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Solving the Genetic Puzzle of AMD

Our vistas and options for treating AMD patients have vastly improved in the past few years. The advent of photodynamic therapy, the ready availability of anti-VEGF and the success of combination therapy are all options to deal with various manifestations of this visually debilitating disease. However none of these therapies have any role to play in altering the basic pathogenesis of this condition.

Brilliant research papers published in the last few years linking immuno biology with AMD have opened an exciting period of research linking the complement proteins, genetics and clinical symptom of AMD.

Ironically the connection between the body's immune system and AMD has been staring back at ophthalmologists each time a clinician examines the fundus of an AMD patient. The earliest finding in AMD- a drusen; has components suggesting that it is the product of a localized inflammatory response and it is here that the recent genetic susceptibility data fit into the overall clinical pictures of AMD.

It has been firmly established that the alternative complement system is a critical player that may help scientists to join the dots between the drusen and symptomatic degeneration of the macula.

In 2005 the factor H gene (known as CFH or HFI) located on human chromosome Iq31 was identified as a major risk factor for AMD. The specific mutation on the CFH allele that predisposes individual to AMD causes an amino acid change (Y 402 H) from tyrosine to histidine on the CFH protein. Several strands of evidence support the correlation between the Y402 H change in CFH and the development of AMD. First, the polymorphism exists within a chromosomal region long suspected of involvement in AMD. Second the CFH gene codes for a complement protein that has been identified in the drusen of AMD patients. The original CFH finding was followed by reports of a locus on 10q 26, other complement players like B (CFBF) and complement component 2 (C2). CFH, BF and 10q26 locus represents the 3 major loci in the human genome that predisposes individuals to AMD.

The variant that has attracted most attention is the Y402 H variant in the CFH locus which has been associated with an odds ratio of 2.45 to 3.33 for all stages of AMD. If you have a homozygous TT in that locus, it means that you have the amino acid tyrosine (Y) in that position on the protein and you are more protected from AMD. Conversely if you have the risk allele, the codon change causes the insertion of amino acid histidine in that position and your risk becomes higher for acquiring the disease later in life.
The same is true for factor B. Analysis of CFH and BF loci showed that in 74% of AMD patients there is at least one risk allele in CFH and/or BF and no protective alleles while in 56% of control subjects there was at least one protective allele in either locus. In other words these 2 loci alone explain up to three quarters of AMD.

So what about diagnostic screening? A population screen conducted at the University of Columbia collected data from approximately 350 controls (disease free individuals > 65 years), 300 early stages and 350 late stage AMD patients. Examination of the variation in the CFH and 10q loci showed that if you are a double homozygous for the major risk allele in these 2 loci you will invariably develop AMD and will most likely develop the late-stage pathology. A fairly accurate prediction of the risk of developing AMD is possible only in a small percentage and further studies are required to develop a more precise diagnostic screen.

Given the very strong correlation between certain gene variants and predispositions to AMD, it should be reasonable to advise anyone carrying the specific risk allele to avoid environmental risk factors such as smoking. So should people who are heavy smokers undergo a genetic screen to determine their susceptibility status to AMD? Although genetic risk may explain 75% of AMDs, modifiable exposures such as smoking and lifestyle may interact with genetic risk and increase the susceptibility to AMD. (Smoking + CFH: 34 fold risk; RPE/ESR inflammatory markers + CFH: 20-28 fold risk; Obesity + CFH: 11 fold risk for AMD).

All this may ultimately and hopefully lead to the development of a nice small molecule that can be easily delivered in to the eye, which can modulate the activity of gene variants in such a way as to tip the balance in favour of dampening down the alternative complement reaction !!! A nice thought to fall back on.

Dr. Meena Chakrabarti MS DO DNB
Editor
Uveitis is a chronic inflammatory disease. The etiopathogenesis of various uveitic conditions are varied. The distinct established entity called uveitis can be further broken up into a myriad subtypes. Several modalities of classifications exist with regard to anatomy, duration, etiology and pathology. However a more crucial differentiation ought to be made between infectious and non-infectious forms as management varies and may even be diametrically opposite. Infectious forms further encompass a spectrum comprising bacterial, spirochaetal, viral, protozoal and fungal diseases.

A description of the clinical characteristics is outlined which will enable the ophthalmologist to adopt a more prudent approach towards the diagnosis. Nevertheless several of the listed features can coexist in the same individual and need to be evaluated in detail.

In a tertiary referral eye care center, uveitis accounted for 1.5% of new cases. Out of 1273 uveitis cases over a three year period at Sankara Nethralaya, anterior uveitis was the most commonly observed [39.28%], followed by posterior uveitis [28.75], intermediate uveitis [17.44%] and panuveitis [14.53%]. The most common cause of posterior uveitis was toxoplasmosis [27.87%]. The incidence of microbiologically proven tubercular uveitis was high as compared to other studies. A few were detected to have intraocular nematodes as the etiology for uveitis.

**Clinical approach of a patient with Uveitis:**

A meticulous examination of a suspected uveitis patient would involve addressing the following issues:

- Establishing a diagnosis of uveitis.
- Determining the visual potential.
- Detecting an existing complication.
- Narrowing down the most likely etiology.
- Confirming the underlying systemic disease.
- Instituting appropriate treatment.

**Diagnosis**

Most of the infectious uveitic conditions have characteristic clinical features which can be diagnostic. One should know the typical signs and symptoms of various infective agents which would help in clinching the diagnosis. Ancillary investigations such as fundus fluorescein angiogram, indocyanine angiography may be helpful in detecting the activity of the lesions such as in choroiditis or ultrasonography in differentiating subretinal abscesses from other mass lesions.

Blood tests, especially, to detect antibodies against the infectious agents such as toxoplasma, toxocara etc. can be very helpful.
Table 1. Classification of Infectious Uveitis

**Anterior Uveitis**
- Granulomatous Uveitis: Tuberculosis, Leprosy, Lyme’s disease
- Non granulomatous Uveitis: Syphilis, Herpes, Toxoplasmosis (spill over from the posterior segment)

**Intermediate Uveitis**
- Tuberculosis
- Toxocariasis
- Lyme’s disease

**Posterior Uveitis**
- Vasculitis: Tuberculosis, Toxoplasmosis, Syphilis, Cytomegalovirus retinitis (CMV Retinitis), Acute retinal necrosis, Rubella
- Vitritis: Toxoplasmosis, Tuberculosis, Syphilis
- Mild Vitritis: Cytomegalovirus (CMV Retinitis)
- No vitritis: Histoplasmosis
- Neuroretinitis: Syphilis, Lyme’s disease, HIV, Cat scratch disease
- Choroiditis and Retinitis: Toxoplasmosis, Tuberculosis, Cytomegalovirus retinitis (CMV), Herpetic uveitis
- PANUVEITIS: Tuberculosis, Syphilis, Leptospirosis, Lyme’s disease, Viral (herpetic)

Table 2. Frequently encountered infections in an immunocompetent patient:
- **Bacterial**: Mycobacterium tuberculosis, Mycobacterium leprae
- **Spirochaetal**: Treponema Pallidum, Lyme’s, Borrelia
- **Viral**: Herpes simplex, Varicella Zoster, Cytomegalovirus, HIV
- **Protozoal**: Toxoplasmosis
- **Intraocular nematodes**: Toxocariasis, Gnathostomiasis
- **Fungi**: Histoplasma Capsulatum, Candida species

When these investigations are non confirmatory, invasive tests such as aqueous tap, vitreous biopsy or fine needle aspiration biopsy can be done. Intraocular fluid can be subjected to special tests such as histopathology or polymerase chain reaction for detecting the genome of the organism.

**PCR in infectious uveitis**

PCR diagnosis renders it possible to detect infectious agents in situations wherein one is confronted with diagnostic dilemmas. It can help in detecting the presence or absence of the genome of various infective agents. Nested PCR is a more sensitive test while RT PCR is a more reliable test to detect the viable organisms in the specimen.

**(A) Bacterial**

1. **Tuberculous Uveitis**

Uveitis correlates with systemic tuberculosis only in 1.39% of patients as per our study at Sankara Nethralaya. It is believed to be predominantly a representative of an immune mediated hypersensitivity reaction in the presence of a few tubercular bacilli in the choroid or retinal pigment epithelial cells, though hematogenous dissemination can occur. Clinical features are granulomatous iridocyclitis, solitary choroidal granuloma, multifocal choroiditis, periphlebitis, and panuveitis. In a study done by Gupta V et al, out of 158 patients of intraocular tuberculosis, the commonest form of intraocular inflammation was posterior uveitis [42%] which was consistent with our study. As high as 52.3% of cases had posterior uveitis as the manifestation of ocular disease.

Tubercular uveitis is the most common tuberculous infection of the eye. The most common presentation of tubercular uveitis is of disseminated tubercular choroiditis (fig. 1) which manifests as choroidal tubercles. The lesions range from 0.5 to 3 mm in diameter and vary in size and elevation. The second most frequently encountered lesion is focal choroiditis (also referred to as solitary granuloma) which occurs predominantly at the posterior pole. The elevated mass may be accompanied by an overlying serous retinal
detachment. A choroidal tubercle may progress to a sub retinal abscess (fig. 2) and may mimic a choroidal amelanotic melanoma. Periphlebitis (fig. 3) with vitreous hemorrhage occurs due to tubercular proteins and causes sudden loss of vision. A serpiginous like pattern of choroiditis is another atypical presentation in the constellation of clinical findings which may be the reason for the delay in the diagnosis. Tuberculous involvement is always associated with vitritis and perivascular cuffing which contrasts with the absence of vitritis in serpiginous choroiditis and may be instrumental in distinguishing the two diseases.

Anterior uveitis is less common and characterized by remission and exacerbation with severe anterior chamber reaction, appearance of nodules on the iris (Busacca nodules and Koepppe nodules) and mutton fat keratic precipitates of varying numbers.

**Investigations:** Complete blood count, Erythrocyte sedimentation rate, Mantoux, X Ray chest, CT chest may prove inconclusive and it has been inferred that polymerase chain reaction by virtue of detection of DNA for mycobacterium tuberculosis and Quantiferon gold tests are diagnostic of the disease.

**Intraocular fluids** such as aqueous or vitreous or a FNAC sample from the abscess itself can be subjected to PCR or histopathology testing when there is a strong clinical suspicion and if non invasive tests are inconclusive.

Newer tests such as QUANTIFERON GOLD TEST can be helpful. It is an in vitro diagnostic aid using peptide mixtures simulating early secretory proteins (antigenic target 6 and culture filtrate protein 10) in heparinized whole blood. This test measures a component of cell mediated immunity and is based on the quantification of interferon gamma released from sensitized whole blood. It detects both active tuberculosis disease and latent tuberculosis infection but however is not interchangeable with tuberculin skin test as they do not measure the same components of the immunologic process.

**Treatment** involves the use of Antituberculous treatment as 4 drugs [isoniazid, rifampicin, pyrazinamide and ethambutol] for an initial 2 months followed by a choice of different options of 2 drugs over the next 4 months according to DOTS (Directly Observed Treatment). Additional anti inflammatory therapy such as topical and systemic corticosteroids along with cycloplegics is required.

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2. **Uveitis in Hansen’s Disease**

The uveal tract involvement is seen commonly in the lepromatous form and its incidence is directly proportional to the disease duration. Early and subtle signs of ciliary body involvement are autonomic dysfunction, including diminished pupillary reactions. Acute iritis may be fulminant and is caused by immune complex deposition in the uvea. Chronic iritis results from direct invasion by bacilli. A pathognomonic sign is the presence at the pupillary margin of small glistening iris pearls, which may enlarge, coalesce and drop into the anterior chamber.

Posterior segment lesions are uncommon as the bacillus has a predilection to lodge itself in the cooler parts of the body.

**Treatment** : Anti leprotic drugs forms the anchor of treatment in association with topical and systemic corticosteroids.
Spirochaetal Uveitis

1. Acquired Syphilis

Uveitis occurs in the secondary and tertiary stages of the disease though it may occur during any stage. Iridocyclitis occurs in about 4% of patients and is bilateral in 50% according to Western studies. The classical presentation of anterior uveitis is the presence of roseolae of iris capillaries, iris atrophy and varying degrees of vitritis.

Posterior uveitis is seen as multifocal chorioretinitis [most common], focal chorioretinitis, neuroretinitis, isolated vasculitis and pan uveitis(fig 4). The fundus in multifocal chorioretinitis displays several active, greyish yellow lesions with a preference for the posterior pole. Intermediate uveitis of Lyme’s disease or sarcoidosis may resemble syphilitic uveitis and serves as a differential diagnosis. Healed lesions assume a salt and pepper retinopathy which resembles retinitis pigmentosa.

Investigations: Diagnostic tests may be specific or non specific.

FTA-ABS is specifically directed against treponemal antigens and becomes positive during the secondary stage remaining so, for a lifetime regardless of the treatment status.

Non-specific tests such as VDRL and Treponema pallidum Immobilisation Test quantify the amount of serum antibodies directed against the antigen.

Treatment : Penicillin in either intravenous or intramuscular forms is administered.

Ocular syphilis is treated like neurosyphilis and recommendation for treatment is as follows.

Intravenous penicillin G 18 to 24 million units daily for 10 to 14 days. Further supplementation is with intramuscular benzathaine penicillin G at a dose of 2.4 million units for 3 weeks. Tetracycline (500 mg) four times daily or Doxycycline (100 mg) twice daily for 14 days is given in patients with penicillin allergy.

2. Lyme’s Disease

Uveitis may take the form of granulomatous iridocyclitis, intermediate uveitis, retinal vasculitis and rarely neuroretinitis. Recommended therapy for early disease consists of tetracycline, penicillin or erythromycin.

3. Leptospirosis

Uveitis herein is underdiagnosed as it occurs several months after the onset of the systemic disease. It can exist as two subtypes:

Acute non granulomatous uveitis which may be associated with a hypopyon.

Posterior uveitis seen as vitritis (vitreal membranes), choroiditis, vasculitis, papillitis and panuveitis.

Investigations: Diagnostic procedures are based on two principles
i) Isolation of the causative organism: ELISA
ii) Isolation of DNA: Microscopic agglutination test (MAT) Polymerase chain reaction (PCR)

Treatment involves administration of oral doxycycline 100 mg two times daily for 14 days. Cephalexin is also used as an alternative.

(C) Protozoal Uveitis

1. Ocular Toxoplasmosis

Toxoplasmosis is an ubiquitous infection with an incidence ranging from 12% - 90%. On the basis of epidemiological studies, most cases of ocular toxoplasmosis are believed to result from congenital infection but may also occur due to infection acquired postnatally. The response to infection correlates with parasitic and retinal antigen levels. Active chorioretinitis is associated with anterior uveitis, which may be granulomatous or non granulomatous. A solitary
inflammatory focus of variable size [focal retinitis], adjacent to an old pigmented scar [satellite lesion] is the most common finding. Severe vitritis may impair visualization of the fundus although the inflammatory focus may still be discernable [Headlight in the fog appearance]. Occasionally vasculitis and papillitis may be seen.

A yellow white or greyish lesion is seen in the posterior pole involving the macula in a vast majority of patients (fig 5). The borders are ill defined with adjacent retinal oedema. A healed scar typically has well defined borders with central chorioretinal atrophy and peripheral pigment epithelial hyperplasia. Active lesions localized to the juxtapapillary region cause a neuroretinitis. Viral necrotizing retinopathy closely mimics toxoplasma infection in immunocompromised patients. In newborns, TORCH group of infections and others such as congenital syphilis are a major source of infection. The important differential diagnosis include other infections such as focal choroiditis due to tuberculosis and non infectious condition like macular coloboma.

Investigations: Diagnosis is based on a compatible fundus lesion and positive serology for toxoplasma antibodies. An antibody titer of raised levels of IgG and IgM are seen. ELISA is a more specific test for detection of antibodies. Polymerase chain reaction is an important tool and using this technique antibodies titres are measured in aqueous humor and serum and Witmer-Goldman coefficient is calculated. Fundus fluorescein angiography and indocyanine green angiography confirm the activity of the lesions and detect complications. Optical coherence tomography can help in detecting complications such as epiretinal membranes, vitreo macular traction, cystoid macular oedema and choroidal neovascularization.

Treatment does not reduce the frequency of recurrences and only limits the size of the scar. The best combination is use of non sulphonamide with a sulphonamide and oral steroids in tapering doses. We commonly use either Clindamycin or Azithromycin with a sulphonamide in combination with systemic corticosteroids for a minimum period of 6 weeks to 3 months based on the response to therapy. It is important to specifically rule out allergy to sulpha drugs before advising those drugs.

The antitoxoplasma agents commonly in use are:

- **Clindamycin** 300 mg 4 times daily orally for a minimum of 6 weeks.
- **Pyrimethamine** 50 mg daily for 6 weeks can be used. However frequent monitoring of blood counts is required. Besides oral folinic acid 4 mg three times daily should be given as supplementation. It is important to check for the tolerability of this drug by the patient. It is known to cause severe nausea, vomiting and other gastrointestinal disturbances.
- **Co-Trimoxazole** 960 mg twice daily can be given alone or in combination.
- **Azithromycin** and **Sulfadiazine** (4 gm daily in divided doses for 6 weeks) are also alternatives.

**D) Nematodes**

### 1. Ocular Toxocariasis

This nematodal infection is seen most commonly as posterior pole granuloma and may be associated with hemorrhage. The lesion simulates a retinoblastoma, sarcoid granuloma, toxoplasmis or focal choroiditis. Long standing masses may have choroidal atrophy, hyperplasia of the retinal pigment epithelium and choroidal neovascular membrane. Various clinical manifestations of the parasite according to decreasing preference are:

Peripheral granuloma, Posterior pole granuloma
Chronic endophthalmitis like picture
Optic nerve head involvement
Anterior segment involvement

**Investigations** reveal an eosinophilia. ELISA detects and evaluates antibodies directed against this organism.

**Treatment** is with Thiabendazole or Diethylcarbamazine. Oral corticosteroids should be used to suppress inflammation.

2. **Intraocular worms – Gnathostomiasis**

Gnathostoma spinigerum is an intestinal nematode. The host for human infections are domestic cats and dogs. Men can acquire the infection by eating raw meat or through skin penetration by the larva during food handling. The most common mode of presentation is anterior uveitis with or without secondary glaucoma. Iris holes may be considered a diagnostic sign. The larvae may migrate into the eye along the optic nerve or directly penetrate the sclera. Once the parasite is removed, inflammation subsides markedly with topical and systemic antibiotics.

(E) **Viral Uveitis**

1. **Herpetic Uveitis**

It is seen to occur commonly in association with active or healed keratitis. Herpetic anterior uveitis presents with fine small keratic precipitates scattered all over the endothelium with a mild anterior chamber reaction. Sectorial iris atrophy due to ischemic vasculitis, blood stained hypopyon and secondary glaucoma are characteristic features.

ACUTE RETINAL NECROSIS is a panuveitis which is caused by herpes simplex virus (1 or 2), varicella zoster virus and also rarely cytomegalovirus.

The characteristic triad includes moderate to severe vitritis, occlusive vasculitis involving both the arteries and veins and peripheral confluent retinal necrosis with scalloped margins.

**Investigations:** Diagnosis is usually clinical due to the typical clinical features but when in doubt, PCR testing for the viruses from the anterior chamber tap can be diagnostic.

**Treatment:** Acyclovir is given intravenously for 14 days. The dose is 750 mg loading dose and 500 mg 8th hourly for 2-3 weeks as a slow intravenous infusion. This is followed by oral acyclovir 800 mg five times daily for 3 to 6 months. Apart from its antiviral effect on the affected eye it reduces the risk of fellow eye involvement.

A newer oral antiviral drug, Valacyclovir [L-valyl ester of acyclovir], has better bioavailability and is used in the doses of 1gm three times a day for 6 to 8 weeks.

Systemic steroids are started a few days after initiation of antiviral therapy. Argon laser photocoagulation is required as prophylactic barrage in areas of potential break formation to prevent risk of RD when inflammation is under control.

(E) **HIV Related Eye Diseases**

The risk of developing atleast one abnormal ocular lesion for a HIV positive ranges from 52-100 %. The frequency of occurrence of opportunistic infections in HIV positive patients in India are: Cytomegalovirus retinitis, toxoplasmosis, tuberculosis, progressive outer retinal necrosis, and acute retinal necrosis due to herpetic viruses, syphilis and pneumocystis carinii. HIV uses a unique viral enzyme, reverse transcriptase to transfer the genetic code from viral RNA to viral DNA. This is then integrated into the host cell DNA. Various drugs used in the treatment of HIV target specific sites in this process. Highly active antiretroviral therapy (HAART) is a combination of any of these agents.

**Cytomegalovirus Retinitis in HIV**

CMV retinitis develops in 15-40 % of HIV positive patients. It is the most common ocular infection in AIDS. It runs parallel to the existing CD4 count wherein less than 50 cells /mm$^3$ is associated with the disease. It is seen as a fulminating retinitis with vasculitis and mild vitritis. The opacification extends alongside the retinal blood vessel in a characteristic “brushfire like” fashion. In the earlier stages the retina shows white granular patches with regular margins and variable overlying haemorrhage. The perivascular distribution gives rise to “Cottage cheese with tomato ketchup” or “pizza pie” appearance (fig 6). Severe vascular sheathing gives rise to “frosted branch angiitis” (fig 7) which is seen in about 6 % of patients. Retinal detachment occurs in 30 % of healed cases.
**Treatment** may be administered individually or in combination.

Ganciclovir is the drug of choice to treat this infection. It is given intravenously as 5 mg/kg every 12 hours for 2 weeks followed by 5 mg/kg once daily as maintenance as slow infusion. Oral drug Valganciclovir, Intravitreal ganciclovir and biodegradable ganciclovir implants are also very effective. Valganciclovir, is a prodrug of ganciclovir and achieves blood levels comparable to intravenous ganciclovir. Induction therapy involves 900 mg twice daily for 21 days followed by 900 mg once daily as maintenance therapy.

**Toxoplasmosis in HIV**: Ocular toxoplasmosis is seen in 1-2% of AIDS patients. Retinochoroiditis lesions are more extensive (fig 8) and multifocal with broad areas of necrosis rendering a hard indurated appearance to the retina. There is more severe visual impairment and serological diagnosis is often difficult due to depressed antibody response. Anterior chamber tap for PCR testing may be helpful.

**Varicella zoster virus in HIV**: Progressive outer retinal necrosis is caused by a variant of VZV and is among the most common opportunistic infections occurring in advanced stages of AIDS. The posterior pole (macula) is involved in the early stages and hence visual prognosis is poor. The outer retinal layers are principally involved with rapid confluence of inflammatory foci leaving large areas of retinal necrosis. The scant or absent involvement of retinal vasculature renders the characteristic “cracked mud” appearance of the fundus. (fig 9)

**Ocular syphilis**

About 1-2% of HIV positive patients are found to have ocular syphilis. Ocular findings include chorioretinitis, optic neuritis, papilloedema. An unusual manifestation of syphilis is acute necrotizing retinopathy and may

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**COMPARISON OF VIRAL RETINITIS**

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<td>HSV, VZV</td>
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<tr>
<td>Presentation</td>
<td>Bilateral (30-50 %)</td>
<td>Bilateral 71%</td>
</tr>
<tr>
<td>Vision</td>
<td>Variable loss depending on the site of involvement</td>
<td>Mild, progressing to severe loss of vision</td>
</tr>
<tr>
<td>Anterior uveitis</td>
<td>Mild non granulomatous</td>
<td>Mild non granulomatous</td>
</tr>
<tr>
<td>Vitreous</td>
<td>No Vitritis to minimal vitritis</td>
<td>Significant vitritis present</td>
</tr>
<tr>
<td>Retinal necrosis</td>
<td>Full thickness-granular border</td>
<td>Full thickness</td>
</tr>
<tr>
<td>Characteristic appearance</td>
<td>Cottage cheese with ketchup or pizza pie</td>
<td>Swiss cheese</td>
</tr>
<tr>
<td>Complications</td>
<td>Hemorrhages</td>
<td>Vasculitis, hemorrhages, RD</td>
</tr>
<tr>
<td>Progression</td>
<td>Slow</td>
<td>Rapid progression</td>
</tr>
</tbody>
</table>
mimic ARN. In HIV positive ocular syphilis a neurological abnormality is found to be more common. The other ocular infections that may coexist in patients with HIV are: Ocular Tuberculosis in AIDS, HIV retinopathy, Atypical Mycobacterial infection, Cryptococcus neoformans, Candida, Molluscum contagiosum and Pneumocystis choroidopathy.

(F) Fungal Eye Diseases

Usually present with endophthalmitis and has to be treated with intravitreal and systemic anti fungi followed by vitreoretinal surgery if not responding.

Conclusion

Crucial facets to be addressed are the complications associated with uveitis such as complicated cataract. Besides, the ideal time to initiate corticosteroid therapy [to suppress associate inflammation] as an adjunct to treatment directed against the infective agent needs to be tailored to the patient's response. The diagnostic procedures and tests are trimmed according to the various suspected infections.

If the etiology remains undetermined: CBC, ESR, Mantoux, VDRL, X-ray chest, Motion for ova/cyst, Urine for albumin/sugar is ordered as part of a routine work up.

Intraocular fluid testing is very helpful in cases of diagnostic dilemma. The ophthalmologist needs to concur with the dermatologist, dentist, physician, rheumatologist and STD clinic. Working in tandem will orient more precise and efficient management.

References

10. David BenEzra Ocular inflammation-Basic and clinical concepts.
11. Foster and Vitale :Diagnosis and treatment of uveitis


An Effective Model for Counseling in Diabetic Patients

Dr. Meena Chakrabarti MS, Dr. Valsa Stephen MS, Dr. Arup Chakrabarti MS, Dr. Sonia Rani John DNB

Recent studies from India report the incidence of diabetes as between 5 % - 10 %. About 25 % of these diabetics will be affected by diabetic retinopathy. Half of them will require intensive follow up, laser treatment, vitreous surgery, and low visual aid rehabilitation. This epidemic increase in prevalence of diabetes is compounded by the fact that this disease can only be controlled and never cured in the life time of the patient. In addition to being a major cause of morbidity from multisystem complications, diabetes is the leading cause of blindness from diabetic retinopathy in the “working age” population.

The real problem lies in the fact that diabetic patients are not aware that diabetes affects the eyes. Physicians and general medical practitioners do not give much importance to this aspect of the disease. Many Ophthalmologists refer cases at a very advanced stage to tertiary centers when nothing much can be done. This is mainly due to the lacunae in the awareness of the available treatment modalities. In addition there is no proven service delivery model for diabetic retinopathy. With the overall aim of controlling diabetes and creating an awareness of its complications we have developed a comprehensive model to screen for diabetic retinopathy.

Materials and Methods

The main objectives of this model are

1. **Eye Health Promotion**
   - To create awareness in the community
   - To create awareness among Ophthalmologists
   - Awareness in all diabetic patients visiting our tertiary eye care centre

2. **Prevention**
   - Develop screening model for diabetic retinopathy in the general population
   - Screen High risk diabetic cases for diabetic retinopathy

3. **Treatment**
   To provide tertiary care in the form of appropriate treatment for diabetic retinopathy patients.
   - FFA and Laser Photocoagulation
   - Vitrectomy
   - Pharmacological therapies

**Rehabilitation**
To provide low vision care using low vision aids for burnt out diabetic retinopathy patients with sub normal vision.

**STAGE I of MODEL**

*Primary Physicians Awareness Programme*

The following were the guidelines given to primary care physicians. They were advised to interact with their diabetic patients and

1. Inform patient about sight threatening complications of diabetes.
2. Educate patients on ophthalmic examination schedule in various type of diabetes.
a. Type I Insulin dependent DM: At least one detailed ophthalmic evaluation (including dilated fundus evaluation) within 5 years of diagnosis

b. Type II Maturity Onset DM: at the time of diagnosis of DM
c. Pregnant Diabetic: Once in every Trimester

3. Create awareness of the results of major multicentric trials on diabetes in their diabetic patients.
   - Glycaemic control and progression: Good and constant glycaemic control can prevent progression of diabetic retinopathy.
   - PDR is an important risk factor for development of myocardial infarction, stroke and amputation.
   - Patients with PDR are at higher risk of developing diabetic nephropathy.
   - Increased blood pressure, anemia (Hb<12g %) elevated lipid and gross proteinuria can accelerate the course of diabetic retinopathy.
   - There is no ocular contraindication to Aspirin therapy when required for cardiovascular diseases.

Primary physician awareness was achieved through
- Seminars and Workshops for Medical Practitioners: 2 per month
- Guest Lectures in clubs and organizations: 1 per month
- Regular Press Meetings: 1 every third month

Brochures depicting stages of Diabetic Retinopathy and treatment modalities were distributed at these meetings after diadic lectures on screening, prevention and management of diabetic retinopathy.

STATE II OF MODEL:

Awareness Programme in Diabetic Patients
- Conducting Diabetes Screening Camps in association with Diabetologists, Indian Diabetic Education Association, Labs, drug companies: 2 per month for the past 3 years (2005 – 2007) = 60 camps conducted over the past 3 years.

o Education of diabetic patients attending our centre (5600 patients in 3 years) using flip charts, educational CD’s and giving educational literature.

o Putting up posters in Malayalam depicting ravages caused by diabetic retinopathy and emphasizing importance of prevention and early treatment at all camp sites.

o Use of Mass Media: TV, Radio, Press.

Model of a Flip Chart: We have made flip charts which lucidly represented the following in the local language
- Magnitude of the problem of diabetes
- Simple representation of multi system complications
- Education on diabetic control
- Signs and symptoms of diabetic retinopathy
- Fundus photographs and angiograms on all stages of diabetic retinopathy
- Education on FFA
- How laser helps in retinopathy
- Scope for surgical treatment and newer pharmacological treatment.
- Expected outcomes of treatment
- Schedule of ocular examination

All diabetic patients who attended our centre were counseled using our Flip Chart.

Stage III of Model: Training of Paramedical Staff to support counseling. The paramedical staff were made to follow a structured 30 days programme where they were taught all the main points to be emphasized during counseling. They attended angiography, laser and had postings in the OT during diabetic vitrectomies to enhance their ability to counsel.

Table 1. References

<table>
<thead>
<tr>
<th>Year</th>
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</tr>
</thead>
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<tr>
<td>2000-2001</td>
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<td>2002-2003</td>
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<td>2003-2004</td>
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<td>2004-2005</td>
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<tr>
<td>2005-2006</td>
<td>725</td>
</tr>
<tr>
<td>2006-2007</td>
<td>1200</td>
</tr>
</tbody>
</table>
To assess the efficacy of this model programme we tried to analyze.

1. The rate of reference of diabetic retinopathy to our tertiary care centre (Table 1).

The number of references to our centre has shown a progressively increasing trend. Analysis of our database showed the effects of the physician awareness programme.

2. Improvement in diabetic control by comparing HbA1C done every 3rd month (in patients who could afford this test) showed that stable diabetic control could be achieved in 78%.

3. Compliance to follow up: The scheduled follow up strategy was discussed with the patients. Analysis of our data on 5600 patients followed up for 3 yrs revealed that we were able to get compliance in 78% of patients who reported regularly at least once in 6 months.

4. Rate of early detection of diabetic retinopathy: We were able to detect early retinopathy in the form of microaneurysms and dot and blot hemorrhages in 10% of the patients who were followed up at our centre. They were again educated on the necessity for strict diabetic control and advised 4 monthly follow up.

5. Progression of existing retinopathy (worsening of the retinopathy status) occurred in 20% of our subjects. These were all patients with preproliferative and proliferative stages of retinopathy at baseline. Depending on the condition they were subjected to either fillin PRP or vitrectomy if there was non resolving hemorrhage. 86 patients with progression of retinopathy underwent vitrectomy.

Sequential scheduled follow up helped in early detection; early initiation of treatment and early stabilization of retinopathy.

This model for counseling diabetic patients can be carried out at all levels of eye care. Efficient and trained paramedicals can act as excellent counselors to the patients.

The results of our study show that this is an excellent model to emulate at other eye care centers. Creating public awareness, screening for early detection; and initiation of treatment at the earliest will go a long way in controlling an epidemic of diabetic retinopathy.

**Discussion**

The WHO has estimated the diabetic population in the world to be about 150 million – 170 million. This population is expected to grow to more than 370 million by the year 2030. While diabetes will continue as a major health problem in the developed world it is estimated that approximately 70% of all new cases will appear in the developing countries.

There is a rising prevalence of diabetes in urban India. Between 1989 and 2004, the prevalence of diabetes increased by 72.3%. The prevalence rate-age standardized for the Chennai census 1991 according to the Cures Study was 14.3%. The diabetic population in India will increase from 19 million in 1995 to 57 million in 2025. The Diabetes Care Asia study, a multi country, multi centre study conducted in 230 centres in 12 South East Asian countries and enrolling 22,000 patients was undertaken to study and compare the diabetes profile and quality of care. It is also noteworthy that patients from India have the lowest mean age of onset of 43.6 years in comparison to other South East Asian countries where the mean age of onset is higher and ranges from 49.6 (Thailand) to 51.5 (Taiwan) in this study.
The reported prevalence of diabetic retinopathy varies in different studies as very few population-based studies had standardized grading and documentation systems. The table given below (Table 3) summarizes the prevalence of diabetic retinopathy reported in various studies.

In the Indian Scenario, JS Jain et al screened a diabetic population attending the diabetic clinic and eye outpatient service of PGI, Chandigarh and reported a prevalence rate of 42.9 %. Two large clinic based Indian studies1,2 have shown prevalence rate of diabetic retinopathy in Type 2 diabetic patients in South India as 34.1 % and 37 % respectively. These studies are biased by the referral of more severe cases to tertiary care centre.

The Andra Pradesh Eye Disease Study (APEDS)3 analyzed the prevalence of diabetic retinopathy in a population of self reported diabetics and reported a prevalence ratio of 26.2 % for diabetic retinopathy. The Chennai Urban Rural Epidemiology Study (Cures I)4,5, a population based survey where 4 field fundus photography was performed on 1382 subjects, reported a prevalence ratio of 17.6 %.

The rising prevalence ratio is attributed to a change in lifestyle due to urbanization, intake of high calorie and high fat diet, decreased physical activity, and an increased longevity. Indians are more prone to diabetes and its attendant complications making India the diabetic capital of the world. Indians have a genetic phenotype characterized by low body mass index, high upper body adiposity, high body fat percentages and a high level of insulin resistance which are risk factors for diabetes.

Considering the large number of diabetic subjects in India6, diabetic retinopathy still poses an enormous public health and economic burden. We need to strengthen our primary healthcare facilities to diagnose and initiate treatment for diabetes and diabetic retinopathy at an early stage.

The DRS and ETDRS have conclusively advocated the role of regular eye examination to ensure early detection and treatment of diabetic retinopathy and thereby prevent severe visual loss. The awareness and adherence to periodic eye checkup is poor even in developed countries. The scenario in developing countries like India is worse. These factors highlight the need for a population based diabetic retinopathy awareness programmes, screening and appropriate counseling. The physicians and ophthalmologists require further awareness and training so that they can educate their own patients. The three level model adopted by our centre was very effective in screening and detecting early cases of diabetic retinopathy. These patients were vigorously counseled and were followed up for 3 years. Any progression in retinopathy could be therefore detected and treated.
References


Evaluation of Interobserver Agreement In Gonioscopy

Dr. Jenny Thomas Jacob DO, Dr. Shima M. MS, Dr. Thomas George T. M.S.

Abstract

Aim: To define the learning curve in gonioscopy.

Materials and Methods: A single blind study using one resident with one year experience in Ophthalmology and another resident with two years experience in ophthalmology and three weeks in Glaucoma speciality clinic was done. Each student independently did gonioscopy on the fellow eye of fifty patients admitted for cataract surgery. Tests of agreement (reliability) was performed and kappa was derived.

Results: Observed agreement for Shaffer's grading: 0.62 (k=0.46), occludability: 0.88 (k=0.45), posteriormost structure: 0.62 (k=0.46).

Keywords: Gonioscopy, interobserver agreement, kappa, glaucoma, Shaffer's grading, occludability, posteriormost structure.

Introduction

Gonioscopy is a clinical technique used to examine structures in the anterior chamber angle, which is essential in the management of different types of glaucoma. But like many other physical findings, radiographic interpretations and other diagnostic tests, gonioscopy too often rely on some degree of subjective interpretation by observers. Thus, like any other diagnostic test, gonioscopy too has its own learning curve effect.

Aims and Objectives

To define the learning curve involved in gonioscopy and to assess the level of agreement between two postgraduate students at different levels of training in Ophthalmology, in the interpretation of gonioscopy findings.
The test of agreement (reliability) was performed and kappa was derived which is the statistical method of analysis adopted in our study.

**Method of Analysis**

**Accuracy versus precision**

When assessing the ability of a test (radiograph, physical finding etc.) to be helpful to clinicians, it is important that its interpretation is not a product of guesswork. This concept is often referred to as ‘precision’ (though some incorrectly use the term ‘accuracy’). For example, if we actually hit the bull's-eye of a target (representing agreement with the gold standard), we are accurate. If all our shots land together, we have good precision (good reliability). If all our shots land together and we hit the bull’s-eye, we are accurate as well as precise. It is however possible to hit the bull’s-eye purely by chance.

Precision, as it pertains to agreement between observers (interobserver agreement), is often reported as a kappa statistic, which, is intended to give a quantitative measurement of the magnitude of agreement between two or more observers. For example, comparing the presence of wheezes on lung examination to the presence of an infiltrate on a chest radiograph assesses the validity of the exam findings to diagnose pneumonia. Assessing whether the examiners agree on the presence or absence of wheezes (regardless of validity) assesses precision (reliability).

**Kappa Statistics**

<table>
<thead>
<tr>
<th>Significance for kappa values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 0</td>
</tr>
<tr>
<td>0.01 – 0.20</td>
</tr>
<tr>
<td>0.21 – 0.40</td>
</tr>
<tr>
<td>0.41 – 0.60</td>
</tr>
<tr>
<td>0.61 – 0.80</td>
</tr>
<tr>
<td>0.81 – 0.99</td>
</tr>
<tr>
<td>+ 1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Significance for kappa values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than chance agreement</td>
</tr>
<tr>
<td>Slight agreement</td>
</tr>
<tr>
<td>Fair agreement</td>
</tr>
<tr>
<td>Moderate agreement</td>
</tr>
<tr>
<td>Substantial agreement</td>
</tr>
<tr>
<td>Almost perfect agreement</td>
</tr>
</tbody>
</table>

Interobserver variation can be measured in any situation in which two or more observers are evaluating the same thing. For example, let us imagine a study in which two medical students are evaluating the usefulness of the lectures. They agree that the lectures are useful 15 percent of the time, while it is not useful 70 percent of the time (in other words the remaining 15 percent of the lectures the two students disagreed with each other i.e., 1 felt it was good and the other called it bad).

The calculation is based on the difference between how much agreement is actually present \( (PO = \text{observed agreement}) \) compared to how much agreement would be expected to be present by chance alone \( (PE = \text{expected agreement}) \). In the example cited above, the observed agreement is the percent of all lectures for which the two residents’ evaluations agree.

Also, kappa is a measure of the difference between the two \( k = \frac{PO - PE}{1 - PE} \). The values are standardized to lie on a scale from -1 to 1 scale, where 1 is perfect agreement, 0 is exactly what would be expected by chance, and negative values indicate agreement less than chance, i.e., potential systematic disagreement between the observers.

Sometimes, we are more interested in the agreement across major categories in which there is a meaningful difference. For example, let’s suppose we had five categories of ‘helpfulness of noon lectures’: ‘very helpful’, ‘somewhat helpful’, ‘neutral’, ‘somewhat a waste’, ‘complete waste’. Here, we may not care if one resident categorizes as ‘somewhat helpful’ and another categorizes as ‘very helpful’, but we might care if one resident categorizes as ‘very helpful’ and another categorizes as ‘complete waste’.

Using a clinical example, we may not care if one radiologist categorizes a mammogram finding as normal and another categorizes it as benign, but we do care if one categorizes it as normal and another categorizes it as cancer.

Here, is where the weighted kappa assumes its significance, which is an appropriate ‘chance adjusted measure of agreement’ between two observers, when there are more than two ordered categories of classification. This statistic ranges from -1 (agreement less than chance) to +1 (perfect agreement). In our previous example, a disagreement of normal versus benign would still be credited with partial agreement, but a disagreement of normal versus cancer would be counted as no agreement.
Limitation of Kappa

It may not be reliable for rare findings. Thus, very low values of kappa in such cases, may not necessarily reflect low rates of overall agreement.

Our Results

Observed agreement (PO)
- Schaffer's grading → 0.62 (k = 0.46)
- Occludability → 0.88 (k = 0.45)
- Posterior most structure → 0.62 (k = 0.46)

<table>
<thead>
<tr>
<th>Shaffer's grading</th>
<th>Occludability</th>
<th>Posteriormost structure</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 20 patients</td>
<td>PO=0.6</td>
<td>PO=0.85</td>
</tr>
<tr>
<td></td>
<td>(k = 0.393)</td>
<td>(k = -0.071)</td>
</tr>
<tr>
<td>Last 20 patients</td>
<td>PO=0.66</td>
<td>PO=0.9</td>
</tr>
<tr>
<td></td>
<td>(k = 0.516)</td>
<td>(k=0.615)</td>
</tr>
</tbody>
</table>

Discussion

We found moderate agreement with Schaffer's grading (k = 0.46), occludability (k = 0.45) and posterior most structure (k = 0.46). Thus, we have demonstrated that a junior resident can achieve moderate levels of agreement with a senior resident, in the gonioscopic evaluation even without specific training. Further consensus training can increase the level of agreement to substantial to almost perfect.

The two observers showed fair agreement (k = 0.393) for Schaffer's grading, less than chance agreement (k = -0.071) for occludability and fair agreement (k=0.393) for posteriormost structure seen, for the first twenty patients seen among the fifty selected for the study. These values improved to moderate agreement (k=0.516) for Shaffer's grading, substantial agreement(k=0.615) for occludability and moderate agreement (k=0.516) for posteriormost structure, seen for the last twenty patients. This demonstrates a learning curve for gonioscopy and suggests that the stage of training might have influenced the degree of improvement. It can also be concluded that occludability showed a steeper learning curve when compared to the other two parameters.

Development of standardized criteria and reporting forms, pilot testing and training of raters through the review of disagreements are some of the methods of maximizing agreement in a wide variety of clinical ratings.

Conclusions

- As with any other diagnostic test, gonioscopy too has its own leaning curve, with a steeper curve for occludability.
- As the junior resident has demonstrated moderate agreement with the senior resident even without specific training in gonioscopy, it can be concluded that with consensus training, the junior resident can be given sole responsibility for assessment of gonioscopy and thus, the patient needs can be addressed in a better way.
- Multiple clinicians involved in clinical trials, should seriously consider pilot training and assessment of the level of agreement in making clinical and diagnostic test ratings, to enhance the power and accuracy of their studies.

References

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Impact of Trypan Blue (TB) Staining of the Anterior Capsule on Capsulorhexis in Various Grades of Cataract

Dr. Arup Chakrabarti MS, Dr. Meena Chakrabarti MS, Dr. Valsa Stephen MS, Dr. Sonioa Rani John DNB

Continuous curvilinear capsulorhexis (CCC) is a critical step in phacoemulsification. An intact CCC, of an appropriate size and shape is mandatory for successful phacoemulsification. Moreover, this also happens to be the most difficult step to learn in phaco and also tricky in certain special situations like a white cataract. Many techniques have been proposed for improving anterior capsular visualization. The most reliable method is the use of a dye to stain the anterior capsule. Indocyanine Green (ICG) first used by Horiguichi et al 1 and Trypan blue reported by Melles et al 2 work beautifully in this regard. They are much superior to fluorescein 3 which being a smaller molecule diffuses into the lens and vitreous. Recently trypan blue (TB) dye has been used with a great degree of success to facilitate CCC in these situations. This paper studies the impact of trypan blue on CCC in various grades of cataract. Staining of the anterior capsule for better visualization is necessary in any situation where either the red reflex is poor or visualization of the capsule is compromised. The presence of the asteroid hyalosis, corneal scarring, corneal oedema or the dark brunescent nucleus are examples of this situation.

Materials and Methods

200 successive cases of phaco emulsification where anterior capsule was stained with trypan blue were included in the study. The patient profile and the type of cataract were noted down. Video recording of the CCC was made in randomly selected cases.

Good mydriasis was achieved in all cases with a combination of tropicamide and phenylephrine eye drops. Flurbiprofen eye drops were used four times one hour prior to surgery. All operations were performed under peribulbar anaesthesia. Temporal clear corneal incision and 2 paracenteses were fashioned.

Staining of the anterior capsule

The anterior chamber was filled with an air bubble through the inferior paracentesis. Trypan blue 0.06 % (0.1ml) was squirted on to the anterior capsule under the air bubble through the same paracentesis wound. After a 20 seconds contact time, the excess trypan blue was washed off from the anterior chamber by bimanual I/A.

CCC was performed using a bent 26 G needle under viscoelastic cover (Fig 1). Capsulorhexis forceps was used in some patients with intumescent or hypermature cataracts.

Video recording was performed with and without the red reflex enhancer

(a) before trypan blue staining (fig 2) (b) after trypan blue staining of the anterior capsule (Fig 3) (c) during the progression of the CCC and (Fig 4) (d) after the CCC and removal of the anterior capsular flap (Fig 5).
Results

200 consecutive patients undergoing phacoemulsification whose anterior capsules were stained by trypan blue were included in the study. There were 115 males and 85 females. The average age was 65 (range 38 to 83). 113 patients (56.5 %) had satisfactory red reflex, 44 patients (22 %) had poor reflex and the rest 43 (21.5 %) with white cataracts had no red reflex. All the patients had well dilated pupils.

Capsulorhexis was complete in all cases. Trypan blue had a dampening effect on the red reflex in patients with good reflex to start with, and had no contributory effect to the success of the capsulorhexis. However the dye was found to be of great aid in eyes with dull reflex or no reflex (white cataracts) whatsoever.

There were some patients (23) with good red reflex who had grade III nuclear sclerosis, though by and large, patients with lesser grades of nuclear sclerosis tended to have brighter red reflex.

Subjectively there was lesser chance of damage to the capsulorhexis margin by the phaco tip owing to enhanced visibility of the stained capsulorhexis margin.

Discussion

Capsulorhexis is a prerequisite for successful phacoemulsification. However this also happens to be the most difficult step to learn during the learning curve, and in certain situations like a white cataract, capsulorhexis may be quite difficult to accomplish even by an experienced surgeon.

Capsulorhexis requires direct and easy visualization of the anterior capsular flap. The visualization of the flap may be difficult (a) by the untrained eye during the early stage of the learning curve, (b) when sub capsular cortex gets disturbed and gets mixed with the capsular flap and (c) when the red reflex is not well visualized by a very basic microscope or due to a very dense or white cataract. Various staining techniques of the anterior capsule have been described in the literature. Fluoroscein, methylene blue, trypan blue and indocyanine green have been used for this purpose. In January 1999, Melles et al reported the use of Trypan blue dye in 30 patients with mature white cataracts. There were no complications reported which were
attributable to the dye. Trypan blue creates a much darker staining and provides superior visualization when compared to ICG. Unlike ICG there is no particulate suspension with trypan blue and it is much more convenient to use because there is no mixing involved.

Because it is supplied in a smaller amount it is less expensive. Trypan blue staining lasts longer usually through all the phacosteps and hence there is less chance of damage to the dye stained and easily visualized capsulorhexis margin.

Trypan blue is commercially available in the Indian market at an affordable price. It is now extensively used and has been found to be very effective with no clinically relevant toxicity to the corneal endothelium. A 20 second contact time was enough to achieve adequate staining of the anterior capsule in all cases.

This study looked into the effectiveness of trypan blue with respect to the density of the cataract. The results and their implications are summarized here.

1. If a given case of cataract is associated with a bright red reflex, staining of the anterior capsule with trypan blue is associated with significant dampening of the red reflex. Hence, capsulorhexis can be achieved quite easily without staining if the red reflex is bright. On the contrary, capsular staining in this scenario may not offer any additional benefit as far as enhanced visibility of the capsular flap is concerned.

2. If the red reflex is dull or absent for whatever reason, the stained anterior capsule greatly enhances the visibility of the anterior capsular flap and facilitates the performance of the capsulorhexis.

3. Trypan blue should be used to stain the anterior capsule by a beginning phaco surgeon if he is using a lower end microscope which may lack in satisfactory red reflex or other user friendly features. Enhanced visibility will also greatly reduce the chances for damages to the capsulorhexis margin by the phaco tip.

**Conclusion**

In conclusion, trypan blue staining of the anterior capsule is strongly recommended for capsulorhexis in all cases during the initial stages of the phaco learning curve. Use of trypan blue should also be strongly considered for capsulorhexis in white cataracts.

**References**

Assessment of Merits of Clear Corneal Incision over Scleral Tunnel Incision in Phacoemulsification

Dr. Sujithra H, Dr. Meenakshi Dhar, Dr. Dhireesh, Robin Shanmugham

Abstract

The study evaluated the surgical outcome of phacoemulsification in clear corneal and scleral section. Patients were divided into two groups. One group underwent phacoemulsification with clear corneal incision and the other group with a scleral tunnel. All patients underwent surgery after comprehensive, routine preoperative assessment. All surgeries were done under local anaesthesia with peribulbar block by a single surgeon. Vision was checked on the first postoperative day, after one week and after one month. Autokeratometry was done preoperatively, after one week and one month. Visual recovery, peri and post-operative patient comfort, congestion, eyelid drooping, postoperative astigmatism and postoperative refraction were compared in the two groups.

Introduction

The refractive aspect of cataract surgery has received considerable attention with the advent of modern small incision surgery, phacoemulsification and microincision cataract surgery. The amount of surgically induced astigmatism can be controlled better, and the faster wound stability reduces the time required for visual rehabilitation. Recently preference has shifted from corneo scleral incision to clear corneal incisions. Clear corneal incisions are well tolerated by the patients and it induces less postoperative astigmatism and results in faster visual recovery.

Aim

To evaluate the advantage of clear corneal incisions over scleral tunnel incisions.

Method

A prospective study was done at our institute to compare scleral and corneal incisions in patients undergoing phacoemulsification with foldable lens implantation. It comprised of 60 eyes. Patients were randomly divided into two groups of 30 each based on the incision types (Group I- Scleral incision and Group II- Corneal incision). All patients had undergone thorough pre-operative evaluation. All cases with obvious corneal opacity and retinal pathology were excluded. Phacoemulsification was done with implantation of foldable acrylic posterior chamber IOL (Acrysof) with 3.5 mm incision. All steps of surgery were similar in the 2 groups except for the incision and putting of the bridle sutures. Superior and inferior rectus suture was put in all cases of the scleral group. Postoperative vision checked on first post operative day, one week and after one month. Auto-refractometry was done before surgery and after six weeks. Against the
rule astigmatism was with axis 80-100°. With the rule
astigmatism was with axis 10-170°. All other axes were
taken as oblique astigmatism. A difference of 0.5D or
more was taken as significant corneal astigmatism.
Amount of postoperative astigmatism and speed of
recovery are compared between the two groups.

All surgeries were done by the same surgeon under
peribulbar anaesthesia. Incision site is supero temporal
quadrant both for the scleral and corneal group.

**Group I - Scleral Tunnel group**

Scleral triplanar incision was made in the supero
temporal quadrant, 1-1.5mm posterior to the limbus
with 11 no. blade after making a fornix based
conjunctival flap. Bipolar cautery was used to cauterize
the bleeding vessels. Scleral tunnel was dissected with
crescent blade (angled bevel up 20 Gauge). Depth was
about 1/3rd of scleral thickness. Dissection extended
into the clear cornea for 1 mm. Anterior chamber was
entered with keratome (angled bevel up 19 Gauge).
Two side ports were made. Capsulorhexis was done
through the side port. Then hydrodissection,
hydrodelineation and phacoemulsification were done
through the main section. After cortical clean up
foldable acrylic posterior chamber IOL was implanted
in the bag. Side ports and main port were hydrated to
seal the chamber. Incision tested for water tightness.
The conjunctival flap was then re-approximated with
bipolar forceps cauterization.

**Group II- Clear Corneal Incision group**

Conjunctiva was not reflected. In corneal incisions we
made a 2-3mm incision furthest away from the optical
axis i.e. supero temporal quadrant. This should affect
the astigmatism minimum. The incision is made just
anterior to the anatomical limbus. Tri planar incision
is made in the cornea extending 1 – 1.5 mm into the
cornea with care taken to maintain lateral margins
radially to the cornea. Corneal incision was made in
the superotemporal quadrant with lance tip [sideport]
24 G blade. Tunnel was dissected with crescent blade
(angled bevel up 20 Gauge). Anterior chamber was
entered with keratome (angled bevel up 19 Gauge).
Other procedures were same as that for scleral tunnel.
Cautery was not used.

**Observations and Results**

**Postoperative Visual Acuity [VA]** was recorded for
each patient in the 2 groups at 1day, 1week and 6 weeks
[Table1 and 2]. 50 % of patients had 6/12 or better
unaided vision on day 1 in group I and 79 % in Group II.
In Group I, 63 % of patients after 6 weeks got 6/6
unaided vision.

In Group II 80 % got unaided vision of 6/6 at 6 weeks.

<table>
<thead>
<tr>
<th>Table 1. Postoperative visual recovery in Group I {scleral}</th>
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<tr>
<td>1 day</td>
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<table>
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<tr>
<th>Table 2. Postoperative visual recovery in Group II {corneal}</th>
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<td>1 day</td>
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[Table 3]: Post op astigmatism in Group I (scleral)

<table>
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<tr>
<th>SCLERAL GROUP [N=30]</th>
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<tr>
<td>With the Rule</td>
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<td>Preop</td>
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<tr>
<td></td>
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<tr>
<td>No Significant Astigmatism</td>
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<tr>
<td>0.5-1.0</td>
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<tr>
<td>1.0-1.5</td>
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<td>1.5-2.0</td>
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In group I (Table 3) preoperative corneal astigmatism ranges from -0.25 to -2.00 D. With the rule (WTR) astigmatism was in 2, against the rule in 6 cases.

In group II (Table 4) preoperative corneal astigmatism ranges from -0.25 to -2.5 D both pre and postoperative. With the rule (WTR) astigmatism was in 8, against the rule in 12 cases and 10 had oblique astigmatism. Postoperative corneal astigmatism with the rule [WTR] astigmatism in 1, against the rule in 6 cases. 9 cases had no corneal astigmatism in the post op period and 14 had oblique astigmatism.

Patients with Corneal incision showed a low induced astigmatism and faster recovery of vision. None had lid swelling, subconjunctival suffusion or lid droop.

In the scleral group healing was slower in the sense of mild swelling of lids, droop of upper lid

**Discussion**

Cornea contributes almost 2/3rd of the refractive power of the eye. The full thickness incision made in the cornea can differentially affect the curvature of the cornea to varying amount, in different meridians. The site, size and types of the incision are the major determinants for this change. The more corneal, and larger the incision, more is the induced astigmatism. The wound apposition of the inner lip of the incision determines the degree of alteration in the curvature of the cornea.

Postoperative astigmatism depends primarily on the size, architecture and location of the incision. Self-sealing tunnel incisions do not induce corneal changes caused by sutures. Thus the incisions are more than a port of access to the anterior chamber. It is the most important step during surgery affecting ocular integrity and corneal stability. Suture less clear corneal incision technique provides several advantages including less ocular tissue manipulation and surgical time. Also a corneal incision means we do not need to cautery the sclera at the limbus. Cauterization can contribute to astigmatism by causing contraction of the adjacent scleral and corneal lamella.

### Advantages of clear corneal incision

- Less surgery time.
- Elimination of the need for cautery and the potential for sub-conjunctival haemorrhage and hyphema
- Preserves the subconjunctival space for future filtering surgeries
- Promotes more rapid recovery and vision restoration.
- Does not disturb the filtering bleb, in an eye with a functional bleb
- Well tolerated by the patients.
- It can be performed under topical anaesthesia
- It is mainly suitable for foldable IOLs.
- Visibility during the phaco emulsification procedure is better due to shorter tunnel.

Average induced astigmatism in both groups was similar.

Superiorly placed corneal incisions cause an ‘against the rule’ (ATR) shift in the pre-operative astigmatism i.e. the vertical meridian becomes flatter. For that reason unsutured clear corneal incision should not be made superiorly in cases with pre-operative ATR astigmatism. Temporally placed incisions induce with the rule shift in pre-operative astigmatism and can reduce pre-existing ATR astigmatism. In the scleral group, more patients had oblique astigmatism both pre (15(50%)) and post operative (15(50%)). Against the
rule astigmatism (n=6) was more common than with the rule (n=2) in both pre and postoperative corneas. 9 patients did not have any astigmatism in the postoperative period. The incidence of no astigmatism was almost same in pre (n=7) and post (n=6) operative period.

In the clear corneal group, more patients had oblique astigmatism both pre (10(33 %)) and post operative (14 (47 %)). Against the rule astigmatism (n=12) was commoner than with the rule (n=8) in Preop corneas. 9 patients did not have any astigmatism in the post operative period.

Previously reported studies with clear corneal incisions developed significant change in corneal curvature with surgically induced astigmatism. Our study in contrast has shown lesser surgically induced astigmatism in the clear corneal group. This may be because our incision site was Superotemporal and incision was triplanar, made more gently in controllable manner rather than a uniplanar clear corneal one. The triplanar incision allowed better wound apposition with healing that altered the corneal curvature minimum, with hydration of the incisions that enabled good apposition of the inner wound on the cornea. Also the small size of the incision alters the curvature minimum in both the groups. Therefore pre-operative evaluation of keratometric astigmatism is important not only for calculating IOL power but also planning the incision site and to control the amount of post-operative astigmatism.

The postoperative keratometric astigmatism can be minimized when one considers the preoperative astigmatism when selecting incision type and location. Preoperative against the rule astigmatism was reduced significantly by temporally placed clear corneal incisions and preoperative with the rule astigmatism, by superiorly placed clear corneal incisions. The predominant factors in incision healing and stability were incision geometry, architecture, and location. Upper lid pressure on the superior corneal incisions led to fluctuating, against-the-rule astigmatism that was significantly higher than that induced by temporal incisions.

Superior corneal incisions produce more astigmatism than temporal in with the rule preoperative astigmatism. No statistically significant difference was found in the surgically induced astigmatism (SIA) between corneal incisions on axis or corneal temporal incision.

References
Fungal endophthalmitis, although uncommon, remains a serious ophthalmologic challenge owing to limited available treatments and potentially devastating ocular consequences. Fungal endophthalmitis can be caused either by exogenous origin, such as ocular trauma or surgery, or by endogenous infection spreading to the eye, such as those in immunocompromised patients. Until recently, intra vitreal injection of amphotericin B has been the principle treatment for fungal endophthalmitis \(^1\) although other potential intravitreal antifungal agents have been uninvestigated \(^2\)-\(^4\). However intravitreal amphotericin B, even at low concentrations 4.1 mg/ml or 8.3 mg/ml (5 mg or 10 mg injection into 1.2 ml of rabbit vitreous) \(^4\)-\(^5\) can cause focal retinal necrosis \(^6\)-\(^7\). Furthermore, resistance to amphotericin B has been documented in a variety of human systemic fungal infections \(^8\). Fluconazole, a triazole agent has been used systematically as a supplement or alternative to amphotericin B to treat fungal endophthalmitis because it can reach effective concentration in the vitreous after oral administration \(^9\)-\(^10\) but it lacks a broad spectrum of coverage against many of the most commonly enumerated organisms found in fungal endophthalmitis \(^11\)-\(^12\). Thus ophthalmologists have had a very limited number of antifungal agents and the current treatment protocols for fungal endophthalmitis are far from optimal.

Recently, a new antifungal agent, Voriconazole, has been approved by the US Food and Drug Administration for systemic fungal infection.

**Trade and Generic Names and General features.**

Voriconazole is a second generation synthetic derivative of fluconazole and it differs from fluconazole by the addition of a methyl group to the propyl backbone and by the substitution of a triazole moiety with a fluoropyrimidine group.

Voriconazole is developed by Pfizer pharmaceuticals. The trade name of Voriconazole is Vfend TM.

**Mechanisms of action**

The structural changes in Voriconazole result in a higher affinity for the fungal 14-demethylase leading to more potent activities \(^12\). Like fluconazole, Voriconazole exerts its effects primarily by inhibiting the fungal cytochrome P 450 CYPZA enzyme lanosterol 14 a – demethylase, preventing the conversion of lanosterol to ergosterol. This is turn causes depletion of ergosterol, which disrupts the integrity and function of the fungal cell membrane, eventually leading to cell lysis \(^13\). Voriconazole also inhibits 24-methylene dihydrolanosterol demethylation in certain yeast and filamentous fungi, explaining its increased activities against moulds \(^14\)-\(^15\).

**Susceptibility Patterns**

Voriconazole has favourable invitro activity against a variety of fungi. These include Candida sp, Asperigillus sp, Cryptococcus neoformans, Blastomyces dermatitidis, Coccidoides immunities, Histoplasma capsulatum, Fusarium sp and Penicillium sp and Penicillium marneffei.

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Chakrabarti Eye Care Centre, Kochulloor, Trivandrum 695 011
Email: tvm_meenarup@sancharnet.in
Voriconazole is generally considered to be a fungistatic agent against Candida sp and Cryptococcus neoformans.

Its enhanced activity against fluconazole resistant Candida krusei, Candida glabrata and Candida guilliermondii is noteworthy. Some fungi which are resistant to fluconazole and /or itraconazole may exhibit cross resistance to Voriconazole. Zygomycetes such as Mucor sp and Rhizomucor sp generate considerably high Voriconazole MICS.

**Pharmacology:** - It has oral and i/v formulations. Voriconazole is well absorbed orally with a bioavailability of 96 %, allowing patients to be switched between intravenous and oral administration.

Being metabolized by hepatic cytochrome P450, Voriconazole interacts with some drugs. Administration is contraindicated with some drugs (such as sirolimus, rifampin, rifabutin and dose adjustments and / or monitoring are required when administered with others (including cyclosporine, tacrolimus, omeprazole and phenytoin). Voriconazole may be safely administered with cimetidine, ranitidine, indinavir, macrolide antibiotics,mycophenolate and prednisolone.

Because Voriconazole is metabolized by the liver the dose should be halved in patients with mild to moderate hepatic impairment. There is no data available for patients with severe hepatic impairment.

No dose adjustments is necessary for renal impairment or advanced age, but children seem to clear Voriconazole faster than adults and drugs levels may need monitoring.

In 2005, Voriconazole was also approved for the treatment of invasive candidiasis.

Intravitreal Voriconazole has been used for drug resistant fungal endophthalmitis. A recent report has compared the minimum inhibitory concentration (MIC) of natamycin, amphotericin and voriconazole against aspergillus species isolated from keratitis. MIC of natamycin was 32 mg/ml, amphotericin B was 2-4 mg/ml and that of Voriconazole was the lowest 0.25 – 0.5 mg/ml. It is to be noted that effectiveness of antifungal agents depends on the concentration of drug achieved locally, in practice; the topical antifungals are given at different concentrations - amphotericin B because of toxicity is prescribed at 0.15 %, Voriconazole at 1 % and natamycin at 5 %. Thus although the MIC level of natamycin is higher, it is administered at five times the strength of Voriconazole and 30 times that of amphotericin B.

Voriconazole eye drops is prepared by reconstituting lyophilized powder used for parenteral administration with 19 ml sterile water for injection to obtain 20 ml of 1 % solution and administered every hour round the clock initially and then gradually tapered as per the response. Voriconazole eye drops should be prepared every alternate day, stability of the solution could be extended up to 48 hours between 20°C and 8°C F. For optimum intraocular drug concentration, both oral and topical administration of Voriconazole is recommended.

Studies in rats demonstrate that intravitreal Voriconazole did not cause any retinal toxicity on either ERG or histologic studies when concentrations were 25 mg/ml or less. When the intravitreal Voriconazole concentration reached 50 mg/ml or more, focal retinal necrosis was occasionally noticed on histologic examination, but ERG was not affected (because ERG is a mass electrical response from the whole retina and focal necrosis may not cause ERG abnormalities). When these results were transferred to human eyes, assuring minimal species variability, Voriconazole 100 mg may be injected into the human vitreous without causing long term ERG or histologic abnormalities, based on the fact that the average human vitreous volume is 4 ml. Voriconazole is much safer to the retina than amphotericin B because very low loses of intravitreal amphotericin B (4.1-8.3 mg/ml) cause focal retinal necrosis in rabbit studies. Since Voriconazole is superior or equal to amphotericin B for common and rare yeast and mould infections, Voriconazole should be considered as a possible first line intravitreal agent for treatment of fungal endophthalmitis. A recent case report showed that endophthalmitis caused by Fusarium solani was successfully treated with intracameral, topical and systemic Voriconazole when the endophthalmitis failed to respond to amphotericin B, fluconazole or itraconazole.

Voriconazole is active following both oral and intravenous administrations. In clinical trials, oral (200 mg twice daily) and intravenous (3-6 mg/kg every 12 hour) doses have produced favourable response. However, typical doses at individual clinical settings are not yet known. Parenteral administration can be followed by an oral course of Voriconazole therapy.
Side Effects

The most common side effects associated with Voriconazole include transient visual disturbances, fever, rash, vomiting, nausea, diarrhoea, headache, sepsis, peripheral oedema, abdominal pain and respiratory disorders.

Unlike most adverse effects, which are similar to other azole antifungal agents, visual disturbances (such as blurred vision or increased sensitivity to light) are unique to voriconazole. Visual disturbance is an interesting and extensively investigated side effect of voriconazole. It seems to be due to the blockage of receptor de excitation by Voriconazole. These visual disturbances have been reported by more than 30% of patients in clinical trials. They generally occur approximately one-half hour after administration and last approximately for 30 minutes. In some patients they may go away after continued use. Studies have shown that there is no damage to the eye or long term effect on vision. However, patients taking Voriconazole should be advised against driving at night or other potentially hazardous tasks.

Though rare, there have been cases of serious hepatic reactions during treatment with Voriconazole. Liver function tests should be evaluated at the start of and during the course of therapy.

This medication may also cause peeling of skin. It is best to apply lotion or coconut oil to help with this side effect.

Current Status:

Clinical use of Voriconazole was approved by FDA in May 2002. It was approved for primary treatment of acute invasive aspergillosis and salvage therapy for rare but serious fungal infections caused by the pathogens Scedosporium apiospermum and Fusarium sp.

Thus intravitreal Voriconazole will offer a significant new treatment option in the management of fungal endophthalmitis.

References

Spectral Optical Coherence Tomography (OCT) has
captured the imagination of Ophthalmic surgeons world
wide and has sought to replace the old Time Domain
OCTs (TD-OCT) which were in vogue in the past
decade.

What is spectral OCT?
The new generation OCT uses Fourier domain
technology which is capable of higher resolution
scanning, 3D image generation and faster acquisition
speeds as compared to the older Time domain scan
employed in the Stratus OCT which we are all used to.

Greater clinical accuracy is made possible due to dense
grid scanning as compared to the sparse radial scanning
method of TD-OCT.

All spectral OCTs use a Super Luminescent Diode source
at a wavelength of 840nm.

A-scan frame rate varies from 23,000 A-Scan/Sec to
26,000 A-Scan/Sec.

Scan depth ranges form 2.0-2.3mm

Outstanding resolution of 5-6 microns is achieved. This
increases the sensitivity of OCT from 83 (in TD-OCT)
to 97 (in S-OCT). Details provided by the Doheny
Institute of Retina (USA) reveal excellent imaging
quality with Vitreous opacities, Epiretinal Membranes,
Photoreceptors and RPE layer.

Common Features of Spectral OCTs
3D image generation, movie capture, RNFL scan, Optic
Disc Analysis, etc

The Parameters Compared
Comparisons is based on Hardware, Resolution,
Analytical capability of the software, Ease of use,
Reproducibility, Reliability, Pricing, and most
importantly the after sales support.

Topcon 3D OCT-1000
The Topcon Corporation, Japan was probably the “first
off the block” to advertise the coming of the Spectral
(Fourier) domain OCTs.

Hardware: The 3D OCT-1000 employs a modified
non-mydriatic fundus camera that offers color fundus
pictures along with the OCT. This is unique to the
Topcon OCT.

The scan rate is 18,000 frames/sec, (which is the least
in all the S-OCTs).

Resolution: Axial - 6 microns, Horizontal – 20
microns (resolution is poor, but Topcon says this will
improve)

Software: Box scan, Circular Scans, Line Scans and
Radial Scans possible with 3D image generation. RNFL
scans are of doubtful reliability as normative data was
late in coming and that too it employs Oriental data
passing off as Asian data. Reproducibility needs
improving as well as the measurement modes.

Lacks SLO and IR images. The fact that it is a modified
Fundus Camera gives the color image that could give
additional information like pin point registration and
retinal mapping which could be used effectively during
patient counseling. Orientation Markers provide quick
orientation of 3D image to fundus image. It is possible
to import ICG pictures and pin point the lesions.
Excellent movie and analytical package like TrueMap which enables viewing of images and data in three viewing modes simultaneously – 3D, fundus image and 2D.

**Pricing:** Sold and serviced by Mehra Eyetech, Mumbai. List pricing is 90,000 – 100,000 USD which is very steep compared to the competition. After-sales support would be good.

**Zeiss Cirrus HD-OCT**

The leader in OCT technology was a bit late in coming with their version of the S-OCT but as the saying goes “better late than never”.

**Hardware:** The Cirrus is unique in that it employs a 90 degree orientation thereby enabling the doctor to observe the patient as he is fixing on the target. All other OCTs have a 180 degree orientation. Also, the Cirrus consumes less floor space as it is a very compact design.

Auto re-call function recalls the instrument setting of the individual patient (like chin rest height) from his last visit. The Line scanning Ophthalmoscope employed by the Cirrus is nothing but the SLO image in the other S-OCTs.

The scan rate is 27,000 frames/sec.

Pupil size requirement is the least at 2mm which is exceptional.

**Resolution:** Axial - 5 microns, Horizontal – 15 microns

**Software:** Only Cube and Raster scans are available (The raster consists of just 5 lines). I think the cirrus could have employed that radial scans as well. The RNFL scans and Optic disc analysis scan are highly accurate and fairly reproducible which we have come to respect Zeiss for.

The Cirrus software is truly outstanding in its layer by layer mapping, Overlay analysis, etc.

The printout format and clarity is by far the best.

**Pricing:** The quoting price is in the region of 80-95,000 USD with the option of an exchange with the Stratus OCT always on the table. After sales support needs careful analysis by end user (proper contracts need to be negotiated).

**OTI SLO-OCT**

OTI has been a leader in the 3D imaging section with their 3D B-scans being in the market for nearly 6 years. The design of the OCT has undergone several modifications and upgrades to reduce the footprint.

**Hardware:** The scan rate is 27,000 frames/sec. Scan depth is 2.0 to 2.3 mm (in power user mode). Connectors are of suspect quality. Frequent design changes make the hardware liable to failure.

**Resolution:** Axial - 5 microns, Horizontal – 15 microns

**Software:** The software is quite powerful and user friendly. It offers a range of alternative measurement modes and also a power user mode. Micro-perimetry option is unique to this system. Whether this perimetry is precise is within the realm of speculation.

RNFL and optic disc analysis programs are not reliable or reproducible. Precise determination of the disc boundary is among the worst I have seen.

**Pricing:** Sold and serviced by Biomedix Optotechnik & Devices. The quoting price is in the region of 65-85,000 USD. After sales support would be excellent.

**RTVue-100**

Optovue was the first to bring out a fully loaded S-OCT nearly 2 years ago. The software and normative data was complete in all respects at that time.

Even today, RTVue is the first to offer a fully functional anterior segment addon package to the Spectral platform.

**Hardware:** The scan rate is 27,000 frames/sec. Scan depth is 2.0 to 2.3 mm

**Resolution:** Axial - 5 microns, Horizontal – 15 microns

**Software:** SLO is available along with color pictures and near IR images.

The color mapping of retinal thickness and Grey scale maps are truly impressive. The details of the OCT image could be a little better though. It offers standard tools like Slicing, Peeling, Difference maps, 3D animations, etc. Glaucoma surgeons would be delighted with the range of measurements offered on the RTVue with power
packed normative data to support it. Glaucoma analysis combines the report of 3 standard glaucoma detection tolls available in the market today. Normative data unique to this system offers the capability to analyze the ganglion cell layer that suffers first glaucomatous damage. Precise determination of the disc boundary is by far the best I have seen.

Cornea and Anterior Module is an additional feature.

**Pricing:** Sold and serviced by IOC (Intra Ocular Care). The quoting price is in the region of 60-80,000 USD which makes it the best to buy. After sales support would be of a little concern.

**Conclusion**

Spectral OCTs have opened a new dimension to retinal imaging. The technology is bound to undergo some improvements, mainly in the user interface and software. The hardware is more or less settled.

My personal preference would be the RTVue 100 if the service commitment can be obtained properly. Cirrus comes a close second but Zeiss would never package their anterior segment module along with it. OTI and Topcon have some hardware issues to settle.

Happy Imaging!
Managing Retained Lens Fragments After Cataract Surgery

Dr. Meena Chakrabarti MS, Dr. Valsa Stephen MS, Dr. Sonia Rani John DNB, Dr. Arup Chakrabarti MS

Phacoemulsification with posterior chamber intraocular lens implantation – the gold standard for managing cataracts results usually in rapid rehabilitation from an anatomic and visual standpoint. However a small percentage of patients develop intraoperative or postoperative complications, the commonest being retained nucleus or lens fragments and dislocated posterior chamber lens.

Although exact figures are not available, the incidence of posteriorly displaced lens fragments is probably 0.3%. However considering the large volumes of cataract extractions performed, this number is considerable. The displaced fragment may involve the entire nucleus or only its miniscule fraction.

This article will discuss in detail the options for management by the anterior segment surgeon as soon as posterior capsular rent is recognized, when the surgeon still has time to resort to nuclear retrieval techniques. Once the fragment or the entire nucleus has dislocated into the vitreous cavity, the clinical characteristics, indications for removal, timing of the vitrectomy, and the surgical techniques employed are discussed in detail.

For the anterior segment surgeon who encounters the complication of posteriorly dislocated lens fragment during surgery, retrieval of the lens fragment should be attempted only if the fragment is readily accessible. The technique of posterior assisted levitation (PAL techniques) can be tried to bring the nuclear fragment into the anterior chamber. However this technique may exert vitreous traction leading on to retinal tears, retinal detachment and vitreous haemorrhage.

Adequate anterior vitrectomy should be performed to clear any vitreous present in the wound.

**Should an IOL be implanted along with the primary procedure when a nuclear fragment was lost posteriorly?**

Most vitrectomy surgeons recommend that if there is sufficient capsular support or if suture fixation techniques are available, the IOL be placed in the posterior chamber and the limbal wound closed in the usual fashion.

Implanting a multiflex open loop design AC IOL into the anterior chamber is a viable option provided there is no corneal endothelial decompensation, compromised angles or loss of iris tissue.

Primary IOL implantation is however not advisable if a large chunk of very hard / black nucleus has dislocated, the best option in this situation is to remove it through the limbal route and perform a secondary IOL implant along with the vitrectomy procedure.

Posterior loss of lens fragments is usually recognized by the cataract surgeon after posterior capsular rupture.

**Common Predisposing Factors for Posterior Capsular Rupture**

Certain types of cataract may be at a higher risk for developing posterior capsular rent. They are...
1) posterior polar cataract (PPC) and the cataract associated with posterior lenticonus or lenti globus, (2) post vitrectomy cataract, 3) traumatic cataract, 4) white cataract, and 5) black cataract. Preoperative counseling of patients with these types of cataract should include a thorough discussion about the potential for a posterior capsular rent and its sequelae.

**Early Recognition Of Zonular or Posterior Capsular Rupture**

If a posterior capsular tear is not recognized in time, subsequent intraocular maneuvers required for phacoemulsification (viz. nuclear rotation, sculpting, cracking) and fluctuations in anterior chamber depth will quickly enlarge the size of the tear. The risks of vitreous loss and dropped nucleus increase, longer the rupture goes unrecognized. Early recognition of a posterior capsular tear and prompt prophylactic measures\(^\text{5}\) will prevent expansion of the tear size\(^\text{15,16}\).

Signs of early posterior capsular tear or zonular dehiscence\(^\text{6}\) include the following.

- Sudden deepening of the anterior chamber with momentary dilatation of the pupil.
- Sudden transitory appearance of a clear red reflex peripherally.
- Newly apparent inability to rotate a previously mobile nucleus.
- Excessive lateral mobility or displacement of the nucleus and loss of nucleus followability.
- Excessive tipping of one pole of the nucleus.
- Partial descent of the nucleus into the anterior vitreous space.

**Rescuing a Partially Descended Nucleus**\(^\text{17}\)

Posterior capsule or zonular rupture should be recognized early enough to avoid a dropped nucleus. Without timely recognition of the capsular rent, the continued phaco maneuvers and forces will expand the initial defect thereby creating a big hiatus to permit the nucleus to drop. A brunescent nucleus may abruptly and rapidly sink through the liquified vitreous without antecedent vitreous loss. However if enough supporting vitreous is present the nucleus will descend only partially, allowing for rescue maneuvers.

No attempt should be made to chase and spear the descending nucleus with the phacotip. The posteriorly directed fluid infusion will flush more vitreous out, expanding the rent and propelling the nucleus away. Additionally, vitreous may be snagged into the phacotip, potentially leading to giant retinal tears and detachment\(^\text{17}\). An alternative strategy is to levitate the nucleus into the pupillary plane or anterior chamber for subsequent management either by extraction through a standard ECCE incision (or rarely by careful phacoemulsification over Sheets glide)\(^\text{18}\). The nucleus may be rescued by injecting viscoelastic behind the nucleus and / or manipulating it with a hook using a limbal approach. However, this technique may be inadequate if the capsulorhexis is small and intact, if the pupil is small, if vitreous has already prolapsed around the nucleus or it has subluxated laterally or posteriorly. The “PAL”\(^\text{5,6,7}\) technique (posterior assisted levitation), first developed by Charles Kelman utilizing a cyclodialysis spatula through a pars plana stab incision to push the nucleus up into the anterior chamber from below, is preferred by many. Richard Packard modified this technique by inserting a Viscoat cannula through a parsplana stab incision located 3.5 mm behind the limbus. Through a combination of injecting Viscoat and maneuvering the cannula tip itself, the nucleus can be elevated through the capsulorhexis and pupil and into the anterior chamber. This minimizes iatrogenic vitreous traction and reduces the chance of touching the retina with a metal spatula tip. If the nucleus fragment has totally disappeared from view the surgeon should not blindly fish for it with the phaco or vitrectomy instruments.

After a thorough anterior vitrectomy and residual lens matter removal (with the vitrector or 1/A instruments) an IOL can be implanted or the patient can be left aphakic (depending upon the size and hardness of the dropped nucleus). If patient has been left aphakic, IOL can be implanted at the conclusion of the second stage three-port vitrectomy and dropped nucleus removal by fragmatome or anterior route\(^\text{19}\).

**Settings for Anterior vitrectomy**\(^\text{20}\): Use of the maximum possible cutting rate, lowest vacuum and flow rates reduces traction on the retina. The vitrectomy cutter should be advanced or held stationary during anterior vitrectomy and never pulled away while cutting.
Testing for vitreous in anterior chamber can be accomplished by

1. Injecting air into anterior chamber through the side port incision and looking for fragmentation of the bubble. Air, if used instead of infusion fluid prevents vitreous from hydrating and coming forwards. Air helps to delineate the surface of vitreous and keeps it confined by surface tension.

2. Using triamcinolone acetonide (preservative free) to stain the vitreous in the anterior chamber

**Infusion Options**

**1) Coaxial Infusion Cannula**\(^{17,18}\) for vitrectomy is possible by slipping the infusion sleeve over the vitrectomy tip. There are several disadvantages and dangers of using a coaxial infusion cannula for anterior vitrectomy.

a) **Enlargement of posterior capsular tear:**
The force of the infusion is in the same direction as the direction in which the vitrector tip is pointing. This means that the infusion will be directed towards the deep areas of the eye. As the tip approaches the torn posterior capsule, the infusion flow will strike the capsular flaps and force them apart. This extends the capsular tear and enlarges the opening resulting in prolapse of more vitreous

b) **Hydrates the Vitreous:** The infusion fluid hydrates the vitreous increasing its volume and causing it to expand. The only direction in which the vitreous is able to expand is towards the anterior chamber through the opening in the posterior capsule.

c) **Flushing the Vitreous:** The force of the infusion acts like a high-pressure hose flushing out the vitreous from the eye into the anterior chamber.

All these factors act together to increase the amount of vitreous that needs to be removed. (Fig: 1, 2, 3)

**2 Bimanual Technique with Separate Infusion Line**\(^ {19,20}\):-

Place the cutter through the PC Rent with the cutting port facing upwards. The strategy is to pull the vitreous from the anterior chamber down to the cutter.

The coaxial sleeve around the vitrector is removed and replaced by a separate infusion line. The AC maintainer or the irrigation port of the I/A hand piece can be used. The vitrector tip becomes less bulky and is able to pass through a paracentesis wound. This facilitates vitrectomy in a closed chamber away from the main phaco wound. The appropriate strategy for vitrectomy following vitreous loss during cataract surgery is to use the bimanual technique.

The vitrectomy tip is inserted through the opening in the posterior capsule and placed a mm or two behind the posterior capsule. The aspiration port is directed upwards towards the cornea. (Fig: 4 & 5)

![Fig. 4 & 5. Technique of Performing Anterior Vitrectomy](image)

The strategy is to pull the vitreous in the anterior chamber down to the vitrectomy tip until no more vitreous is there in the anterior chamber. The offending vitreous in the anterior chamber should be removed down to the level of and just below the posterior capsule. The rest of the vitreous in the vitreous cavity should not be touched.\(^ {17}\) (Fig: 5). It is advisable to begin the vitrectomy dry and then infuse BSS gently, if the chamber tends to collapse, through the side port. More vitreous will prolapse if the pressure in the anterior chamber is low when the aspiration continues.

The vitrectomy tip should not be placed through the primary phaco incision because the incision is the wrong size for it.\(^ {18}\) Instead the eye should be made firm with
viscoelastics and a new 1 mm incision be made a few millimeters away from the main phaco incision. The incision is the right size for the vitrector and the original side port incision, the correct size for the chamber maintainer. The pressure in the anterior chamber from the viscoelastics or chamber maintainer irrigation will close off the self sealing corneal or corneoscleral incision previously used for phacoemulsification.

Another portal of entry for the vitrectomy instrument is the pars plana through a sclerotomy 3 mm behind limbus. Infusion is performed using the AC maintainer. The vitrector is used to cut the vitreous at and behind the posterior capsule taking care to retain as much of the posterior capsule as possible.

**Performing Vitrectomy Without Irrigation (DRY Vitrectomy)**

This is a useful technique in performing a small vitrectomy. If the eye softens the pressure in the eye can be equalized by putting more viscoelastic into the anterior chamber. This has the added advantage of pushing vitreous towards the back of the eye and reducing the amount of vitrectomy that has to be performed.

When irrigation is used, the irrigating fluid might hydrate the vitreous in the anterior chamber, but this is acceptable because the vitreous in the anterior chamber is going to be removed anyway. We do not want the fluid to hydrate much of the vitreous below the vitrectomy tip. This is avoided by keeping the infusion cannula parallel to the iris so that the infusion is directed towards the AC and the vitrectomy tip can remove the fluid before it escapes into the body of the vitreous. The force of the infusion can act as a high power hose flushing out the vitreous from the eye. All this movement causes the vitreous to be flushed out of the back of the eye into the AC. This increases the amount of vitreous that needs to be removed. This is what happens when what looks like a small amount of vitrectomy turns into a large one. This is therefore not surprising that vitrectomy following vitreous loss in cataract surgery has a postoperative complication rate of 30% to 50%.

The best strategy when performing a vitrectomy is to avoid violating more vitreous than is actually needed. If you can remove the vitreous from the AC, without disturbing the rest of the vitreous especially that which overlies the vitreous base, you should have very few postoperative problems.

**PRE – PPV ASSESMENT**

Patients with retained lens fragments present with varying degrees of inflammation depending on the size of the fragment, amount of time elapsed following cataract surgery and extent of intraocular manipulation performed.

Clinical signs may include corneal oedema, glaucoma, uveitis and vitreous opacities causing profound visual loss. Frequently however, signs are mild especially in the immediate postoperative interval.

**Preoperative Evaluation**

- BCVA
- Slitlamp Biomicroscopy (pre and post dialation)
  - Degree of corneal oedema
  - Uveitis
  - Cortex at pupil
  - Asses extent of posterior capsular rupture and integrity of capsular zonular apparatus
- Applanation Tonometry
- Fundus examination/ B Scan USG
- In patients with severe corneal oedema, uveitis retained lens material at the pupil or associated vitreous haemorrhage percluding visualization into the vitreous cavity, a preoperative B Scan

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**Table 1. Major steps in managing posteriorly dislocated lens fragments**

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Attempt retrieval only if fragments are easily accessible.</td>
</tr>
<tr>
<td>2.</td>
<td>Perform anterior vitrectomy to avoid vitreous prolapse.</td>
</tr>
<tr>
<td>3.</td>
<td>Insert IOL when safe and indicated, preferably by capsular fixation or into the anterior chamber if necessary.</td>
</tr>
<tr>
<td>4.</td>
<td>Perform standard wound closure and viscoelastic removal</td>
</tr>
<tr>
<td>5.</td>
<td>Prescribe frequent postoperative topical NSAIDS and IOP lowering agent.</td>
</tr>
<tr>
<td>6.</td>
<td>Provide referral for prompt VR consultation</td>
</tr>
</tbody>
</table>
Ultrasonography is essential. Associated pathologies like retinal detachment, choroidal detachment, vitreous haemorrhage can also be picked up (fig 6 and 7).

- Document all findings and discuss options in detail with patients and attender
- Get informed consent

**Management**: If the crystalline lens or lens fragment dislocation is not associated with other problems, it may not lead to any complications on follow-up in a few cases. The eye can be rehabilitated by contact lenses or aphakic glasses. However, in some eyes the dislocated crystalline lens or retained lens matter has to be removed because of complications.

In general, the indications for removal of a dislocated crystalline lens includes impaired visual acuity, resulting from obstruction of the visual axis by the dislocated lens, development of complications such as phacolytic uveitis, or glaucoma, retinal detachment, and vitreous herniation into the anterior chamber resulting in a cystoid macular oedema. A relative indication for surgery is severe monocular diplopia.

Indications for removal of lens fragments.

1. Eyes with small retained lens fragments may be observed without treatment. Careful observation for 1-2 weeks may be safely pursued, especially with smaller fragments and can be extended beyond this depending on clinical progress.
2. Surgery is indicated in eyes with moderate or severe inflammation or retention of 25% or more of lens material.
3. Another indication for prompt surgery is elevated IOP. Medical measures can result in adequate IOP control. If IOP remains uncontrolled despite adequate medical therapy, surgical treatment should be considered.
4. Associated retinal detachments, retinal tears or endophthalmitis are all urgent indications for surgery.

Numerous surgical techniques have been described for the management of dropped nucleus, however, most have been abandoned because of their limitations, complications and complexities. The common techniques currently used to remove a crystalline lens dislocated into the vitreous cavity includes performing a three port pars plana vitrectomy. With this technique a through pars plana vitrectomy with removal of as much of the basal vitreous gel is performed using vitrectomy cutter (Fig. 8). The nucleus is lifted into the midvitreous cavity and fragmented. Frequently it may be necessary to crush the lens between the endoilluminator and the fragmatome into smaller fragments, which can be easily emulsified and aspirated. This procedure, though easy to perform can be hazardous in view of the mechanical retinal damage from falling lens fragments, or due to high energy of...
the ultrasonic probe. There is also an added danger of vitreous traction as the vitreous gets sucked into the probe, if the vitrectomy has not been completed. Retinal damage is particularly likely to occur if these maneuvers are performed when the retina is detached and mobile.

Therefore to remove the dropped nucleus safely and effectively, the use of prefluorocarbon liquids $^{28,29,30}$ have been recommended. The advantage of using perfluorocarbon liquid in removing dropped nucleus into the vitreous cavity are as follows,

1. The perfluoro carbon liquid lifts the dislocated lens from the retinal surface into the anterior vitreous.
2. In the presence of a retinal detachment, their high specific gravity mechanically flattens out the retina.
3. The PFCL bubble $^{31}$ forms a cushion which supports the lens and prevents mechanical retinal damage from falling lens fragments.

Therefore with the use of PFCL $^{31}$, the potential for retinal damage is reduced. The surgical technique includes performing a pars plana vitrectomy with removal of as much basal vitreous gel, prior to lens removal (Fig. 8). After the completion of vitrectomy, PFCL is injected into the vitreous cavity over the optic disc floating up the dislocated lens into the anterior vitreous (Fig. 9). If the dislocated lens is associated with a retinal detachment $^{29}$, the PFCL injection mechanically flattens the retina against the retinal pigment epithelium, displacing the subretinal fluid through a pre-existing inferior retinal break into the vitreous cavity. The dislocated lens is then fragmented in the anterior vitreous cavity while floating on the PFCL (Fig 10). Small fragments of lens matter that drop, float on the surface of the perfluorocarbon bubble and are easily aspirated. Lens particles can get entangled in the basal vitreous making its removal difficult. Damage to the peripheral retina can occur in an attempt to remove these entangled fragments.

If the dislocated crystalline lens $^{32,33,34,35,37}$ is very hard it is preferable to remove it through the anterior (limbal) route using either a cryo or an irrigating vectis (Fig 11).

If the eye with the dislocated crystalline lens also has an associated rhegmatogenous retinal detachment $^{31}$, the scleral buckle is placed prior to the lens removal.

After removal of the lens, endolaser retinopexy is performed around the tear and a PFCL air exchange is carried to achieve pneumohydraulic retinal reattachment.

### Timing of Vitrectomy for removal of retained lens fragment

Surgery to remove the retained lens fragments is performed within 2 weeks of the original cataract
surgery to expedite visual rehabilitation, to break the cycle of progressive lens induced inflammation, and to avoid long term glaucoma 38.

A higher incidence of glaucoma following vitrectomy48 for dropped nuclear fragments have been reported in cases where the vitrectomy was delayed for more than 3 weeks 39.

The ideal timing would be to perform the vitrectomy immediately. This is however possible only in larger set ups where the VR facility is readily available.

**Surgical Technique:** There are three basic approaches to removing lens fragments by pars plana vitrectomy 40,41.

1. By Ultrasonic fragmentation
2. Other viable options when a fragmatome is unavailable or when the nuclear material is extremely hard involves (a) crushing the nuclear fragments between the endo illuminator and cutter (b) retrieval through the limbal route.
3. Using the Vitrectomy cutter for soft nucleus or cortical matter.

Perform adequate vitrectomy prior to use of an ultrasonic fragmatome to avoid vitreous fibrils being sucked into the fragmatome hand piece, causing vitreous traction. Using triamcinolone acetonide to stain the vitreous ensures easy visualization, easy PVD induction and ensures a more complete vitreous removal 42,43.

Reducing fragmentation power to only 5 -10 % 44,45 facilities nuclear extraction by continuous occlusion of the suction port and avoidance of projectile fragments. This manoeuvre minimizes the risk of projectile fragments falling back to strike the retina causing damage. Using a small bubble of PFCL ensures that there is a cushion between the nuclear fragment and retina enabling safe phacoemulsification and minimizing the risk of projectile fragments. Use of PFCL is also recommended when a retinal detachment is associated with dislocated nuclear fragments. Finally at the conclusion of vitrectomy, the peripheral retina should be examined in detail for possible retinal tears or detachment 35, 36.

A dropped nucleus or dislocated IOL is one of the most serious complications of phacoemulsification. Parsplana vitrectomy in these patients lead to improved vision and is the procedure of choice. Shields showed that 44 % to 71 % of patients achieve a visual acuity of 20/40 or better. Complications associated with vitrectomy includes retinal detachment and cystoid macular oedema 3,42,43.

The incidence of retinal detachment varies from 0 % to 45.4 % (median 15.9 %) and of cystoid vascular oedems from 5 % to 22 % 44,45 (median 20 %). CME can occur several months after cataract surgery and hence adequate follow up is essential. This condition becomes chronic in 20 % eyes. Sulcus placement of a PCIOL 73 at the time of cataract surgery was associated with a lesser incidence (8%) of CME than when the eye was left aphakic (46 %) or when an ACIOL implanted. This could be due to the fact that sulcus placement of the IOL was associated with lesser traumatic cataract surgery, more amount of intact posterior capsule and a reduced iris irritation and inflammation.

Elevated intraocular pressure occurred in 46.3 % 44,45 of cases (25 % to 52 %). A clear association between anterior vitrectomy and decreased incidence of elevated IOP has been validated in various studies.

Review of recent literature 27,33,42 shows that the incidence of retinal detachment before vitrectomy was very low. This may indicate that cataract surgeons have become more experienced in dealing with complications. Resorting to use of lens loop, vectis, forceful irrigation, cryoprobe and phaco inside vitreous cavity are rarely seen in the present day.

Older studies have reported a higher incidence of retinal detachment following vitrectomy to retrieve the lost nuclear fragment (15.9 %). However recent studies show a trend towards a much lower incidence of 5.15 %. Smiddy et al has provided a likely explanation for the lower rates of RD (retinal detachment) after PPV; that is a meticulous examination of fundus periphery with scleral indentation at conclusion of vitrectomy for open iatrogenic breaks and use of ultrasound only when necessary. The proposed association between delayed vitrectomy and retinal detachment has not been validated.

Thus irrespective of its timing, pars plana vitrectomy for dropped nuclei is associated with a good clinical out come as well as a low incidence of complications such as retinal detachment and endophthalmitis.
Conclusion

With the available vitreoretinal microsurgical techniques, successful and safe management of dislocated nucleus or lens fragments is possible. Effective visual rehabilitation in these patients is possible with the availability of better designs and more bio-compatible intraocular lenses.

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Viral keratitis is a common cause for unilateral blindness in both developing and developed countries. The frequency of viral keratitis has become greater because of the role of newer antibiotics in eliminating the bacterial flora.

Even though both DNA and RNA viruses are responsible for keratitis, common corneal infections are caused by DNA viruses-(herpes group viruses- (Type I, Type II, Type III -VZV, Type IV-CMV, Type V-EBV) and adenoviruses).

Herpes Type I- and Type II (Fig. 1) cause labial infection and keratitis which are its commonest manifestations.

Prevalence
Studies show that 90% of adults have positive herpes simplex antibodies but all are not symptomatic. Transmission occurs by direct contact with lesions, secretions or sub clinically.

After entry into the host and primary infection with viral replication within the end organ, HSV travels in a retrograde fashion to various sensory ganglia, most commonly the trigeminal ganglion and possibly the brain stem.

Clinical Manifestations
1) Congenital Ocular Herpes
Is rare and is acquired from genital herpes in the mother during birth. Ocular lesions include lid lesions, conjunctivitis, epithelial and stromal keratitis and cataracts.

2) Primary Ocular Herpes
It is the first infection of a non immune patient. By the age of 5 years, 60% population has been infected by HSV. But only 6% of these develop clinical manifestations which typically affects perioral region rather than the eye. May present as typical epithelial lesions such as A/C follicular conjunctivitis, keratoconjunctivitis with pre auricular lymphadenopathy, punctate keratitis evolving into microdendrites etc. Condition is self limiting and the usual course of the disease is 10 days.

3) Recurrent Ocular Herpes
33% of recurrence is reported within two years in patients with two prior infections.

A clinical manifestations of infectious keratitis and immunological disease can affect all levels of the cornea. Bilateral disease is noted in immuno compromised patients.

Whether primary infection is asymptomatic or symptomatic, all patients once infected with HSV at
any site become viral carriers with the virus residing in a latent stage in the sensory ganglia or cornea.

**Trigger factors for reactivation-**

- Fever, systemic illness
- UV light
- Intraocular surgery
- Ocular trauma
- Trigeminal ganglionectomy
- Laser treatment on the eye
- Topical steroids, latanoprost, etc.

**Clinical types of recurrent ocular herpes**

1) Infective epithelial keratitis
   a) Corneal vesicles
   b) Dendritic ulcers
   c) Geographical ulcers

2) Non infectious epithelial keratitis
   Neurotrophic keratopathy

3) Stromal keratitis
   a) Necrotising stromal keratitis- Infective
   b) Immune stromal (interstitial) keratitis- Non-infective
   i) Antigen antibody complement mediated
   ii) Lymphocyte mediated

4) Endothelitis
   a) Disciform
   b) Diffuse
   c) Linear

**1) Infective Epithelial Keratitis**

**Superficial Punctate Keratitis :** Earliest manifestation of viral infection of cornea are corneal vesicles/superficial punctate keratitis with decreased corneal sensation (Fig. 2a & 2b).

Symptoms include pain photophobia, watering, decreased vision

S/L biomicroscopy shows multiple white sub epithelial non staining infiltrates

**Dendritic ulcer**

This is a common presentation. It is a branching linear lesion with terminal bulbs and swollen epithelial borders that contain live virus. It extends up to the basement membrane. Even after healing of the ulcer, the dendritic ulcer may result in abnormally appearing epithelium for several weeks. This epitheliopathy is dendritic in shape but is not ulcerated and does not require treatment (Fig. 3a & 3b).

**Geographic ulcer**

This is a widened dendritic ulcer with swollen epithelial borders that contain live virus (Fig. 4a and 4b). It extends through the basement membrane and has scalloped borders. May be associated with previous use of steroids.

**Sequelae of Infective Epithelial keratitis**

Infective epithelial Keratitis may resolve completely or may progress to stromal disease.

**2) Neurotrophic keratopathy**

Factors contributory:-
   a) Impaired corneal sensation
   b) Decreased tear secretion
   c) Excessive use of antivirals
Patients who have had epithelial disease are at risk to develop this entity. This is neither immune nor infectious. The epithelial defect is oval in shape with smooth borders and eventually leads to stromal ulceration (Fig. 5).

Complications are stromal scarring, corneal neovascularisation, secondary bacterial infection, necrosis and perforation of cornea.

3) Stromal Keratitis

Rare in primary infection. Stromal disease accounts for 20-48 % of recurrent ocular disease.

a) Necrotising stromal keratitis

The clinical findings are necrosis, ulceration and dense infiltration of the stroma with an overlying epithelial defect (Fig. 6). The combination of replicating virus and severe host inflammatory response leads to destructive intrastromal inflammation. The ulcer resembles microbial keratitis.

b) Immune stromal (interstitial) keratitis

i) Non infective antigen antibody complex mediated stromal keratitis which manifests as a recurrent disease (Fig 7).

Retained viral antigen within the stroma triggers an intrastromal inflammation. Punctate stromal opacities with intact overlying epithelium is characteristic. The stromal infiltration may be focal, multifocal or diffuse accompanied by anterior chamber reaction and ciliary flush. It may lead to disciform keratitis at any level of the cornea. Stromal neovascularisation may occur.

ii) Wesseley ring

It is an immune phenomenon seen in the cornea due to deposition of immune complexes and inflammatory cells around the antigen.

iii) Limbal vasculitis

is often confused with staphylococcal marginal keratitis associated with infective blepharitis

Symptoms in limbal vasculitis are pronounced, neovascularisation is common. Corneal sensation is diminished and the lesions progress centrally, and can affect any meridian (Fig. 8).

b) Immune stromal (interstitial) keratitis

ii) Non infective lymphocyte mediated limbal vasculitis (Fig. 9)

Endothelitis

Many patients with HSV disease develop stromal oedema without stromal infiltration. They present with
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KPs, overlying stromal and epithelial oedema and iritis. Disciform keratitis is actually an endothelitis as the inflammatory reaction is not a reaction of stroma but at the level of the endothelium. May clinically present as disciform, diffuse or linear lesions (Fig. 10).

4) Electron microscopy
5) Growth of virus in tissue culture
6) Detection of viral antigen
7) Serological tests
8) PCR-Specific but costly

Treatment of IEK (Infective epithelial keratitis)

1) Debridement of the involved epithelium with a sterile applicator followed by local antiviral treatment- 3 % Acyclovir eye ointment 5 times daily for 2 weeks

2) In recurrent cases oral ACV 200mg 5 times daily for 2-3 weeks

Occasional patients develop more than 2-3 recurrence/year.

Such patients require oral Acyclovir 400mg bid 6 months to 2 years or newer antiviral drugs like Famcyclovir (Famvir) 500 mg 3 times a day or Valacyclovir(valtrex) 500mg 3 times a day are also effective and more convenient.

In the absence of stromal disease corticosteroids are contraindicated in infective epithelial keratitis

In Epithelial trophic ulcers(meta herpetic ulcers) structural damage occurs in basement epithelium and anterior stroma due to recurrent scarring and loss of sensation which results in persistent sterile ulceration. Damaged basement membrane takes atleast 12-15 weeks to heal.

Management of trophic ulcers

Antiviral therapy is not indicated in patients with trophic ulcers.

Cauterization can worsen the condition by further damaging the basement membrane

Lubrication with artificial tears, patching, therapeutic contact lenses can be tried.

Tissue adhesive with soft contact lenses till the epithelial healing closes the defect may help.

Conjunctival transplantation, tarsorrhaphy, conjunctival flapping or KP are the other therapeutic options.

Iridocyclitis

It may develop concomitantly or subsequently with HSV infection. A trabeculitis is a common accompaniment leading to secondary glaucoma, iris atrophy, corneal decompensation etc. (Fig. 11)

Fig. 9. Viral endothelitis

Fig. 10. Disciform endothelitis

Fig. 11. Zoster ophthalmicus

**DIAGNOSIS OF VIRAL KERATITIS**

1) Clinical presentation
2) Decreased or absent corneal sensation-in herpetic keratitis
3) Exam of infected samples cytology for inclusion bodies

4) Electron microscopy
5) Growth of virus in tissue culture
6) Detection of viral antigen
7) Serological tests
8) PCR-Specific but costly
Cyclopentolate is avoided since this drug tend to attract polymorphs

**Treatment of herpetic immune stromal / endothelial disease**

1) Artificial tears if disease is mild and not involving the visual axis.

2) Cycloplegics atropine/homatropine

3) Topical steroids 0.1% dexamethasone or 1% prednisolone eye drops every 3 hours for severe disease to 0.12% prednisolone eye drops daily QID for less severe cases and tapered slowly.

4) Topical antibiotic prophylaxis at higher steroid dosage

5) Topical Acyclovir 5 times if steroids are used more than 2 times/day.

6) If the epithelium is ulcerated topical steroids are replaced by 1% medroxy progesterone

7) Antiglaucoma therapy as and when needed

8) Consider systemic Acyclovir 400mg bid in place of topical ACV in recurrence IEK.

9) “Kerato plasty SHOULD BE DEFERRED”-until the eye has been quiet on little or no steroid treatment for several months. HSV – IK is the commonest form of ocular herpes to recur in a new graft.

Penetrating Keratoplasty should be considered when there is corneal perforation that cannot be treated with other modalities, especially in the presence of a central stromal scar.

PK helps to eliminate the corneal antigenic materials responsible for immune mediated keratitis.

Post operatively Acyclovir 400mg bid for 1 year is given along with frequent topical steroids to improve graft survival.(12-19% recurrence)

**Herpes Zoster Keratitis**

Varicella zoster virus is also known as Herpes virus III differs clinically and antigenically from herpes simplex. Chicken pox and zoster are different entities caused by the same virus.

**Aetiopathogenesis**-Chicken pox represents primary infection after exposure of a non immune person to varicellazoster virus. This virus then becomes latent in the cells of dorsal root ganglion through out the body. Reactivation may occur resulting in characteristic dermatological eruption of the herpes zoster, common in older age group. Herpes Zoster in younger age indicates immunosuppression.

Commonly involves ophthalmic division of 5th cranial nerve. Nasociliary involvement in herpes zoster ophthalmicus is associated with 76% chance of ocular complications. Herpes Zoster virus lies latent in trigeminal ganglion, reactivation is preceded by a prodrome of malaise, fever and head ache which represents viremia.

HUTCHINSON’s sign describes vesicles at the side and tip of nose. It indicates ocular involvement which occurs in 50% cases (Fig. 12).

Clinical manifestations are variable. These include punctuate epithelial keratitis, associated with follicular conjunctivitis, pseudodendritic keratitis, anterior stromal infiltrates or nummular keratitis, kerato uveitis, endothelitis, serpiginous ulceration, sclerokeratitis, corneal mucous plaques, disciform keratitis, neurotrophic keratopathy, exposure keratopathy and interstitial keratitis

**Pseudo dendrites**-are broader, more plaque like and without central ulceration. Varicella Zoster Virus is present in these lesions.

**Nummular keratitis**

Represent stromal reaction to soluble viral antigen diffusing into the anterior stroma/or direct viral toxicity. These lesions appear as hazy granular infiltrate just under the Bowmans membrane, they are markers of previous Herpes zoster virus inflammation (Fig. 13).
Management

To prevent-severe ocular hazards and promote healing of skin lesions as the crust formation leads to severe trigeminal neuralgia.

- high doses of oral Acyclovir 800mg 5 times/day 10 days started within 72 hours of onset of disease
- For severe cases parenteral Acyclovir 5-10 mg/Kg IV 8th hourly for 8-10 days
- Topical antibiotic to skin lesion

Nummular keratitis, disciform keratitis, sclero keratitis all respond to topical steroids. 1% prednisolone acetate eyedrops 6th hourly along with cycloplegics and antibiotic cover is given.

Neurotrophic keratitis is managed conservatively using lubricants, patching, therapeutic contact lens, tarsorrhaphy and conjunctival flapping

Corneal mucous plaques requires the use of topical steroids supported by lubricants and mucolytic agents like acetyl cysteine 10%

Penetrating Keratoplasty has a poor success rate and hence a period of 1 year after the disease become quiescent is advised. Healthy donor tissue, postoperative protection of epithelium with lubricants, and tarsorrhaphy increase the rate of success of penetrating keratoplasty.

Live attenuated varicella zoster vaccine is used in the prevention of varicella zoster infections.

Adenoviral Keratitis

This is commonly associated with fever and upper respiratory infection

Keratitis is seen associated with pharyngo conjunctival fever caused by type 3,4,7 adenoviruses. (30 %), or more commonly with epidemic keratoconjunctival fever (80 %)

Clinical Manifestation

Stage I - punctate epithelial erosions (7-10 days)
Self limiting in 2 weeks. Antivirals are not useful.
Stage II - focal white subepithelial opacities which represent an immune response to the virus (Fig. 14).

Stage III - anterior stromal infiltrates which fade over months or years.
These are associated with preauricular lymphadenopathy.
This needs supportive and symptomatic treatment. Steroids suppress inflammation but lesions can recur on tapering steroids.

Epstein Barr virus-infection can produce infectious epithelial keratitis, microdendritic ulcers, stromal keratitis etc. Epithelial keratitis responds to topical Acyclovir or trifluoro thymedine while stromal keratitis needs topical steroids as in herpes disease.
In case of *Molluscum Contagiosum* (pox viruses) the umbilicated wart like growths along the lid margins are associated with punctate keratitis as a result of virus shed into the tear film. Untreated patients develop pannus. Therapy is surgical removal of growths by excision, chemical cautery or cryotherapy. HIV patients are particularly affected by this virus.

Cytomegalovirus infection is the most common virus transmitted in utero. In AIDS patients it causes opportunistic infection of retina. Corneal manifestations include linear stellate endothelial deposits in reticular pattern most often in inferior cornea. These do not resolve even after effective treatment for CMV retinitis.

Most of the RNA viruses like mumps, measles viruses produce only minimal epithelial keratitis most of which are insensitive to acyclovir. They need only symptomatic and supportive treatment.

**Conclusion**

Keratitis is the most serious manifestation of viral infection of the eye especially due to herpetic viruses. All layers of cornea can be involved by the acute or relapsing forms of the disease.

Now specific antiviral drugs are available with less toxic effects on the normal cells.

Long term therapy seems to be beneficial in chronic cases. Only herpes group virus respond to most of the antivirals.

Topical antiviral therapy can be stopped in two weeks. Topical corticosteroids are beneficial for inflammatory components like moderate to severe stromal keratitis, uveitis, trabeculitis, endothelitis etc. than infective epithelial disease. Once you start the patient on topical steroids prolonged maintenance dose may be required.

Role of systemic steroids are controversial. But oral antivirals combined with oral steroids relieves pain more than oral antiviral alone in severe form of the disease.

Severe corneal scarring requiring keratoplasty may be undertaken if there is no active disease and under cover of prophylactic oral antivirals for a minimum of one year with initial frequent steroid drops to reduce inflammation.

Reactivation of viral keratitis can be initiated by any type of laser treatment, antiglaucoma drugs like latanoprost, topical steroid therapy, exposure to uv light, intraocular surgeries etc. So a proper history and thorough examination is mandatory to rule out pre existing viral disease prior to any invasive procedure on the eye.

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In a lighter vein

…………By Degrees

RRV

There was a trilogy of Malayalam movies featuring two job seekers – turned – C.I.D.s. One of them boasts that he had a ‘first class in B.Com.’, while the other says that ‘pre-degree is not a bad degree at all’. They mockingly reflect the mentality of our society, where the number of alphabets suffixing your name is given too much importance. The more, the better, it seems.

When I came out of the Medical College in the second half of seventies, post-graduates were not as common as was later. But people who had passed B.Sc. used to add that too before the M.B.,B.S. I had a class-mate who used to put B.Sc.(Spl.), because he had passed ‘Special’ B.Sc., with two main subjects (totally irrelevant to his profession). But that is nothing compared to another veteran whose qualifications, M.A.(Hum.), M.B.,B.S., used to puzzle me every time I passed his clinic. When I got to know him better, I asked him about the M.A.(Hum.) part. It was M.A. in Humanities form Aligarh Muslim University! In the early eighties, when P.G. degree was becoming more and more desirable (nee essential), lots of ‘Academies’ and ‘Colleges’ cashed in on that. Many are the G.P.s who still add F.I.C.A. (Fellow of International College of Angiology, nothing less) to their name.

You know, they are not to be blamed. The general public associates more degrees with more expertise. In our hospital we used to have a physician who had taken M.R.C.P. from Edinburgh and Glasgow, and wrote both with an (E) and (G) suffixing them. Once I heard a patient comment to another. “Dr. Menon has passed M.R.C.P from England and ‘Germany’.”.

I used to think that this affinity was peculiar to our society. But one of my class-mates who has returned from ‘States’ recently gave a notification in the Kerala Government Gazette about the change in his signature wherein he had added “MBBS” at the end. He says it is the custom there to add ‘MD’ to ones signature.

I had a relative who has passed M.Sc. and M.A. and went on to get an M.B.A. and later added L.L.B. and L.L.M. to them. While working on his doctoral thesis, he fell ill and died. They say he killed himself by degrees!

**Tail piece**: My eccentric friend says he is planning to start an Academy of Medically and Ophthalmologically Useful Specialties. For five thousand rupees, he says he will make me a ‘fellow’ and then I can add F.A.M.O.U.S. to my suffixes. Tempting, isn’t it?
The Omega – 3 Revolution

Dr. Meena Chakrabarti MS, Dr. Valsa Stephen MS, Dr. Sonia Rani John DNB, Dr. Arup Chakrabarti MS

Omega – 3 fatty acids are considered as essential fatty acids. They are essential to human health, but cannot be manufactured by the body. Hence these essential fatty acids are obtained from food. A rich source of these essential fatty acids is found in fatty fish such as salmon, tuna and halibut, other marine life like algae and krill, certain plants and nut oils. Also known as long chained poly unsaturated fatty acids (LCPUFA), they form a family of unsaturated fatty acids that have in common a carbon- carbon double bond in the n – 3 position (i.e. the third bond from the methyl end of the fatty acids).

Nutritionally important omega- 3 fatty acids include.
1. α Linolenic acids (ALA)
2. Eicosapentaenoic acid (EPA)
3. Docosahexaenoic acid (DHA)

Although the human body cannot synthesize n-3 fatty acids denovo, it can form 20 and 22 carbon unsaturated n-3 fatty acids from the 18 carbon n-3 fatty acid, α linolenic acid. These conversions occur competitively with n – 6 fatty acids which are essential closely related chemical analogues that are derived from linolenic acid. Clinical studies indicate that the ingested ratio of n-6 to n-3 fatty acids is important to maintain cardiovascular health. Both n-3 and n-6 fatty acids are essential and human beings consume them in their diet n-3 and n-6 competes for the same metabolic enzymes. Thus the synthesis of the longer n-3 fatty acids from linolenic acid is competitively slowed down by the n-6 analogues. Accumulation of LCPUFA (n-3 fatty acids) is more effective when they are directly obtained from food or when competing amounts of n-6 analogues do not greatly exceed the amounts of n-3 fatty acids. Thus the ratio of n-6: n-3 will significantly influence the ratio of ensuing eicosanoids (hormones).

Generally grassfed animals accumulate more n-3 than do grain fed animals that accumulate more of n-6. It is necessary that the n-6: n-3 be balanced with the healthy ratios ranging from 1:1 to 4:1. The typical western diet provides a ratio of 10:1 to 30:1 dramatically skewed towards n-6. The eicosanoids produced from omega – 3 fatty acids are anti inflammatory in function when compared to those produced from n-6 arachidonic acid (prostaglandins, prostacyclin, thromboxane, leukotrienes). If both n-3 and n-6 fatty acids are present, they will ‘compete’ to be transformed, and so the ratio of n-3: n-6 directly affects the type of eicosanoids produced.

Omega 3 fatty acids have been found useful in treating a wide varieties of disorders. These include
1. High Cholesterol:- Administration of Omega 3 fatty acids has been reported to lower the triglyceride levels by 45 % and the VLDL cholesterol by more than 50 %.
2. Coronary Heart Disease: - Consumption of EPA and DHA fatty acids may reduce the risk of coronary heart disease. Fish oil stimulates blood circulation, increases breakdown of fibrin, a compound involved in clot and scar formation, reduces blood pressure, significantly reduces triglyceride levels and thereby reduces the risk of secondary and primary heart attack.
3. Reduces the risk of ischemic and thrombotic stroke\textsuperscript{9}: However, very large amounts of omega-3 polyunsaturated fatty acids should be used with caution as they increase the risk of developing a hemorrhagic stroke \textsuperscript{10}.

4. Link between n-3 fatty acids and cancer prevention: Review of studies failed to find clear effects of n-3 fatty acids on prevention of cancer \textsuperscript{11}.

5. In patients with recent myocardial infarction, treatment with 1 gram per day of n-3 fatty acids reduced the occurrence of death due to a cardiac event by 20\% - 45\% \textsuperscript{12}.

6. n-3 fatty acids supplementation has been found useful in depression, autism, and attention-deficit hyperkinetic (ADHD) disorders \textsuperscript{13}.

7. n-3 fatty acid supplementation in children from 9-12 months resulted in an improvement in immune function maturation with no apparent reduction in immune activation \textsuperscript{14}.

8. Anti-inflammatory action of n-3 fatty acids translate into clinical benefits in patients with neck pain, rheumatoid arthritis and, menstrual pain etc.

9. Beneficial effects also have been seen in achieving weight loss, combating depression and osteoporosis, healing of burns and reducing sensitivity in patients with photodermatitis and also in reducing the bowel inflammation when used along with sulfa salazine in patients with inflammatory bowel disease.

Although this meta analysis suggests that consumption of fish and foods rich in omega-3 fatty acids may be associated with a lower risk of AMD, there is 1) insufficient evidence in current literature, 2) Few prospective studies, 3) No randomized trials to support their routine consumption for AMD prevention.

The role of Omega-3 long chained PUFA in health and diseases of retina was studied extensively by Sangiovanni et al \textsuperscript{16}.

- Omega-3 long chained PUFA (LCPUFA) exhibit cytotherapeutic actions contributing to a number of anti angiogenic and neuroprotective mechanisms within the retina Omega-3 PUFAs modulate metabolic process and attenuate effects of environmental exposures that activate molecules implicated in vasoproliferative and neurodegenerative diseases of the retina. These processes include ischemia, chronic light exposures, oxidative stress, inflammation, cellular signaling mechanisms and ageing.

- Many biologically active molecules are affected by these effects. These molecules include compounds classified as eicosanoids, angiogenic factors, matrix metalloproteinases, reactive O\textsubscript{2} species, cyclic nucleotide neurotransmitters and neuromodulators, proinflammatory and immuno regulatory cytokines and inflammatory phospholipids.

**How Is Omega – 3 Fatty Acids (LCPUFA) Status Related To Retinal Structure And Function?**

Docosahexaenoic acid (DHA) a major dietary LCPUFA is a major structural lipid of the retinal photoreceptor outer segment membranes.

Biophysical and biochemical properties of DHA may affect photoreceptor membrane function by altering permeability, fluidity, thickness and lipid phase properties.

They affect retinal cell signalling mechanisms involved in phototransduction and operate in signalling cascades to enhance activation of membrane bound proteins. They may also be involved in rhodopsin regeneration. Thus tissue DHA insufficiency which is associated with alteration of the retinal functions can be ameliorated by DHA supplementation.

**Omega 3 fatty acids consumption and retinal disease**

Elaine W T, Chong\textsuperscript{15} and colleagues conducted a systematic review of studies published before May 2007 evaluating the fish consumption and overall Omega-3 fatty acid intake for the prevention of AMD.

A total of 9 studies were identified with 89,974 participants including 3,203 individuals with AMD.

When the result of all 9 studies were combined, a high dietary intake of Omega-3 fatty acids was associated with a 38\% reduction in the risk of late (more advanced) AMD, while eating fish twice a week was associated with a reduced risk of both early and late AMD.
What Evidence Exists To Suggest That LCPUFA Modulate Factors And Processes Implicated In Diseases Of Vascular And Neural Retina?

On a basic level Omega – 3 fatty acids influence 1) Retinal gene expression, 2) Cellular differentiation and Cellular survival.

Omega – 3 PUFA activates nuclear hormone receptors (peroxisome – proliferation – activator receptor alpha and retinoid X receptor) that act as transcription factor for molecules that modulate reduction – oxidation sensitive and pro-inflammatory genes which prevents vascular endothelial cell dysfunctions and vascular remodeling. Inhibition of vascular smooth muscle cell proliferation, inducible nitric oxide synthase production, interleukin – 1 induced cyclooxygenase (COX-2) production, thrombin induced endothelin-1 production also act to prevent vascular remodeling.

EPA, the parent fatty acid and substrate for DHA is converted into Arachidonic acid derived eicosanoids which are responsible for abnormal retinal permeability and retinal inflammation. EPA downregulate a VEGF specific Thyrosine Kinase receptor NF Kappa B (Thyrosine Kinase Nuclear Factor Kappa B ) activation and expression resulting in VEGF expression, endothelial cell migration, endothelial cell proliferation, microvascular permeability, endothelial cell release of MMPs, interstitial collagenases and endothelial cell tube formation. The mechanism of VEGF receptor down regulation is believed to occur at the tyrosine kinase nuclear factor Kappa B (NF Kappa B) within a nuclear transcription factor that upregulates COX-2 expression, intracellular adhesion molecule, thrombin and nitric oxide synthase all 4 factors associated with vascular instability. The anti inflammatory activity of omega – 3 fatty acids is mediated through decreased activity of Tumour Necrosis Factor Alpha (TNFα). The retina of mouse fed on Omega – 3 fatty acid diet shows increased production of anti-inflammatory compounds which also potentially protect against neovascular proliferation. These compounds include Neuroprotectin D1, resolvin E1 etc. Omega – 6 fatty acid produce the reverse effect releasing proinflammatory eicosanoids like thromboxane, prostacyclins and leucotrienes. This action is also mediated through TNF. Omega – 3 fatty acids may help decrease vision loss in patients with wet or neovascular AMD, a disease that also involves abnormal vessel growth. This possibility is now being explored by the NEI in the AREDS 2 study coordinated by Emily Chew J, Paul Giovanni, both co –authors of the animal study. AREDS 2 will test the roles of oral supplementation with lutein / zeaxanthine and or omega – 3 long chain poly saturated fatty acids in the management of AMD.

Research on mice model led by Lois Smith et al studied retinopathy after feeding the mice diets that emphasised either omega -3 fatty acids (Comparable to a Japanese diet) or Omega – 6 fatty acids (Comparable to a western diet).

Mice on Omega – 3 diet rich in DHA and its precursor EPA have less initial vessel loss in the retina than Omega – 6 fed mice (the area with vessel loss was 40 – 50 % smaller). As a result the Omega – 3 group had a 40 – 50 % decrease in pathological vessel growth. Their studies suggest that after initial loss, vessels regrow more quickly and efficiently in the Omega – 3 fed mice. This increases the O2 supply to retinal tissue, resulting in a dampening of the inflammatory alarm signals that lead to pathologic vessel growth. Because Omega – 3 fatty acids are highly concentrated in the retina, a mere 2 % change in dietary Omega – 3 intakes was sufficient to decrease severity by 50 %.

Premature infants especially lack in Omega – 3 fatty acids because they miss this nutrient from their mothers – a transfer that normally happens in the 3rd trimester of pregnancy. Clinical trial at children’s hospital, Boston will soon begin testing the effects of Omega – 3 supplementation in premature babies who are at risk of vision loss due to Retinopathy of Prematurity.

**Health risks:** - Suspected risk of EPA and DHA n-3 fatty acids may include

1. Increased bleeding if over used (normally over 3 gms per day) by a patient who is also taking aspirin or warfarin
2. Hemorrhagic stroke (only in cases of very large doses)
3. Reduced glycaemic control among diabetics
4. Suppression of immune and inflammatory responses and consequently decreased resistance
to infection and increased susceptibility to opportunistic bacteria.

5. An increase in concentration of LDL cholesterol in some individuals.

**Cardiac risk:**
Persons with congestive heart failure, and chronic recurrent angina should be prudent before taking n-3 fatty acids.

**Possible Drug Interactions:**
If you are currently being treated with any of the following medications you should not use omega-3 fatty acid supplements including EPA, DHA and ALA without first consulting your health care provider.
1. Blood thinners
2. OHA like glipizide, glyburide, glucophage or Insulin
3. Cyclosporine
4. Cholesterol lowering medications.
5. NSAIDS

**Dietary Sources:**
Fish, plant and nut oils are the primary dietary sources of omega-3 fatty acids. EPA and DHA are found in cold water fish such as salmon, mackerel, tuna, halibut and sardines ALA is found in flaxseeds, canola (rapeseed) oil, soybeans, soybean oil, pumpkin seed oil, walnuts and walnut seed oil. Other sources of n-3 fatty acids include sea life such as krill and algae.

**Available Forms**
EPA and DHA can be taken in the form of fish oil capsules. Dosing with fish oil supplements should be based on the amount of EPA and DHA in the product and not on the total amount of fish oil. Supplements vary in the amount of Omega-3 fatty acids. Recommended dosage is 170-560 mg EPA and 72-310 mg of DHA.

In young children and pregnant women, dosing with fish oil supplements should be performed with caution due to the presence of free heavy metals such as mercury, lead and cadmium.

**Precautions:**
Because of the potential for side effects and interaction with other medication, dietary supplements should be taken only under the supervision of a knowledgeable health care provider.

Omega-3 fatty acids should be used with caution in people who bruise easily, have a bleeding disorder, or are taking blood thinning medications like warfarin, clopidogrel as excessive amounts of n-3 fatty acids may cause bleeding.

In fact people who eat more than 3 gm of n-3 / day (equivalent to 3 servings of fish / day) may be at an increased risk of hemorrhagic stroke.

Fish oils can cause flatulence, bloating, belching, diarrhea etc. Time release preparations may reduce these side effects. People with Type 2 diabetes may experience increase in fasting blood glucose levels while taking fish oil supplements.

Although regular consumption of EPA and DHA may reduce the risk of AMD, a recent study has shown that ALA may substantially increase the risk of this disease. Further research is warranted in this area and till that time EPA and DHA supplements may help.

Similar to the findings in macular degeneration, fish and fish oil products may protect against prostate cancer, but ALA may be associated with increased risk of prostate cancer in men. More research is required in this area also.

Fish and fish oil may contain potentially harmful contaminants such as heavy metals, dioxins, poly chlorinated biphenyls (PCBs). Certain sea fish (Mackerel, swordfish, shark, tile fish) contain higher levels of mercury and excess consumption should be avoided. Unrefined fish oil preparation may contain pesticides also.

There is a wealth of research that supports the health benefits of LCPUFAs. Its health benefits are multifarious and almost all disease conditions benefit. Definite cardio protective and cholesterol lowering effect, reduction of macular degeneration, prevention of depression and improvement in muscle mass, protection from cancer......... to recount a few of its actions, makes fish and fish oils a “panacea of cures” for all age-related disorders in the senior citizens. You would be wise to stock your fridge with tuna and mackerel! A fishy
revolution is in progress despite a few pinpricks by way of negative study results demonstrating contrary findings (.......... is there something fishy about fish oils!!).

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An Unusual Presentation of Pituitary Macroadenoma

Dr. Indu N MS, Dr. Anuradha Rao MS

Abstract

It is rare for a pituitary adenoma to present with cranial nerve palsy. Prolactinomas and non-functioning tumors are the common pituitary tumors which attain large sizes (macroadenomas). We are presenting two such cases where third cranial nerve palsy was the presenting symptom. A thorough ocular evaluation was done in both cases. One patient presented with unilateral ptosis, a dilated pupil and restriction of elevation in right eye. Another patient presented with unilateral ptosis and ill sustained pupillary reaction in the left eye. MRI evaluation in both cases revealed pituitary macro adenomas. One was an ACTH producing clinically nonfunctioning tumor, and other was a prolactinoma. It is rare for both the tumors to present as cranial nerve palsy. The proximity of the anterior pituitary to the third cranial nerve in cavernous sinus makes it more vulnerable to ocular motor nerve impairment.

Key words – pituitary adenoma, ptosis, third nerve palsy.

Introduction

Pituitary adenomas account for 10-15 % of intracranial neoplasms. They can cause anterior pituitary hormonal imbalance or symptoms and signs related to invasion of surrounding structures. Pituitary tumors are classified by Hardy according to size and invasive characteristics. Stage I are microadenomas (less than 10mm in diameter). They may cause hormonal oversecretion but are not associated with structural problems. Stage II tumors are macroadenomas (greater than 10mm) with or without suprasellar extension. Stage III tumors are macroadenomas that locally invade the floor of the sella and may cause sellar enlargement and suprasellar extension with multiple cranial nerve palsy. We report this case because it is rare for a pituitary adenoma to present as isolated third nerve palsy.

Case 1

45-year-old male presented to us with complaints of drooping of right eye of 2 weeks duration (Figure: 1). He was not a known diabetic or hypertensive. There was no history of visual disturbances, headache or vomiting as well as hormonal imbalance. The best-corrected vision in both eyes was 6/6. The right eye showed restriction of elevation. There was mild ptosis of 4mm with good LPS function. The pupil was 5 mm, sluggishly reacting to direct light and not reacting to consensual. Results of examination of all other cranial nerves were within normal limits. Fields, color vision and fundus examination in both eyes were normal. MRI revealed features suggestive of large pituitary macroadenoma filling sphenoid sinus and extending into nasopharynx, 3rd ventricle, right hypothalamus, right posterior cerebral artery and right crus cerebri. Both cavernous sinuses and internal carotid artery were also involved.
On histopathological analysis, the subepithelium showed tumor cells arranged in sheets and trabeculae with intervening fibrovascular core. Cells were round to oval exhibiting moderate amount of eosinophilic cytoplasm with vesicular nucleus and prominent nucleoli showing minimal pleomorphism that are PAS stain positive (Figure: 2). Immunohistochemical studies showed that ACTH was strongly positive (> 85% cells) and both GH and prolactin were negative.

He underwent endoscopic assisted trans-sphenoidal decompression. Postoperatively his ptosis improved remarkably.

**Case 2**

A 37 years old lady presented with progressive dimness of vision since last two months involving left eye along with occasional headache and inability to open the eye completely over a period of two months (Figure: 3). She also had amenorrhoea for the last six months and lactation for two days. No history of vomiting, headache, seizure and loss of consciousness. She was a known diabetic for four years and hypertensive for two months. Unaided vision in both eyes was 6/12 improving to 6/6. There was moderate ptosis with mild fullness in LE. Extra ocular movements were normal in both eyes. The pupil was dilated with ill sustained pupillary reaction. Fundus was within normal limits. She had bitemporal field loss. MRI brain showed sellar lesion suggestive of pituitary adenoma with parasellar extension. Serum prolactin levels were elevated.

She was put on medical management with oral Cabergoline (Dopamine agonist) 0.5mg twice a week with periodic hormonal assay and follow up 2.

**Discussion**

It is rare for a pituitary adenoma to present with third cranial nerve palsy. There are only a few reports in literature 3-7. It could be due to tumor invasion or apoplexy 7. The onset is acute in apoplexy and in tumor invasion it is gradual. When correctly diagnosed and treated, the third nerve dysfunction appears to be reversible 6.

In both our cases, third nerve palsy had pupillary involvement unlike diabetic third nerve neuropathy in which pupillary reactivity is usually preserved 6. Lateral extension of pituitary tumor is usually associated with involvement of fourth and sixth cranial nerves, pain or numbness in the distribution of fifth cranial nerve and symptoms of compression of internal carotid artery 1. It is the third nerve being singularly involved that makes these cases different 8,9. Third nerve is liable to be easily compromised because of its close proximity to the anterior pituitary, which is the commonest site of pituitary adenoma (Figure: 4).
Prolactinomas and nonfunctioning tumours are the commonest pituitary macroadenomas. Though this is a case of ACTH producing tumour, it is clinically nonfunctional. ACTH producing adenomas account for about 10-15 % of all pituitary tumours. Most ACTH producing pituitary tumours are relatively small microadenomas less than 5mm in diameter but macroadenomas are also seen. Some ACTH secreting adenomas are clinically silent but may present unusually with a third nerve palsy as seen in our case. Only less than 10 % of pituitary tumors present with visual loss. Most of these are nonfunctioning tumors. Prolactinomas usually present with visual complaints, the most frequent objective finding being bitemporal hemianopia. However cranial nerve palsies are rarely seen in prolactinomas.

**Conclusion**

Invasive pituitary adenomas with extension to the cavernous sinus are rare and comprise 6-10 % of all pituitary tumors. Among these, third nerve palsy is very rare. Nevertheless, any patient presenting with an isolated third nerve palsy should make one suspect a pituitary adenoma.

**References**

Subcutaneous *Dirofilaria Repens* Infection of the Eyelid - A Report of Two Cases

Dr. KV Raju MS, Dr. A Anju MS, Dr. Vijayalakshmi MS

**Introduction**

Dirofilariasis is a parasitic disease of domestic and wild animals that occasionally occur in humans. Ophthalmic infection with Dirofilaria is documented all over the world, including North America, Europe, Australia, Africa, the Middle East, and Asia. Reports of this infection from India are however limited. The involvement of the eye may be periorbital, subconjunctival or intraocular. In this report we describe two cases of *Dirofilaria repens* presenting as a subcutaneous swelling of the upper eyelid.

**Case 1**

A thirty three year old female presented with a six-week history of painful swelling of the upper eyelid. There was no history of preceding trauma, injury, or visual impairment. Her medical history did not disclose any general health problems and she had no close association with domestic animals. The lesion persisted after systemic antibiotic and anti-inflammatory treatment. Ocular examination showed a tender, nodular swelling of size 3 x 2 cm over the left upper eyelid with surrounding periorbital odema. Routine laboratory tests were within normal limits. On excision, an encysted nodule was removed and a long, thin, dead worm was extracted. Histopathological examination showed inflammatory lesion with plenty of eosinophils. The worm was 9 cm in length, with a maximum width of 320 μm (Fig 1a). The anterior rounded end of the worm was observed to be wider than its posterior end, and parasite had a thick unsegmented cuticle with characteristic longitudinal ridges and cross striations (Fig 1b). Based on the morphologic features, the worm was identified as *Dirofilaria repens*. The patient had complete recovery after surgery and is asymptomatic on follow up.

**Case 2**

A forty six year old female presented with painful swelling and itching of the left eye of 4 months duration. There were no systemic symptoms or signs associated with the lesion. Physical examination revealed a mobile swelling 2 x 1cm in the upper eyelid. A complete blood count was within normal limits for all parameters. An adult white worm 8 cm in length was extracted from the nodule. Histopathology of nodule revealed dense
inflammatory infiltrates composed of eosinophils and lymphocytes. Parasitological examination identified the worm as a member of *Dirofilaria* species (Fig. 2).

**Discussion**

Human dirofilariasis is a cosmopolitan zoonosis. The dirofilaria are natural parasites of mammals and are accidentally transmitted to man by bite of zooanthrophilic mosquitos carrying infective larvae acquired from microfilaria rich blood of animal hosts parasitized with dirofilaria. Man is a suboptimal host. Dirofilaria cannot mature fully in human tissue and dies before producing microfilaria. Though nearly forty species of dirofilaria have been identified, only a few have been reported to cause human infection. *Dirofilaria immitis* and *Dirofilaria* of the subgenus *Nochtiella* (*repens, tenuis, ursi, subdermata*) are the two subgenera. The species vary according to the geographical area, with *D. tenuis* transmitted by raccoons being common in United States and *D. repens* mainly transmitted by dogs, cats and foxes in Europe, Middle east and Southeast Asia. Clinical manifestations after infection include nodules in subcutaneous tissues, muscles, and visceral organs. Ophthalmic dirofilariasis is transmitted to humans by common insect vectors like *Anopheles*, *Culex* and *Aedes* mosquitoes. Most cases with ophthalmic infection present with pain in the eye, redness, blurred vision, localized pruritis, hyperemia of conjuctiva, swelling of eyelids and sometimes sensation of movement under the skin or conjunctiva. However allergic reaction with urticaria, facial oedema and fever may occur. In an extensive review of about 400 cases of human infection with *D. repens*, the majority of worms presented within nodules in subcutaneous tissues on the upper half of the body, with the largest number localized around the eyes, in the eyelids or under the conjunctiva. Rarer ocular presentations masquerading as subcutaneous tumor of the eyelid and intravitreal location have also been reported. Symptoms appear mostly weeks or months after infection with microfilaria. The first case of human ocular dirofilaria was reported by Addario in 1885 from Milan, Italy. These worms were earlier referred to as *Filaria conjunctiva* and later as *D. conjunctiva* because of their frequent association with orbit. Cases of *D. repens* infection have been reported in Italy, France, Greece, Spain, Turkey and Israel. Reports of human ophthalmic dirofilariasis from India are very few. The first three cases of human ocular dirofilarial infection in India were reported from same part of India (Kerala). The correct diagnosis of the parasite is usually made with typical gross morphological features and histological examination. *D. repens* have rounded anterior end with buccal cavity and the longitudinal ridges are broader than long, less distinctly raised and appear to have a more branching effect. The dyes used in examining the transverse sections are hematoxylin-eosin (HE) and periodic acid-Schiff (PAS). In majority of instances the parasites are found in excised nodule and tissue biopsies. Less commonly they are removed intact from the tissues. The diagnosis is usually established with the surgical removal of the adult worm. The only cure currently known is surgical excision. In our case, both patients made good recovery after surgery.

**References**


Henri Parinaud (1844 - 1905)
An Unassuming Lovable French Ophthalmologist...

Prof. Padmaja Krishnan MS

Henri Parinaud, one of the ‘greats’ of French Ophthalmology, came of humble stock. He was born to a poor locksmith on May 1, 1844 at Bellac, Haute-Vienne in France.

At 13, he was sent to study at the seminary in Ajain, but the death of his father in 1863 put a temporary halt to his education. He started giving private tuitions to make money for his studies while providing for his mother and brothers.

The money he made enabled him to begin his medical education at the University at Limoges in 1865. He moved to Paris in 1869 but the Franco-Prussian War which broke out in 1870 once again interfered with his studies.

Parinaud joined the Red Cross ambulance service and saw action at Metz. His role in evacuating the wounded from Château d’un earned him a medal for unusual bravery from the prime minister. This was used by the writer Ludovic Halévy in one of his stories.

After the war, Henri returned to Paris to continue his studies. His thesis at medical school, “A study on the optic nerve in meningitis of infants”, earned him the respect and recognition of many in the field including Charcot.

He worked under Charcot at the Salpetrière and developed an interest in Neurology working on multiple sclerosis, ophthalmoplegic migraine, hysteria, supranuclear lesions and concomitant squint. He is best known for describing a syndrome of vertical gaze palsy, convergence-retraction nystagmus and light-near dissociation, caused by dorsal midbrain lesions, typically pinealomas. This has come to bear his name.

Henri worked on the physiology of vision- the role of the visual receptors, light sense, night-blindness and colour vision.

He also described a unilateral conjunctivitis with pre-auricular and cervical adenitis and fever often associated with cat-scratch disease. This was in 1889 and more than two decades before the agent of tularemia was discovered. The eponymic term Parinaud’s oculoglandular syndrome was given to this condition by the American ophthalmologist Harold Gifford in 1898.

Parinaud was a good man without interest in either fame or fortune. He was modest and endeared himself to colleagues, students and patients alike. His free clinic attracted poor patients and students from far and near. He also published extensively and was an active member of several societies of both neurology and ophthalmology.

He devoted his spare time to composing and publishing music under the pseudonym of Pierre Erick.

Henri Parinaud was never physically strong and throughout his life suffered from indifferent health. After the death of his wife in 1904, his own health deteriorated rapidly. He died in Paris on March 23, 1905 of bronchopneumonia.
Ocular Myiasis - A Case Report

Dr. Rajiv Sukumaran MS DO FRCS, Dr. Jayasree Rajiv MBBS DO

A 25 year old postgraduate student presented with complaints of marked pain, burning, itching, redness, and tearing of a week’s duration. He had consulted three other Ophthalmologists before consulting us. Right from non-specific anti-histamines to steroids like Prednisolone acetate eye drops to Acyclovir eye ointment were prescribed with no benefit.

On examination his visual acuity was 6/6 in both the eyes. There was more of fornicial congestion with no corneal staining in the right eye and the left eye was normal. Anterior chamber was normal. On high magnification a tiny mobile shining creature of less than a millimeter was seen in the inferior fornix. It was carefully removed and photographed under a microscope. Thereafter antibiotic drops was prescribed and he was reviewed the next day.

He was very comfortable and profusely thanked us. On further questioning it was revealed that he was a Veterinary postgraduate student and comes into contact with animals frequently.

Discussion

Myiasis is defined as the invasion of living animal tissue by fly larvae (maggots). When larvae invade the eye, this condition is termed ocular myiasis (OM) or Ophthalmomyiasis. Larvae most commonly attack the lids or conjunctiva (external ophthalmomyiasis). In rare instances they may penetrate into the eyeball itself (internal ophthalmomyiasis). External OM can usually be remedied without complications; however, internal OM is very serious and often results in serious damage including blindness.

In the majority of cases, OM is caused by larvae of the sheep nose bot fly (*Oestrus ovis*), although other species such as the human bot fly (*Dermatobia hominis*) are occasionally involved. The sheep nose bot fly is a large, hairy, yellowish-brown, bee-like fly. It resembles a honey bee, but is slightly smaller in size. Unlike most flies, *O. ovis* gives birth to live young larvae which are capable of parasitizing hosts immediately. In its normal life cycle, the adult female fly deposits larvae around the nostrils of sheep and goats and the larvae migrate into the sinuses. There, they mature by going through three progressively larger larval stages (instars). After a few months, the fully mature larvae (third instar) pass out of the nostrils and pupate on the ground. Adult flies emerge from the pupae approximately 3 – 6 weeks later and live for about a month.

Occasionally, the sheep nose bot fly deposits larvae near the eyes of humans living or working in close proximity to livestock. In humans, *O. ovis* larvae generally do not develop past the first instar stage, although other species may grow much larger. An interesting feature of *O. ovis* is that it can deposit larvae while still in flight. The fly darts close to the eyes or nostrils and ejects a stream of larvae into the target area.

OM is characterized by a condition similar to conjunctivitis, marked by pain, burning, itching, redness, and tearing in the affected eyes. Often these symptoms are accompanied by the sensation of a foreign body moving in the eye. Many patients report having had an insect buzzing around their face or striking them in the eye immediately prior to the onset of symptoms. In extreme cases the larvae may penetrate the mucosal sinus causing swelling, pain, and frontal headaches, or may invade the globe of the eye, causing retinal damage and blindness.
body segments, each with spines or hooks which allow them to maintain their hold on the host tissue while moving about by means of peristaltic contractions. A pair of enlarged oral hooks on the anterior end (mouth) anchors the larva firmly while it feeds on eye secretions and bits of broken tissue. The larvae are readily visible to physicians examining the eye. In some cases they can be seen traveling through the cornea. Early growth stage larvae can often be carefully extracted from the eye with fine forceps. Anaesthetic drops may be useful to immobilize the larvae during removal. Antibiotic ointments have also been used to help suffocate the larvae, thereby facilitating removal. Antibiotic ointments or drops, as well as topical corticosteroids, can be used to prevent secondary bacterial infection and reduce inflammation. Follow-up examination is advisable to rule out complications or the existence of additional larvae. If the larvae have burrowed more deeply into the conjunctiva, sinuses, or eyeball, surgery may be required.

Reference:

Occasionally, if a larva dies in the eye, a permanent nodule resembling a stye may develop.

The larvae of the sheep nose bot fly are grey-white in color and measure about 1 mm long. They have eleven
An Unusual Case of Post Vitrectomy Hypopyon

Dr Meena Chakrabarti MS, Dr Valsa Stephen MS, Dr Sonia Rani John DNB, Dr Arup Chakrabarti MS

Ghost cell glaucoma is a clinical entity that was first described in 1976 by Campbell and colleagues in eyes with long standing vitreous hemorrhage. Theoretically the process involves degradation of the red blood cells present in the vitreous haemorrhage. Rigid spherical khaki coloured cells 4-7 μm in size with clumps of haemoglobin at their periphery called Heinz bodies are formed. They find their way through the anterior hyaloid into the anterior chamber and become trapped in the trabecular meshwork leading to obstruction of aqueous humor outflow with secondary intra ocular pressure rise. Clinically a double layered khaki coloured or candy – stripe pseudo hypopyon is a pathognomonic sign.

We report a case of khaki coloured pseudohypopyon in the anterior chamber in a phakic patient who underwent vitrectomy for suspected peripheral exudative and haemorrhagic chorioretinopathy (PEHCR) in the elderly. Although the anterior chamber was filled with degraded blood products, the intra ocular pressure remained normal.

A 67 year old male presented to our out-patient department with history of sudden onset of visual blurring of 15 days duration. He was a long standing hypertensive on regular treatment and under good control. Ocular examination revealed a visual acuity of hand movements right eye with inaccurate projection of rays and 6/18 improving with pin hole to 6/6 N6 in his left eye. Slit lamp biomicroscopy revealed a sluggishly reacting pupil and blood stained aqueous filling the anterior chamber (Fig. 1). The intraocular pressure measured by the non contact air puff tonometer was 10 in the right and 11 in the left. Indirect ophtalmoscopy showed dense fresh vitreous haemorrhage precluding visualization of the fundus in the right eye. The fundus of the left eye appeared normal. Fluorescein fundus angiography of the left eye was normal (Fig. 2a &b). B Scan ultrasonography showed evidence of vitreous haemorrhage with large subretinal haemorrhage not involving the macula (Fig. 3). A diagnosis of peripheral exudative and haemorrhagic chorioretinopathy in the elderly (PEHCRE) with subretinal and vitreous haemorrhage was made. The patient was advised conservative treatment with advice to restrict physical activity. The guarded visual prognosis and the need for follow up with serial B scan USG was explained.

The patient reported for review after a month. He had a visual acuity of hand motion with inaccurate
projection in the right eye, clear aqueous, no anterior chamber reaction, a tension applanation reading of 8 mm Hg in the right and the fundus view was obscured by organised vitreous blood. B Scan ultrasonography showed total posterior vitreous detachment, no evidence of retinal detachment and peripheral subretinal blood. He underwent parsplana vitrectomy right eye. The procedure was uneventful. There was total PVD and organised yellow subretinal blood peripherally. The macula appeared normal and hence a good prognosis was expected. Over the area of organised subretinal blood peripherally a retinal tear was noticed through which khaki coloured degenerated blood products were coming out. Laser barrage as well as peripheral cryo was performed although the resultant retinal reaction was mild due to the presence of subretinal blood.

On the first post operative day, the vision was hand motion, there was 3 + flare and cells in the anterior chamber and a 1 mm khaki coloured hypopyon (Fig. 4). Tonometry was 15 mm Hg right eye and only fundal red glow could be obtained on indirect ophthalmoscopy. Anticipating ghost cell glaucoma, topical dorzolamide was started along with the routine postoperative medications. The hypopyon progressively increased and by the 5th postoperative day it filled half of the anterior chamber (Fig. 5). There was no fundus view and the intraocular pressure was 11 (RE). Conservative treatment was continued with the decision to perform an anterior chamber wash and vitreous lavage if the level of the khaki coloured hypopyon increased.

Five days later the patient reported with the hypopyon filling the anterior chamber, a tonometry value of 11 on a single antiglaucoma medication and no pain. AC wash and vitreous lavage was performed under local anesthesia. There was good red glow both intraoperatively and for two weeks into the post operative period (Fig. 6 a, b, c). This quiescent period was followed by recurrence of khaki coloured deposits in the anterior chamber although the patient is asymptomatic at 3 months follow up (Fig. 7 a & b).
Fig. 6. (a, b, c) showing khaki coloured cells in AC after 2 weeks of AC wash

Fig. 7. (a, b) Khaki coloured cells presenting in the anterior chamber at 3 months postoperatively

Discussion

Peripheral exudative haemorrhagic chorioretinopathy (PEHCR) was first described by Annesely 5 and later by Shields 6. Classical description includes lesions characterized by blood either subretinal or at the sub RPE level located anterior to the equator and may be associated with massive vitreous blood 7. 8 % of cases with a diagnosis of uveal melanoma have PEHCR 6 as the simulating lesion. Hence this condition is more common than is generally thought of and can lead to temporary or permanent loss of sight in its more advanced stages. This condition should be kept in mind while evaluating spontaneous vitreous haemorrhage or any posterior segment lesion in the elderly. The anticoagulative status of these patients should be taken into consideration if active disease is identified, as there is a risk of massive subretinal haemorrhage. Our patient with acute onset of massive vitreous haemorrhage and peripheral subretinal blood was advised conservative treatment on the diagnosis of PEHCR. Preoperatively itself he had blood staining of aqueous indicating migration of blood cells into the anterior chamber.

The exact incidence of ghost cell glaucoma following vitreous haemorrhage surgery has never been assessed. It may occur a few days after the procedure and has been reported to occur even up to 4 years after surgery 3. The precise mechanism by which the degenerate RBC gains access to the anterior chamber is unclear in a phakic eye 7.8.9. Typically the patients present with pain and a mildly inflamed eye. Perilimbal congestion, variable degree of corneal oedema, khaki coloured cells in the anterior chamber that are out of proportion to the flare and a classical candy – stripe double layered pseudohypopyon are pathognomonic of their condition. Keratic precipitates are absent but large clumps of khaki coloured cells can stick on to the corneal endothelium. Tan coloured cells in the anterior chamber, angle and vitreous are seen. When the diagnosis is unclear, pathological evaluation may be extremely helpful in confirming the diagnosis. Examination of wet preparations of aqueous and vitreous fluid by phase contrast microscopy 9 can provide immediate confirmation of the clinical diagnosis.

The disease is often self limited. Medical treatment of increased IOP is normally sufficient. Surgical options include anterior chamber lavage, vitrectomy and trabeculectomy 10. A rise of intraocular pressure was not observed in our patient probably due to the fact that he had been or topical Dorzolamide eye drops from the first post operative day.

Regardless of its low incidence rate (0.05 %) 11,12 post vitrectomy endophthalmitis should never be overlooked. When in doubt it is better to err on the side of postoperative infection and proceed with intravitreal antibiotics after a vitreous tap.

The presence of the pathognomonic multilayered hypopypon in a patient with history of longstanding vitreous haemorrhage preoperatively should raise the suspicion of ghost cell glaucoma.

References

1977; 83: 63.


The Retinal Spectrum of Ocular Tuberculosis

Dr Gopal S Pillai MS, Dr Abhijeet Khake MS, Dr Natasha Radhakrishnan MS

Tuberculosis is a chronic infection caused by Mycobacterium tuberculosis and Mycobacterium bovis. Ocular TB, involving any tissue of the eye, is a rare event. It occurs in 1% of all cases of TB. Mycobacteria can hematogenously disseminate to the eye and choroid is the most common initial site of intraocular tuberculosis.

Tuberculous panophthalmitis was common before the era of antitubercular treatment. Recently tuberculosis has come into focus because of its association with HIV and AIDS.

Anterior segment involvement can be seen as
1. Palpebral conjunctival ulcerations
2. Granulomatous or nongranulomatous anterior uveitis with or without keratitis
3. Scleritis

Retinal involvement usually present as
1. Choroidal tubercles
2. Choroidal tuberculomas
3. Suprachoroidal abscess
4. Retinal vasculitis, ischaemia, and venous occlusions

**Choroidal tubercles**

Choroidal tubercles are seen in 1.4% to 60% of patients with different forms of tuberculosis. Size ranges between 0.3-3.0 mm. Choroidal tubercles are frequently unilateral close to the posterior pole. They appear as polymorphic yellow white lesions with indistinct borders (Fig. 1). They appear slightly hyperfluorescent on fluorescein angiography (Fig. 2). The tubercles are initially flat, variable in size and exhibit variable pigmentation. Vitritis, papillitis and an overlying serous retinal detachment may be seen with choroidal lesions.

**Choroidal tuberculomas**

They may involve retina and retinal vessels over it. This can lead to formation of hard exudates around the mass. Choroidal tubercles appear hypofluorescent in early phases and hyperfluorescent in late stages (Fig. 9-11).

**Miliary tubercles**

Appears as pale yellow spots and are found in acute miliary tuberculosis, especially tuberculous meningitis, usually as a late event. It is the most important diagnostic evidence of tuberculosis in cases of meningitis and obscure general disease (Fig. 8).

**Exudative retinal detachment**

Can very rarely be caused by large choroidal tubercles due to inflammatory exudation (Fig. 6, 7).

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Fig. 3. Choroidal tubercle with surrounding retinitis with gross macular destruction

Fig. 4. FFA-Choroidal tubercle with surrounding retinitis with gross macular destruction

Fig. 12 (a) Large supra-choroidal abscess

Fig. 12 (b) Fluorescein angiography of large suprachoroidal abscess

Fig. 5. FFA - Late phase Choroidal tubercle with surrounding retinitis with gross macular destruction

Fig. 6. Tuberculous exudative retinal detachment

Fig. 13. Montage view of the same abscess

Fig. 14. Retinal vasculitis, hemorrhages and inferotemporal venous occlusion

Fig. 7. FFA-Tuberculous exudative retinal detachment

Fig. 8. Multiple miliary tuberculosis of choroid

Fig. 15. FFA showing gross peripheral retinal ischaemia of the same patient

Fig. 16. Post papillitis optic atrophy

Fig. 9. Choroidal tuberculoma

Fig. 10. Choroidal tuberculoma (fluorescein angiography)

Fig. 17. Neuroretinitis with macular star appearance

Fig. 18. Disc leakage seen on FFA in the same patient

Fig. 11. Choroidal tuberculoma (fluorescein angiogram)

Fig. 19. Multifocal choroiditis

Fig. 20. Resolving multifocal choroiditis after starting ATT
Suprachoroidal abscess
Large choroidal tuberculomas may undergo caseous necrosis and lead to abscess formation which may be very difficult to drain (Fig. 12 a & b; Fig. 13).

Retinal vasculitis, Venous occlusions and Neovascularisation
TB has also been associated with Retinal vasculitis, ischemia, and venous occlusions. Finally neovascularisation occurs with a high risk of intraocular hemorrhage (Fig. 14, 15).

Papillitis
Affection of optic disc can lead to papillitis. This is a rare presentation. In this case, the patient came with post papillitis optic atrophy (Fig. 16).

Neuroretinitis
Sometimes, neuroretinitis with a macular star can be due to tuberculosis which may be complete or incomplete (Fig. 17, 18).

Multifocal choroiditis
Rarely multifocal choroiditis can also be seen. This case presented with multifocal choroiditis which resolved after anti-tubercular treatment as seen in 2nd photo (Fig. 19, 20).

Rare presentations
- There has been a report of generalised miliary tuberculosis with retinal haemorrhages
- Presumed ocular tuberculosis presenting as a branch retinal vein occlusion in the absence of retinal vasculitis or uveitis has recently been reported
- Macular subretinal neovascularisation can also occur in choroidal tuberculosis
- Peripheral multifocal choroiditis has also been observed

Treatment
Treatment of ocular tuberculosis is similar to that of pulmonary disease. A four drug regimen of isoniazid, rifampicin, pyrazinamide, and either streptomycin or ethambutol for 2 mths is followed by isoniazid and rifampicin for next four months. Treatment should be coordinated with the physician

References
To Treat, or Not to Treat - A Dilemma in Glaucoma

Dr. Murali Ariga MS 1, Dr. Radha Ramanan MS 2, Dr. Ronnie George MS 3, Dr. Saikumar MS 4, Dr. Vinay Nangia MS 5, Dr. Rajesh Radhakrishnan MS 6

To treat or not to treat – a dilemma

Glaucoma undoubtedly is the leading cause of irreversible blindness. The population of human beings predicted to be diagnosed with glaucoma is reaching alarming proportions. It is estimated that there will be 60.5 million people affected by the year 2010 and increasing to almost 80 million in the next decade 1. Not surprising, since over 90% of recently detected glaucoma patients were unaware of their condition 2.

The results of the various randomized controlled trials 3,4 have proved that initiation of appropriate therapy at an appropriate time contributes significantly to retarding the progression of the disease process.

The following case is presented here since the family history of glaucoma is a significant risk factor that contributes to development of disease in a hitherto unaffected individual. Poor compliance is a part of glaucoma management due to the asymptomatic nature of disease and unrealistic patient expectation. This family perhaps represents a group of patients most glaucomatologists will encounter at some point in their practice.

Background:

A 37 year old male patient walked into our glaucoma clinic requesting an evaluation for glaucoma. He worked in a remote area in Saudi Arabia and had returned home on leave, only to find that both his parents had been diagnosed with glaucoma recently. His parents had been told about the hereditary nature of the disease and they had advised him to get screened. He decided to get himself evaluated in his home town since the place he was working did not have sufficient health care facilities and leave was available only once in two years.

Examination findings:

Patient 1: SON, 37 years. No known ophthalmic or systemic disorders till date.
- Visual acuity – 6/6 & N6, unaided (OU)
- Anterior segment – unremarkable
- Fundus – Average size disc with asymmetrical cupping; CDR – 0.65 – 0.7 (OD) & 0.45 – 0.5 (OS) (Figure 1: Composite ONH recording)
- Baseline IOP (GAT) – 17 mm Hg (OD) & 15 mm Hg (OS)
- Gonioscopy – open angles
- Standard white-on-white automated perimetry – Normal VF (OU)
- Nerve fiber layer thickness assessment by OCT – reported as borderline (Figure 2: HRT)

Patient 2: FATHER, 70 years. On treatment for COPD since 20 years.
- Visual acuity – BCVA 6/9 & N6

1 Sundaram Eye Foundation, Chennai 2 Little Flower Hospital, Angamaly, 3 Sankara Nethralaya, Chennai, 4 Giridhar Eye Hospital, Cochin, 5 Suraj Eye Institute, Nagpur 6 Adithya Kran Eye Hospital, Palakkad
Anterior segment – Grade 2 nuclear sclerosis

Fundus – Average size disc; CDR – 0.6 (OD) & 0.75 – 0.8 with inferior polar notch (OS) (Figure 1: Composite ONH recording)

Baseline IOP (GAT) – 18 mm Hg (OD) & 19 mm Hg (OS)

Gonioscopy – open angles

Standard white-on-white automated perimetry – Generalized threshold depression (OU) with significant superior arcuate defect (OS) (Figure 3)

Medication – Brimonidine 0.2% twice daily

IOP with medication – 15 mm Hg (OU)

Patient 3: Mother, 56 years. On treatment for hypertension since 5 years.

- Visual acuity – BCVA 6/9 & N6
- Anterior segment – AC shallow; Grade 1 nuclear sclerosis
- Fundus – Average size disc; CDR – 0.75 (OD) & 0.8 (OS) with inferior polar notch and RNFL defect inferiorly (OU) (Figure 1: Composite ONH recording)
- Baseline IOP (GAT) – 22 mm Hg (OU)
- Gonioscopy – narrow angles widening on indentation
- Standard white-on-white automated perimetry – Superior arcuate defect (OU) (Figure 4 & 5)
- Medication – Latanoprost once daily at night
- IOP with medication – 14 mm Hg (OU)

All patients underwent slit lamp examination, gonioscopy followed by optic nerve head (ONH)
examination using an Ocular 78D indirect lens. Detailed recording of the ONH was made including vertical disc size, rim-to-disc (RDR) ratio in all clock hours, vertical cup-to-disc ratio, disc vasculature and red free examination for RNFL defects.

Gonioscopy was done using the Sussman four mirror lens. ONH was staged using the most recent Disc Damage Likelihood Scale (DDLS) scoring. A correction factor of 1x (manufacturer’s value) was applied to the measured vertical disc diameter when assessing disc diameter.

Visual field was recorded on the OCTOPUS 301 perimeter using the G1 Dynamic program with Peritrend software (Version 6.07).
The consultants were asked to outline their strategies in managing this patient and to comment on the following points.

1. What further investigations will the "SON" require?
2. Will the HRT or GDx provide more information on the NFL?
3. Does CCT have a role in the diagnosis?
4. Since white-on-white perimetry was normal will a SWAP or Flicker perimetry throw up any early defects in this patient?
5. What are the patient’s chances of developing glaucoma?
6. If he developed glaucoma, how long might it take to cause a significant field defect?
7. Should the patient be started on medication?
8. If medication is required, what is the first choice monotherapy option?
9. What should the ideal target IOP range be?
10. How frequently will he require monitoring, especially since he lives and works in a place with inadequate eye care facilities?

**Dr. Murali Ariga**

**Summary**

This 37 year old asymptomatic male with a family history of glaucoma has IOP in the normal range (15 and 17 mmHg) with open angles and normal visual fields. Optic discs in both eyes show asymmetric cupping with no obvious neural rim abnormalities or NFL loss. DDLS has been calculated to show a score of 4 in the right eye and 3 in the left eye. Disc size in both eyes is 1.7 mm. Cirrus OCT assessment of the NFL in both eyes shows near normal measurements.

**Assessment**

I would consider that this patient has a likelihood of developing glaucoma given the fact that at least one parent has definite glaucoma in both eyes. At present however he does not seem to show any definite glaucomatous changes in either the optic disc or fields. A DDLS score of 5 or more is suggestive of an abnormal disc and it has also been stated by Spaeth that field changes are usually noted with DDLS scores of more than 5. The DDLS score may be more useful to document change or progression rather than as a one time diagnostic aid.

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**Figure 5:** Visual field of left eye of Mother
To document findings, I suggest taking good quality disc and NFL photos (preferably stereoscopic) and recording his diurnal IOP (if not feasible at least IOP recordings 2-3 times in a day both mornings and evenings). A one time central corneal thickness measurement would also be useful as baseline documentation. I would recommend a SWAP or blue on yellow perimetry if the facility is available. This is said to show up field changes almost 5 years earlier than standard perimetry. With regards to imaging there is no evidence that one technology is superior to the other in earlier detection of glaucoma. In this situation it would suffice to use any imaging technique that one has access to and do a serial exam (say every year) to detect change. The HRT appears to be the proven technology to detect such changes over time.

There is no way to exactly predict this patient’s chances of developing glaucoma. I would not treat this patient now as he does not have glaucoma at present. He would definitely require follow-up to check IOP, visual fields and if possible to image his optic nerve and NFL every 6 months.

**Dr. Radharamanan**

I would like to treat this 37 year old young male because,

- Very strong family history of glaucoma, both parents getting treatment for Glaucoma.
- He has asymmetry of CDR.
- Both parents come in the category of ‘Normal Tension Glaucoma’.
- He is going back to a remote place and he won’t be available for regular follow up.

A normal white on white perimetry in this patient is what prevents me from treating him. But I like to check his CCT and SWAP. HRT or GDx may not be that useful in this patient. I like to record his blood pressure especially the night BP. I don’t expect an immediate field loss in this patient since both his parents had very slow defects and retain fairly good vision in 70 and 56 years. So I am for treating this man after CCT recording. If the CCT is low, I will treat him and aim to achieve minimum of 30% IOP reduction with latanoprost and if the CCT is normal range I will treat him less aggressively. I like to review him frequently but that may not be possible in this patient. But I will definitely ask him to review within one year.

**Dr. Ronnie George**

This is an interesting case of a 37 year old disc suspect with a family history of both primary angle closure glaucoma and open angle glaucoma. There is also a not uncommon social problem with inability to have a regular follow up.

Based on the clinical records available I would classify the Son as a disc suspect, on the DDLS the inferior neuro-retinal rim in both eyes is equal to or thinner than the superior NRR (the left more than the right) in the presence of a vertical cup-disc ratio of 0.65-0.7 in the right eye and 0.45-0.5 in the left eyes. The combination of an increased cup to disc ratio with a 0.2:1 CDR asymmetry between the two eyes needs further evaluation. The IOP (uncorrected for CCT) is normal and white on white perimetry is also normal. The OCT images are essentially normal. The dip in the TSNIT graph seen in both eyes in the superior region is probably related to the retinal vessels and not a localized NFL loss.

This person would require periodic follow up for the rest of his life and additional baseline measurements, if available, would be helpful. These would include, measurement of the central corneal thickness in both eyes and daytime diurnal measurements of IOP would rule out large fluctuations in IOP. HRT measurements of the disc would be useful for follow up since the maximum progression data is available with this instrument. A baseline stereoscopic optic disc photo is a reasonable, easily available substitute (Ideally a stereo-pair of 20 degree disc centered colour photographs and a stereo pair of red free disc centered 30 or 50 degree NFL images).

SWAP would be helpful in this case, a negative test practically rules out the development of a white on white defect in the next 2 years. I would, however, hesitate to start treatment based on only a SWAP defect since the optic disc is only marginally abnormal and IOP’s are statistically normal. Glaucoma is a very slowly progressive disease with the average rate of progression is estimated to be approximately a mean deviation loss of 1dB per year after onset of disease. Even if this patient had pre–perimetric glaucoma a two year delay in
starting treatment is unlikely to result in significant visual field loss.

The family history of glaucoma needs to be taken into account, the lifetime risk of POAG a family member has been estimated to be 22%, a recent report suggests that it may be higher. There is no information about the heritability of angle closure disease. However, since parameters such as anterior chamber depth and angle width show high heritability it is likely that the risk of angle closure glaucoma in family members is higher than in others. The total risk of a family history of glaucoma (combination of angle closure disease in one parent and POAG in the other parent) is likely to be the additive risk for both together. It is unlikely to be any higher because both are probably mediated by different genetic variation.

It is necessary to explain the nature of the disease, the potential risk of glaucoma and the need for follow up to the patient. I would not advise any medications in this case since there is no evidence of perimetric disease at the present and IOP's are in the mid range. In ideal circumstances a review after 6 months would be appropriate. If all other investigations (SWAP, CCT) are normal, the risk of progression in 2 years is minimal.

Dr. S. J. Saikumar

1. **What further investigation will the patient require?**
   I feel all the relevant investigations have been done. Probably a CCT and diurnal variation will throw some additional light on the clinical scenario.

2. **Will the HRT or GDx provide more information?**
   A composite printout of OCT plus GDx is already provided. Looks like a reliable and good printout with just a bit of nasal thinning in the left eye. I don’t think an HRT will give any additional information.

3. **Does CCT have a role in the diagnosis?**
   A CCT should definitely be done, not to diagnose Ocular Hypertension, but to look for a thin cornea, something less than 500, which will definitely add to the risk of development of glaucoma in future.

4. **Will SWAP or Flicker show any defects?**
   With almost no RNFL defects in OCT, I don’t think any other Perimetry will show any additional field defect.

5. **Chances of developing Glaucoma?**
   The risk factors are pretty high. Both parents have typical glaucomatous field changes. The patient himself has asymmetry in CDR. But I am unable to mention any percentage of risk.

6. **When will he develop field defects?**
   Studies show that field defects appear a few years after typical RNFL changes in one of the pre perimetric machines. Here OCT RNFL is normal. So my guess is that he is not going to develop any significant field loss for 5 – 6 years.

7. **Should medication be started?**
   I wouldn’t start treatment for the son with the available data. His risk increases if the corneal thickness is less than 490. Even if that is the situation I would prefer to wait. No catastrophe is going to happen in 2 years. If, during his next visit to India, he develops any pre perimetric change, then treatment can be started.

8. **Choice of drug**
   My first choice would be a PG analogue. This gives a substantial reduction of IOP. Considering his age, we should aim at a target of 10 to 12.

9. **Follow up**
   There is no doubt that he requires close follow up. Even if Perimetry or OCT may not be available, a proper examination of the optic disc can be done by his local Ophthalmologist. Methods like Disc Damage Likelihood Scale comes in very handy in such situations.

Dr. Vinay Nangia

1. **What further investigations will the patient require?**
   Answer: One may perhaps do a 50 degree and 20 degree colour photo to assess the retinal nerve fiber layer which sometimes may give a definite clue to an early wedge shaped defect or reduced visibility of the retinal nerve fiber layer. Short wave length automated perimetry may also be suggested. In addition a morning diurnal or a 24 hour diurnal variation may be performed.

2. **Will the HRT or GDx provide more information on the NFL?**
   Answer: The HRT does provide more information on the retinal nerve fiber layer. In terms of thickness, and in terms of asymmetry between the two eyes. However
this by itself may not be sufficient to determine whether
one may wish to label the son as having glaucoma,
indicating thereby that therapy for glaucoma be started.
Visual evaluation of the retinal nerve fiber layer along
with HRT data may strengthen the clinical evaluation
and impression.

3. Does CCT have a role in the diagnosis?
Ans: CCT ought to be done in this situation, since he is
a glaucoma suspect. A lower CCT may explaining a
lower IOP. This would indicate an even greater need
for detailed follow up, Since subjects with lower CCT
are known to present with greater amount of visual
field loss because of the lower IOP measurements.

4. Since white-on-white perimetry was normal will a SWAP
or Flicker perimetry throw up any early defects in this patient?
Ans: A SWAP may be done and the possibility exists
that an early defect may show up, keeping in mind the
obvious asymmetry between the two eyes.

5. What are the patient’s chances of developing glaucoma?
Ans: Family history is always important and taken into
consideration. He has a higher chance than normal of
developing glaucoma.

6. If he developed glaucoma, how long might it take to
cause a significant field defect?
Ans: There is no hard and fast rule for developing of a
visual field defect. One may not note a visual field defect
even after losing a significant percentage of the retinal
nerve fiber. I assume that a significant visual field defect
is one that meets the definition of a glaucomatous visual
field defect. If this patient had retinal nerve fiber layer
changes in association with the obvious optic disc
asymmetry, then even with the earliest consistent visual
field change (even one that does not meet the classic
criteria of a visual field defect) one may consider
labeling a patient as having glaucoma.

7. Should the patient be started on medication?
Answer: For the time being, he ought to be followed
up. A threshold of glaucoma diagnosis is needed to start
a person on antiglaucoma therapy, which is for life.

8. If medication is required, what is the first choice
monotherapy option?
Answer: One may opt for prostaglandins or beta blockers
with due regard to systemic factors and local acceptance.

9. What should the ideal target IOP range be?
Answer: If we assume that minimal diagnostic criteria
have been met at his current IOP, then the target
pressure would be in the low teens. This is a
hypothetical situation and this answer must be
understood in that context.

10. How frequently will he require monitoring, especially
since he lives and works in a place with inadequate eye
care facilities?
Answer: He may be followed up once in six months.

Editor’s comments:
This patient, seen in isolation, may be uninteresting
to a general ophthalmologist except for the asymmetrical
cupping. However, when viewed in a background with
a strong family history of glaucoma it is bound to
instantly draw the attention of the ophthalmologist.

Management Plan
When assessing this patient, equal importance has to
be given to the following factors.

1. Both parents have significant glaucomatous optic
neuropathy, though by different mechanisms.
Heredity plays a significant role in OAG.

2. Patient is employed at a place in Saudi Arabia
where monitoring facilities are not easily accessed
and leave is available only once in two years.

3. Compliance is a major issue as all three members
of the family are defaulters.

   - Neither have the parents returned for follow
   up nor did the patient come back after the OCT.
   (The imaging report was obtained directly
   from the referral center).

   - He has not come back for his CCT measurement
   and diurnal phasing as he has returned to his
   work place, cutting short his leave.

1. Assembling the parameters
Assuming that the patient was available for further
investigation the first priority would be to measure the
central corneal thickness. A thinner cornea would be
an additional risk factor besides the family history and
asymmetrical cupping. A diurnal phasing, at least during the day may throw light on the fluctuation in baseline IOP. In the event of an indication for medical therapy the target range of IOP will be decided by the diurnal curve and the CCT. If possible, a SWAP or Flicker perimetry to rule out early damage (unlikely with a normal OCT) can be done.

Once that is done, the following parameters will be available.
1. Angle status
2. Base line intraocular pressures, including diurnal fluctuation
3. Central corneal thickness
4. Optic nerve head parameter
   a. Disc size
   b. Cup disc ratio
   c. Rim with in all quadrants
   d. DDLS score
5. Standard achromatic visual field
6. Nerve fiber layer status

2. Analysis and assessment

As mentioned earlier, this patient viewed in isolation, may not warrant any therapy except an annual monitoring. However, with both parents having been diagnosed with glaucoma, the patient has been subjected to a battery of tests. With the available parameters, no definite pointer is readily available to suggest initiation of therapy. Perhaps, serial fundus photos and optic nerve head parameter monitoring would have been sufficient. If the patient had been available for a diurnal phasing and CCT measurement the findings could tip the scales in favour of therapy.

3. Decision to monitor

Assuming that CCT were normal and diurnal curve did not show an IOP of more than 20 to 22 mm Hg we would not be far wrong in just monitoring him. Optic nerve head assessment and intraocular pressure monitoring every six months; annual perimetry (if required SWAP or Flicker) and nerve fiber layer analysis on HRT would be the ideal protocol. This will hold if the patient is complying with instruction for follow up. We need to keep in mind his distant work place and lack of infrastructure for proper monitoring locally.

4. Decision to treat

With a strong family history of, perhaps, late onset glaucoma of low pressure type in one parent and the relative inability of patient for follow up a less aggressive strategy maybe adopted if therapy is to be initiated. With a normal CCT we could aim for a target IOP about 20% less than baseline. However, if the CCT is less than 480–490 microns the chances of development of significant glaucomatous damage in the patient's life time increases and therefore we may need to be more aggressive. An IOP reduction of at least 30% from baseline should be our target.

5. Follow up & monitoring

Monitoring this patient without therapy is a tricky affair considering his inaccessible location and a query regarding compliance. The first step is to counsel the patient regarding the very real possibility of his developing the disease a few years down the line. The role of family history the predominant risk factors needs to be explained. The asymptomatic nature of the disease and its potential to cause significant irreversible damage has to be highlighted. The counseling has to also involve other family members, both those already affected and those not affected. Explaining the nature of the disease with the visual field defect of parents will emphasize the serious nature of the disease and need for regular voluntary monitoring.

One of the easiest ways to detect, assess and monitor a manifesting glaucomatous neuropathy will be the Disc Damage Likelihood Scale (DDLS). It is a simple but versatile tool that needs only clinical skills and minimal instrumentation. The first clue to progression lies in the narrowing of the rim and appearance of nerve fiber layer defects. Most optic nerve head parameters can be monitored with the DDLS and its sensitivity and specificity has been proven to as good as any imaging device. If the patient is accessible this can be done every six months and can substitute a fundus photograph in places without fundus camera.

Standard automated perimetry should be an annual affair interspersed with SWAP or Flicker perimetry if suspicious optic nerve head or nerve fiber layer changes are noticed. Pre-perimetric glaucoma can be detected by progression recorded by serial optic disc stereo
photographs and DDLS. They have been proven to be
more sensitive or equally sensitive to analysis by the
imaging devices 7. DDLS has also been proven to
correlate well with HRT and visual field defect 8.

6. Conclusion

First degree relatives of those diagnosed with OAG are
10 times more likely to develop glaucoma than those
with no family history. In the Barbados Eye Study,
23% of relatives of families with glaucoma had manifest
OAG at examination. We can’t be far wrong in pursuing
family members of our patients diagnosed with
glaucoma especially when there is evidence to show
that 90% of glaucoma diagnosed in our country is
accidental.

Rather than wait for a patient to walk into our offices
with advanced disease, it is our responsibility to “Chase
the Family” 6 and halt the relentless progression of the
disease among our vastly uninformed population.

Imaging devices aid in detecting and monitoring disease
but they can at best support clinical findings and cannot
be the basis for therapy. Currently their availability and
accessibility is limited and till such time they are freely
available we need to trust our clinical and observational
skills to pick up subtle changes that herald the onset
and progression of this sight stealing disorder.

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Compiled by Dr. Rajesh Radhakrishnan MS, Adithya Kiran Eye Hospital, Palakkad
Long Term Results Of Viscotrabeculotomy In Congenital Glaucoma: Comparison To Classical Trabeculotomy

N Tamcelik, A Ozkiris
Br J Ophthalmol 2008; 92:36-39

Management of congenital glaucoma is surgical and current classical procedures in congenital glaucoma include goniotomy, trabeculotomy ab externo and trabeculectomy. To increase the success rate of trabeculotomy, some modifications such as combined trabeculotomy-trabeculectomy and trabeculotomy with newly designed probes were applied. The authors considered the use of viscoelastics during trabeculotomy, and named this modified technique viscotrabeculotomy.

The purpose of the study was to evaluate the outcomes of viscotrabeculotomy and classical trabeculotomy, and to compare these two techniques. 64 patients with primary congenital glaucoma who presented at Istanbul University Cerrahpasa, Turkey before the age of 12 months were divided into two groups. Group 1 consisted of 58 eyes of 34 patients who underwent viscotrabeculotomy, and group 2 consisted of 51 eyes of 30 patients who underwent classical trabeculotomy.

Pre- and postoperative IOPs, mean antiglaucoma medication, mean corneal diameter, success rates, intra- and postoperative complications were compared between two groups.

Classical trabeculotomy was performed as described by Allen and Burian. In viscotrabeculotomy, the Schlemm canal was cannulated on either side and high –viscosity sodium hyaluronate was injected into the Schlemms canal. Trabeculotome was passed into the canal; the tip of the probe was gently rotated into the anterior chamber. The probe was removed and repeated in the opposite direction. Sodium hyaluronate was injected into the anterior chamber, if a shallow anterior chamber developed, then a small amount was injected to prevent adhesion of incision lips.

Complete surgical success was determined by an IOP <18 mm Hg under general anaesthesia without medication or resurgery, with no progression of disc cupping or corneal diameter and with no devastating visual complications. Failure was defined as IOP >/= 18mm Hg in patients with medication, resurgery or sight –threatening complications. Postoperatively, the mean IOPs and antiglaucoma medications were significantly lower in group 1. The percentage of mean reduction in IOP from baseline to the last follow-up was 47% in group 1 and 42.1% in group 2. At the last visits, the success rate of group 1 was statistically higher when compared with group 2. 10 eyes in group 1 and 18 eyes in group 2 required antiglaucoma medications to control IOP. Additional surgery was performed in five eyes in group 1 and 13 eyes in group 2. During surgery, only one eye with severe bleeding and iridodialysis in group 2 required injection of viscoelastic into the anterior chamber.

The disadvantage of viscoelastic material is that when left in anterior chamber, they cause a temporary IOP elevation. Viscoelastic materials remain in the canal.
for 4-6 days, prevent collapse of the Schlemm canal and create a barrier to the migration of fibrinogen released by the ciliary body during surgery.

The success rate of classical trabeculotomy at the last visit was 68.6 % and 91.3 % for viscotrabeculotomy, and the difference between two groups was statistically significant. The most common early postoperative complication was transient IOP elevation in group1, the incidence of hyphema in group1 was only 6.8 % - these low rate of hyphema may only be explained by using viscoelastic materials.

In conclusion, the authors say that viscotrabeculotomy is safer and more effective than classical trabeculotomy. Intra and postoperative complications are very rare. Dilation of the possible narrow Schlemm canal, keeping away the lips of trabeculotomy incision, possibly prevention of the postoperative haemorrhage and fibroblastic proliferation by means of high-viscosity sodium hyaluronate are the possible factors that play important roles in the overall success of the procedure.

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### Capsular Block Syndrome After Cataract Surgery: Clinical Analysis And Classification

**Hyon Kyun Kim, Jae Pil Shin**

*J Cataract Refract Surg 2008; 34:70-75*

Capsular block syndrome (CBS) is a rare complication. It is characterized by distension of capsular bag and accumulation of a liquefied substance inside the capsular bag. Slitlamp biomicroscopic examination shows forward displacement of the intraocular lens optic. Patients have a myopic shift, anterior displacement of iris-lens diaphragm, and increased IOP.

This study conducted at Kyungpook National University Hospital, Republic of Korea evaluated the clinical characteristics and risk factors for postoperative CBS. They reviewed the clinical records of 1100 patients who had undergone phacoemulsification and PCIOL implantation, and evaluated 8 cases of postoperative CBS. They suggest a new classification of postoperative CBS according to its pathogenic mechanisms. This study did not include cases of intraoperative capsular block. Longer axial length (>=25 mm) was a significant risk factor for the development of postoperative CBS. Although the OVD used intraoperatively was not a risk factor, the type of PCIOL, had a significant influence on the development of the syndrome. In particular, postoperative CBS is more prevalent in eyes with a 4-haptic IOL than in eyes with a modified C-loop IOL. Former lens is not posteriorly angulated, it results in relatively larger contact area between the IOL and the anterior capsule than with other PCIOLs.

The CBS was classified into 3 groups based on distinct clinical characteristics. The groups were noncellular CBS, inflammatory CBS, and fibrotic CBS. Noncellular CBS was characterized by a distended capsular bag within a day to several days after surgery (very early postoperative period). These cases have few cellular reactions and fibrotic adhesion between IOL and anterior capsule. Retained OVDs play a major role in the pathogenesis. Treatment includes disruption of capsular bag or aspiration of retained OVDs. Inflammatory CBS developed several days after surgery (early postoperative period). They did not have retained substance on the first postoperative day. They have a cellular reaction around the anterior capsule margin and the IOL optic was attached to the overlying anterior capsule. A distended capsular bag subsequently developed. In this case distended capsular bag can be deflated by anti-inflammatory medication without any surgical trial. Fibrotic CBS occurred in the late postoperative period (several months to years after surgery). It was characterized by fibrosis over the entire circumference of the anterior capsule opening. Main cause of this condition is the proliferation and metaplasia of LECs, which produces numerous types of collagen and extracellular matrix that accumulate in the bag. This type was treated by Nd:Yag laser posterior capsulotomy.
Efficacy of Intravitreal Bevacizumab in Treating Postoperative Pseudophakic Cystoid Macular Oedema

Martin S. Spitzer, Focke Ziemssen, Efdal Yoeruek, Kartin Petermeier, Sabine Aisenbrey, Peter Szurman
J Cataract Refract Surg 2008; 34:70-75

Postoperative pseudophakic cystoid macular oedema (CME) is one of the main causes of suboptimal visual acuity after cataract surgery. The goal of any treatment is to reduce macular oedema, however there is no widely accepted technique to treat chronic macular edema. Recently, Mason et al reported 2 patients with persistent CME who had been successfully treated with a single intravitreal injection of 1.0 mg bevacizumab. This analysis of a small interventional case series was designed as an exploratory investigation of an intravitreal treatment to test short-term safety and effectiveness in patients with postoperative CME. This retrospective case series conducted at University Eye Clinic Tuebingen, Eberhard-Karls University, Germany comprised 16 eyes of 16 patients with CME after cataract surgery refractory to current standard treatment who received an injection 1.25 mg intravitreal Avastin. The main outcome measures were visual outcome, retinal thickness on OCT, and complications related to treatment.

The mean duration of CME before treatment with intravitreal Avastin was 14 wks. Although the mean retinal thickness decreased slightly after intravitreal Avastin, the mean visual acuity remained unchanged. Visual acuity improved by 2 lines in 1 patient, remained unchanged in 12 patients, and decreased by 2 lines in 2 patients. Repeated Avastin injections did not result in a better outcome. Other than mild ocular irritation, there were no adverse effects of the intravitreal injections. They concluded that intravitreal Avastin, although safe, did not result in improved visual function in patients with postoperative CME. In contrast to findings in a previous case report, the beneficial effect of vascular endothelial growth inhibition in Irvine-Gass syndrome was negligible with respect to improvement in visual function.
In 1982, a book authored by one Milton Bruce Shields and entitled *A Study Guide for Glaucoma* made its debut. It was readily embraced for its breadth of knowledge combined with a clear and concise writing style. Succeeding edition, of the now familiar *Textbook of Glaucoma*, have solidified the position of this remarkable resource for students of glaucoma at all levels of training.

Our understanding of glaucoma has evolved immensely over the three decades since the first edition was released. The mechanism responsible for most forms of glaucoma were only beginning to be understood. Computer analysis of the optic nerve head and retina and its attendant technologies were a distant dream. Remarkably, Bruce Shields kept pace with the advancement of the subspecialty of glaucoma for over 25 years, an astonishing achievement. Now, with the passing of the guard, the editors and authors of *Shields’ Textbook of Glaucoma* have dedicated themselves to this remarkable legacy.

The expansion of knowledge in glaucoma, has been truly remarkable, as evidenced by the advances and new body of literature in each of the five editions. This is testimony to the sustained commitment and skills of the scientists and clinical investigators in our profession. The outset, the focus of this textbook has been not so much to express one view point on the science and management of the glaucoma, but rather to present a balanced review of the literature that was felt to be pertinent at the time. The five authors who continue the revisions of the book share this focus, which will hopefully remain the guideline for whatever future editions may be written. This edition is better than any of the previous four, because of the contributions of these five authors. They have done an excellent job of eliminating much of the materials that is no longer pertinent, updating on the new literature, and providing an element of science that was progressively lacking.

This revised fifth edition has four sections and forty four chapters. The basics aspect of glaucoma is described in section one in six chapters. Section two elaborately cover the various clinical forms of glaucoma in twenty chapters. Section three covers the various aspects of management of glaucoma in eighteen chapters.

This latest revised edition of ‘Shields’ Textbook of Glaucoma’ is recommended as a reference book and also as a final word about glaucoma for general ophthalmologists and post-graduate students.
Fundus Fluorescein & Indocyanine Green Angiography

Edited by: Amresh Chopdar
Published by: Jaypee Brothers New Delhi
First Edition: 2007
Price: Rs 1295/-

Fundus fluorescein angiography is a well established investigative procedure in ophthalmic practice. However, indocyanine green angiography is a relatively new one. Following the success of the author's previous two books on fluorescein angiography he has now included indocyanine green angiography in this new edition. The book describes the features of indocyanine green angiography alongside those of the fluorescein angiography so that the reader is acquainted with the additional benefit of the indocyanine green angiography.

The book has twelve chapters dealing with Development of Fluorescein Angiography, Basic principles, Techniques and Pitfalls of Fundus Fluorescein Angiography, Normal Angiogram, Abnormal Fluorescence, Retinal Vascular Disorders, Macular Degenerations, Macular Dystrophies, Chorioretinal Disorders, Disease of Optic Nerve Head, Intraocular Neoplasms, Diabetic Retinopathy etc.

This book presents a comprehensive coverage of the subject in a concise manner useful for practising ophthalmologists, post-graduate students and paramedical staff connected with retinal imaging and fundus photography.

Atlas on Optical Coherence Tomography Of Macular Diseases and Glaucoma

Edited by: Vishali Gupta, Amod Gupta, MR Dogra
Published by Jaypee Brothers, New Delhi
Second Edition: 2006
Price Rs 2995/-

The emergence of Optical Coherence Tomography (OCT) in the recent years has changed forever, the way we 'look at' or shall we say 'look through' the retina. The OCT provides, in real time, high-resolution cross-sectional images of the macula very similar to obtaining in vivo histopathological sections. It represents a major advance in the diagnostics of retinal disease and has found rapid acceptance among the retina specialists.

In this 'Atlas', the authors have attempted to share their experience of Stratus OCT (Tm) in various macular disorders where they found it helpful in diagnosing and monitoring the response to various therapies and interventions and above all identifying the correct therapeutic approach in a given patient. It finds extensive application in diagnosis, management and follow-up of diabetic macular edema, macular hole, taut
posterior hyaloid membrane, vitreofoveal traction, idiopathic central serous chorioretinopathy, submacular pathology and many more areas that are divided into 28 chapters. For ease of comprehension, they provide with brief case summaries, fundus photographs, fluorescein angiography and the OCT images and the follow-up images for most of the patients that they share with the readers.

In recent years, OCT has also emerged as a valid tool for assessment of retinal nerve fiber layer and optic disc evaluation in pre-perimetric glaucoma. The role of OCT in various neuro-ophthalmological disorders is still emerging. Based on the clinical experiences, the authors have contributed a new section on ‘Glaucoma’ in second edition. Since the last edition in 2004, the upgraded software for image analysis and normative data has become available. The techniques in the first section have accordingly been revised. The case presentations have been revised with newer pathologies.

The revised second edition has 28 chapters in three sections. Section one is introduction to OCT, second section describe the OCT patterns in various macular diseases and the new section three entirely on OCT findings in glaucoma and neuro-ophthalmology. This new edition, of the atlas on OCT of macular diseases and glaucoma is of great use for day to day practice as a reference book.

The Sankara Nethralaya Atlas of Neuro-Ophthalmology

Edited by: Satya Karna, Ambika S, Padmaja S, Smitha Menon, Nikhil S Choudhari
Published by: Jaypee Brothers, New Delhi
Price Rs 1795/-

Many neurological disorders are reflected in the eyes and speciality of neuro-ophthalmology lies at the confluence of two major disciplines of medicine-ophthalmology and neurology.

The last 25 years have seen an explosion in neuroimaging technology paralleling the growth in information technology. The last decade has seen neuro-ophthalmology grow into a rich and complex field of study. There are very few atlases of neuro-ophthalmology available to ophthalmologists world wide and none from Asia. This atlas is a guide to the interpretation of clinical symptoms and signs in neuro-ophthalmology.

Each section is color coded and the chapters in each section are in alphabetical order for easy cross-referencing and navigation through the book. For every topic, a small number of recent classic reviews have been added. Original images of patients seen in the neuro-ophthalmology department of Sankara Nethralaya constitute this atlas. Selected disorders are illustrated using several different images that present the early and late stages of the condition as well as variations of presentation.

The information explosion in neuro-ophthalmology since the first edition motivated the authors to plan for this second edition. The aim was to make this edition current and comprehensive. Hence the Atlas has been updated with 95 chapters in 9 sections, 335 new photographs, 9 new chapters and the latest references for further reading. A special section on illustrative multiple choice questions with explanatory answers has been added. Authors have organized and edited many of the existing chapters. The new chapters include diabetic papillopathy, Leber’s hereditary optic neuropathy, normal tension glaucoma, blepharospasm and hemi facial spasm, orbital apex syndrome, skew deviation, HIV and neuro-ophthalmic disorders, cerebral venous thrombosis and orbital varices.

The purpose of this atlas is to provide ophthalmologists and the other physicians concerned with eye disease with a carefully selected collection of quality illustrations that review the spectrum of disease.
commonly seen in neuro-opthalmic practice. The Atlas of Neuro-Ophthalmology that is based on experience with patients seen at Sankara Nethralaya will play a key role in acquainting the ophthalmologist with neuro-opthalmic disorders. The photographs are augmented by succinct textural descriptions and useful references that allow deeper investigation into the subject. It is for these reasons that this book may be recommended to all students and practitioners of ophthalmology.

The second edition of this book will be used frequently in the day-to-day practice of ophthalmologists, radiologists, neurologists, neurosurgeons and neuro-ophthalmologists world wide.

Dr. C. V. Andrews Kakanatt, JMMC Thrissur

ERROR

KJO June 2008. Consultation Section - Managing a Cosmetic Blemish - pg. 196

The formula to calculate the implant size is $\frac{4}{3} \pi r^3$ (4/3 pi r cube). The pi was missing. The error is regretted.
CME Programmes

STATE CONFERENCES

TACOPSIA 2008
21st September
Dr. Rani Menon
Venue: Thrissur
Mob: 9447 284008

Drishti 2008
21-23rd November
Annual Conference of Kerala Society of Ophthalmic Surgeons,
Venue- Gokulam Convention Centre, Cochin
Organizing Secretary: Dr. Revathy Ramesh
Ph: 0484 2334905

NATIONAL CONFERENCE

32nd Andhra Pradesh Ophthalmological Society
Annual Conference
26, 27,28th September 2008
Vijayawada, Andrapradesh
Dr. K. Madhukar Reddy
madhukar_dr@rediffmail.com
Ph: 09848018170

XVII Annual Conference of Glaucoma Society of India
31st Oct.- 2nd Nov 2008
Advanced Eye Centre, Post Graduate Institute of Medical Education and Research, Chandigarh
Dr. S.S. Pandav.
Ph: 0172-2756112, 2747837
e-mail: ss.pandav@yahoo.com

XVIIth Vitreo Retinal Society of India Conference
4th -6th December 2008
Venue: Raichak, West Bengal
Contact: Dr. Mangat R. Dogra
0172-2756111

67th All India Ophthalmological Conference (Maru Jyothi)
5th – 8th February 2009,
Venue: Jaipur
Dr. BK. Mathur (Organising Secretary)
C-126, Moti Marg, Baper Nagar, Jaipur
Ph: 0141-5131236/37
Fax 0141-2221695

INTERNATIONAL CONFERENCE

XXVI Congress of the European Society of Cataract and Refractive Surgeons
13-17th September
Berlin 2008
Tel: +3531209110 Fax: +3531209112
www.escrs.org

2008 SEAGIG and AACGC Joint Congress
September 25-27, 2008
Venue: Seoul, Korea
Organised by Organising Committee of SEAGIG 2008 and Seoul AACGC 2008
www.seagig-aacgc.org

2008 Joint Meeting of the American Academy of Ophthalmology and the European Society of Ophthalmology (SOE)
November 8-11, 2008
Venue: Georgia World Congress Centre, Atlanta Georgia
Web: www.aaao.org.

MEACO 2009:
Middle East African Council of Ophthalmology
26-30 March 2009
Bahrain
www.meaco.org
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VIRAL RETINITIS

ACUTE RETINAL NECROSIS

- ARN Syndrome
- Fulminant Necrotising Vasocclusive Retinitis with a high incidence of multiple retinal breaks & RD In 86%

ARN : A BRIEF HISTORY

- Kirisawas Uveitis
- 6 Patients
- Severe intraocular inflammation
- Retinal Vascular Sheathing
- Large, white, confluent retinal infiltrates
- Progression to Rheg RD
- Poor visual potential
- No aetiological agent identified
- No therapy effective

EPIDEMIOLOGY

- Any race
- Either sex (? Male Preponderance)
- Any age (Typically young adults)
- Immunocompetent / Immunosuppressed

CLINICAL FEATURES

- Usually unilateral
- 30% of ARN: Bilateral involvement
- 2nd Eye diseases delayed by ? 20yrs
- Immunocompetent without systemic symptoms
- Associated herpes infection at another site
  (Aaberg TM et al AJO 84:209_219, 1977)
- Associated with viral meningitis

CLINICAL FEATURES

- H/o: pain, redness, floaters, D/V
- Progressive vitritis
- Small patchy yellowish full thickness retinal lesions
- Mid peripheral lesions / post pole involvement Rare
- Enlargement & Coalescence of lesions
- Areas of clearing (Swiss Cheese Appearance)
- Asso RPE Perturbation

CLINICAL FEATURES

- RETINAL VASCULITIS
- Severe Retinal Arteritis
- Periphlebitis
- Retinal Vein Occlusion
- Retinal Haemorrhages

OPTIC NERVE INVOLVEMENT

- Optic Neuropathy
- Intraorbital ON Enlargement
- Vaso Occlusion
- Viral Infiltration of ON
- ON Distension (? ON Sheath Fenestration)

ATYPICAL PRESENTATION

- Hypopyon Uveitis
- Painful Orbitopathy
  (Clinical & Exp Ophthalmol June 2003)
- Diffuse Orbital Cellulitis

CLINICAL COURSE

- Resolves spontaneously with and without Therapy
- Course of untreated disease 2-3m
- Course predicted by clock hours of retinal inv
- TRD / Rheg RD with large breaks (86%)

CLINICAL FEATURES

- Usually unilateral
- 30% of ARN: Bilateral involvement
- 2nd Eye diseases delayed by ? 20yrs
- Immunocompetent without systemic symptoms
- Associated herpes infection at another site
  (Aaberg TM et al AJO 84:209_219, 1977)
- Associated with viral meningitis
STANDARD DIAGNOSTIC CRITERIA FOR ARN
(American Uveitis Society : Executive Committee)
- Clinical Appearance
- Course of Infection
  - One/more foci of retinal necrosis with discrete borders in peripheral retina
  - Rapid progression of disease if AntiViral Therapy is not instituted.
  - Circumferential spread of disease
  - Evidence of Oclusive Vasculopathy with Anterior Involvement
  - Prominent inflammation in Vit & AC

DIFFERENTIAL DIAGNOSIS
- PORN
- Endophthalmitis
  (Bacterial / Fungal)
- Behcets
- Intraocular Lymphoma
- CMV Retinitis
- Syphillis, Sarcoid
- Toxoplasmosis

THERAPY FOR ARN
ACYCLOVIR THERAPY:
Advantages
- Good activity against HSV
- Activity against VZV at higher conc
- Hastens resolution in infected eye
- Prevents contralateral spread
  87.1% Vs 35.1% AJO 1991, Palay et al)
Cautions
- Therapy does not diminish vitritis
- Does not reduce incidence of RD/RT
- SIDE EFFECT : Renal Function & Liver Function abnormalities, nausea vomiting & headache (3%)

ACYCLOVIR THERAPY
- 10-14 Day Course
- I/V 500mg / m² every 8h hourly for 10-14 days
- ORAL 800mg 5 times for 6 weeks
- VALACYCLOVIR / FAMICYCLOVIR
- Oral 1 gm TDS
- Same Efficacy, Lesser Side Effects

ALTERNATIVE THERAPY
- Severe : progressive disease despite Acyclovir : Ganciclovir or Foscarnet
- Systemic Corticosteroid Therapy
  0.5mg / kg within 24-48 hrs of Acyclovir Therapy
- Aspirin: Extensive Arteritis & Retinal Vascular Occlusions
- Intravitreal Injection Of Antivirals

ALTERNATIVE THERAPY
- PROPHYLACTIC LASER BARRAGE
  Demaricates involved and uninvolved areas
  Reduces Incidence of RD
  (Sternberg et al Ophthalmol 1988)
- Prophylactic SB –PPV –Endolaser
  (Blumenkraz; Retina 1989)
- RD Repair with Silicone Oil
- ON Disease
  : ON Fenestration / Steroids

PORN (PROGRESSIVE OUTER RETINAL NECROSIS)
- IMMUNOCOMPROMISED
- 2nd Most frequent opportunistic infection in AIDS
- Rapidly progressive visual loss
- Asymptomatic in 11 %
- Median CD4 + Count 21/mm³
- Definite history of cutaneous Zoster
- Patchy Choroidal & Deep Retinal Lesions
- Early Posterior Pole Involvement
- No Vasculitis
- No Vitritis
- ON Involvement 17 %
- RAPD 38 %

CRITERIA | ARN | PORN
--- | --- | ---
VISUAL LOSS | MILD TO GROSS | GROSS EARLY
ANT SEG | PANuveitis | MILD NGU
VIT Rn | SEvere | ABSENT
RETINAL INV | PERIPHERAL, FULL THICKNESS | DEEP MULTIFOCAL, MACULAR
 | SWISS CHEESE | CRACKED MUD
VASCUlitis | COMMON | RARE
ON INV | COMMON | RARE
PROGRESSION | RAPID | RELENTLESS
SPREAD | CIRCUMFERENTIAL | NO DEFINITE DIRECTION
SUPPORTIVE EVIDENCE | SCLERITIS, PAIN | PERIVENULAR CLEARING
CMV RETINITIS
- Most common opportunistic infection in AIDS Patients
- 15% - 40 % Of AIDS Patients
- 95% of all viral retinitis
- Strong correlation with low CD4+ Count
- 50% Heterosexual males & 95% Gays (+) for CMV Antibodies :Prior systemic systemic infection & mononucleosis like picture
- Opportunistic infection in immunocompromised patients
- Infants with congenital CMV infection

CLINICAL PRESENTATION
- Asymptomatic / DV / Floaters
- Early CMV Retinitis :CWS
- FULMINANT LESION : Full thickness perivascular fluffy white retinal opacification with haemorrhages & exudates : Cottage-Cheese with red tomato ketchup / Pizza- pie appearance
- INDOLENT LESION : Slowly spreading lesion with active advancing border & central atrophic area with pigment & scanty haemorrhages
- 6% Frosted Branch Angitis
- 30% RD In Healed Stage

PROGRESSION
- Haematogenous spread : New lesion
- Advancing active spreading border

VISUAL LOSS
- Macular involvement by active lesion
- Macular edema by adjacent retinitis
- Optic nerve involvement
- Peripheral field loss & preservation of central island
- RD

RD IN CMV RETINITIS
- 20% -30% PATIENTS
- 50% Develop RD in fellow eye
- 11% Develop RD within 6 Months of diagnosis
- 25% Develop RD within 1 Year
- Risk increases If > 25% peripheral retina is involved and if patient is Myopic
- Tractional Breaks & Atrophic Breaks
- Multiple, Large, Posterior breaks

BEFORE HAART ERA
- Relentless progress & blindness
- TIME INTERVAL
  Onset Of AIDS & Diagnosis Of CMV Retinitis : 9 months
  With AntiCMV therapy : progression of CMV
  I/V Ganciclovir / Foscarnet : 2 Months
  I/V Cidofovir : 2 – 4 Months
  Sustained release Ganciclovir Implant : 7 Months
  Mean survival time after diagnosis Of CMV :6wks ( Not On Anti CMV Therapy) – 10 Months (on Anti CMV Therapy)
- Specific Anti CMV therapy continued For 3 Months After CD4 Count has increased with HAART

SPONTANEOUS RESOLUTION
- Associated With Immune Recovery
- Evidenced by CD4 Count > 400
- General feeling of wellbeing
- Reports of spontaneous regression in HIV patients on HAART Regimen

CMV RETINITIS IN THE ERA OF HAART
- Viral Replication
- CD4 + Count
- Mortality
- Incidence of CMV Retinitis reduced by 50 %
- Altered clinical manifestations
- Dramatic change in clinical course
- Can control CMV even in absence of specific Anti CMV therapy
- No recurrence on discontinuing maintenance therapy
- Several months of HAART before sufficient immune response against opportunistic infection is restored

IMMUNE RECOVERY UVEITIS
- Vitritis
- Macular Oedema
- Uveitis
- Cataract
- ERM
- Optic disc oedema
- ? Better immune response
- ? Strict adherence to HAART regimen
- No new RD / IO inflammation & pigment hypertrophy
**IMMUNE RECOVERY UVEITIS**
- Does not occur without CMV Retinitis
- Inflammation related to infection
- Subclinical viral replication in the eye
- Related to amount of infected retina
- Amount of CMV antigen in the eye
- Degree Of BRB breakdown due to retinal necrosis

**Treatment strategy**
**HAART ERA**
- Can consider Stopping Maintenance Anti CMV therapy in patients with stable CMV Retinitis
- Stable CD4 Counts
- Increasing CD4 Counts
- CD 4 Counts >100 cells for 3 months
- Improved QOL
- Close F/U For Reactivation / EO disease
- CD4 Count <100 : Consider Anti CMV Therapy

**ANTI CMV THERAPY**
- **GANCICLOVIR**
- **INTRAVENOUS**: INDUCTION 5mg /kg BID FOR 14 – 21 DAYS
- 5-10 mg/kg body weight I/V as maintenance Dose
- Oral : 1 gm 3 times daily
- Intravitreal : 400- 2000mcg/0.05 -0.1ml
- Side effects: Neutropenia, thrombocytopenia, anaemia, elevated liver enzymes

- **NUCLEOSIDE Analogue :acts as competitive inhibitor & faulty substrate for CMV DNA Polymerase**
- **Virustatic**
- **Effective in 90% -100% Viral Retinitis**
- Clinical effect apparent in 2-3 Weeks
- Inactive border achieved in 4-6 wks
- CMV Retinitis relapses without maintenance in 3 weeks

- **GANCICLOVIR**
- **INHIBITS DNA Polymerase**
- **INHIBITS reverse transcriptase**
- **DECREASES HIV replication**
- **INTRAVENOUS**: 90mg/kg bid,14-21 days (Induction)
- Maintenance : 90mg/kg/day
- **INTRAVITREAL**: 2400 mcg /0.1 ml
- Side effects: Nephrotoxicity; GIT Disturbance; Electrolyte Imbalance

**CIDOFOVIR**
- Cytosine derived nucleotide analogue
- **INDUCTION Dose**: 5mg/kg weekly for 2 weeks
- **Maintenance 5mg/kg every 2 weeks**
- **Intravitreal Dose**: 20mcg/0.1ml

**FOMIVIRSEN**
- **Antisense oligonucleotide**
- Complimentary to a sequence on CMV viral mRNA
- **INTRAVITREAL**: 330 micrograms weekly for 2 weeks
- **Maintenance Dose**: once every 4 weeks
- Indicated in pts intolerant or resistant to other AntiCMV
- **Side Effects**: Uveitis, Vitritis, IOP, RPE Degeneration

**SOCA TRIALS**
(Studies on Ocular Complications of AIDS)
- Compared Ganciclovir & Foscarnet
- Equally effective in preventing progression
- Median time of progression of CMV Retinitis : 8 Weeks
- Survival time longer with foscarnet (12m vs 8m)
- More neutropenia with Ganciclovir
- More infusion related problems, gut problems, Nephrotoxicity, Electrolyte abnormalities with Foscarnet

**SOCA TRIALS**
- Both drugs become less effective with time
- Rate of field loss :30 0/ month
- Rate of 3 line visual acuity loss : 94 – 100 persons/year

**COMBINATION THERAPY**
- I/V Ganciclovir + I/V Foscarnet
- IVT Formivirsen + IVT Cidofovir
- Ganciclovir Implant + Oral / I/V Ganciclovir

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GENERAL INSTRUCTIONS TO AUTHORS

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Authors submitting materials to this journal are requested to adhere STRICTLY to the norms laid down below. The matter must be typed on one side of the paper. A margin of 1” must be left all around and the material must be double-spaced. A page should contain not more than 25 lines. Two copies of the text in paper and one copy in a CD must be submitted to the Editor and the corresponding author is advised to keep another copy with him. The corresponding author must give it in writing in his covering letter that the same matter will not be submitted elsewhere if accepted. He must also enclose the copyright transfer of his work to this journal. The papers sent will be subjected to peer review. The accepted manuscripts become the permanent property of this Journal. The author is informed that, if his work is returned to him for correction / clarification after peer review, he should effect the same and send the manuscript back to the Editor within one month. Each manuscript component mentioned here under must begin with a new page and the pages are to be numbered at the right tip corner starting from the Title page.

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2. ABSTRACT: The abstract is to be given in the beginning itself. It should not exceed 200 words. It must contain the aim, methodology, results and conclusion. For case report, summary / conclusion alone is to be given.

   KEY WORDS (maximum five) in capitals are to be included at the end of Abstract.

3. INTRODUCTION: Describe the aim of the study, along with the hypotheses that were tested. Only necessary references are to be given

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6. DISCUSSION: The discussion should be to the point and relevant to the subject under discussion. This section can be combined with the previous one if the author desires. Avoid speculations. Use only standard abbreviations or the abbreviations already introduced.

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