tortuosity in a 9 year old girl with biclonal gammopathy: Mallika Goyal

www.tsos.co.in

Second Annual TOS Conference

@ HICC, Hyderabad, August 5-7, 2016

From The Editor's Desk

Dear Friends,

Best wishes for a wonderful New Year from the Editorial Board & Executive Committee of Telangana Ophthalmological Society.

We hope you were able to attend and enjoy the 2nd annual conference of the Telangana Ophthalmological Society in August 2016 at HICC organised by Hyderabad Ophthalmologists Association under the stewardship of the organizing secretary Dr KB Nandkishore. That was a mega event with several International and National Guest Faculty. Scientific Program was astounding, thanks to the efforts of the dynamic Scientific Committee headed by Dr Pravin Krishna.

An important issue concerning ophthalmic practice this year has been the off label use of drugs in ophthalmology. Off label use is not just a catchy term.... it carries with it greater responsibility on part of the service providers for ensuring quality and sterility of the product as what is good for systemic use is not necessarily safe for intraocular use.

Drugs manufactured forintravenous use may not be subjected to the same stringent standards of sterilisation as those made specifically for intraocular use. Unlike blood, vitreous is an excellent culture medium and lacks any antibodies or immunity to challenge introduced microorganisms. The small volume of aqueous and vitreous are also inadequate to dilute the injected medication (containing any microorganisms) unlike the 5-6 litres of blood available for dilution, circulation and inactivation of microorganisms following intravenous injection.

Presence of preservatives and other adjuvants that maybe safe for systemic use but not for cornea, retina, optic nerve and other ocular tissues further complicates the issue.

A case in point is the devastating haemorrhagic occlusive retinal vasculitis (HORV) observed in 22 patients following the off label use of intraocular vancomycin(including intravitreal, intracameral bolus, and irrigating solution containing vancomycin) leading to joint alert from ASCRS- ASRS regarding its intraocular use. According to the alert, 22 cases of HORV have been identified, 14 of which were bilateral. 12 of the cases occurred in 2015-2016, five in 2013-2014 and five before 2013. These were not related to any one manufacturer. Onset of problem could be from first post op day to upto 4 weeks after the injection, hence bilaterality was common as other eye had also been treated before the first eye showed the complication.

Even syringes and needles manufactured for systemic use may not be entirely ocular safe as silicone oil droplets have been seen in the vitreous following intravitreal injections of anti-VEGF agents and steroids[1,2].

In our own country, recent widespread problems from use of avastin and ringer lactate have made it imperative that patients be informed of the off label use of the most important drugs and the alternatives available; and physicians avoid choice of drug based solely on cost saving for their patients.

With best wishes for a glorious 2017!

Mallika Goyal, MD
Editor Publications
Telangana Ophthalmological Society

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From The Desk of the President, TOS

TOS Presidents' Vision 2016-2019 - A 3 year Mission

Dear Colleagues,

The Telangana Ophthalmological Society came into existence officially last year. The founder President Dr Harikishan and other members of the Executive committee gave suggestions for the future of our society. These would be amalgamated with my / our own Vision and we shall aim for some concrete actions during the three year tenure this year onwards. We shall achieve this through a few Advisory Adhoc sub committees with a time bound action plan. We would like to solicit cooperation from all members in this endeavor. This mission shall begin now and each year a revision made by the respective president for that year.

1. **Membership**: With all formalities of Registration of the Society being completed, we have now distributed the Life member and Non Resident Life member certificates and ID Cards to all members. The Nonresident LM certificates were well appreciated by most members and this resulted in developing bonhomie with all. The strength of any society is related to the number of members. In order to expand the membership of our society, we propose to appoint a Membership sub -committee to enroll members from the remote areas of our state. We propose the name of Dr C Sharat Babu, Past President to take-up this responsibility and also nominate a few members.
2. **Headquarters**' for TOS: In order to have a permanent address for our society I submitted a representation to the Superintendent SDEH to explore the possibility of getting a suitable premise within Sarojini Devi Eye Hospital on Long-term Lease from the Government to set up our office and a meeting room for our Executive Committee. Dr V Rajalingam is assisting me in following up with this issue and we have received a favorable response from the authorities.
3. **TOS Website**: Efforts shall be made to upgrade the website of the society to make it more interactive and act as a one site resource centre for
   A. Online membership and payment.
   B. Abstract Submissions on a continual basis.
   C. Future conference registrations, including payment.
   D. A SBI Collect payment portal to be incorporated for the aforesaid purpose.
   E. A page can be provided to each member to upload their profile.

   We request the secretary Dr Ravindra to coordinate with the website vendor to update accordingly. We can even consider opting for a bigger vendor if so required.
4. **District Chapters**: One of the important aims and objects of the society is to develop camaraderie among its members. Apart from the daily professional routine, our members need to develop social interactions along with families. They should be regularly meeting atleast once a quarter at District level. Of all the newly reconstituted districts, only about 4 have a functioning District Chapter. It shall be our endeavor to personally visit the underserved districts and encourage them to constitute district Chapters under TOS. In order to achieve this objective, the first step is to arrange a list of members' district wise. Request the Secretary to get this done ASAP.
5. **OFWS**: The Family Benefit Scheme of the erstwhile APOS is rechristened as Ophthalmologist's Family Welfare Society. TOS shall extend all support for this Society which aims to promote its social Aims and Objects. The new secretariat has started functioning at Warangal and we wish its EC all the very Best.
6. **Career Guidance for YOs'**: Every year our state produces around one hundred new Ophthalmologists. While it is outside the purview of our Society to interfere with the academic curriculum, it is certainly our responsibility to give proper guidance to these Young Ophthalmologists' about their future career. They should be aware about the pros and cons of practices in government and private institutions and personal clinic setups. For this purpose, I would like to constitute a Career Guidance Sub-committee. This committee shall organise career guidance sessions as part of the CME activities of the ARC. Additionally this Committee shall also be guiding youngsters who approach them personally. We propose the name of Dr GVBK Gangadhar Reddy to Chair this Committee and Chairman ARC to assist.
7. **Hands on Training for YOs'**: Ophthalmology has made rapid advances over the last two decades in Diagnostics. All medical

colleges may not be having this advanced equipment/s. Apart from a few premier institutes in our state who cater to this need at a national level; the majority is deprived of this exposure. We would like to coordinate and request Eye centre's in the private sector in our state to permit these young professionals to get a hands on training for 2 weeks. The list of centre's offering this exposure shall be listed on the website and interested youngsters can apply through TOS for these training slots. For this purpose a Hands on Training Committee shall be constituted. We propose to request Dr Vidyavati M to Chair this Committee with the coordination helps from the Chairman ARC.

8. **Ophthalmic Diagnostic Centres**: We would like to encourage formation of Ophthalmic Diagnostic and Laser Centre's on a Cooperative basis at each District headquarter for the benefit of practicing doctors, who are also stake holders as also the community at large. These centre's can be managed by trained YO's.
9. **Scientific Temper**: The relevance of any scientific body depends upon the quality of scientific deliberations among its members, as also its activities on the administrative front. To encourage the members to continuously strive for academic and administrative Excellence, I would like to propose constitution of "TOS Achievement Awards". Members who attain adequate cumulative credits for their contribution for a minimum of three years shall be awarded with a Medal and a certificate. The credits shall be counted from our First TOSC and the first awards can roll out from the Fourth TOSC, subject to the terms of these awards. The details shall be presented in the next EC
10. **TOS Today**: The official publication of our society, TOS Today needs to be indexed for the benefit of our members. All efforts shall be made to get it indexed during this year. Request the editor Dr Mallika to look into this.
11. **Public Relations**: Our medical Profession is considered worldwide as the toughest profession. Like in mathematics, in medicine "2+2 need not be always 4". Despite having taken all precautions and doing the best for the patient, the outcome may be abnormal due to various factors. This is picked up by the media and instantly projected as a botched up surgery or even gets extrapolated as the so called Eye Bazar or Eye Business. This causes considerable strain on the psyche of the surgeon. In any such situation while the Surgeon does take collective responsibility, the media should equally exercise restraint and ascertain facts from other professionals before reporting. To fill up this need I would like to form a PR Committee to whom the media and the public can approach to get technical details before reporting. An additional role that is proposed for this subcommittee shall be to represent and coordinate with the diverse State Government agencies in framing suitable policies for the benefit of the majority of the members including those working in remote areas.
    We propose the names of
    Superintendent SDEH as Convener &
    Dr Pradeep Swarup
    Dr Vishwanath and
    Dr RaviKumar Reddy
    As members of the PR Committee of TOS.
12. **Elimination of Quackery**: We shall form a Vigilance Committee to liaise with the official agencies to eliminate the menace of Quackery. I would like to request all our members to bring relevant info to the notice of this committee who shall forward it to the concerned agencies for further action. We propose to request Dr G Harikishan to chair this committee with assistance of our Joint secretary Dr Sree Kumar as Coordinator.
    We would like to solicit blessings from all seniors, Constructive cooperation from our colleagues and best wishes from all friends in discharging our duties as the President/s for the year 2016-19. Our endeavor shall be to be in Real time touch with all TOS members through various social media options.

    Together we shall make it happen.
    Jai TOS, Jai Hind

    Manoj Chandra Mathur / KB Nandkishore / Madhukar K Reddy

From The Desk of the Secretary, TOS

Dear Colleagues,

Telangana Ophthalmological Society wishes all its members a very Happy and Prosperous 2017.

The Second Telangana State Ophthalmological Conference held at HICC Hyderabad hosted by Hyderabad Ophthalmologists Association was a stupendous success. Overcoming all initial apprehensions to conduct a state conference at such a prestigious venue, the Local Organising Committee did very well surpassing all expectations. Kudos to all members of LOC and in particular Dr. KBNand Kishore, Dr.MallikaGoyal , Dr. Manoj Mathur and Dr Praveen Krishna.

Our membership now stands at around 865. Life Membership certificates along with ID cards were issued at Hyderabad Conference to both Resident & Non-resident members. Those who have not received can collect them from my office.

Dr. Rajalingam, Chairman ARC, organised a weeklong CME Workshop at Sarojini Devi Eye Hospital in October 2016, for post graduates of Telangana and neighbouring states was well attended by more than 150 delegates.

Very soon our Website will be made more interactive, informative and one site resource center for all our activities.

We will shortly be allotted a room in Sarojini Devi Eye Hospital to set up our permanent office . Thanks to our President and Superintendent SD Eye Hospital for their efforts.

Now eye hospitals need only 20 beds alongwith other requirements to get empaneled under Aarogyasri- EHS scheme. Many are getting benefitted under the new scheme.

The Third Telangana State Ophthalmological Conference will be held at Warangal in July 2017 under the aegis of Warangal Ophthalmological Association. All members are requested to extend their cooperation in making it a big success.

With regards,

Dr. AitharajuRavindra, MS, DO
Hony. General Secretary
Telangana Ophthalmological Society

From The Desk of Organizing Secretary TOC 2016

Dear Colleagues,

From when the 2nd TOSC was allotted to HOA and an LOC was constituted it was our honest endeavour that we should make this as a model event and took the bold and courageous step to host it in the prestigious venue, HICC. Right from the dates clashing with TNOC, the huge funds that were required, the doubts about attendance,the reluctance of trade to support and the shortage of working hands we saw and went through all!

From a position of possible deficit in all areas we could navigate to a total turnaround. Apart from boasting of one of the best scientific programmes to date, thanks to our dynamic SC. Committee Chair Dr Pravin Krishna, we had a phenomenal 650 registrations apart from trade.

The conference garnered praise from all corners of our country. It is to our credit that the President of AIOS spent one day at TNOC even though he belongs to that state and two days at our conference.

To top it all we managed to contribute a record amount of over ten lakhs to TOS which even many state societies could not contribute to AIOS while conducting a national conference.

I am deeply indebted Drs NS Reddy, KP Reddy, Pradeep Swarup and Viswanath for their guidance and for my team of LOC Drs Milind Naik, Rishi Swarup, Sai Kiranmayi, Alpa Poorabia, Shiv Kumar, Rajesh Agarwal, Ravikumar, Umashankr, Venugopal, and Sachindra.

No words can express my gratitude for the amount of responsibility shouldered by Dr Mallika Goyal and Dr Manoj Mathureven though they did not carry any official position. It is their support which realised our goal of making this a Model Conference.

Wishing the 3rd TOSC at Warangal a grand success

With warm regards

Dr KB Nandkishore
Organising Secretary 2nd TOSC
Hyderabad

TOS TODAY

Wishes

you and your Family

Bright & Prosperous

New year - 2017

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Guest Article

Advances in gene therapy for retinal diseases

Authors: **Padmashree Dr. S. Natarajan** *and* **Dr. Vishwas B. Chavan,** *Aditya Jyot Eye Hospital, Mumbai*

It has been more 50 years since the fundamental principles behind gene therapy were established. In the 1940s, Nucleic acids were identified as the carriers of genetic information. In the 1960s, virus-based methods for delivering therapeutic genes to patients were developed. In the 1970s, came recombinant DNA technology. Many years of scientific efforts resulted in the first approved gene therapy clinical trial in 1990 for children with severe combined immunodeficiency.[1]

Gene therapy has several ups and downs in last few years. After years of promising advances, the research in gene therapy almost came to a halt in 1999, when a death in a gene therapy trial to treat an inherited metabolic disorder caused fears about the technology's safety. It was during this time that some gene-therapy researchers began to see a glimmer of promise in treating eye disorders. The eye is protected from immune system to some extent, reducing the possibility of an immune attack on the virus used to introduce the genes. The eye is also relatively easy to access, allowing surgeons to inject the virus near to the cells where the gene is required. In retinal diseases, more than 200 genes are involved, the opportunities for genetic correction are enormous.[2]

How retinal gene therapy works?

The vertebrate neural retina contains many different types of cells, like retinal ganglion cells, rod and cone photoreceptors etc. In retinal gene therapy, AAV enters these various types of cells and expresses the DNA sequence of intended therapeutic value. Retina cells are non-dividing, therefore AAV persist and provide therapeutic expression of DNA sequence over a long time period.

In 2008, three independent research teams demonstrated that patients of Leber's Congenital Amaurosis (a rare genetic retinal disease) had been successfully treated by gene therapy which used a virus called adeno-associated virus (AAV). In all three studies, an AAV virus delivered a functioning copy of the RPE65 gene. RPE65 is short form for retinal pigment epithelium-specific protein. Replacement of faulty gene with a functioning gene restored vision in LCA-affected children.

AVV as a vector for gene therapy

In gene therapy of retina, commonly used vectors are AVV. One benefit of the using AVV for gene therapy is that it creates very small immune response and can infect many types of retinal cells leading to long term transgene expression, such as cells in blood-retina barrier which protect the retina from micro-organisms and immune-mediated injuries.

Initial studies with AAV in the retina have used AAV serotype 2. Nowadays new variants of AVV are being developed to increase the efficiency of gene transfer process. Many naturally-occurring serotypes of AAV have been isolated that can infect retinal cells. After intravitreal injection, AAV-2 and AVV-8 can transfer genes to retinal ganglion cells. AAV-2, AVV-8 and AVV-9 can transduce occasional Muller cells. After subretinal injection, AAV-2, AAV-5, AAV-7 and AAV-8 serotypes efficiently transduced photoreceptors, and AVV-1, AVV-2, AVV-5, AVV-7, AVV-8, and AVV-9 serotypes can efficiently transduce cell of RPE. Modification of AVV by chemical, immunological or genetic changes enables the AAV with cell specificity and increased delivery speed. Specific promoter sequences can determine that which gene is expressed in which type of retinal cell types. To control the expression to a specific type of cell, a promoter sequence which is either tissue-specific or cell-type specific can be used.

How gene therapy is administered to retina

Generally AAV is administered by two routes: intravitreal or subretinal. In the intravitreal route (targeting retinal ganglion cells and a few Muller glial cells), AAV is injected in the vitreous humor of the eye. In the subretinal route (targeting photoreceptors and RPE cells), AAV is injected underneath the retina. These different routes are necessary for different cells, because the inner limiting membrane (ILM) and the various retinal layers are physically obstructed delivery of drugs and vectors to the deeper layers of retina. Generally, subretinal AAV is more efficient than the intravitreal route.

Most importantly, the retina is partly protected from immune system, and thus does not cause a major immune-response when AAV is injected. Recent results indicate that the subretinal route offers higher amount protection than the intravitreal route.

Regulation/Control of transferred gene: Since continuous/strong expression of newly transferred gene is sometimes harmful for long-term retinal health, it is necessary that such gene expression should be regulated by some means. One way is using a promoter system in AVV vectors, which can be regulated externally. One example is tetracycline-inducible expression system, which uses a silencer/transactivator AAV2 vector and a different inducible doxycline-responsive co-injection.[3]

Retinal gene therapy holds great promise in the treatment of different types of inherited and non-inherited blindness. Let's have a look at eye diseases for which retinal gene therapy hold promise:

Mitochondrial Leber hereditary optic neuropathy (LHON): It is a retinal disease of ganglion cells that leads to blindness in young adults. In this disorder, a mutation occurred in mitochondrial DNA at location 11778. At this position, a single G-to-A change in the gene encoding subunit 4 of NADH dehydrogenase (ND4) causes a arginine-to-histidine substitution at position 340. This mutation caused around fifty percent of LHON cases. Studies showed an effective therapy for LHON can be allotopic mitochondrial gene delivery.[4]

Leber's congenital amaurosis (LCA): In 2008, results of clinical trials of using AVV for Leber's Congenital Amaurosis was reported. The researchers delivered the RPE65 gene by using an AAV vector through a subretinal injection, where therapeutic fluid is injected underneath the retina. These studies indicate that AAV is safe with no dose-limiting toxicities, because in all the three studies, no major adverse events were observed. Furthermore, patients in all three studies showed improvement in their visual function and continuing to do well after more than 1.5 years.

Age-related macular degeneration: After success of gene therapy in treating LCA, similar treatments for treating age-related macular degeneration (AMD) are being developed. Currently, wet AMD is treated with frequent injections of recombinant protein into the eyeball. Therefore scientists are working on long-term disease management by a single dose of drug.

Choroideremia: One clinical trial, reported in January 2014, used subretinal AAV to restore the REP gene in affected patients. Initial results stated that all patients had better vision.

Color Blindness: Recent research has found out that AAV can successfully restore color vision to treat color blindness in adult monkeys.

Juvenile retinoschisis: It is an X-linked recessive degenerative disease of the central macula and affects the nerve tissue in the eye. The disease is caused by mutation in the RSI gene encoding the protein retinoschisin (important for maintaining the synaptic integrity of the retina). Therapy using AAV-5 vector containing the wild-type human RSI cDNA driven by a mouse opsin promoter showed recovery in functional and structural aspects of retina, which is long-standing.

Retinitis pigmentosa (RP): It is an inherited disease which leads to a gradual night blindness and loss of peripheral vision due to photoreceptor cell death. Most people who suffer from RP are born with dysfunctional rod cells. Rod cells help us to see in low light levels and hence these patients cannot see at night. After that cone cells death occurs which are responsible for color vision and acuity. Loss of cones leads to full blindness in children. The disease can be inherited as autosomal dominant type, autosomal recessive type or X-linked type. When mutations occur in the rhodopsin molecule, the protein movement is affected because the mutations can affect folding of protein molecule, it's stability, and intracellular trafficking. Gene therapy to this is introducing AAV-delivered ribozymes designed to target and destroy a mutant mRNA.

Another mutation in the structural proteins, especially in peripherin 2, can lead to recessive RP and macular degeneration in humans. In an animal study, subretinal injection was given to mice, which contain AAV-2 carrying a wild-type peripherin 2 gene directed by a rhodopsin promoter. Consequently, the function and structural ability of photoreceptor were improved.

If the disorder is caused by some unknown mutation, then gene replacement therapy cannot be administered accurately. Since apoptosis can cause the death of photoreceptors in most of the retinal dystrophies, survival factors and antiapoptoic reagents may be administered as an substitute therapy. When scientists injected the rod derived cone viability factor (RdCVF) protein into the eye of the animals (dominant RP mutation rat models), it promoted the survival of cone activity, and prevent disease progression by improving cone functions.

Ocular neovascularization (NV): It is the abnormal formation of new capillaries from blood vessels of the eye,which is a distinguishing feature of eye diseases like diabetic retinopathy, retinopathy of prematurity and wet age-related macular degeneration. The main culprit in these diseases is VEGF (Vascular endothelial growth factor) which induces vessel leakage and which is angiogenic. Many molecules can neutralize the effect of increasing local VEGF, such as soluble Flt-1, and pigment epithelium-derived factor (PEDF). It was speculated that PEDF loss plays a primary role in the ischemia-driven NV development. One study shows that the levels of PEDF in aqueous humor of human are reduced with advancing age, suggesting that the reduction may cause AMD development. These findings suggested that the expression of these protective factors by using AAV as vectors can be implemented to treat NV. This can be an alternative to multiple eye injections of recombinant protein.[3]

Retinal degeneration (RD): It is an inherited eye disease, in which there is permanent loss of photoreceptors, with activation of the resident microglia secondary to the disease. Treatments based on stem cells have recently made rapid improvement in treating RD. It has been demonstrated that when neural stem cells (NSCs) are transplanted in the retina, they exert immunomodulatory effects on microglia cells, thus delaying the degeneration of photoreceptors .[5]

In summary, vision loss from inherited retinal diseases is a debilitating condition. However, with many gene therapies available to ophthalmologists, there is hope for patients.

The principal aim for ocular gene therapy focuses on the retinal pigment epithelium (RPE) or photoreceptors cells. To deliver viral vectors to these cells many methods are used, such as subretinal injection, intravitreal injection, or suprachoroidal space access. Recombinant adeno-associated virus can be engineered to increase gene delivery to specific targets. At present, many clinical trials of gene therapy are in progress and their initial results are encouraging. Thus, recent advances in gene therapy have allowed for a better knowledge of many types of retinal diseases, including inherited and proliferative. These novel treatments can be a life-changing event for thousands of patients affected with these ailments.[6]

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AIOS Election News

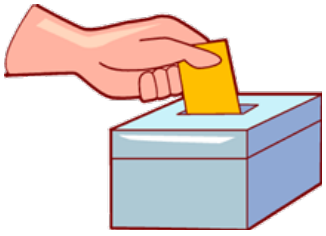
All the AIOS members from our society are requested to use their voting rights in the forthcoming AIOS elections.

Two candidates from our society are in the fray.

1. **Dr. Mallika Goyal** for Member - Scientific Committee
2. **Dr. Santosh G Honavar** for Editor, Indian Journal of Ophthalmology

TOS Executive & its members wish them success in the forthcoming elections and also in all their future endeavours.

Dr. AitharajuRavindra, MS, DO
Hony. General Secretary
Telangana Ophthalmological Society



Guest Article

Recent updates on Ocular Sarcoidosis

Dr. Kalpana Babu, *MD, MRCOphth (Lon)*
Vittala International Institute Of Ophthalmology & Prabha Eye Clinic and Research Center, Bangalore

Abstract:
Sarcoidosis is a chronic multisystemic granulomatous disorder caused by an exaggerated cellular immune response to a variety of self or non-self antigens in a genetically predisposed individual resulting in non caseating granulomas. Ocular involvement occurs in 25-60% of systemic sarcoidosis at some point of time. In this article, we look at the emerging literature on epidemiology, pathogenesis, clinical profile and management in ocular sarcoidosis.

Sarcoidosis is a chronic multisystemic granulomatous disorder caused by an exaggerated cellular immune response to a variety of self or non-self antigens in a genetically predisposed individual. It is characterized by noncaseating granulomas affecting many organs including the lung, lymph nodes, skin, heart, liver, muscles and the eye.[1,2] The diagnosis of sarcoidosis is usually made on the basis of a biopsy and a combination of typical clinical features and radiology findings in the absence of any condition that can cause similar clinicoradiological and pathological changes.

Ocular sarcoidosis is believed to occur in 25-60% of systemic sarcoidosis at some point of time. In 10-20% of patients, ocular involvement is the initial presentation before any other systemic manifestation.[3] Ophthalmological clues help to search for any occult systemic sarcoidosis. In this article, we look at the emerging literature on epidemiology, pathogenesis, clinical profile and management in ocular sarcoidosis.

Epidemiology:
Sarcoidosis has an overall incidence of 6-10 per 100,000. Although sarcoidosis occurs world wide, it is predominant in certain ethnic and racial groups like the Afro-caribbeans, Scandinavian and Irish populations.[4,5,6] Literature shows that the highest incidence of ocular sarcoidosis is in the 20-40 years age group. Some studies show two peaks of incidence for ocular sarcoidosis, the first at ages 20-30 years and the second at ages 50-60 years.[7] There is also a higher incidence of ocular involvement in women.[6] The reported incidence of ocular sarcoidosis varies according to the geography, patient population, diagnostic criteria employed and referral patterns of the reporting ophthalmologists. Earlier considered a disease of the developed world, ocular sarcoidosis is increasingly recognized from the developing world in recent years from countries like India,[8] Singapore, Thailand, Taiwan, Malaysia, Kuwait, Lebanon and Turkey.[9,10] This may be due to the increasing awareness of this disease and availability of improved diagnostic modalities like computed tomography and trans bronchial lymph node biopsies.

More than 25-60% of patients with sarcoidosis have ocular involvement with some reports being as high as 90%.[8] The actual incidence of ocular findings may be higher if all patients with sarcoidosis (asymptomatic and symptomatic) were examined on the slit lamp. Ophthalmic presentation may be an initial manifestation of a systemic disease. Most patients with ophthalmic findings showed evidence of systemic involvement. Clustering of sarcoidosis in families have been reported in literature.[9]

Pathogenesis: Sarcoidosis is caused by antigenic stimulation. The antigen activates a cascade of immune responses in genetically susceptible individuals. The severity of the disease depends on the disease modifying genes and the interplay of regulatory immune responses mediated through regulatory cells and cytokines. The process involves presentation of the antigen by antigen presenting cells, activation of CD4 + T cells, production of cytokines, recruitment of immunocompetent cells, compartmentalization at the site of inflammation sealing off the antigen and production of a granuloma.[11]

Etiology of Ocular Sarcoid: Although bacterial, viral and environmental antigens have been studied, none have been proven to be the cause for sarcoidosis. Current literature evidence states that sarcoidosis may result from not a single agent, but multiple agents capable of generating a Th1 mediated response in a genetically susceptible individual[11]

Mycobacteria: Clinical, radiological and histopathological similarities of tuberculosis and sarcoidosis and the coexistence of both diseases have implicated mycobacteria as a cause of sarcoidosis. Although studies have demonstrated mycobacterial DNA and RNA being detected in sarcoid tissue, it has not been isolated in culture from the sarcoid tissue. Recent detection of high frequency of mycobacterium tuberculosis catalase peroxidase (mKatG) reactive, IFN-g expressing T cells in patients with active sarcoidosis has renewed interest in mycobacteria as a causative agent.[11,12,13]

Propionibacteria: Recent studies reporting the occurrence of rRNA and DNA of propionibacteria in sarcoid tissue have supported the theory of propionibacteria as a causative agent.[14]

Other Infectious agents: There are reports of the association of hepatitis C with sarcoidosis. Reports of sarcoidosis following the use of IFN-g and antiviral therapies for hepatitis C are known. At the same time, reactivation of sarcoidosis in patients with preexisting disease have been reported during treatment of hepatitis C with IFN-g suggesting that the interferon is important in the pathogenesis of sarcoidosis. Antibodies against Epstein Barr virus, herpes virus and helicobacter pylori have also been found higher in sarcoid patients.[11,15]

Immunogenetics: Although no gene has been demonstrated to be responsible for sarcoid, HLADR17 and TNF polymorphisms play a role in the disease severity and prognosis. High levels of TNF-α, associated with TNFA2 allele are found in Lofgren's syndrome, which is associated with good prognosis. Another gene implicated as a susceptibility gene is the butyrophilin-like 2 gene, which is located near the HLA-DRB1 region.[15]

Histopathology: The classic sarcoid granuloma is made up of modified macrophages or epithelioid cells surrounded by a rim of lymphocytes and fibroblasts. This is similar to that of other granulomatous inflammations like tuberculosis, fungal infections like histoplasmosis and talc or beryllium granulomatosis. Necrosis or caseation, which is usually seen with tuberculosis and fungal infections, is absent in sarcoid granulomas. Occasionally, small foci of fibrinoid necrosis may be present in sarcoidosis. Immunohistochemical studies demonstrate the presence of CD4+ Tcells admixed with the epitheloid cells in the center of the cellular infiltrate. This localized accumulation of CD4+ Tcells in seen in the bronchoalveolar fluid and also in the eye. The compartmentalization of CD4+ Tcells at sites of the disease leads to markedly high CD4/CD8 ratio of more than 10. An elevated BAL CD4/CD8 T-cell ratio of greater than 3.5 predicts the diagnosis of sarcoidosis with 94% specificity and 53% sensitivity. This is also associated with a favorable prognosis. Recent reports[16] of increased CD4/CD8 ratio of lymphocytes in the vitreous greater than 3.5 provided a diagnosis of ocular sarcoidosis with a sensitivity of 100% and a specificity of 96.3%.[16] Thus sarcoidosis is mediated by an exuberant T cell immune response and not an immunodeficiency as was thought earlier.

Clinical manifestations:[15-21]

The course of sarcoidosis ranges from asymptomatic to severe disease. Clinical manifestations and the severity of the disease are strongly associated with racial and ethnic factors. Acute and more severe disease is seen in black patients. The relative prognosis of the disease is relatively benign. However factors associated with poor outcome include black race, late onset, disease persisting longer than 6months, involvement of more than 3 organs and stage 3 pulmonary disease.[5]

Patients may be asymptomatic or present with blurred vision, floaters, redness or discomfort. Ocular involvement is usually a chronic disease and has an insidious onset. It can involve the lacrimal glands, orbit, eyelids, conjunctiva, uveal tract and the optic nerve. (Figures 1-9)The ocular findings in sarcoidosis are summarized in table 1.[4-10, 15-21]

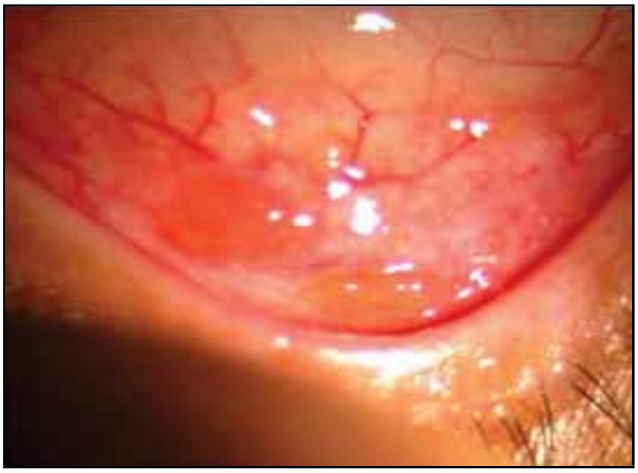


Figure 1: Slitlamp photograph showing the conjunctival granulomas in a patient with thoracic sarcoid.

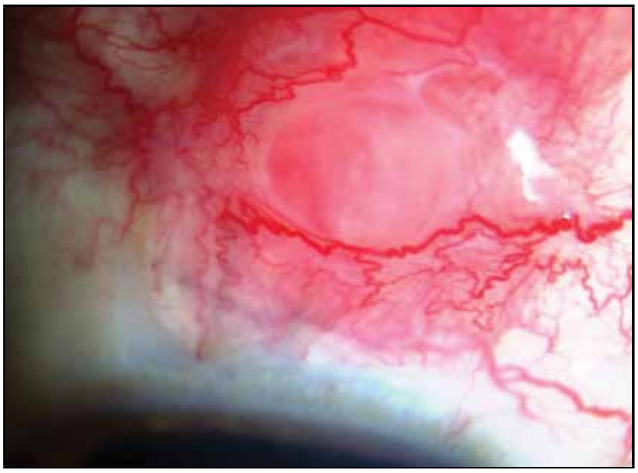


Figure 2: Slitlamp photograph showing the scleral nodule in a patient with sarcoidosis.

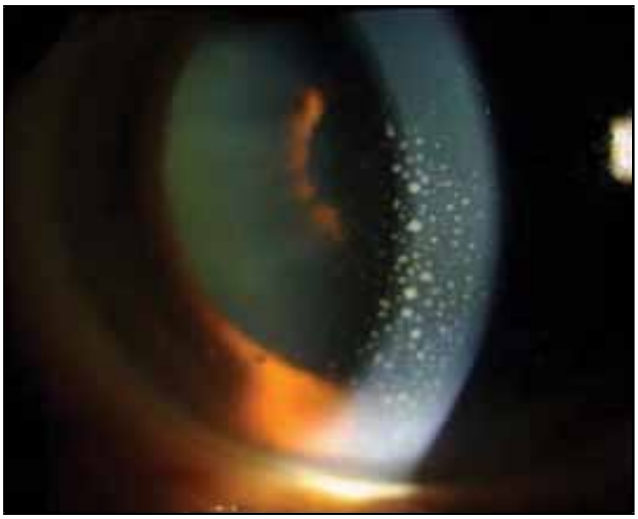


Figure 3: Slitlamp photograph showing the granulomatous anterior uveitis

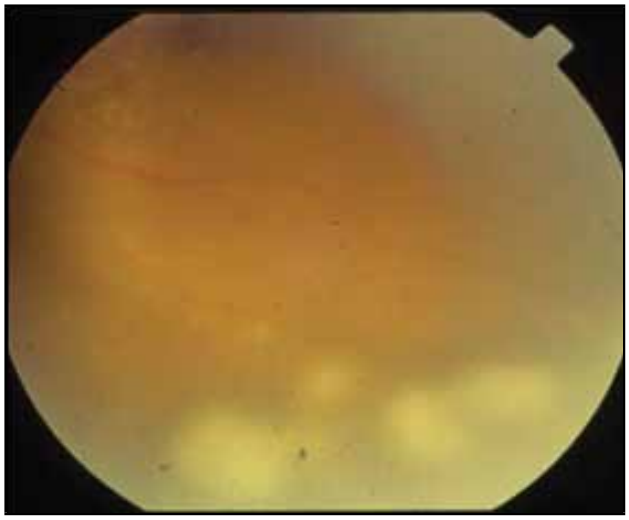


Figure 4: Fundus photograph showing vitritis with snow ball opacities in the peripheral retina

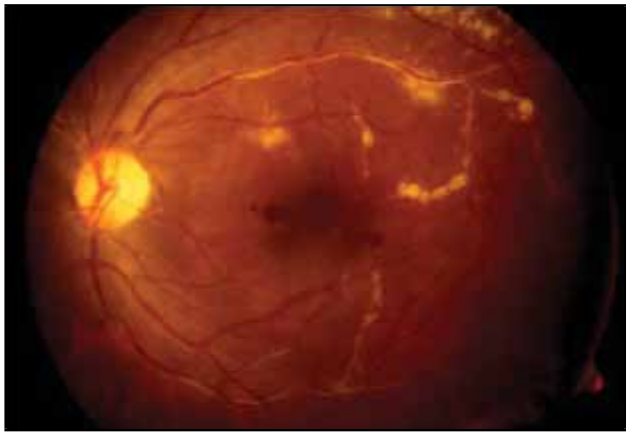


Figure 5: Fundus photograph showing the perivasculitis

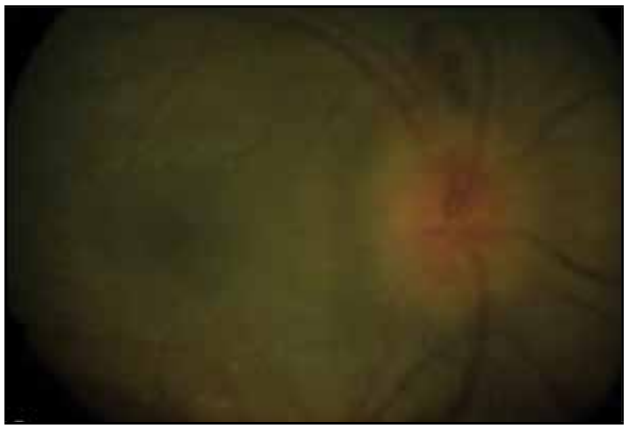


Figure 6: Fundus photograph showing the disc edema

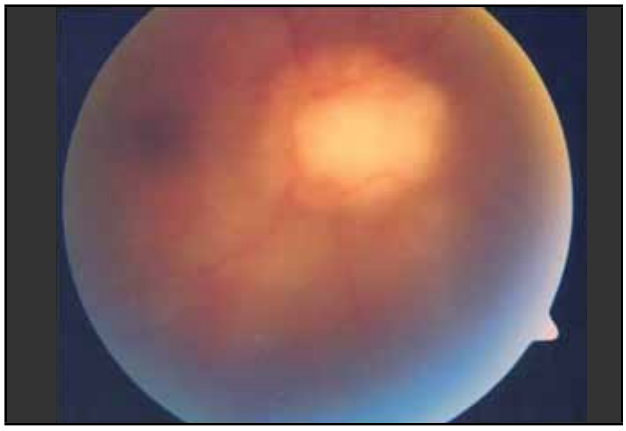


Figure 7: Fundus photograph showing the optic nerve granuloma

Figure 8: Fundus photograph showing the choroidal granuloma

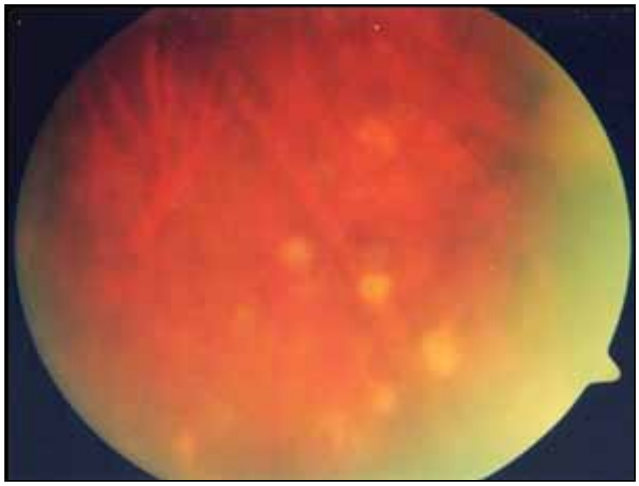


Figure 9: Fundus photograph showing the peripheral chorioretinal punched out scars in a patient with sarcoidosis

Table 1: Ocular Findings in Sarcoidosis

Bilaterality
Eye lid and conjunctival granulomas
Lacrimal gland involvement
Keratoconjunctivitis sicca
Nongranulomatous anterior uveitis
Mutton Fat keratic precipitates
Iris and pupillary nodules/iris mass
Increase in IOP
Tent shaped PAS
Nodules in trabecular meshwork
Intermediate uveitis
Snowballs/strings of pearls vitreous opacities
Multifocal peripheral chorioretinal lesions (active and atrophic)
Nodular and /or segmental periphlebitis (with or without candle wax exudate)
Retinal Macro aneurysm
Choroidal granuloma
Hemorrhagic retinopathy with branch or central retinal venous occlusions

Acute posterior multifocal placoid pigment epitheliopathy and retinal pigment epithelial detachments
Optic disc nodules/ granuloma /optociliary shunts/ dilated collateral veins on the optic nerve head
Neurological manifestations: Cranial nerve palsies, encephalopathy, chiasmal syndromes, motility disorders, disorders of the hypothalamus and pituitary gland, optic atrophy either due to direct sarcoid tissue infiltration or compression by cerebral mass.

Lacrimal gland and orbits: Lacrimal gland involvement is common and may present as a dry eye. Severe keratoconjunctivitis sicca is not uncommon. Reports of sarcoid induced myositis have been reported. Sarcoidosis coexisting with grave's disease has been reported.[4,5,7]

Eyelids and conjunctiva: Conjunctival granulomas are millet shaped to large cream to brown nodular lesions, which can occur in sarcoidosis. They are usually asymptomatic. However large conjunctival granulomas can cause diplopia. They respond generally to topical steroids. Eyelid granulomas are also seen in sarcoidosis.[17] Long standing uveitis may lead to corneal band degeneration especially in children.

Uveitis: It is typically a chronic bilateral uveitis and characterized by granulomatous inflammatory reaction in the anterior segment which includes mutton fat keratic precipitates, iris nodules, trabecular meshwork nodules and tent shaped peripheral anterior synechiae; snowball and / or "string of pearls" opacities in the vitreous; and in the posterior segment, nodular periphlebitis, multiple peripheral active or atrophic chorioretinal lesions, optic disc nodules/ granuloma and choroidal nodules.[18] Reports of Scleral involvement have also been described in sarcoidosis.[19] In acute onset sarcoidosis, non granulomatous anterior uveitis may be seen. The Ist International workshop on ocular sarcoidosis in Tokyo in 2006 laid down diagnostic criteria for ocular sarcoidosis, consisting of 7 ocular signs (Table 2), 5 laboratory investigations (Table 3) and diagnostic criteria based on a combination of ophthalmic signs and laboratory investigations (Table 4). [20]

Table 2: Clincial Signs suggestive of ocular sarcoidosis

1	Mutton fat keratic precipitates and / or iris nodules at papillary margin or on stroma
2	Trabecular meshwork nodules and / or tent shaped peripheral anterior synechiae
3	Snowballs/strings of pearls vitreous opacities
4	Multifocal peripheral chorioretinal lesions (active and atrophic)
5	Nodular and /or segmental periphlebitis (with or without candle wax exudate) and/or macroaneurysm
6	Optic disc nodules/ granuloma and / or solitary choroidal nodule
7	Bilateral inflammation (evident on clinical examination or on investigational imaging)

Table 3: Laboratory investigations in suspected ocular sarcoidosis

1	Negative tuberculin test in a patient who either had BCG vaccination or previously had a positive tuberculin test
2	Elevated serum angiotensin converting enzyme and /or elevated serum lysozyme*
3	Chest Xray: Bilateral hilar lymphadenopathy
4	Abnormal liver enzyme tests(any 2: of alkaline phosphatase; aspartate transaminase; alanine transaminase
5	Chest CT scan in patients with normal chest X-ray

*Lysozyme is required in patients treated with ACE inhibitors

Table 4: Diagnostic Criteria for ocular sarcoidosis

1	Biopsy supported diagnosis with compatible uveitis	Definite Ocular sarcoidosis
2	Biopsy not done; bilateral hilar lymphadenopathy with compatible uveitis	Presumed ocular sarcoidosis
3	Biopsy not done; Chest Xray normal; 3 suggestive ocular signs and 2 positive investigational tests	Probable ocular sarcoidosis
4	Biopsy negative; 4 suggestive ocular signs and 2 positive investigations	Possible ocular sarcoidosis

Note: All other causes of uveitis- in particular, tuberculosis- are excluded. The term ocular sarcoidosis implies sarcoidosis within the eye, with or without associated systemic disease.

The advantages of the new ocular sarcoidosis diagnostic criteria are the standardization for research purposes, comparisons between centers and allow diagnosis in the absence of a biopsy. However the disadvantages of the new criteria include the need for validation of these criteria in different ethnic groups other than the Japanese population. They are also based on the CT scan findings, which may not be available at all times.

Complications: Most frequent complications include cystoid macular edema (76%), cataract (49%), glaucoma (36%), retinal ischemia (16%) and neovascularisations (11%). Corneal band shaped keratopathy occurs in longstanding cases of uveitis. Reports of Inflammatory choroidal neovascular membranes have been described in sarcoidosis.

Investigations/Laboratory tests supportive for diagnosis of ocular sarcoidosis[21-27]

Indocyanine angiography: ICGA allows detection not only of bilaterality of disease but also of occult choroidal lesions.

Conjunctival biopsy: The biopsy yield is 66.7% if conjunctival follicles are present in comparison to 31.4% from normal conjunctiva. The yield is also increased with presence of ocular findings and pulmonary infiltrates on CT. Multiplane sectioning also gives a higher yield.

Tuberculin test: Depression of delayed type hypersensitivity manifesting, as a cutaneous anergy to tuberculin is a clinically important phenomenon occurring in sarcoidosis. Tuberculin sensitivity is depressed in sarcoidosis even in the background of high prevalence of TB. Thus negative tuberculin test in a BCG vaccinated patient or in a patient with a previously positive tuberculin skin test is a useful test in diagnosis of sarcoidosis. A negative tuberculin test excludes TB, except in seriously sick or otherwise immunosuppressed individuals (were the diagnosis of tuberculosis needs to be supported by strong bacteriological evidence).[24]

Serum Angiotensin converting enzyme& Lysozyme level: Elevated Serum ACE is advocated by some as a good measure of granuloma activity but it is elevated in only half the patients with sarcoidosis. It is nonspecific and is also elevated in children and other diseases like tuberculosis, leprosy, Gaucher's disease, chronic pulmonary disease, rheumatoid arthritis and histoplasmosis. Serum ACE in patients taking ACE inhibitors is known to be undetectable. In such cases, serum lysozyme is helpful. Lysozyme also reflects the activity of macrophages and giant cells and may be elevated in the absence of a concomitant serum ACE elevation.

Abnormal Liver enzyme tests: The international criteria for the diagnosis of ocular sarcoidosis included abnormal liver enzymes: three times the upper limit of normal values for alkaline phosphatase or elevation twice over the upper limit of two of the following liver enzymes: aspartate aminotransferase (ASAT), alanine aminotransferase (ALAT) or alkaline phosphatase.

Radiology: A Chest radiograph is helpful in making the diagnosis of sarcoidosis. Bilateral hilar lymphadenopathy is the most common finding in systemic sarcoidosis and is present in 50-89% of systemic sarcoidosis. Computed tomography of the thorax is more sensitive than a chest X-ray. High resolution CT scan detects presence of interstitial infiltrates and small lymph nodes. Contrast enhanced CT scan helps to distinguish the tubercular lymph node enlargement from that of sarcoidosis. (Figures 10-12)

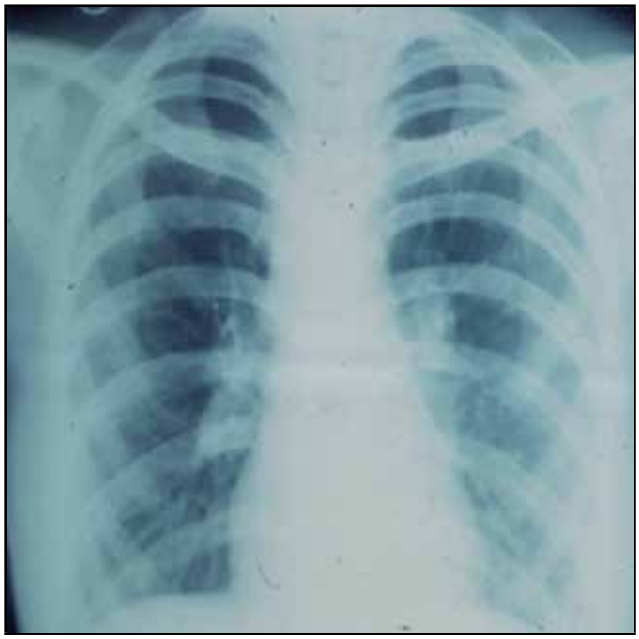


Figure 10: Chest X-Ray showing bilateral hilar lymphadenopathy in a patient with sarcoidosis

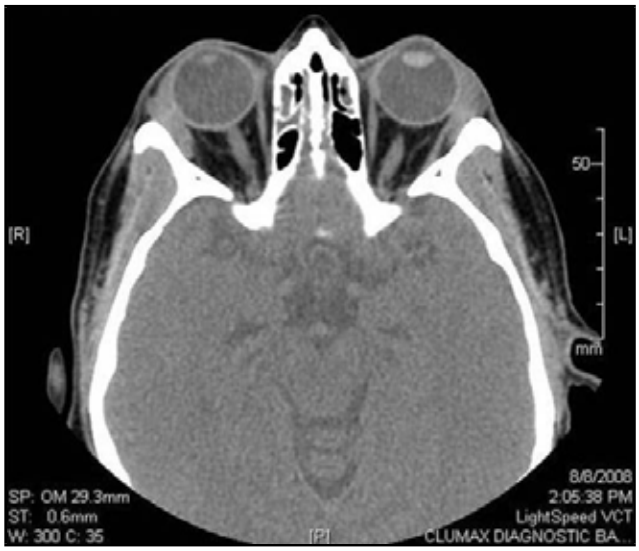


Figure 11: High resolution computed tomography of the orbits showing bilateral lacrimal gland enlargement

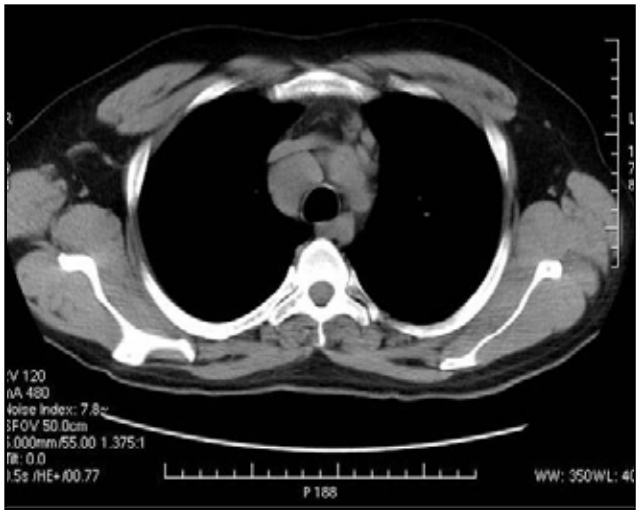


Figure12:High resolution computed tomography of the thorax showing mediastinal lymphadenopathy in a patient with grade 2 sarcoid with respiratory symptoms

Gallium citrate scanning: Gallium -67 is a radioactive isotope, which is taken up by activated macrophages and marks areas where epitheloid cell granulomas are formed. Gallium uptake is assessed and graded in the lacrimal gland, salivary glands, thorax, spleen, liver and eyes 48 hrs. after injection. However this is nonspecific and is also increased in tuberculosis and malignancies.

Bronchoalveolar lavage fluids: Examination of BAL fluid includes cytology. Lymphocytosis and an increase of CD4/CD8 ratio in nonsmoking individuals are considered to be characteristic in sarcoidosis. Smears and cultures may help to differentiate from tuberculosis.

Biopsy: Transbronchial lymph node biopsy is a most commonly performed biopsy in sarcoidosis. However EBUS-TBNA (Endobronchial ultrasonography with trans bronchial lymph node aspiration) is better in comparison to TBLB & BAL.[27,28] Non-caseating granulomas are present in sarcoidosis.

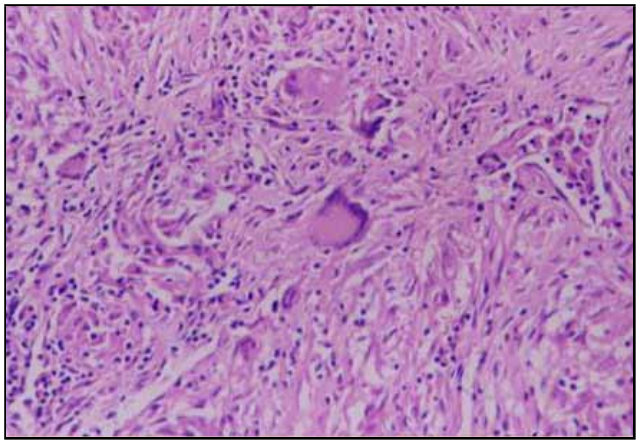


Figure 13: 18F-FDG PET/CT is a recent diagnostic modality for diagnosis & adjustment of therapy in systemic sarcoidosis.

Hypercalcemia: Hypercalcemia occurs in fewer than 10% of patients with sarcoidosis and is of limited diagnostic value.

Differential diagnosis of ocular sarcoid

In addition to the clinical signs and laboratory tests indicative of sarcoidosis, exclusion of other entities that can be mistaken for sarcoidosis is equally important for the diagnosis of sarcoidosis. Granulomatous uveitis may be seen in tuberculosis, syphilis, vogt-koyanagi Harada syndrome, toxoplasmosis, herpetic uveitis and multiple sclerosis.[29]

Chorioretinal granulomas may also be seen in tuberculosis, syphilis, vogt-koyanagi Harada syndrome, birdshot retinochoroidopathy, and primary intraocular lymphoma.

Treatment:[30-33] There is no standardized therapy for ocular sarcoidosis associated uveitis. The mainstay of treatment is corticosteroids and is given in topical, periocular and systemic routes. Periocular injections or intravitreal injections of steroids may be useful in unilateral disease in the absence of a systemic disease. Indication for systemic steroids includes optic nerve involvement (pulse intravenous route)[30]and severe or resistant posterior uveitis. The dosage depends on the disease severity. Often higher doses of oral steroids with slow tapering are required to achieve remission. For corticosteroid resistant or steroid intolerant cases, steroid sparing immunosuppressive agents like methotrexate are effective.[31,32]Other immunosuppressives in the treatment of sarcoidosis include mycophenolate mofetil, azathioprine and cyclosporine.[32] TNF–α blocking drugs have been used in resistant cases of sarcoidosis. Reports of treatment with rituximab in aggressive cases of sarcoid have been reported in literature.[33]Neovascularization may regress with systemic anti-inflammatory treatment and in some cases may require laser treatment. Intravitreal steroids or sustained steroid drug delivery devices like dexamethasone implants, fluocinolone acetonide implants are very useful in cystoid macular edema. Associated complications like cataract and glaucoma may be needed appropriately to prevent visual morbidity. Reports of intravitreal injections of anti VEGF like bevazizumab and ranizumab in inflammatory choroidal neovascular membranes and neovascularization in sarcoidosis are known.

Conclusions:

Sarcoidosis has been increasingly diagnosed in recent years, due to increased awareness of the disease and better availability of diagnostic modalities. Ocular evaluations contribute in making a diagnosis of systemic sarcoid. Early diagnosis and appropriate, adequate treatment reduces the visual morbidity in ocular sarcoidosis.

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Management of Optic Disc Pit and Maculopathy

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Introduction

Optic disc pits (ODP) refer to focal cavitations in the substance of the optic discs which constitute a part of the atypical optic nerve coloboma complex.[1,2] This condition was first described way back in 1882.[3] ODP is a very rare condition with an incidence rate of 1/11,000 and typically present unilaterally.[4] Though usually singular in presentation, presence of two or three simultaneous ODP is known. The occurrence has no gender predilection with a possible autosomal dominant inheretance.[5]

Pathophysiology of ODPs

ODPs occur due to an incomplete closure of the optic fissure during embryological development. This results in an anomalous communication between the subarachnoid space and the disc pit.[6] Histological examination of a disc pit reveals herniation of dysplastic retinal tissue into the subarachnoid space through the lamina cribrosa.[7]

Pathophysiology of Optic disc pit maculopathy (ODP-M)

As high as 3/4th of all cases of ODP are associated with concurrent macular changes leading to serous macular detachment, cystic degeneration and pigmentary changes.[8,9] The origin of the fluid and the mechanism by which the fluid causes the ODP-M are both debatable. The various proposed origins of the fluid range from the sub arachnoid space around the optic nerve, the vitreous cavity, the peripapillary leaky blood vessels and the peridural orbital space.[10-13] Although the intraretinal fluid communicates with the optic disc pit, the subretinal space usually does not.

The mechanism of occurrence of ODP-M was well elucidated by Lincoff in 1988.[14] According to the hypothesis, initially fluid from the ODP accumulates in the inner retinal layers causing a schisis-like separation. This further progresses to cause an outer layer macular hole. The fluid progressively accumulates in all the retinal layers causing a retinal pigment epithelial detachment and finally a serous retinal detachment like picture. As the occurrence of ODP-M is a late phenomenon in life, it has been proposed that, the abnormally developed optic nerve head has a pocket of liquified vitreous over it. Over the subsequent years, tractional forces develop at the surface of the retina leading to ingress of the liquified vitreous into and under the retina.[15]

Clinical features and diagnosis

The cases with ODP alone are asymptomatic but majority of them progress to ODP-M. Cases with ODP-M usually occur in pits which are temporally located. The patients usually do not get symptomatic till the third or the fourth decade. In the natural history, spontaneous recovery is rarely reported.[16] Majority of cases though have a poorer prognosis without treatment with a natural history of worsening of visual acuity to 20/200 or worse.[17]

The diagnosis of ODP and ODP-M is largely clinical. Fundus examination clearly shows a temporal greyish-black depression on the temporal part of the optic disc with or without an overlying diaphanous membrane consistent with the diagnosis of ODP.(Fig 1a) In cases of ODP-M one may see a clinically elevated looking macula with cystic changes at the fovea. The investigation used to confirm and quantify the diagnosis is optical coherence tomography (OCT). OCT helps in confirming the presence of the disc pit, demonstrates classical macular schisis and the subretinal fluid. (Fig1b)OCT may also show outer retinal hole with retinal pigment epithelial detachment.

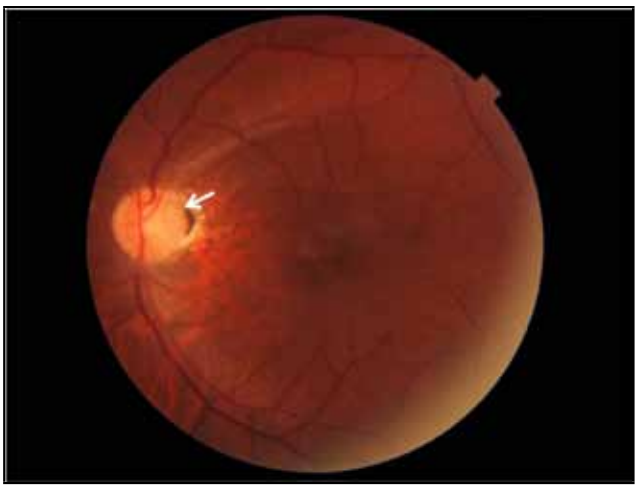

Figure 1a. Fundus photo showing optic disc pit

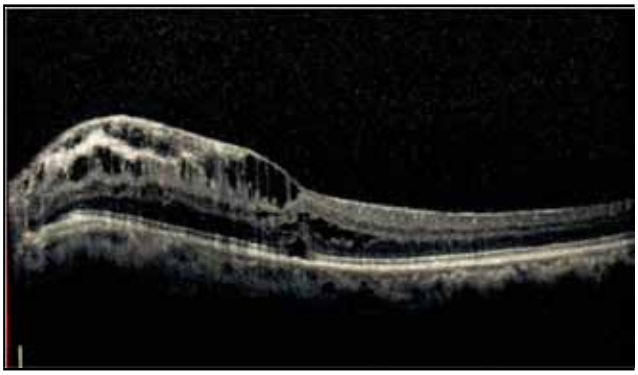

Figure 1b. OCT showing macular schisis

Management

Incidentally diagnosed ODP without vision loss or maculopathy can be observed. Prophylactic treatment is not warranted as no study has demonstrated any beneficial effect of prophylactic therapy. As the natural history of ODP-M demonstrates progressive visual loss, treatment is warranted in all cases of ODP-M to preserve vision. Over the years various modalities have been attempted

to treat ODP-M with no consensus as to which is the best therapeutic technique.

The earliest reports of management described the usage of oral steroids.6 However, with this the effect was inconsistent and short lasting and is no longer recommended. Gass, et al recommended laser photocoagulation at the temporal disc margin of the disc pit.[18] The laser was hypothesized to cause a chorioretinal scarring with subsequent creation of a barrier between the ODP and the sub retinal space. The effect though is known to be very slow and unpredictable.[19] Lincoff, et al described the technique of pneumatic displacement of the sub retinal fluid in ODP-M to result in macular attachment.[20] This technique was also combined with laser photocoagulation with reasonable success.[21]

Among surgical treatment options, macular buckling was proposed by Theodossiadis, et al.[22] In this technique, an exoplant is sutured to the posterior globe from 6 o clock to 12 o clock position. Though over 85% cases were shown by the authors to have achieved resorption of subretinal fluid in these cases, the technique did not get popular due to the tedious nature of the surgery. With advancement in the vitreous surgery techniques in the past decade, sutureless pars plana vitrectomy (PPV) has become the most popular procedure for treatment of ODP-M. The most essential step is proposed to be posterior vitreous detachment (PVD) induction as it is hypothesized that it relieves traction that allows for reattachment of the detached retina.[23] (Fig 2).

Figure 2. Triamcinolone-assisted PVD induction

The associated steps of added endolaser and fluid gas exchange have not been conclusively proven to be of benefit. Various studies have also proposed the importance of internal limiting membrane peeling (ILM) as an adjunctive step to treating ODP-M. Cases which have a multi-layered macular schisis are more difficult to treat than those who do not and additional ILM peeling has been advocated for such cases.[24] (Fig 3) In cases where additionally glial tissue is present over the disc pit, it is recommended that the glial tissue be peeled off.[25] Anecdotal case reports have also described sealing off of the pit by sealants like autologous platelets, inverted peeled ILM and fibrin glue.[26,27,28] Recently, Ooto, etal [29] reported a large case series of 18 eyes treated by the technique combining PPV with inner partial thickness retinotomies just temporal to the optic disc. In their series, complete resolution of the subretinal fluid was seen in 17 (94%) eyes with 10 (56%) eyes achieving a final visual acuity of 20/30 or better.

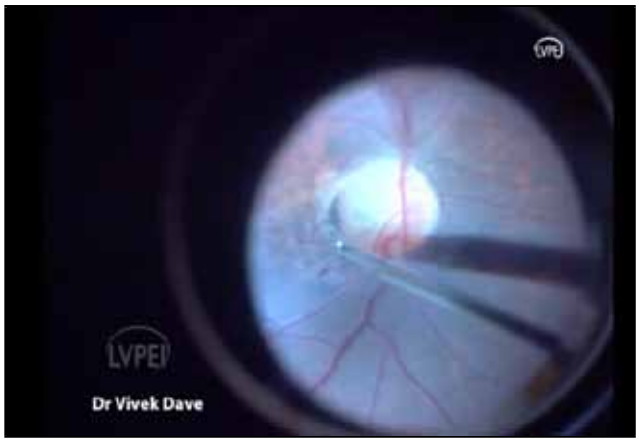

Figure 3. ILM peeling over the edge of the optic disc pit

Conclusion

In conclusion, there is no consensus on the optimal surgical technique or a consensus on management protocol for ODP or ODP-M. Asymptomatic ODP should be just observed and explained about the risk of progression to ODP-M. They should be advised to regularly follow up annually or earlier if visual deficit is noted. No prophylactic treatment is advisable for these eyes.

For cases presenting with ODP-M, the treatment of choice is PPV. Laser photocoagulation can be reserved for those who cannot undergo surgery for any reason. Literature suggests PVD induction to relieve traction and gas tamponade to avoid fluid movement across the hole as key steps to achieve success during PPV. The role of ILM peeling is unclear and can be reserved for cases with significant macular schisis. The key point to be well discussed with the patient is that the visual recovery is a very slow process after ODP-M surgery with most studies describing a recovery period as long as 6-12 months.[30] (Fig 4)

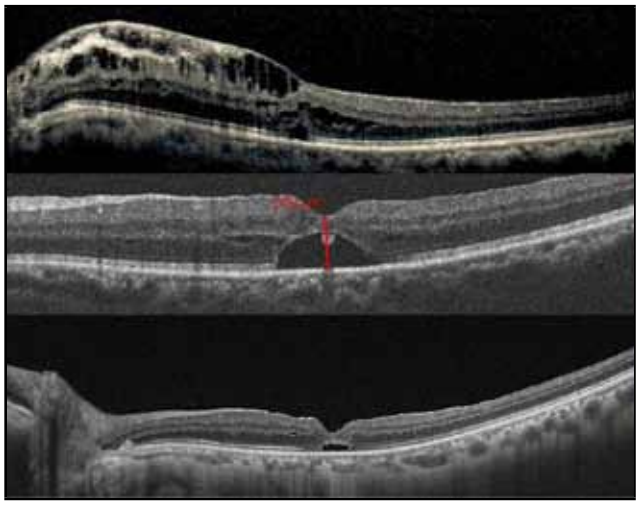

Figure 4. Serial OCTs at presentation, Month 1 and Month 3 post -operative

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Sutureless & Glue Free Technique of Conjunctival Autograft in Primary Pterygium Surgery: A Series of 52 Eyes

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Keywords: Pterygium, fibrin glue, glue free, sutureless

Purpose: To study the efficacy and complications of sutureless and glue free conjunctival autograft for the management of primary pterygium over a period of one year.

Methods: This was a retrospective case series and patients who were operated between November 2012 and June 2014 were analysed. 52 eyes of 50 patients were included in the study. Pterygium excision with limbal conjunctival autografting without using glue or sutures was performed in all the patients followed by bandaging for 12 hours. The patients were followed up post operatively on 1st day, 1 week, 6 weeks, 6 months and 12 months. They were examined for haemorrhage, wound gape, graft shrinkage, chemosis, graft dehiscence, recurrence or any other complication.

Results: The mean age of the patients was 48.7 +/- 13.6 years (range 19 – 77 years). 2 eyes had biheaded pterygium, 2 had cystic pterygium and the remaining were primary nasal pterygium. Total graft dehiscence occurred in 1 eyes (1.92%),partial graft retraction in 2 eyes (3.84%), graft edema in 3 eyes (5.76%) and recurrence was seen in 1 eye (1.92%) on one side in a patient who had biheaded pterygium. No other complication was noted.

Conclusion: Sutureless and glue free limbal conjunctival autografting following pterygium excision is a safe, effective and economical option for the management of primary pterygium.

Introduction: Pterygium is a common disorder in many parts of the world with reported prevalence rates ranging from 0.3 – 29%.1 It is wing shaped conjunctival encroachment generally situated on the nasal side but at rimes both nasal and temporal and rarely it can be seen only on the temporal side.2 Pterygium is most frequent in areas with more ultra violet radiation.3 Hot, dry windy dusty and smoky environments also seem to play a role. Hereditary factors have been postulated as a possible cause for pterygium.4 The exact cause for pterygium still remains unclear.

Over the years various treatment strategies such a simple excision with or without adjunct measures (post op B irradiation, intraoperative and post-operative mitomycin) and various techniques of conjunctival grafting have been tried to decrease recurrence after surgery.3 Kenyon et al in 1985 proposed that a conjunctival auto graft of the bare sclera could be used in the treatment of recurrent and advanced pterygium.5 While doing conjunctival auto graft initially sutures were used with associated complications of infection, granuloma formation and chronic inflammation .6 Fibrin glue has been used in place of sutures while doing conjunctival autograft with reported advantage of less pain, shorter operating time, and less pterygium recurrence.1,7 Postulated etiology of reduced recurrence include immediate adherence of the graft and lack of post-operative inflammation which inhibits fibroblast ingrowth. Bahar et al reported greater patient acceptance with the use of fibrin glue compared to sutures in addition to shorter operating time.8 Major concerns of use of fibrin glue include the cost and also the risk of transmitted infection. The cost of glue can be overcomed by scheduling multiple pterygium surgeries on a single day. Potential risk of complications like transmission of virus infection with the use of bio adhesive have been found in patients undergoing thoracic surgery but similar cases have not been reported following pterygium surgery. The other rare complication of use of plasma derived products includes hyper sensitivity reaction.

D De Wit et al in 2010 reported the simple method of achieving conjunctival autograft adherence during pterygium surgery without the use of fibrin glue or sutures.6 This study involved 15 eyes of 12 patients. After pterygium excision and fashioning of conjunctival autograft the recipient bed is encouraged to achieve natural haemostasis and relative desiccation before graft placement. Excessive haemorrhage in the bed was tamponaded. Graft adherence and positioning was examined 20 minutes after surgery. The mean follow up time was 9.2 months. Cosmesis was excellent in all cases. There was no intra operative or post-operative complication requiring further treatment.

We present our experience of above technique in a series of 52 eyes of 50 patients with a average follow up of 13.7 months (range 7 – 20 months).

Materials & Methods: Retrospective chart review of all consecutive cases of primary pterygium who had undergone suture less and glue free conjunctival autograft performed between November 2012 and June 2014 in the cornea service of Maxivision eye hospital was done. All these patients were operated by a single surgeon. All cases were operated under peribulbar anaesthesia. The blunt spatula was used to dissect the plane at the neck of the pterygium and with a sweeping fashion it was peeled off from the cornea. The pterygium

along with its body is then dissected down to the bare sclera avoiding the insertion of the medial rectus and extensive dissection of tenons followed by careful 15 number blade dissection of conjunctival remnants on the cornea. The size of the defect is then measured with Castroviejo Calipers (Bausch & Lomb Storz, Storz instruments, St Louis, MO, USA). Careful dissection is then done between the donor graft conjunctiva and tenon's layer to get a thin graft which is 1 mm oversized from the superior conjunctival limbal area. The orientation of the limbal area is maintained of the donor graft while covering the recipient area. Fresh bleeding is then initiated by fracturing small veins and capillaries to induce bleeding to provide autologous fibrin between the graft and the recipient bed. The stabilisation of the graft is tested with a forceps on all the sides after haemostasis is achieved which usually takes 5-7 min. None of the patients were on anti-coagulating agents pre operatively which usually cause lot of bleeding and delayed haemostasis as well. All these cases were examined after half an hour to see if the graft is in place. Then patch was again applied after instilling 0.3 % Moxifloxacin eye drops (Vigamox, Alcon, USA). The patch was removed the next day. On post op day 1, the position of the graft and presence of epithelial defect on the cornea was noted. Post operatively patient was continued on 0.3 % Moxifloxacin eye drops 6 times a day for 1 week along with 1% Prednisolone eye drops (Prednisolone, Alcon, USA) in a tapering dosage over 1 month and artificial tears as needed. All these patients were followed up on post op day 1 after surgery, day 4-5 till the epithelium heals, every week for the next 2 weeks, every month for the next 3 months and then every 3 months till the last follow-up.

Patients were examined post operatively for haemorrhage, wound gape, graft shrinkage, chemosis, graft dehiscence, and recurrence. Recurrence was defined as any growth across the limbus onto the cornea. The pre – op, surgical steps and post – op images of a patient is shown in Figure 1.

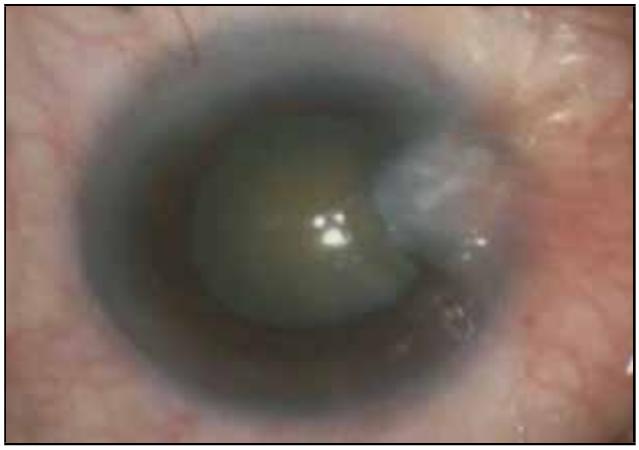
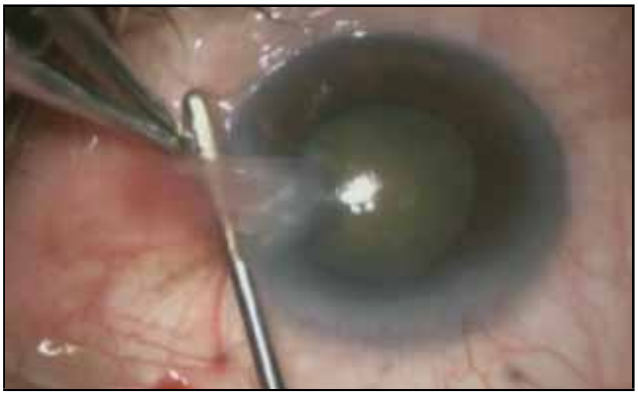
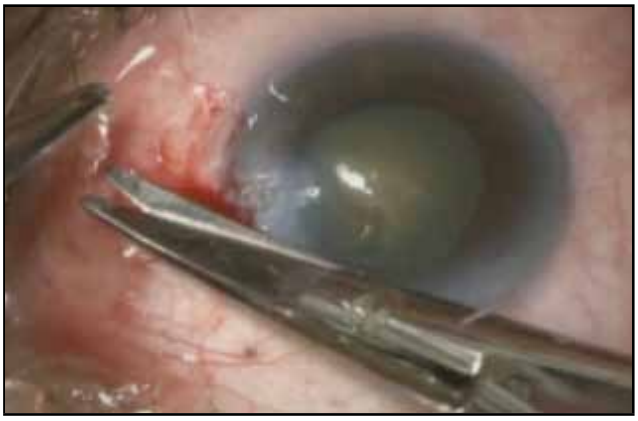


Figure 1. Surgical steps of gluefree and suture less pterygium surgery.

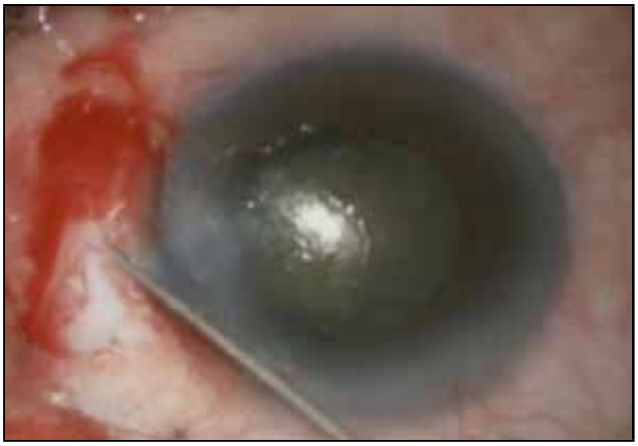
1A. Pre op clinical image of a patient with primary nasal pterygium.



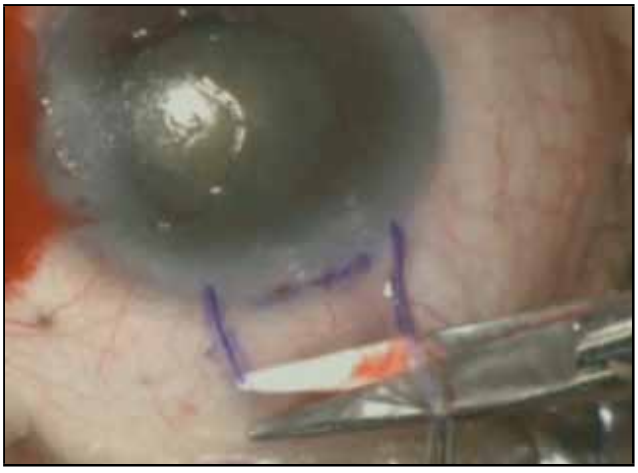
1B. Blunt iris spatula is used to free the neck of the pterygium at the limbus and subsequently the pterygium is peeled off from the cornea.



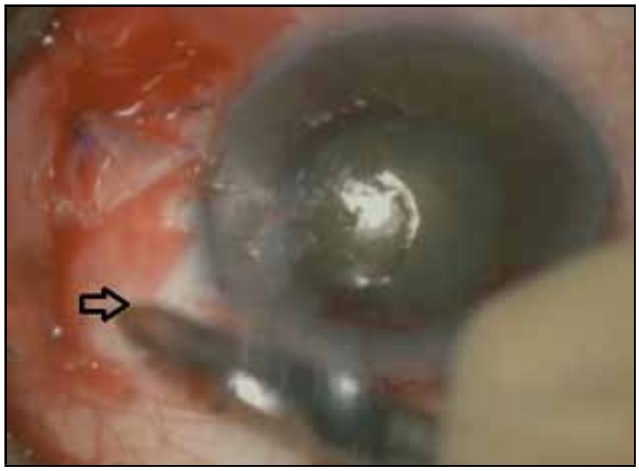
1C. Dissection is done down to the bare sclera to remove the sub conjunctival tissue with Wescott's scissors.



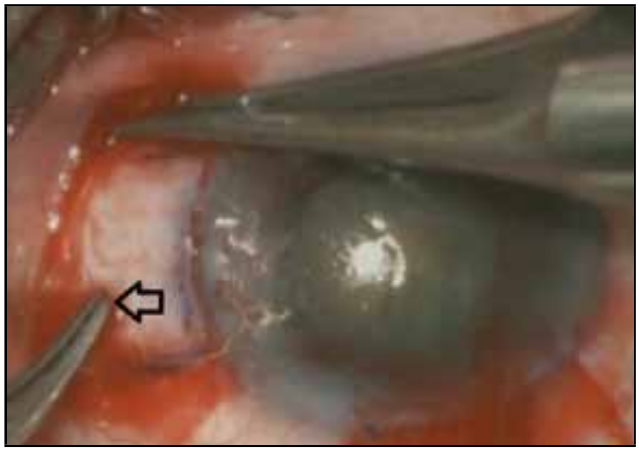
1D. 15 No. Bard parker blade is used to smoothen the cornea.



1E. Donor graft being prepared from the superior limbus, care is taken to achieve a thin conjunctival graft avoiding the Tenon's.

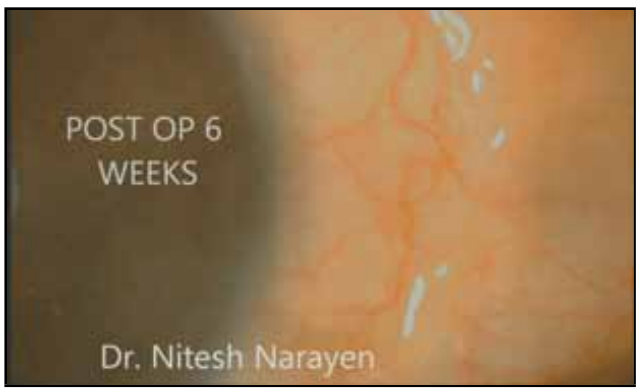


1F. Graft is then placed on the recipient bed maintaining the limbus to limbus orientation, the arrow head indicates the point of bleeder.



1G. The graft being placed under the thin layer of fibrin created by the bleeder.

1H. Clinical photograph of Day 1 post op patient showing well opposed conjunctival graft.



1I. Clinical photograph of the patient at 6 weeks follow up.

Results: The study involved 52 eyes of 50 patients (26 Males and 24 Females). The mean age of the patient was 48.7 +/- 13.6 years (range 19 – 77 years). 48 patients had unilateral pterygium and 2 had both nasal and temporal pterygia.The pie chart showing the relative distribution of nasal primary pterygium, nasal and temporal pterygia and cystic pterygium is shown in Table 1. The post-operative complications seen in this series is shown in table 2.

Post operatively graft dislodgement was seen in 1 eye (1.92%). This patient was managed by suturing the graft with 8-0 vicryl sutures. Partial graft retraction was seen in 2 eyes (3.84%, Figure 2) and overriding of the graft onto the cornea was seen in 1 eye (1.92% , Figure 3). Both had no consequence and the surface healed well with regular post-operative medications. Graft edema was seen in 3 eyes (5.76 %, Figure 4) which was managed by stepping up the frequency of steroid drops. None of these eyes led to the recurrence of pterygium. Recurrence was seen only in 1 eye(1.92%, Figure 5). This was a patient of both nasal and temporal pterygia. Recurrence was noticed during the 2nd month follow-up. The recurrence remained stationary after extending to about 1.5 mm on the cornea. The visual acuity of this patient was 20/20 with the refraction of 0.50 x 90 at 17 months of follow up.

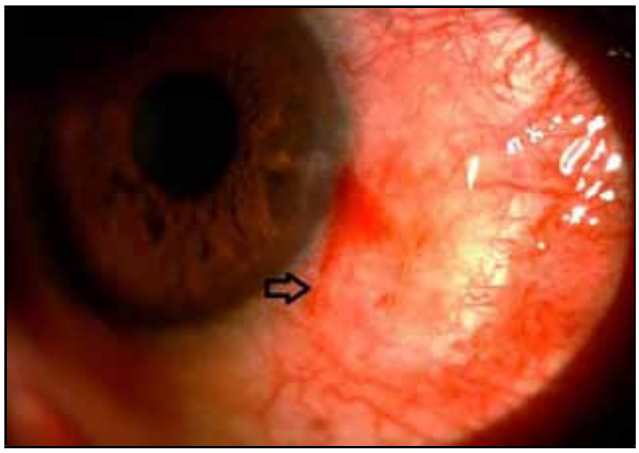


Figure 2 : Diffuse slit lamp view showing graft retraction inferiorly

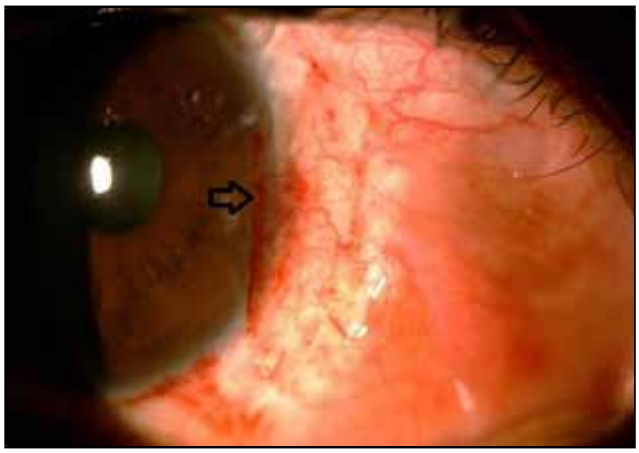


Figure 3 : Diffuse slit lamp view showing over riding of the graft onto the cornea to the extent of 1 mm.

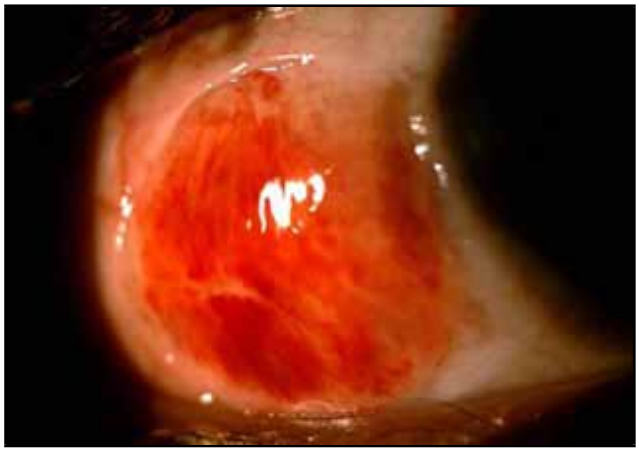


Figure 4. Diffuse slit lamp view showing graft edema which resolved with topical steroids.

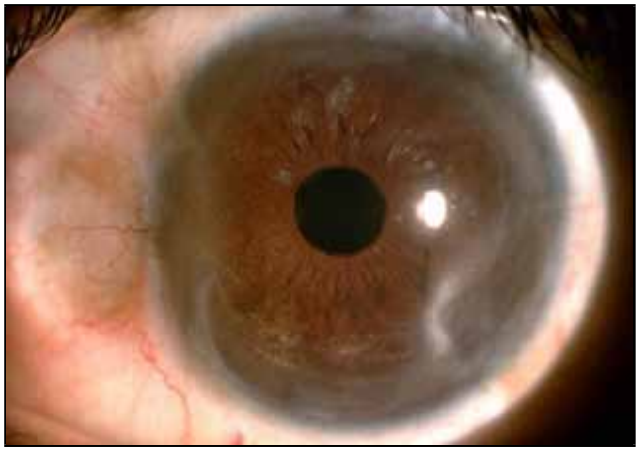


Figure 5. Slit lamp view showing partial recurrence temporally with a case with bi- headed pterygium

Discussion: Operative complications related to pterygium surgery are uncommon and generally related to the surgical technique. This includes excessive bleeding, button hole of the graft, and rare injury to the medial rectus. Early post-operative complications include persistent epithelial defect, pyogenic granuloma and dellen formation. Of greater concern is potentially serious sight threatening complications that have been associated with the use of Mitomycin C and Beta irradiations such as scleral necrosis, 9 infectious scleritis, 10,11 serious secondary glaucoma,12 iritis, cataract, corneal edema,13 corneal perforation14 and endophthalmitis.16The main post-operative complication is recurrence.

Fibrin glue has been used as an to sutures for securing conjunctival grafts.2,7,16,17 The use of fibrin glue shortens operating time significantly and is associated with less post-operative discomfort. It is claimed that the use of fibrin glue also provides a more even attachment of the graft to the scleral bed. There is minimal inflammation and sporadic cases of graft dislodgement or loss. Korannyi et al 2005 demonstrated recurrence rate of 5.3 % with glue versus 13.5% with sutures.7

The idea of suture less and glue free conjunctival auto graft in pterygium surgery is promising and also safe with no glue related complications like hypersensitivity reactions and possible risk of viral transmission. In the series of D vit et al no intra-operative or post-operative complications were seen in 15 eyes.6 The mean graft area was 24 mm2 and the mean follow up time was 9.2 months. The primary outcome measures in this series were graft dislocation and pterygium recurrence. Secondary outcome measures included size of the graft used, patient comfort on visual analogue scale and visual acuity. In this series there were no transplant dislocations or failures. The visual acuity was not affected in majority of the patients. Post-operative pain on Day 1 after surgery was consistently rated as less than or equal to 2 out of 10 on a visual analogue score. Pain did not increase after the first post- operative day. Patients rated their cosmesis as excellent in all cases. The authors concluded that the operating time, post-operative symptoms, recurrence and complication rate of suture less and glue free conjunctival autograft appeared to be equivalent to conventional suture and glue technique of a similar follow up duration. The risk of graft retraction appears to be no greater without suturing as long as meticulous dissection of sub epithelial graft tissue was respected. The authors opined that in this small series surgical time appeared no greater than published literature though the possibility of longer operation time compared to sutures or fibrin glue is possible.

The similar technique of suture less and glue free limbal conjunctival autograft for pterygium surgery was reported by Malik KP et al in 2012.18 This was a prospective case series of 40 consecutive eyes with primary nasal pterygium. Total graft dehiscence occurred in 2 eyes (5%), graft retraction in 3 eyes (7.5%) and recurrence was seen in 1 eye (2.5%).

In this series we report 52 eyes of 50 patients; the same technique reported by De Vit et al and Malik KP et al was adoped. This is the largest series of suture less and conjunctival autograft reported in literature. All these patients had a minimum follow up of 7 months. No serious intraoperative or post-operative complications were seen. Graft dislodgement was seen only in 1 eye which was managed by suturing. Partial graft retraction, over riding of the graft on to the cornea and graft edema were minor complications seen but had no consequence as all these 3 resolved with regular post-operative medications. Recurrence was seen only in 1 eye around 2 months after surgery; this was a patient of both nasal and temporal pterygia. Recurrence required no further management as it remained stationary after extending 1.5 mm onto the cornea with no significant astigmatism and had good unaided visual acuity.

Table 3 summarises the results of published literature on use of glue, sutureless and gluefree technique in comparison with the present series.

To conclude we present excellent results after sutureless and gluefree conjunctival autograft in pterygium surgery in a series of 52 eyes of 50 patients. Prospective randomised comparative study involving patients with pterygium using sutures, fibrin glue and sutureless, gluefree technique with conjunctival autograft will provide more evidence regarding the efficacy of the presented technique.

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Case Report

Aicardi Syndrome

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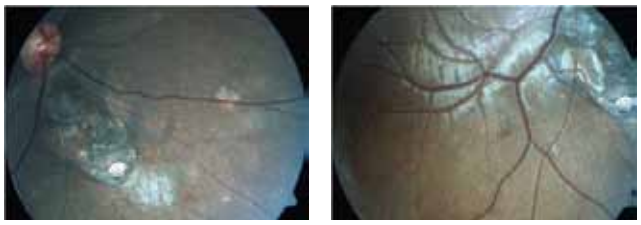
Introduction: Aicardi syndrome (AS), identified first by Jean Aicardi in 1965, is a rare genetic disorder characterized by infantile spasm (IS), corpus callosal agenesis (CCA) and chorioretinal lacunae (CRL).[1]. This is a X-linked dominancondition and occurs almost exclusively in females, because of early embryonic lethality in hemizygous males. Cerebral malformations like microgyria, heterotopias, intraventricular cysts and choroid plexus papillomas coexist at times. The other occasional findings are abnormalities of ribs and spine, microphthalmia, optic nerve coloboma and asymmetry of cerebral hemispheres.

Key Words: Aicardi syndrome, Chorioretinal lacunae, Infantile spasms.

Case Report: A 11 year old girl presented to our OPD for refraction. Her mother gave history of seizures from since when she was three months old for which they had consulted a neurologist .She was on anti-epileptic treatment since then .She has 5-6 episodes of seizures in a month till date inspite of treatment with sodium valproate 1000 mg and lamotrigine 500mg BD and ketogenic diet. She had an average intellectual ability. No history of gastrointestinal problems.

Ocular Evaluation: Her ocular examination showed 20/20 vision in each eye. Anterior segment in both the eyes was normal. IOP was 14 mm Hg both eyes measured with Goldmans applanation tonometer. Gonioscopy revealed open angles in both eyes.

Fundus examination in the right eye (Figure 1a & 1b) showed chorioretinal lacune in the inferonasal quadrant pathognomic of Aicardi syndrome. Fortunately her vision was not affected as the chorioretinal lacunae did not involve the macula.



Figures 1 a& 1b: Inferonasal quadrant chorioretial lacunae

Fundus examination in the left eye revealed optic disc coloboma with partial morning glory syndrome (Figure 2).

Figure 2: Optic disc coloboma with partial morning glory disc.

Investigations: MRI Brain: showed dysgenetic corpus callosum, focal polymicrogyria with csf cleft, band heterotopias extending till right lateral ventricular margin thus confirming her diagnosis (Figure 3).

Figure 3: MRI Brain with features suggestive of Aicardi Syndrome.

EEG: Her EEG report showed attenuated waves in frontal lobe.

USG Abdomen & X-Ray Spine: Normal study.

Discussion: Aicardi syndrome is thought to be an X-linked dominant disorder lethal to males. Except for 2 male children, all reported instances have been in females. Both males had XXY genotypes, which further supports an X-linked male lethal genetic substrate. This mutation appears to be de novo. [2,3]

The diagnosis of Aicardi Syndrome is based upon the classic triad of Corpus callosum agenesis/dysgenesis, Chorioretinal lacunae and Infantile spasms. Classic chorioretinal lacunae do not enlarge or progress. Although other ocular lesions are present in Aicardi syndrome, this manifestation is pathognomonic for diagnosis. [4]

There is a range of costovertebral, ocular and cerebral abnormalities associated with this disorder. The cerebral gray-matter heterotopias and other cortical malformations act as epileptogenic foci. Their seizures typically start in early childhood and are usually intractable. Besides Infantile spasms other seizure types are also demonstrated. Dissociated burst-suppression or burst suppression pattern appearing asymmetrically in either cerebral hemisphere is a characteristic EEG finding in AS. The developmental delay in AS is generally profound, involving both motor and language skills. Chevrie and Aicardi in their analysis of 184 patients of AS observed that none had acquired meaningful speech. But of late a larger spectrum of the disease has been recognized and it had been found that higher functioning AS individuals do exist. Most of the AS cases die at an early age primarily due to aspiration pneumonitis. But some do live into their adolescent years and even in their twenties. Good visual function in AS patients does occur if the fovea is uninvolved with chorioretinal lacunae as in our case.

Cerebral heterotopias, inter hemispheric cysts, optic nerve coloboma, microphthalmia, thoracolumbar kyphoscoliosis may also be present as associated features in AS. Severe psychomotor impairment and absence of meaningful speech have also been noted.

In our case seizures were of early onset and intractable.. A recent report observed good results with vigabatrin and recommends this as the first-line drug in the treatment of seizure in AS cases.

Aicardi syndrome is indeed a rare neurological illness. Therefore, any female child with recurrent seizure occurring in early childhood should be investigated appropriately to exclude AS. Vigabatrin may be considered among the first-line AED; however, one should keep in mind the risk of retinal toxicity with this medication.

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Case Report

Anterior Segment Optical Tomography Findings in Vortex Keratopathy

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Abstract: Fabry disease is an X-linked lysosomal disorder. The ocular manifestations of Fabry disease includes cornea verticillata which is a "vortex whorl" like corneal opacity. Three patients with vortex keratopathy in Fabry disease were subjected to slit lamp photography and OCT. Slit lamp biomicroscopy of the three patients showed the presence of bilateral whorl-like opacities located in the epithelium. On Anterior segment Optical Tomography (ASOCT) there are areas of hyperreflectivity of corneal epithelium corresponding to the areas of the whorl like opacities.

Keywords: Fabry disease, vortex keratopathy, AS OCT.

Introduction: Fabry disease is an X-linked lysosomal disorder that has a prevalence of 1 in 40,000 males .[1]. Deficiency of alpha-galactosidase A activity leads to lysosomal accumulation of glycosphingolipids, predominantly the cerebroside trihexosides. Diffuse, abnormal accumulation of glycosphingolipids occurs in all tissues, producing swelling and proliferation of endothelial cells accounting for the associated clinical abnormalities of skin, eye, kidney, heart, brain, and peripheral nervous system. The ocular manifestations of Fabry disease includes cornea verticillata which is a "vortex whorl" like corneal opacity. The cornea verticillata is the outcome of the deposit of GB3 in the basal layer of the corneal epithelium. We report the characteristics of three cases of vortex keratopathy in Fabry disease by segment ASOCT.

Case History: Three patients who have come to our OPD for a routine eye check up were examined. Slit lamp examination was done for them. There was appearance of bilateral whorl like opacities seen on the cornea. The anterior segment module of optivue RTvue fourier domain OCT with a resolution of 5 microns was used to perform ASOCT.

Case 1: A 42 year old female patient with best corrected visual acuity 20/20 in both eyes. Slit lamp biomicroscopy showed bilateral vortex keratopathy involving the epithelium (Figure 1). An ASOCT was done, which showed hyperreflectivity in the areas of whorl like opacities involving the entire thickness of the epithelium (Figure 2). The central corneal thickness was 551 microns in the right eye and 547 microns in the left eye. The thickness of epithelium was measured at the central 3 mm zone. The average epithelial thickness was 41 microns in the right eye and 42.5 microns in the left eye.

Figure 1: Slit lamp photograph of vortex keratopathy

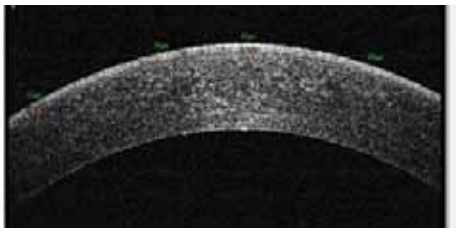

Figure 2: AS OCT of the same patient with hyperreflective epithelium

Case 2: A 17 year old boy with best corrected visual acuity 20/20 in both eyes. Slit lamp biomicroscopy showed bilateral vortex keratopathy involving the epithelium. An ASOCT was done, which showed hyperreflectivity in the areas of whorl like opacities involving the entire thickness of the epithelium(Figure 3). The central corneal thickness was 568 microns in the right eye and 551 microns in the left eye The thickness of epithelium was measured at the central 3 mm zone. The average epithelial thickness was 42.5 microns in the right eye and 45.7 microns in the left eye (Figure 4).

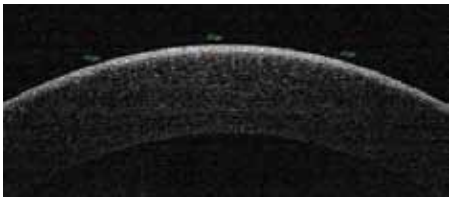

Figure 3: AS OCT showing hyperreflective epithelium

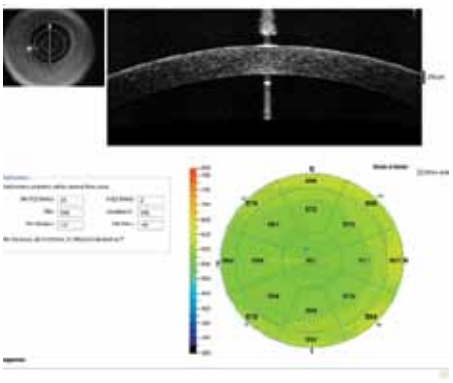

Figure 4: Corneal thickness measurements

Case 3: A 44 year old male patient with best corrected visual acuity 20/20 in both eyes. Slit lamp biomicroscopy showed bilateral vortex keratopathy involving the epithelium (Figure 5). ASOCT showed hyperreflectivity in the areas of whorl like opacities involving the entire thickness of the epithelium The central corneal thickness was 568 microns in the right eye and 566 microns in the left eye. The thickness of epithelium was measured at the central 3 mm zone. The average epithelial thickness was 41 microns in the right eye and 42.5 microns in the left eye.

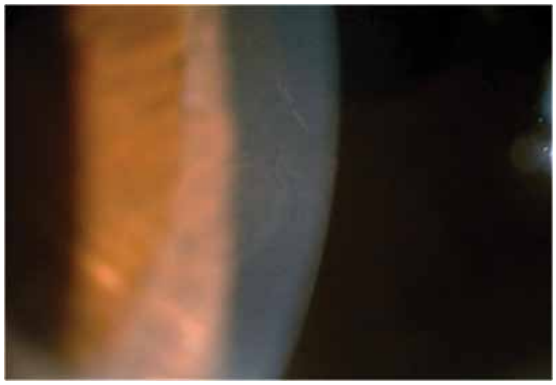

Figure 5: Vortex keratopathy in slit lamp photograph

Discussion: Fabry disease is an X-linked lysosomal storage disease that is caused by deficient activity of lysosomal enzyme α -galactosidase A (α -Gal A) affecting approximately 1 in 40,000-60,000 males. Most males with no α -Gal A activity develop the classic phenotype of Fabrys disease, which affects multiple organ systems.

Ocular Manifestations: Ocular abnormalities in Fabrys disease result from the deficient activity of the lysosomal hydrolase, α-galactosidase A. This deficiency leads to a progressive deposition of glycosphingolipids in some ocular structures. [2-4]. The most specific ocular manifestations of Fabry disease are:conjunctival vascular abnormalities, corneal opacities (cornea verticillata) and lenticular opacities.

Cornea verticillata which is a "vortex whorl" like corneal opacity. It is the most common finding and is seen in all hemizygotes and majority (up to 70%) of hetrozygotes.[5]. The cornea verticillata is the outcome of the deposit of GB3 in the basal layer of the corneal epithelium.

On slitlamp examination vortex keratopathy appears as bilateral whorl-like opacities located in the corneal epithelial layer, most commonly in the inferior corneal area. These opacities are typically cream coloured, ranging from whitish to golden-brown. They are termed cornea verticillata because the deposits are distributed in a vortex pattern.

We are reporting ASOCT findings in three cases with vortex keratopathy in Fabrys disease. The ASOCT picture is showing hyper-reflectivity in the entire thickness of epithelium in the areas of vortex keratopathy. The average corneal thickness in these patients was also less than the normal average corneal thickness.

Significance: Assessment of eye abnormalities in patients with Fabry disease forms a significant part of Fabry Outcome Survey(FOS) an international database for all patients with Fabry disease who are receiving, or are candidates for, Enzyme Replacement Therapy(ERT) with agalsidase alfa. Few instances have shown apparent regression of corneal opacities after ERT, detected using a slit lamp. However, quantification of the vortex opacities is highly subjective, and reliable methods of imaging cornea verticillata are still awaited.

Can OCT become a useful tool to quantify these changes in cornea in future??

We performed a Medline search with keywords "fabry disease with cornea verticellata", "vortex keratopathy AS OCT findings", "whorl keratopathy" and various combinations. To the best of our knowledge, AS OCT findings in Vortex Keratopathy has not been reported previously .

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A rare case of visual field loss in a young female patient

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Abstract: A 37 year old female presented to our Out Patient Department with complaints of visual loss in LE for 4 months. Best corrected visual acuity was RE 6/6 and LE 6/60. Anterior segment examination in RE was within normal limits; LE revealed 0.3 log units RAPD. A routine confrontation test showed inferotemporal field defects in each eye. 30-2 HFA visual field test showed bitemporal hemifield defects. Fundus examination with 90 D in RE was normal and LE showed temporal pallor of optic disc. MRI scan revealed pituitary macroadenoma with mass effect over left optic chiasma and left optic nerve. Endocrinological reports revealed raised prolactin hormone levels suggestive of prolactin secreting pituitary tumour.

Keywords: Temporal pallor, Pituitary macroadenoma, Optic chiasma, Prolactinoma , Cabergoline

Introduction: Pituitary adenomas represent from 10% to 25% of all intracranial neoplasms and the estimated prevelance rate in the general population is approximately 17%. [1]

Based on its size a pituitary adenoma can be classified as a microadenoma (<10mm) or a macroadenoma (>10mm).However this classification has now been augmented by a more comprehensive system based on immunohistochemistry and electron microscopy. [2]

Patients with pituitary tumours commonly present to clinics with the following symptoms:

- Fifty to sixty percent present with visual symptoms due to compression of optic nerve structures.
- The presentation of a pituitary macroadenoma relates to its mass effect and pressure on surrounding structures.
- Nonspecific headache can be seen.
- Lateral extension can result in compression of the cavernous sinuses and may cause ophthalmoplegia, diplopia, and/or ptosis.
- Talkad et al recently reported an isolated, painful, postganglionic Horner syndrome as the initial sign of lateral extension of a large prolactinoma. [3]
- Extension into the sphenoid sinuses can cause spontaneous cerebrospinal fluid (CSF) rhinorrhea.
- In addition to visual symptoms, endocrine dysfunction may be seen. Tumours that grow more rapidly, even if they are hormone inactive, are capable of producing symptoms of an intracranial mass, such as visual field disturbances. [4]

Lesions of the optic chiasma can produce a variety of visual field defects including bitemporal hemianopia, junctional scotoma (anterior chiasmal defect), quadrantanopia and bitemporal, or unilateral temporal scotoma depending on the site and extent of the lesion.[5]

Visual field improvement following resection of the pituitary tumor occurs in three stages.[6] Stage one is the early fast phase of recovery seen within few days to a week of the surgery. In a few individuals, there can be complete normalization of the visual fields. Stage two is the phase of slow recovery which is seen within a few weeks of the surgery to a few months. During this stage, the visual fields show significant and presumably slow and sustained improvement. Stage three is the late phase starting a few months after decompression to a few years. During this stage, there is minimal improvement of the visual fields. Some studies have identified improvement as long as 5 years following surgical resection. Poor prognostic signs for improvement of visual fields include dense and extensive preoperative visual field deficit,[7] pituitary tumor volume greater than 5 cc[8] and the postoperative development of a surgically "empty sella"[9]

Treatment of choice must be individualized and is dictated by the type of tumor, the nature of the excessive hormonal expression, and whether or not the tumour extends into the brain around the pituitary. [10,11]

Standard treatments for patients with pituitary tumors include:

- Surgery
- Radiation therapy
- Medical therapy
- A combination of surgery, radiation therapy and medical therapy

Transphenoidal microsurgical approach to a pituitary lesions and represents a major development in the safe surgical treatment. Progressive deterioration of visual fields is often the primary neurological criterion on which surgical management is based. [12]

Conventional radiation therapy is an effective adjunct to the treatment of pituitary tumours. [13]

Most prolactinomas respond to medical therapy. Surgery should be considered if medical therapy cannot be tolerated or if it fails to reduce prolactin levels, restore normal

reproduction and pituitary function, and reduce tumor size. If medical therapy is only partially successful, this therapy should continue, possibly combined with surgery or radiation treatment

Cabergoline commonly prescribed drug for treating prolactinoma is an ergot derivative, a potent dopamine receptor agonist on D_2receptors. Rat studies show cabergoline has a direct inhibitory effect on pituitary lactotroph (prolactin) cells.It is frequently used as a first-line agent in the management of prolactinomas due to its higher affinity for D_2 receptor sites, less severe side effects, and more convenient dosing schedule than the older bromocriptine.

The goal of treatment in prolactinomas is to return prolactin levels to normal, reduce tumor size, correcting any visual abnormalities, and restoring normal pituitary function. Careful monitoring of clinical signs and symptoms, coupled with pituitary imaging and with serial measurements of serum hormone levels remains the cornerstone of follow up for these patients. [14]

Clinical Course

A young 37 year old female presented to our OPD with loss of left eye vision. BCVA was RE 6/6, LE 6/60. Anterior segment was normal right eye; LE ha RAPD 0.3 log units. Fundus with 90D was normal in right eye; left eye had temporal pallor of disc (Figure1). Confrontation test revealed field loss on inferotemporal quadrant each eye. HFA 30-2 revealed bitemporal field loss (Figure 2). Lesion involving optic chiasm was suspected and MRI brain was requested.

The MRI report showed evidence of well defined 4.23(HF) x 2.87(RL) x 2.64(AP) cms round to oval T2 isointense & T1 iso to hyperintense lesion noted arrising from sellar region (Figure 3). The lesion was extending out of the sella extending anteromedially towards the left orbit causing compression of left optic chiasma & left optic nerve. It was encasing left carotid artery on left side. Post contrast lesion showed intense heterogenous enhancement**.**

After the MRI revealed a pituitary macroadenoma, the patient was referred to an endocrinologist & neurologist to salvage her vision. Patient's history of weight gain, prolonged amenorrhea of 1 year was elicited and her serum hormonal levels showed prolactin levels raised i.e; 250 ng/ml (4-23 ng/ml) suggestive of a prolactin secreting pituitary macroadenoma. Patient was provisionally put on medical treatment of tablet cabergoline 0.5 mg twice a week for 2 months. The patient is yet to follow up to our OPD after 3 months. After starting the treatment patient's regular menstrual cycle has resumed. A repeat visual fields was done for monitoring the treatment (Figure 4) and patient was advised to follow up every 2-3 months.

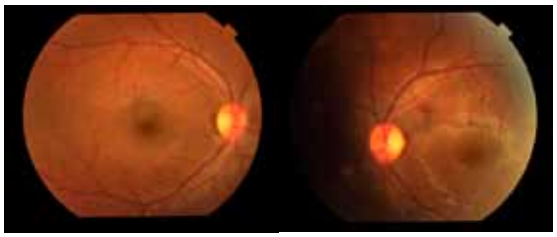

Figure 1. Fundus photographs of the two eyes

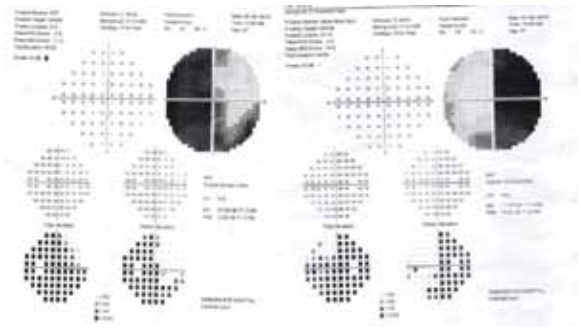

Figure 2. HFA 30-2 of the two eyes

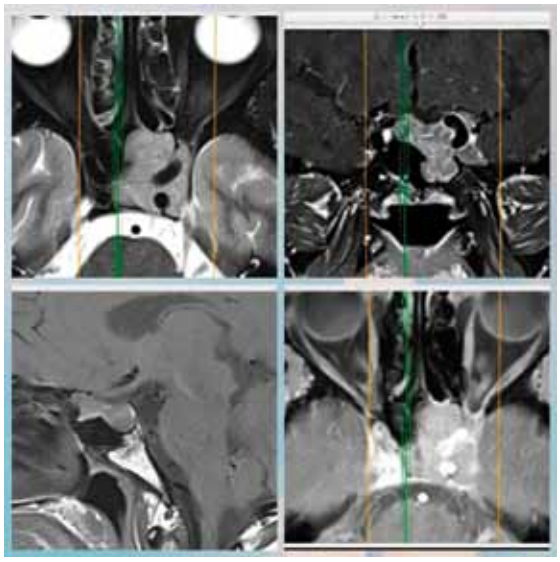

Figure 3. MRI-Brain & Orbit

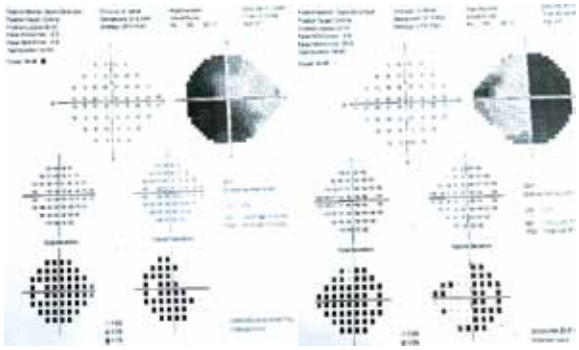

Figure 4. Follow up HFA 30-2 after 3 months

Conclusion: Commonly presenting symptoms to an ophthalmology OPD is visual loss. Visual acuity recording along with a routine confrontation test is a simple and yet a very important test that could give a clue to get the visual field testing done in such patients.

90 D examination in this patient at the slit lamp prior to dilatation helped us to assess the patient's optic nerves.

Temporal pallor in left eye prompted us to do a visual field test.

The patient's left optic nerve and chiasma were most affected evidenced by the visual acuity loss in OS and temporal field defect with preservation of visual acuity in OD. Neurological fields showing bitemporal hemianopia is a classical feature of optic chiasmal compression.

MRI Scan is a superior diagnostic modality than a CT Scan in detecting sellar and suprasellar lesions. Endocrinologist and neurologist opinion is valuable and of utmost urgency in a case of sellar or suprasellar lesions.

Visual fields should be monitored periodically (1–3 months or more frequently) based on the clinical presentation, type of intervention (surgical, medical, or radiation), lesion characteristics and visual complaints of patient. Pituitary imaging and serial measurements of serum hormone levels is the mainstay in treating these patients.

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Lens Induced Glaucoma in Oculocutaneous Albinism with Foveal Hypoplasia

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Abstract: We discuss a rare case with oculocutaneous albinism, foveal hypoplasia, nystagmus and presenile cataracts which presented as lens induced glaucoma which is not reported in the literature so far.

Introduction : The term oculocutaneous albinism (OCA) describes a group of inherited disorders of melanin biosynthesis that exhibits congenital hypopigmentation of ocular and cutaneous tissues. The clinical spectrum of OCA ranges from a complete lack of melanin pigmentation to mildly hypo pigmented forms. OCA1A is the most severe type with a complete lack of melanin production throughout life; the milder forms OCA1B, OCA2, OCA3 and OCA4 show some pigment accumulation over time.[1]

Reduction of melanin in the eyes result in reduced visual acuity caused by foveal hypoplasia and misrouting of the optic nerve fibres.[2]

Presenile cataracts are usually seen in patients less than fifty years of age[3,4.] The primary morbidity of both oculocutaneous albinism and ocular albinism is eye related. Signs and symptoms include photophobia, refractive errors, color vision impairment , strabismus, pendularnystagmus, iris transillumination defects, foveal hypoplasia, reduced pigmentation of the retinal pigment epithelium and abnormal decussation of the optic nerve fibres. However, the degree of their presentation can vary depending on the type of albinism and the racial background of the patient.

Oculocutaneous albinism is of two types, namely tyrosine negative and tyrosine positive. Foveal hypoplasia is typically associated with *PAX6* mutations, albinismand retinopathy of prematurity, whereas atypical forms of foveal hypoplasia have been associated with achromatopsia.

Etiology : Oculocutaneous albinism is a group of autosomal recessive conditions associated with mutations in several enzymes or membrane proteins that contribute to melanin synthesis. The phenotype arises from a problem of melanocyte differentiation that renders synthesis or transport of melanin dysfunctional within the cells. Because the color of both skin and iris are determined by the size and number of melanosomes (organelles that contain melanin), any defect in melanin production or transport of melanosomes (intracellular vesicles that store melanin) affects skin and iris pigmentation. The phenotype in albinism depends on whether or not residual enzyme activity is present. Reduced tyrosinase activity will confer the ability to acquire pigmentation in skin, hair (white hair at birth may become blonde or reddish brown, and skin may tan), whereas patients having no tyrosinase activity will have white hair and white skin throughout their lifetime. Similarly, iris color tends to fall on the spectrum from very light blue to light hazel. The severity of a patient's visual function is often dependent on the degree of nystagmus and amount of pigmentation; hence, patients with no tyrosinase activity (Tyrosinase-negative Oculocutaneous albinism) have poor visual outcome. Ocular albinism is distinguished from oculocutaneous albinism by less of skin and hair involvement, an X-linked recessive inheritance pattern, and the mutation in a distinct loci (*GPR143*) whose gene product is required for melanosomal maturation.

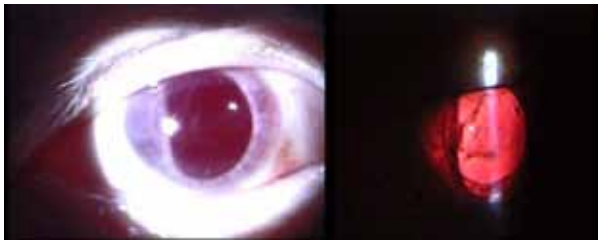
Differential diagnosis of OCA includes ocular albinism, Hermansky-Pudlak syndrome, Chediak-Higashi syndrome, Griscelli syndrome, and Waardenburg syndrome type II.[5]

Case Report: A 40 year old ladypresented with sudden onset ofpain, redness and watering of right eye for 7 days. History of photophobia&diminished vision &nystagmus since childhood was present. No history of bleeding diathesis, recurrent infections or breathlessness.She was the offspring of a consanguineous marriage; there was no relevant past and family history.

Right eye VA was light perception only; left eye had CF 3M. Intra ocular pressure (IOP) was 40.2mmHg with 10 gms; 17.3 mm Hg with 5.5 gm in left eye .Colour vision was defective in both eyes.

There was poliosis, lids were oedematous; horizontal nystagmus was present in both eyes.slit lamp examination showed hazy cornea, shallow anterior chamber, mid-dilated pupil with sluggish reaction to light stimulus. Iris showed transillumination(Figures 1a & b) and lens was mature cataractous(Figure 2) .Fundus examination showed bilateral fovealhypolplasia (Figure 3).OCT showed blunting of foveal reflex and RPE changes (Figure 4).

Left eye findings were unremarkable except for a cataractous lens.



Figures 1a & b: Iris transilluminationpositive (postoperative photograph)

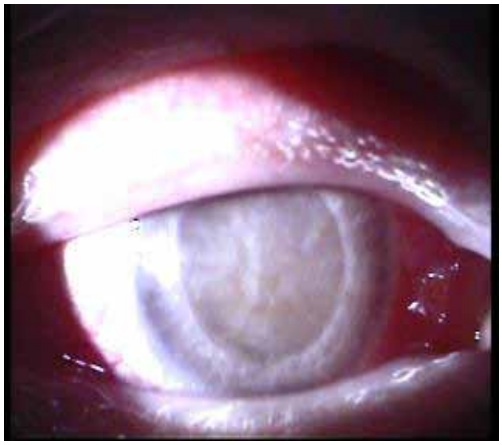


Figure 2: Mature cataract (preoperative)

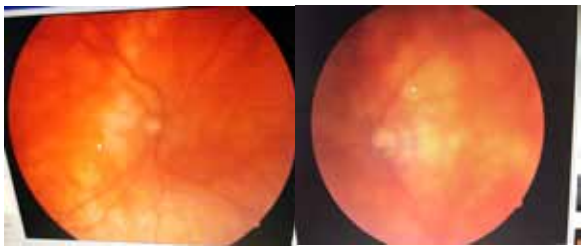


Figure 3: Foveal hypoplasia with albinotic fundus

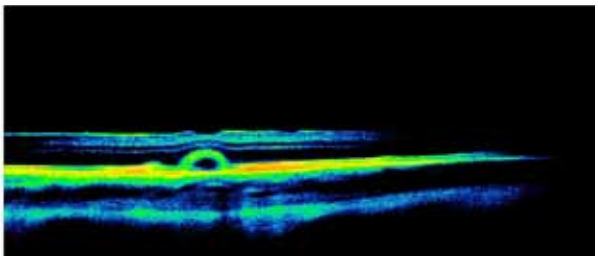


Figure 4: OCT

Skin Generalized depigmentation of the body with diffuse leucotrichia (Figure 5).

All othersystemic examinationand investigations were normal.

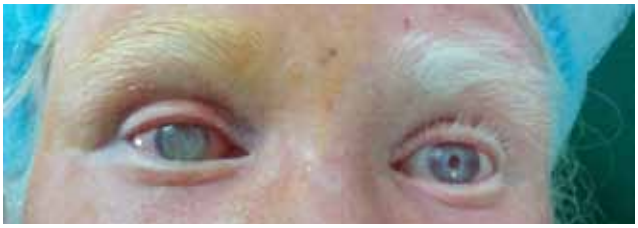


Figure 5: Generalized depigmentation of skin

Systemic examination was normal.

Patient underwent cataract surgery with posterior chamber IOL implantation successfully. Her postoperative visual acuity is 6/24.

Conclusion : We present for the first time inliterature Oculocutaneous Albinism which presented with cataract and lensinduced glaucoma.

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Ocular Tuberculosis

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The term ocular tuberculosis is described as an infection by the mycobacterium tuberculosis species that can affect any part of the eye with or without systemic involvement.

Epidemiology: Ocular TB may not be associated with clinical evidence of pulmonary TB, up to 60% of patients with extra pulmonary TB, may not have diagnosed pulmonary TB.Ocular TB may be an initial presentation of extra pulmonary dissemination of infection. Incidence of TB uveitis is 0.6 – 1% in India. Incidence of ophthalmic manifestations in patients know to have systemic TB is 1 – 2%. The average prevalence of all forms of TB in India is 5.05 per thousand. Although ocular TB was initially recognized by **Maitrejan** in 1740,the credit for the firstauthentic description of disease went to **Jayer** in 1855.

Pathophysiology: It's a granulomatous inflammation with caseating necrosis.

Ocular Manifestations

Lupus vulgaris: Most common form of cutaneous TB is reddish brown nodules, an apple jelly colour, which blanch onpalpation. Usually in children.

Conjunctival tubercles, episcleritis&scleritis occur rarely.

Phlyctenulosis: Due to delayed hypersensitivity reaction to mycobacterium antigens. Othermanifestationsare sectoralintravitreal keratitis, tubercularpannus.

Uvea

Anterior Uveitis: Unilateral or bilateral,granulomatousor non-granulomatous, mutton fat KPs, iris nodules (koeppes nodules) near pupillary border or on iris surface. Broad posterior synechiae, often accompanied by vitreits.

Intermediate Uveitis: Chronic low grade vitritis, bilateral, sometimes associated with discrete perivascular choroiditis may indicate TB etiology. Most frequent complication with TB uveitis is CME, Cataract.

Posterior Uveitis: Unilateral or bilateral, it it is the most common presentation of intra ocular TB, with lesions predominantly present in the choroid. Tubercles are the most well recognized manifestations. Others are serpiginouslike choroiditis and retinal vasculitis. These are the most common clinical presentation of TB posterior uveitis in north India.

ChoroidalTubercles: These are multiple greyish white-yellowlesions with indistinct borders, elevated centrally, usuallylocalizedtoposteriorpole(Figures1-4).Inflammatory cells may be present in AC&vitreous. Sub retinal fluid may be present. Untreated choroidaltubercles may grow into a largetumor like mass called tuberculoma. It is a yellowish, elevated mass- like lesion mimicking an abscess, that is sub retinal with surrounding exudative retinal detachment. Choroidal tubercles are localized in the choroid, but may rarely rupture the Bruch'smembrane and invade the sub retinal space and vitreous causing widespread intraocular inflammation necessitating vitrectomy; alternatively, these may extend to sclera causing scleral granulomas (Figure 5). B scan features are unremarkable (Figure 6). On FFA-hypo florescence in early & hyper florescence in late phases (Figures 7, 8).

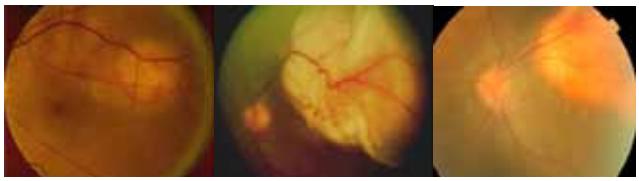


Figure 1: Left eye fundus photograph of a 32-year-old male with choroidalgranuloma (A) at presentation (B) non-responding to first line antitubercular therapy at 2 months; (C) found to be multi drug resistant TB, treated with 7 drug second line anti TB therapy with good response.

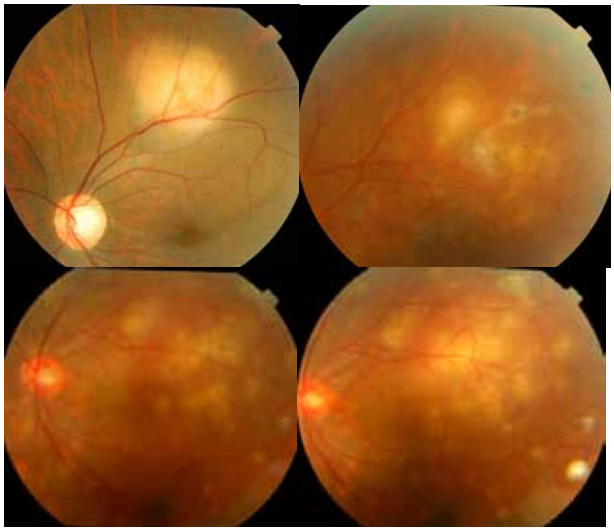


Figure 2: Tubercular choroidal lesions.

Figure 3: Tubercular choroidal lesions at macula and inferior midperiphery. New lesions continue to appear even 6 weeks after starting antitubercular therapy, and even while the initial lesions become inactive.

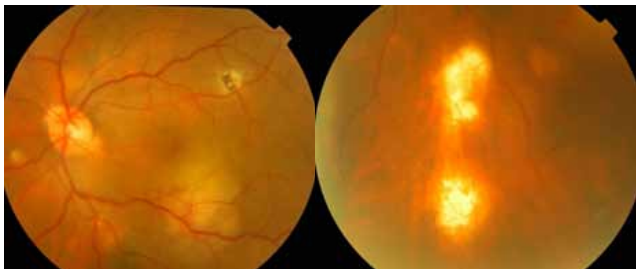


Figure 4: Tubercular choroidal lesions; active paracentral lesion, healed inferior mid-peripheral lesions.

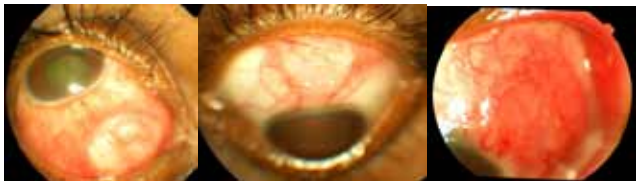


Figure 5: Tubercular scleral granulomas from external extension of untreated choroidal granulomas.

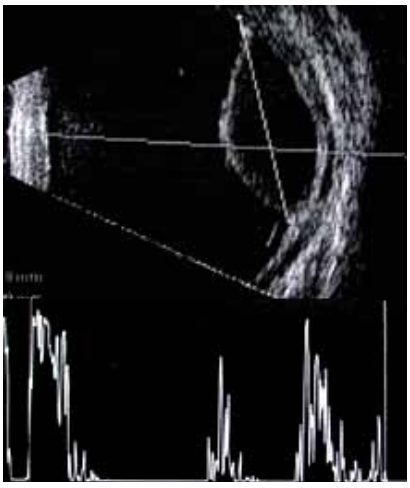


Figure 6: Ultrasound of tubercular choroidal granuloma.

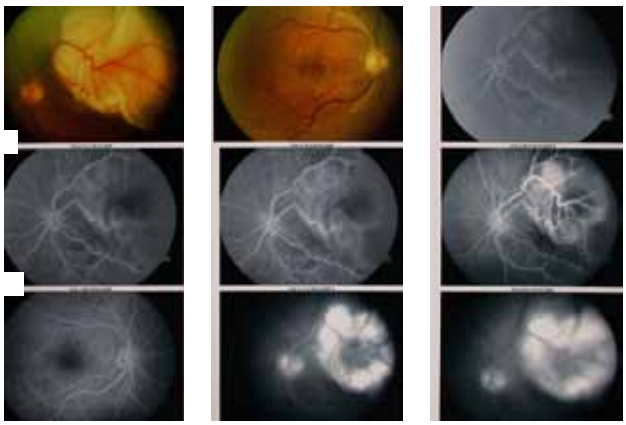
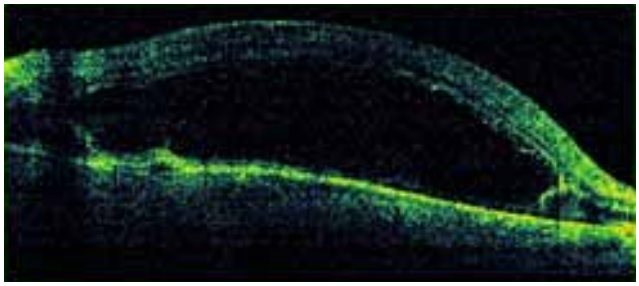


Figure 7: Fundus fluorescein angiogram of tubercular choroidal granuloma. The lesion is hypo fluorescent in early and hyperfluorescent in late phases.



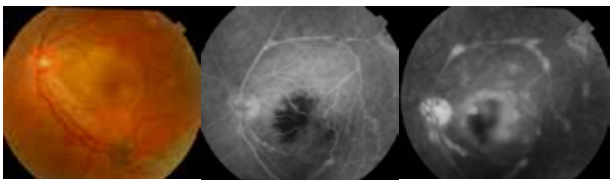
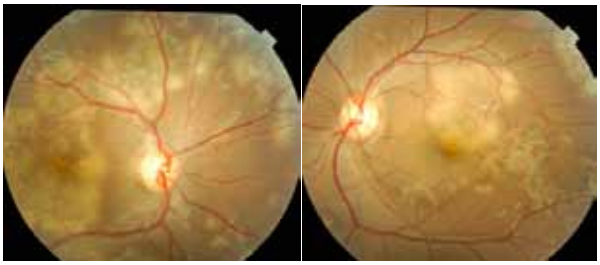


Figure 8: Tubercular retinitis-vasculitis with macular detachment. FFA of same eye.

Serpiginous like choroiditis: - Two types of are seen: 1. multifocal , discrete choroiditislesions that are initially noncontiguous later progress to form diffuse lesions with an active edge resembling serpiginouschoroiditis (Figure 9). 2. Less commonly a solitary diffusive plaque like lesion with an amoeboid extension maybe seen (Figure 10).



Figures 9&10: Tuberculosis-associated immune-mediated choroiditis different patterns

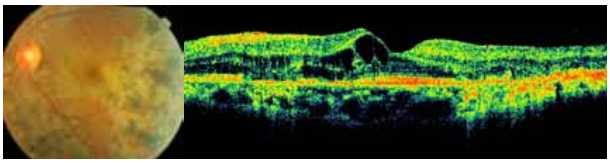


Figure 11: Tuberculosis-associated immune-mediated choroiditis.

The lesions heal with significant scarring. Despite macular involvement fovea tends to be spared, often leading to a good final visual acuity.

FFA helps indifferentiating active from inactive lesion: the active edge ishypo fluorescent and shows hyper fluorescence in the late phase.ICG-the active lesions appear hypo fluorescent in early as well as late phases. Fundus auto fluorescence pattern reveals characteristic changes as the lesions evolve from an initial acute stage to healed stage. OCT reveals progressive changing pattern. In acute lesions a localized fuzzy area of hyperreflectivity is seen in outer retinal layers involving RPE to ONL.As the lesions start healing OCT shows irregular hyperreflective elevation of outer retinal layers with loss of RPE to ELM.

Retinal vasculitis: Inflammation of retinal vessels is a well known association of systemic TB,more commonly veins than arteries (Figure 12). In eyes with evidence of intraocular TB (+vePCR) active vasculitis was seen as severe perivascular cuffing infiltrates, retinalhemorrhages, moderate vitritis, snow ball opacities,neuroretinits and focal choroiditis. On FFA extensive peripheral capillary non-perfusion areas that lead to retinal/opticdisc neovascularization characterize TB retinal vasculitis.

Immune recovery uveitis can occur among patients with HIV and TB infection during antiretroviral therapy.

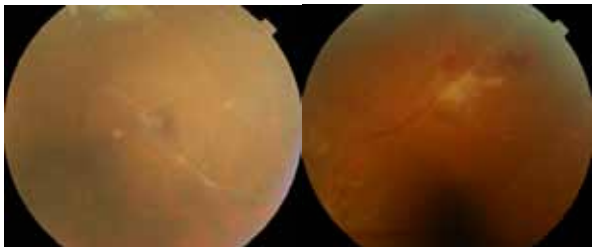


Figure 12: Tubercular retinal vasculitis.

Retina: TB involvement of retina alone is uncommon. Retinochoroiditis and subretinal abscess can occur. Exudative retinal hemorrhagicperiphlebitis in association with uveitis is highly suggestive of TB etiology.

Scleral granulomas, Orbit and lacrimal gland involvement is rare.

Diagnostic TestsMicrobiological and histological evidence of MTB from intraocular fluids or tissue constitutes the gold standard for diagnosing ocular TB. However, the diagnosis of intraocular TB remains largely presumptive as ocular tissue is rarely sampled.

DIRECT EVIDENCE: Evidence of bacilli by ziehl-neelson staining or culture form ocular fluids (Figure 13) or a positive PCR (Figure 14).

Microscopic and histopathological examination: Demonstration of AFB on direct smear or culture of MTB from ocular samples constitutes a definitive diagnosis.

PCR: It's an insertion sequence to detect MTB in peripheral blood, intraocular fluids and tissues.

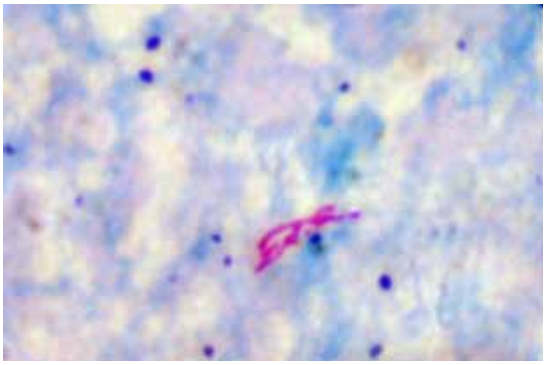


Figure 13: Tubercular acid fast bacilli on Ziehlneelson staining

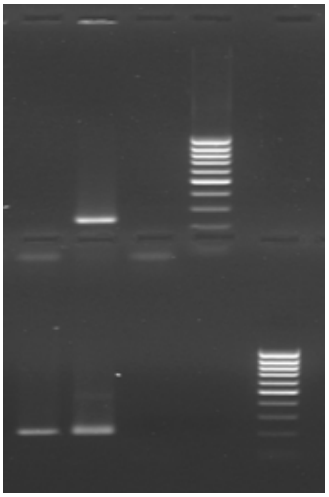


Figure 14: Nested PCR for M. tuberculosis.

Chest X-ray and CT: ChestX-ray for suspected intraocular TB and CT for active pulmonary TB.

PET/CT: Lesionsappropriate for biopsy.

Indirect Evidence:

1 **TST/PPD/ Mantoux test:**It is to detect latent TB,that is a person who is infected with MTB but has no active TB disease.

2 **IGRAS:**It is an immunological diagnostic test such as Quantiferon-TB Gold and Elispot plus.It is a blood test that quantifies the IFNG response of T cells after invitro stimulation of patient lymphocytes by MTB antigen.

FFA, ICG: Can aid in the diagnosis of ocular tubercles. Activechoroidaltubercles, SLC, retinalvasculitis.

OCT: For Tuberculouschoroidal granuloma (Contact Sign) and for cystoid macular edema.

U/S B scan: To differentiate tuberculoma from tumors.

Summary for Diagnosis: The current criteria of making a presumptive diagnosis of intraocular TB include presence of suggestive clinical symptoms and signs, corroborative evidence such as positive TST, positive IGRAS, Chest X-ray, exclusion of known non-tubercular uveitic entities and a positive response to conventional antitubercular therapy.

Differential Diagnoses:

1. Infectious (Toxoplasmosis, Syphilis, Leprosy, Leptospirosis, Brucellosis)
2. Non-Infectious (Sarcoidosis, Auto immune Vasculitis, Metastasis)

Management:

TB drugs can treat latent TB and decrease a person's life time risk of developing active TB by 90%.

Standard ATT: 6 months based on WHO recommendation. First 8 weeks Rifampicin, INH, Ethambutol, Pyrazinamide. Next 18 weeks Rifampicin, INH.

Dose: INH- 5mg/Kg/day, Rifampicin 450 mg/day if BW<50 Kg, 600mg/day if BW>50 Kg, Ethambutol 15mg/Kg/day and Pyrazinamide 25-30mg/Kg/day.

INH, Pyrazinamde and rifampicin cause hepatotoxicity, liver function test to be monitored.

Patients receiving Pyrazinamide should undergo baseline and periodic serum uric acid levels .

Ethambutol toxicity can cause optic neuropathy; those on Ethambutol should be cautioned about reporting vision drop and undergo baseline and periodic visual acuity, red green color perception.

Steroids:May limit damage from delayed hyper sensitivity given 6-12 weeks with ATT therapy.

Topicalsteroid therapy is for 6-12 weeks (Prednisolone Acetate 1% e/d). Periocular (+/- Systemic cortico steroids) for anterior and intermediate uveitis.

Oral/Intravenous steroids for posterior and pan uveitis. Oral steroids dose is 1 mg/KgBW.

Periocular therapy for severe anterior or intermediate uveitis and/or uveitic macular edema. Sub tenontriamcinolone acetonideinjection of 20-40 mg (0.5-1 ml) is given. Duration of effect maybe about 2-3 weeks; can be repeated a few times. Complications include scleral necrosis, cataract, raised IOP, bagginess of lids.

Intravitrealtherapy with triamcinolone acetonide1-4 mg (0.025-0.1 ml) works for 3-6 months and can be used in intermediate, posterior and pan uveitis. Complications include scleral necrosis, cataract, raised IOP, bagginess of lids.

Anti VEGF: Should not be considered as first line of therapy for uveitic macular edema.

For retinal Vasculitis, laser photocoagulation can be done and for orbital TB surgical intervention for diagnostic and therapeutic purpose.

Rifabutin Uveitis: Rifabutin is used for treatment & prophylaxis of mycobacterium in HIV patients and can cause uveitis.

Prognosis : With appropriate diagnosis and treatment, prognosis is often good.

Conclusion: Intraocular TB can occur in absence of pulmonary disease and the patients may present with wide variety of clinical entities. Internist or Infectious Disease specialist help is taken for ATT management. Since the disease is treatable and eyes can be saved using ATT if detected early considerable stress should be laid on its early diagnosis and treatment to prevent ocular morbidity and blindness.

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MODERATORS Dr Jadav Pandurang Dr Nanda Kishore

9.30 a.m - 10.00 am	Inaugaration	by the Chief Guest Dr Manoj Mathur
10.00 am - 10.30 am	Bacterial Infection	Dr Prashant Garg
10.30 am - 11.00 am	Viral Infections	Dr Praveen Krishna
11.00 am - 11.30 am	Fungal Infections	Dr K.Madhukar Reddy

TEA BREAK 11.30 - 11.45

INFECTIONS RELATED TO CATARACT 11.45 a.m - 1.15 pm

MODERATORS Dr Manoj Mathur Dr Vijay Kumar

11.45 a.m - 12.00 pm	Pre Op Preparation	Dr V. Sree Kumar
12.00 p.m - 12.15 pm	Lid & Adnexal Infections	Dr A. Ravindra
12.15 pm - 12.45 pm	Post Surgical Endophalmitis	Dr Ravi Kumar Reddy
12.45 pm - 1.15 pm	Infection control practices in Ophthalmic Operating Room	Dr Joveeta Joseph

LUNCH 1.15 PM - 2.00 PM

ENDOPHTHALMITIS 2.00 p.m - 3.30 pm

MODERATORS Dr Hari Kishan.G Dr Badrinath

2.00 pm - 2.30 pm	Microbiological diagnosis of eye infections	Dr Joveeta Joseph
2.30 pm - 3.00 pm	TASS & Cluster Endophalmitis	Dr V.Raja Lingam
3.00 pm - 3.30 pm	Management of Endophalmitis	Dr Mallika Goyal

TEA BREAK - 3.30 PM - 3.45 PM

QUIZ.

3.45 pm - 4.45 pm	MODERATORS	Dr A. Ravindra Dr V. Sree Kumar Dr R. Arvind

4.45 pm - 5.00 pm VALEDICTORY FUNCTION

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TELANGANA OPHTHALMOLOGICAL SOCIETY

ORUGALLU EYECON 2017

3rd TOS ANNUAL CONFERENCE

21st, 22nd & 23rd JULY 2017

HIGHLIGHTS:

- LIVE SURGERIES
- NATIONAL & INTERNATIONAL GUEST SPEAKERS
- P.G QUIZ SESSION
- BREAKFAST SYMPOSIA
- COMPETITIVE SESSIONS
- INTERACTIVE SESSIONS
- HANDS ON WORKSHOP FOR PG'S

THEME:

SIGHT - IN SIGHT 2017

VENUE: HOTEL VAISHNAVI GRAND, FATIMA COMPLEX, KAZIPET, WARANGAL, TELANGANA.

HOST: WARANGAL OPHTHALMOLOGICAL ASSOCATION

CHAIRMAN
DR.JADAV PANDURANG
SUPERINTENDENT REH, WARANGAL.
7799822292

ORG.SECRETARY
DR.G.R. BHARATH KUMAR
ASST. PROF. REH, WARANGAL
9849191090

ADDL. ORG. SECRETARY
DR.V. SREE KUMAR
ASST. PROF. REH, WARANGAL
7989254646

TREASURER
DR.K.HARIDEV KUMAR
ASST. PROF. REH, WARANGAL
9704115725

CONFERENCE SECRETARIAT :
DR.G.R. BHARATH KUMAR
Regional Eye Hospital, Warangal.
E-MAIL- drbhasha@yahoo.com 9849191090

WORLD DIABETES WEEK(14-21st Nov)

DIABETES -GET SCREENED FOR DIABETIC RETINOPATHY- FREE

AN IMA-AIOS PROJECT

Organised By

Telangana Ophthalmological Society & IMA Warangal

WORLD DIABETES WEEK

Diabetic Retinopathy Screening Camp at Kakatiya Eye Hospital,
Warangal on 17th November, 2016.
Under aegis of Diabetes Blindness Prevention Committee-AIOS chaired by Dr NSD Raju.
Patients were screened for Diabetic Retinopathy
Warangal IMA President Dr T Ravi and other office bearers like
Dr Mahender Reddy also participated in the camp.
Dr. A Ravindra, Dr. P Praveen, Dr Neeraja Praveen,
Dr. T. Ravi, Dr. Mahender Reddy are in the photographs.

APPLICATION FOR LIFE MEMBERSHIP OF TELANGANA OPHTHALMOLOGCAL SOCIETY, HYDERABAD.

PHOTO OF MEMBER

Applied For Life Member ☐ Member in waiting ☐

Existing APOS members need not apply.

Name (In Block Letters) .....

Father's / Husband's Name : .....

Age : ..... Sex : ..... Date of Birth .....

Native District .....

Address (Present) .....

.....

.....

Address (Permanent) .....

.....

.....

Mobile : ..... E-mail: .....

Designation : .....

Academic Qualification : .....

MBBS Year : PG DO. MS DNB Year (For Life Member)

Joined PG in Ophthalmology Year : (for member in - waiting)

Note : Existing APOS members need not apply.

Date : ..... Signature of the candidate

Membership Fee : Rs. 2,000 for Practitioners
Rs. 1,500 for PGS

DD/At Par Cheque No. :

Remarks of Secretary :

DD/Cheque in favour of "**TELANGANA OPHTHALMOLOGICAL SOCIETY**" Payable at Hyderabad.

Kindly send the completed forms to : **Dr. A. RAVINDRA,** Teja Eye Hospital, H.No. 6-2-58, Kakaji Colony, Hanamkonda- 506001. Cell: No. 98664 26367

TELANGANA OPHTHALMOLOGICAL SOCIETY

HYDERABAD.

Mail the duly filled in Registration Form along with your cheque/DD to:

Dr A RAVINDRA
Teja Eye Hospital,
H.No. 6-2-58,
Kakaji Colony, Hanamkonda- 506001
Cell: No. 98664 26367

Second Annual TOS Conference

@ HICC, Hyderabad, August 5-7, 2016

CIPLA ADVT
(to come)