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ADDRESS FOR ALL CORRESPONDENCE: Dr. Meena Chakrabarti, Editor KJO, Chakrabarti Eye Care Centre, Kochulloor, Medical College PO, Trivandrum 695 011, Ph-0471-2555530, 2449599 Fax:- 0471-2558530, E-mail: tvm_meenarup@sancharnet.in

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KJO – CHANGING TIMES

We are all aware of the fact that the KJO is an excellent scientific journal. When I took charge as the new editor I was aware of the implications and the heavy burden of the responsibility that I will have to shoulder. This implied that I have to shoulder that heavy but welcome burden of maintaining the present high standards set by my predecessors, and , at the same time, strive harder to raise these standards even higher by enhancing the quality and content of the journal. In this era of "Changing times" where the state and level of knowledge is constantly changing, we have to work hard to uphold traditional aims and goals and at the same time bring about an evolution to meet the needs of the morrow.

While retaining the basic structure of the journal we have tried to incorporate certain changes. Good scientific material, based on research done in our institutions should first find a forum of expression in the state journal. I send a heartfelt and sincere request to all my colleagues to use this forum and publish their research work. I hope the next few years will witness the publication of original research work by our own young researchers.

We are planning to have a section each on Community Ophthalmology, Ocular Pharmacology and Ophthalmic Instrumentation. A section on Ophthalmic Surgery will deal with specific aspects and step by step methodology adopted for a particular surgical procedure. Current Practice Patterns to be introduced from this issue onwards will focus on the prevailing practice pattern for a particular condition focusing on the emerging, current and controversial practice aspects. This section is included for the benefit of the practicing ophthalmologist and the post graduate student. We are also planning to bring out a tear sheet which will contain in synopsis the essence of managing a particular problem. Brief reports on interesting cases, a photo essay capturing the various stages in the natural history of a disease, book review and journal review have all been planned as part of each issue of the Journal.

The editorial board shall endeavour to meet the high expectations of every reader ranging from the academicians of top institutes to the silent dedicated clinical ophthalmologists working in remote areas who depend solely on this journal as the primary source of information.

I fully understand that it will not be possible to achieve what has been outlined above on my own. I am fortunate to have an enthusiastic editorial and advisory board consisting of a perfect blend of young researchers, experienced teachers, reputed ophthalmologists and excellent strategy makers. With their help – and yours! – KJO will surely make long strides in the coming years.

Let us pledge to transform the KJO into an excellent scientific publication, keeping in mind the need to change and evolve for the better. Let us pledge to raise the status of this journal to a point where each one of us will be proud to be a contributor!

Editor

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Neuroretinitis - Review

Dr. Renuka Srinivasan, MS, DO, Professor

Dr. Subashini Kaliaperumal, MS, DNB, FRCS (Glasg), Senior Resident, Department of Ophthalmology, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry-605 006, India

Address for correspondence:
Dr. Renuka Srinivasan, MS, DO, Professor, Department of Ophthalmology,
Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry-605 006, India
E-mail:renuka@hotmail.com

Introduction

Neuroretinitis is a particular form of optic neuropathy characterised by acute unilateral visual loss in the setting of optic disc swelling and hard exudate arranged in a star figure around the fovea.[1] It affects persons of all ages, although it occurs more often in the third and fourth decades of life, with no gender predilection.[2,3] It is mostly unilateral and may be precipitated by various, known and unknown factors.

Neuroretinitis is a rare clinical entity often confused with the more common papillitis or papilledema. The fundus pictures have several common features and can be mistaken for one another by ill-experienced clinicians and sometimes even by ophthalmologists and neurologists. However, there are diagnostic features distinctive for neuroretinitis. It is a distinct clinical entity with a different etiopathogenesis. Likewise its management and prognosis too differs from fundoscopically similar entities such as papilledema and papillitis, which are encountered more often in our clinical practice.

Though the term neuroretinitis emphasizes clinical involvement of both disc and retina, the pathogenic locus is within the optic nerve head and macula is not the primary disease locus.

Clinical picture

The clinical picture of neuroretinitis is characteristic and clinically distinct from other optic neuropathies. The condition is usually painless but some patients complain of eye pain that may worsen with eye movements as seen in optic neuritis. If the neuroretinitis is due to an infectious process, there may be associated fever, malaise or headache. Visual acuity at presentation can range from 20/20 to light perception. The degree of colour deficit is usually worse than the degree of visual loss would suggest. The most common field defect is a cecocentral scotoma, but central scotomas, arcuate defects, and even altitudinal defects may be present. A relative afferent pupillary defect is present in most patients, unless the condition is bilateral. This is indicative of optic disc involvement. Absence of afferent pupillary defect indicates a primary macular involvement.[4] The degree of optic disc swelling ranges from mild to severe, depending in part on the timing of the first examination. In severe cases, splinter haemorhages may be present. Segmental disc swelling has been reported. A macular star figure composed of lipid (hard exudates) may not be present when the patient is examined soon after visual symptoms begin, but tends to become more prominent as the optic disc swelling resolves.[5] Small, discrete, usually white, chorioretinal lesions may occur in both the symptomatic and asymptomatic eyes.[6] Posterior inflammatory signs consisting of vitreous cells and venous sheathing as well as occasional cells and flare may occur.

Fluorescein angiography

Fluorescein angiography in patients with acute neuroretinitis demonstrates diffuse disc swelling and leakage of dye from vessels on the surface of the disc. The retinal vessels may show staining in the peripapillary region. But the most important point to note is the absence of leakage from the macular vasculature.

Pathogenesis:

The pathogenesis of neuroretinitis is obscure. It is related to direct involvement by an infectious process or inflammation leading to edema of the optic nerve and cellular and fluid exudation from the inflamed area of peripapillary retina. There is abnormal permeability of capillaries deep within the optic disc, with no abnormality of retinal vasculature. Leakage of lipid-rich exudates into the adjacent subretinal space and plane of the outer plexiform layer results. With reabsorption of serum, lipid precipitates in a stellate pattern.

Clinical course:

Neuroretinitis is usually a self-limited disorder with a good visual prognosis. Typically over 6 to 8 weeks, the optic disc swelling resolves, and the appearance of the disc becomes normal or mildly pale. The macular exudates appear late and progress over about 7 to 10 days, then remain stable for several weeks before gradual resolution occurs over 6 to 12 months. Most patients ultimately recover good visual acuity, although some complain of persistent metamorphopsia or nonspecific blurred vision from mild disruption of the macular architecture. Most patients do not experience a subsequent attack in the same eye, and only a few patients develop a similar attack in the fellow eye.

Recurrent Idiopathic Neuroretinitis is an uncommon condition in which repeated acute episodes lead to progressive and permanent visual loss.[4] This disorder usually affects young adults and has no predilection with regard to sex. The interval between attacks is quite variable ranging from 1 month to 9.8 years. Treatment of the acute attack with either oral or intravenous corticosteroids has not appeared to alter the visual prognosis of this condition. Although the cause of recurrent Idiopathic Neuro retinitis has not been elucidated, an autoimmune disorder has been proposed that involves occlusive vasculitis affecting the optic disc. Long-term immunosuppression has been tried in some of these patients. [7]

Etiology: (Table 1)

Neuroretinitis is thought to be an infectious or immune-mediated process that may be precipitated by a number of different agents. The common infections that cause neuroretinitis are cat-scratch disease, and the

Table 1. Causative Agents Implicated in Neuroretinitis

Infectious

Viruses- Herpes, Hepatitis B, Mumps, Herpes Zoster, HIV, HBV.
Parasites- Toxoplasma, Toxocara
Fungi- Histoplasmosis
Bacteria- Syphilis, Leptospirosis, Cat scratch disease, Lyme disease, Tuberculosis

Parainfectious (Immune mediated)

Idiopathic

Leber's stellate neuroretinitis

Differential diagnosis of optic disc edema with macular star

AION
Hypertensive retinopathy
BRVO/CRVO, rarely papillophlebitis
Papilledema
Compressive optic neuropathy
Infiltrative optic neuropathy
Nonspecific uveitis

spirochetoses especially syphilis[8], Lyme disease, and leptospirosis.[2] Cat-scratch disease accounts for two thirds of cases.[9,10] Additional causes include toxoplasmosis,[11] mumps,[12] salmonella,[13] tuberculosis,[14] and histoplasmosis. Rarely, a toxocaral granuloma within the optic nerve head produces a similar ophthalmoscopic picture.[15] Despite thorough evaluation, approximately one quarter of cases remain idiopathic.

Neuroretinitis is commonly associated with an antecedent viral syndrome, suggesting a possible viral etiology for upto 50% of the cases; however viruses are rarely cultured from the CSF of such patients, and serological evidence of a concomittant viral infection is usually lacking. Proposed causative viral agents include herpes simplex, hepatitis B, mumps, and the herpes viruses associated with the acute retinal necrosis syndrome. HIV with opportunistic infections especially syphilis and hepatitis viruses have been implicated in neuroretinitis.[16]

Cat scratch disease

Cat- scratch disease, a systemic infection caused by the pleomorphic gram-negative bacillus Bartonella henselae, is the most common infectious process associated with neuroretinitis. Patients present following cat exposure with fever, malaise, headache, eye pain and blurred vision. Examination typically reveals local lymphadenopathy. Some patients also have symptoms of arthritis, hepatitis, meningitis, or encephalitis. Decreased visual acuity (ranging from 20/40 to counting fingers) is often associated with dyschromatopsia and afferent pupillary defects. Ophthalmoscopic findings include neuroretinitis, cottonwool spots, multiple discrete lesions in the deep retina, and stellate macular exudates. *B. henselae* infection is confirmed with positive blood cultures or elevated immunofluorescent antibody titers or both. Therapy is aimed to promote resolution of neuroretinitis, restoration of visual acuity, and clearance of bacteremia.[9] Electrophysiologic studies show that when compared to the fellow eye, affected eyes have subnormal contrast sensitivity, abnormal color vision, and abnormal visually evoked potentials. However electroretinograms may be normal. Recently polymerase chain reaction has been found to be a valuable method of diagnosing cat-scratch disease when serology is considered negative or borderline.[17]

Lyme disease

Neuroretinitis in Lyme disease may be unilateral or bilateral, but when bilateral is usually simultaneous and symmetric.[18] The patients usually live or work in an endemic area and may give a history of a tick bite within the last 6 months. They often have cutaneous, cardiac and neurological manifestations. Cardiac manifestations include atrioventricular block, myocarditis, cardiomyopathy, and pericarditis. Neurological manifestations include meningitis, myelitis, encephalitis, cranial and peripheral neuropathies. Although ocular manifestations of Lyme disease have long been noted, they remain a rare feature of the disease. The spirochete invades the eye early and remains dormant, accounting for both early and late ocular manifestations. A nonspecific follicular conjunctivitis occurs in approximately 10% of patients with early Lyme disease. Keratitis is characterized by nummular nonstaining opacities. Inflammatory syndromes, such as vitritis and uveitis, have been reported; in some cases, a vitreous tap is required for diagnosis. Neuro-ophthalmic manifestations include neuroretinitis, multiple cranial nerves involvement and optic atrophy. Criteria for establishing that eye findings can be attributed to Lyme disease include the lack of evidence of other disease, other clinical findings consistent with Lyme disease, occurrence in patients living in an endemic area, positive serology, and, in most cases, response to treatment. Management of ocular manifestations often requires intravenous therapy. [18]

Leber's stellate retinopathy

When there is no proven etiology to the disease, a diagnosis of Leber's idiopathic stellate retinopathy is made.[19] Thus; it is a diagnosis of exclusion made after other known causes of neuroretinitis are ruled out. It usually affects children or young adults. This diagnosis is mostly not assigned to patients aged more than 50 years until treatable causes of neuroretinitis or a macular star have been excluded. Most cases are unilateral. The incidence is equal in both sexes. Patients present with acute loss of vision with or without ocular pain. A nonspecific viral illness precedes or accompanies the visual loss. Presenting visual acuity may be 20/20 to LP. But, most cases are in the 20/40 to 20/200 range.

In children, Leber's neuroretinitis must be distinguished from anterior optic neuritis and papillitis, since multiple sclerosis occasionally develops in children with these diseases.[20] A distinguishing feature is the development of macular star. In Leber's disease, the target tissue as suggested by Gass [21] is vascular whereas in anterior optic neuritis caused by demyelinating disease, the target tissue is primarily neural. Leber's neuroretinitis usually resolves without treatment within 6-12 weeks.[19] However the macular star may persist beyond this period. Most patients recover good visual acuity with over 90% returning to 20/50 or better. Recurrences are very rare although in bilateral cases, involvement of the fellow eye may follow the first. Fluorescein study demonstrates intense hyperfluorescence due to leakage from capillaries within the disc. There is no leakage from the retinal vessels in the macula.

Idiopathic retinal vasculitis aneurysms and neuroretinitis (IRVAN)

IRVAN syndrome is the acronym for idiopathic retinal vasculitis, aneurysms and neuroretinitis. This syndrome typically affects young, healthy individuals; it has a female predominance, is usually bilateral and is not associated with any systemic abnormalities. The most characteristic feature is the presence of macroaneurysms seen as dilatations of the retinal and optic nerve head arterioles. Exudative retinopathy and capillary non-perfusion is usually seen adjacent to retinal and optic nerve head aneurysms and is concentrated in the peripapillary location. This condition is not a true neuroretinitis as there is no clinically evident neuropathy but only late diffuse staining of the optic nerve head due to local vascular changes. There is little role of steroids and panretinal photocoagulation is advocated if retinal neovascularisation occurs.[22] Central retinal vein occlusion and hypertensive retinopathy may also have disc edema and macular star figure but have associated multiple flame-shaped haemorrhages and soft exudates.

Diffuse unilateral subacute neuroretinitis (DUSN)

DUSN is a progressive parasitic disease affecting the outer retina and retinal pigment epithelium (RPE). This syndrome is primarily unilateral, although bilateral cases have occurred. The ocular findings include visual loss, vitreous cells, optic disc inflammation and leakage, and transient recurrent crops of gray-white outer retinal lesions.[23] Stationary or migrating nematodes have been identified deep in the retina or in the subretinal space. DUSN is a condition in which prompt identification and destruction of the infecting nematode can result in the cessation of symptoms and the preservation of good visual acuity. If untreated, the disease progressively damages the retina and the optic nerve leading to severe visual loss. Laser photocoagulation of the nematode is the treatment of choice.[24] Visual acuity may not improve significantly unless the worm is killed soon after onset of visual loss. It has been found that thiabendazole is effective in the treatment of some patients when the worm cannot be found and when DUSN is accompanied by a moderate degree of vitritis that is associated with a breakdown in the blood-retinal barrier.[25] But by and large, antihelminthics have not been found to be that effective in confirmed cases of DUSN.[26] Regardless of the nature of the causative nematode, DUSN should always be suspected in healthy patients with unilateral ocular signs of persistent vitritis associated with papillitis, retinal vasculitis, and multifocal lesions involving the outer retinal layers.

Mutiple sclerosis

Multiple sclerosis is one condition that is not associated with neuroretinitis.[2] It is a well known fact that patients who develop typical optic neuritis are prone to develop multiple sclerosis but there is no similar increased tendency for patients who experience an attack of neuroretinitis.[27] Thus, when a diagnosis of an attack of acute optic neuropathy as an episode of neuroretinitis rather than anterior optic neuritis is made, it substantially alters the neurologic prognosis in the patient being evaluated. Nevertheless, there have been anecdotal reports of patients with multiple sclerosis who developed neuroretinitis .[28]

Conditions mimicking neuroretinitis (Table 2)

Certain noninfectious and noninflammatory conditions mimick neuroretinitis as they are characterised by optic

Table 2. Differentiating features between Neuroretinitis and other closely resembling entities.

	Neuroretinitis	Papillitis	Papilledema	CRVO	AION
VA	6/60-6/12	Light perception to 6/12	No visual loss	Moderate to severe visual loss	Moderate to severe impairment
Pupillary reactions	Relative afferent pupillary defect (RAPD+)	RAPD+	Normal	Normal/RAPD+	RAPD+
Laterality	Unilateral, rarely bilateral	Unilateral/bilateral	Always bilateral	Unilateral	Typically unilateral
Eye pain	Nil	Pain especially on upgaze	Nil	painless	painless
Visual fields	Centrocaecal scotoma	Central/ centrocaecal scotoma	Enlarged blind spot	Normal	Altitudinal field defects
Color vision	Severely impaired, disproportionate to visual loss	Severely impaired, disproportionate to visual loss	Normal	Normal	Diminished in proportion to level of v/a
Systemic symptoms	Fever, rash	Weakness of limbs	Headache, vomiting	-	Headache, jaw claudication, polymyalgia rheumatica
Fundus findings	Disc swelling of 2D,hyperemic with macular star figure	Disc swelling rarely above 2D, venous engorgement and haemorrhages less marked	Disc swelling frequently higher, upto 8-9D, more venous engorgement, macular star may develop	Disc edema, macular star along with haemorrhages and soft exudates	Pale disc edema
Fluorescein Angiography	Leakage from disc and peripapillary retina	Leakage from disc and peripapillary retina	Leakage from disc and peripapillary retina	Shows areas of capillary non-perfusion	Unequal choroidal filling in arterial phase
VEP	Decrease in amplitude, increase in latency	Decrease in amplitude, increase in latency	Normal	-	Decrease in amplitude
Specific associations	Syphilis, cat-scratch disease, Lyme's disease	Multiple sclerosis	-	hypertension, diabetes, glaucoma	Giant cell arteritis, hypertension, diabetes
Prognosis	Good	Good	Good with relief of raised ICT	Depends on initial visual acuity	Poor

disc swelling that may on occasion be associated with the development of a macular star figure. These mimicking conditions include papilledema, anterior ischemic optic neuropathy, and infiltration of the optic disc by tumor.[14] Systemic hypertension may also produce both optic disc swelling and a macular star figure. The disk edema and retinopathy resolves after the hypertension is controlled.[29] Optic disc swelling in patients with systemic vascular disease like diabetes and hypertension can be differentiated form neuroretinitis by the absence of abrupt visual loss, background retinopathy and a medical history of such conditions. Spontaneous resolution of the disc edema and recovery of visual acuity serve as distinguishing features of neuroretinitis from papilledema and ischemic optic neuropathy.

Investigations: (Table 3)

Investigation into the etiology of neuroretinitis should begin with a careful history including questioning regarding sexually transmitted diseases, cat-scratches, skin rashes, tick bites, lymphadenopathy, fever, and flu-like illnesses. Complete physical and ocular

Table 3. Investigations into the etiology of Neuroretinitis

Ocular
Color vision, contrast sensitivity, Central fields, Fluorescein angiography, VEP
Systemic
Blood culture-cat scratch disease
VDRL and FTA-ABS- Syphilis
Viral serology
Mantoux , chest X ray
ESR
Lumbar Puncture- opening pressure, cells, proteins, glucose,
CSF culture for bacteria especially leptospirosis and fungi
Immunofluorescent antibody test- cat scratch disease
ELISA- Toxoplasmosis, Toxocariasis
Polymerase chain reaction- cat scratch disease
Neuroimaging

examinations are essential. Screening with serological testing for treatable diseases such as cat-scratch disease, syphilis, and Lyme disease, analysis of CSF, neuroimaging may be desirable in the appropriate setting. In the absence of a proven etiology a diagnosis of Leber's idiopathic stellate neuroretinitis may be entertained.

VEP is useful in the setting of multiple sclerosis where there is a latency of the P100 wave and a decrease in amplitude. It may be abnormal in neuroretinitis. ERG assesses the functional integrity of the retinal layers and hence normal in disorders involving ganglion cells and optic nerve as in optic neuritis and neuroretinitis.

Treatment

Treatment of neuroretinitis depends on whether there is an underlying infectious or inflammatory condition that requires therapy. No treatment is required in the idiopathic group as the disease is self-limiting. Cat-scratch disease is usually described as a benign, self-limited illness.[30] Patients with neuroretinitis associated with cat scratch disease have been treated with prednisolone, dexamethasone, clindamycin, ciprofloxacin, trimethoprim-sulfa, or tetracycline and all had improved vision.[31,32] Doxycycline and rifampicin appear to shorten the course of disease and hasten visual recovery. Long-term prognosis is good, but some individuals may acquire a mild postinfectious optic neuropathy.

Patients with neuroretinitis and secondary or late syphilis should be treated with intravenous penicillin, and patients with Lyme disease should also be treated with an appropriate antibiotic such as ceftriaxone, amoxycillin, or tetracycline. Though systemic steroids have been tried, there is no definite evidence that such

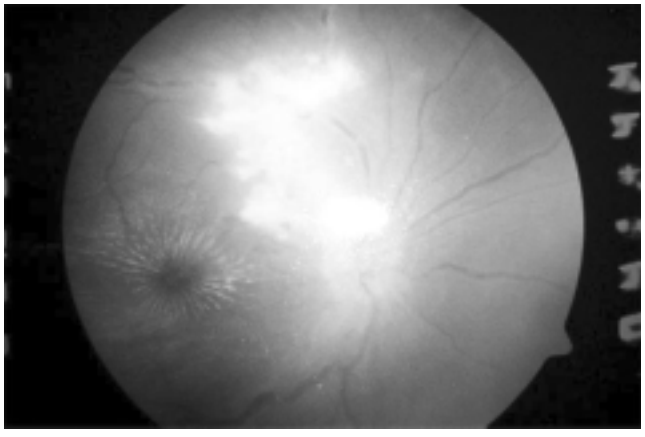


Fig 1. Neuroretinitis with vasculitis involving the superotemporal vessel.

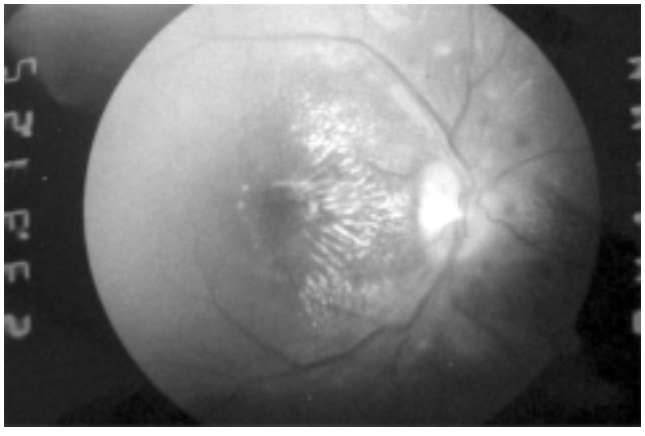


Fig 2. Grade IV hypertensive retinopathy mimicking as Neuroretinitis.

treatment alters either the speed of recovery or the ultimate outcome.[19] The prognosis in most cases of idiopathic neuroretinitis is excellent as it is self limiting.

Conclusion

Thus in most cases, neuroretinitis represents a self-limiting, benign, systemic inflammatory process with rarely a specific etiology being identified. The extent of diagnostic examination should be predicted based on the presence or absence of associated constitutional symptoms. Vision should be expected to recover within weeks to months. Nevertheless, the ophthalmologist should use caution in predicting ultimate visual prognosis.

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ORIGINAL ARTICLE

Chengamanad Diabetic Retinopathy Awareness Study (CDRAS)

Dr. Mahesh G, MS, DO, DNB, FRCS (Ed)[1]
Dr. Anna Elias, DNB[1]
Dr. Sandhya N, MS,DO[1]
Dr. A. Giridhar, MS,DO[1]
Dr. S.J. Saikumar, MS,DO,DNB,MNAMS[1]
Mr.Sankaranarayanan, MSc[1]
Mr. Thomas, MSW[1]

[1]Giridhar Eye Institute, 28/2576(A), Ponneth Temple road, Kadavanthra, Cochin, Kerala, India. E-mail: girieye@vsnl.com
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[1-4]Diabetic retinopathy is one among the leading causes of blindness in developed countries. Though it ranks behind cataract in magnitude in developing countries the number of blindness cases due to diabetic retinopathy is catching up. There is increasing prevalence and incidence of diabetes with increase in age. The number of diabetic population in India will increase from 19 million in 1995 to 57 million in 2025. There are about 12 million blind people in India and the increase in diabetic population will add to this number in the coming years. The situation is worsened by illiteracy and lack of awareness of largely preventable diabetic retinopathy complications in a country like India. There are few well-conducted population based studies from western countries. From India data is available on diabetic retinopathy status in urban population and hospital based studies.

All diabetic patients will develop retinopathy over time, the prevalence being directly proportional to the duration of diabetes. The diabetic retinopathy study and early treatment diabetic retinopathy study have conclusively advocated the role of regular eye examination to ensure early detection and treatment of diabetic retinopathy and prevention of severe visual loss. Individuals with type 2 diabetes are recommended to have a dilated eye examination at the time of diagnosis and annually there after. The awareness and adherence to this periodic eye check up is poor even in highly literate and educated population in developed countries like Japan and USA. So the scenario in developing countries like India where the literacy level is much lower is expected to be worse. These factors highlight the need for population based diabetic retinopathy awareness and prevalence detection studies in developing countries. These studies will help in the proper planning and allocation of funds in an useful manner for prevention of diabetic retinopathy complications. [12]

Unlike other states in India, Kerala has high health care indices and literacy rate. There is no strict division of urban and rural areas in Kerala. The statistics show that the literacy rate is 90.92% and infant mortality rate is 14 per 1000 live births in Kerala. This is at par with developed countries. Even with this background the diabetes related ocular morbidity is on the rise. There are plenty of health related programmes and reports in visual media on diabetes and its complications. Still we find a lot of patients reporting with vitreous haemorrhage and proliferative diabetic retinopathy who says they did not know that periodic check up of eye was essential. To evaluate the points leading to this ignorance among diabetic patients we

conducted a pilot study in our institute, which is a tertiary referral center for treatment of retinal disease. Optometrist or ophthalmologist interviewed 1000 diabetic patients. A questionnaire based on knowledge, awareness and practice (KAP) was used. This pilot study had the disadvantage that the target patients did not represent the true population in Kerala. There was bias because the patients were referred for retinal examination by other doctors. The need for a population based survey was evident.

We conducted a population based door-to-door survey of 24500 people in 5752 houses in Chengamanad Panchayat of Ernakulam district in Kerala state in the southern most part of India. 1096 self-confessed diabetic patients were identified and their awareness of diabetic retinopathy was assessed by a questionnaire.

Objective

(1) To determine the level of awareness of diabetic population regarding the blinding complications of diabetic retinopathy. (2) To evaluate the factors responsible for non-adherence to eye examination in diabetic patients.

Method:

Design: Population based epidemiological study.

Setting: Chengamanad Panchayat in Ernakulam district in Kerala state, India with 14 wards and a population of 25400.The study was done in Chengamanad Panchayat in Kerala state.

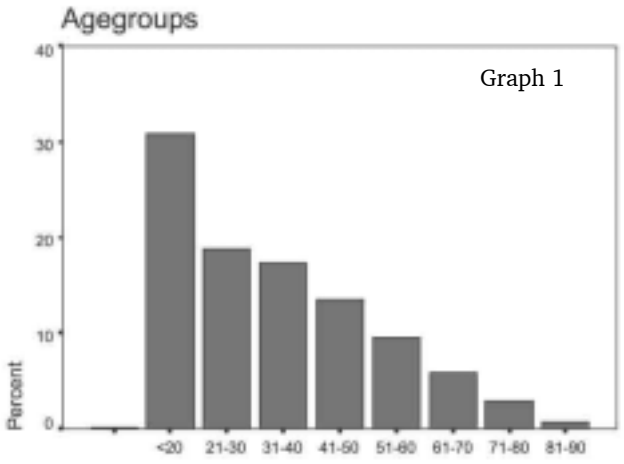
Recruitment and trial enrolment: The voters' list of the whole Chengamanad Panchayat was taken from the Panchayat office. Panchayat is divided into 14 wards. The total population of the Panchayat was 25400 with 5752 houses. The next step was to identify the volunteers. Kudumbasree units are a very well knit network of social workers and self-employed group with a minimum educational qualification was 10^{th} standard. All the volunteers were able to read and write Malayalam, which is the local language. Selected 30 volunteers underwent training by ophthalmologist regarding diabetic retinopathy and how to ask the questionnaire. After the training classes were over the volunteers were interviewed to check the basic knowledge of the disease. 30 volunteers were divided into groups and assigned 14 wards. A total of 5762 houses were covered in 4 weeks. Weekly review meeting was done. Each volunteer was given an incentive for each house visited. Double-checking was done randomly by one of the investigators who visited already covered area. In the case of any disparity in the data that volunteer's service was terminated and the resurvey was done. All the houses were covered in 4 weeks between June and July 2004. The questionnaire had two parts. One general questionnaire was filled in all the houses and self confessed diabetic patients were identified. They were given the main questionnaire based on knowledge, attitude and practice, which was a 24-point questionnaire. The decision to participate in the study was left to the patients and there were no refusals. The data was entered into a computer in the main center for analysis by a trained data entry operator and analyzed by a biostatistician. SPSS version 11.0 statistical package was used for analysis. The whole study was approved by the ethics committee of our institute thus sticking to the Helsinki declaration.

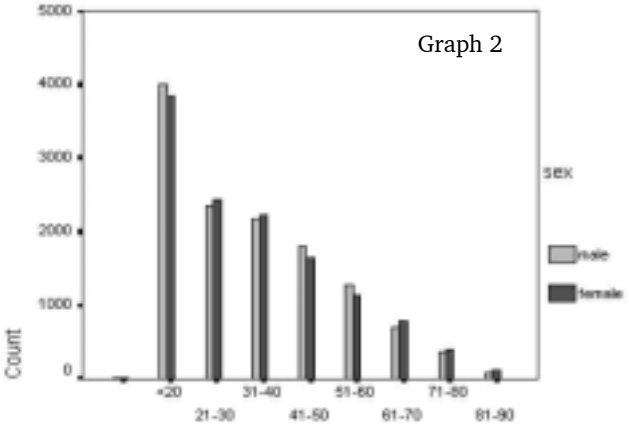
Results

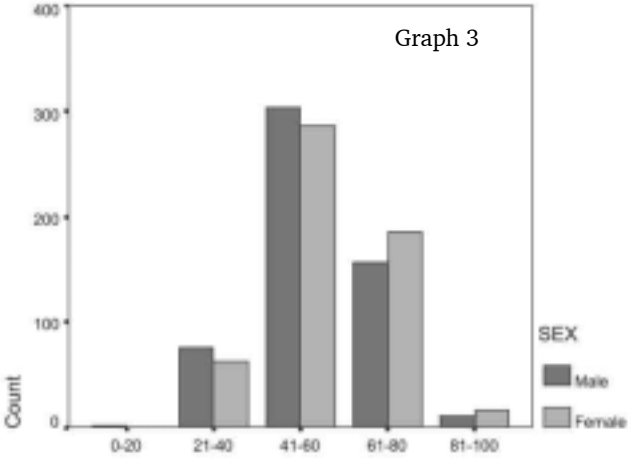
The total study population was 25400. The following observations were made in the cohort. There were 12785 males and 12615 females. The graph shows the more or less equal distribution of the male and female population. The pattern shows the preponderance of young population in this cohort. (Graph 1&2) There were 1096 self-confessed diabetes mellitus people in the population. They were given the questionnaire regarding the awareness of diabetic retinopathy. There were 547 males and 549 females in the target population. The age wise splitting of the groups is given below (Graph 3)

Table 1.

		SEX Male	Female	Total
Age group	0-20	1		1
	21-40	76	62	138
	41-60	303	286	589
	61-80	156	185	341
	81-100	11	16	27
Total		547	549	1096







Though the majority of the diabetic patients were in the age group of 41-80 a striking 139 patients were under the age of 40 years. This young diabetic population contributed 12.68 %.

The educational status of the patients were as follows (See Graph 4 & Table 2)

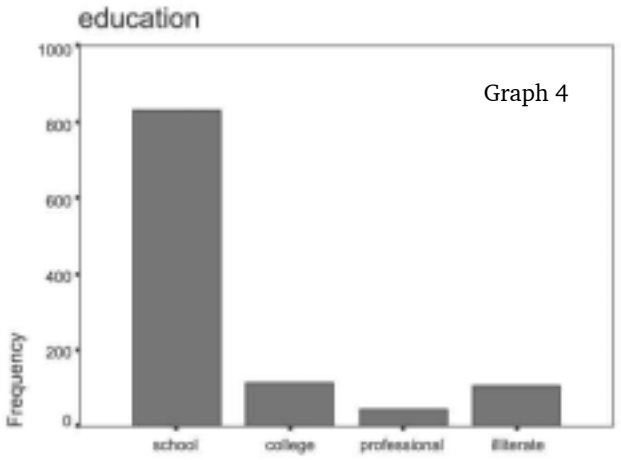


Table 2: Education

	Frequency	Percent
School	831	75.8
College	113	10.3
Professional	44	4.0
Illiterate	108	9.9
Total	1096	100.0

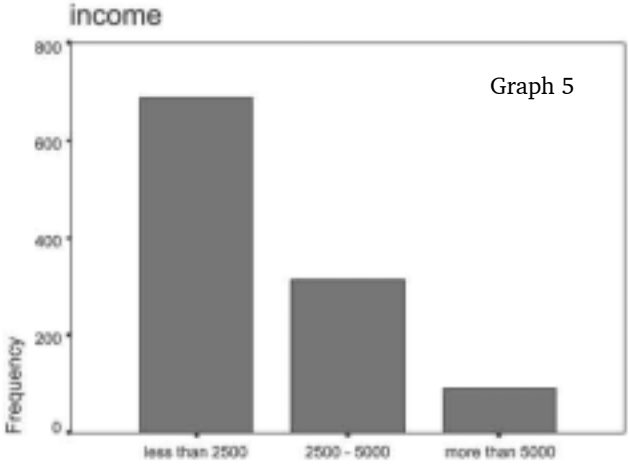
Table 3: Economic background

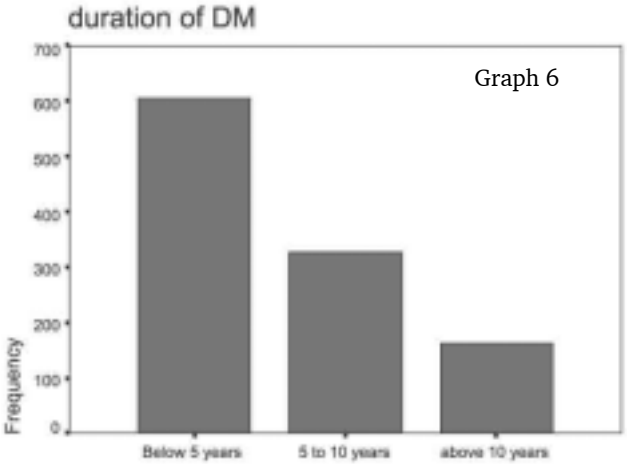
	Frequency	Percent
Less than 2500	688	62.8
2500 - 5000	316	28.8
More than 5000	92	8.4
Total	1096	100.0

The economic background of the patients were shown above (Graph 5 & Table 3)

The duration of diabetes mellitus is shown in the graph 6.

701 patients knew that diabetes could affect many organs in the body. (Graph 7)

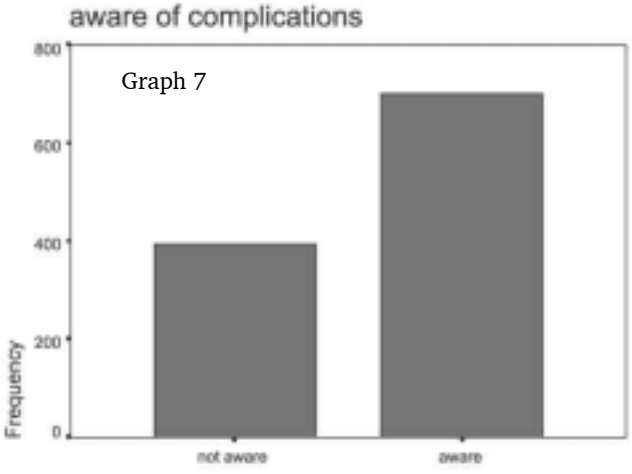




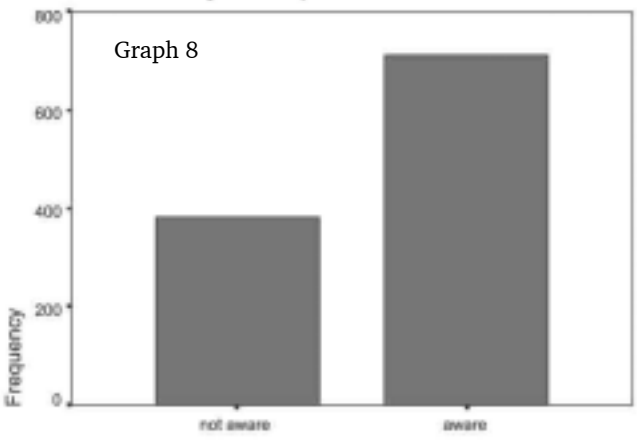
Graph 6

713 patients were aware that diabetes mellitus could affect the eyesight. (Graph 8)

698 patients felt that they were not educated about the complications of diabetes mellitus especially diabetic retinopathy. (Graph9)

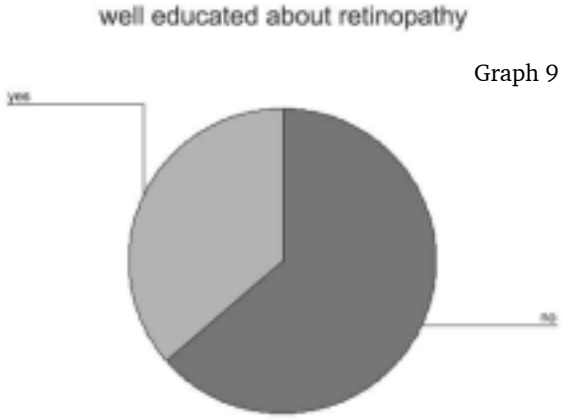


Graph 7

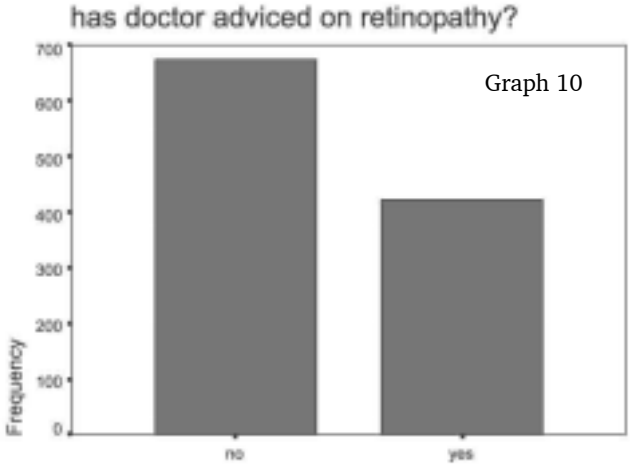


Graph 8

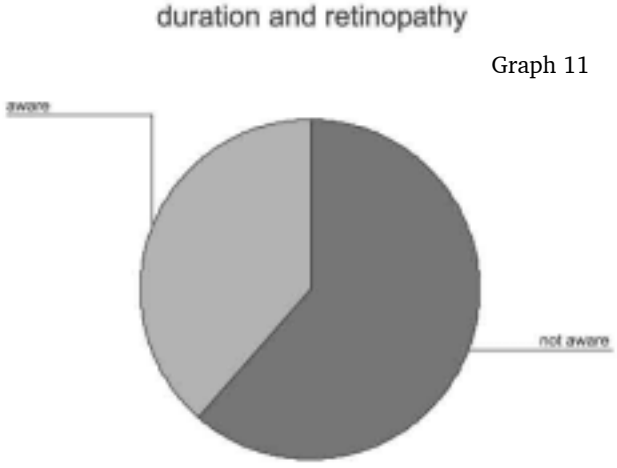
674 patients said the treating physician did not tell them about eye related problems. (Graph10)



Graph 9



Graph 10



Graph 11

Regarding the source of information about diabetes related eye problems in those people who knew about it, 77 patients got the information from other patients, 453 from medical doctor, 42 from eye specialist and

126 from media. 674 patients were not aware that duration of the diabetes has any relation with retinopathy (Graph 11)

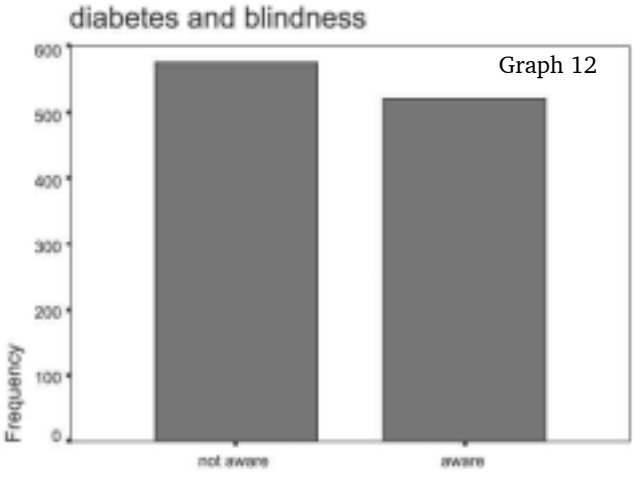
576 patients were not aware that diabetes could result in blindness. (Graph12)

Only 479 patients were aware that frequent eye check ups are necessary for all diabetic patients.(Graph 13)

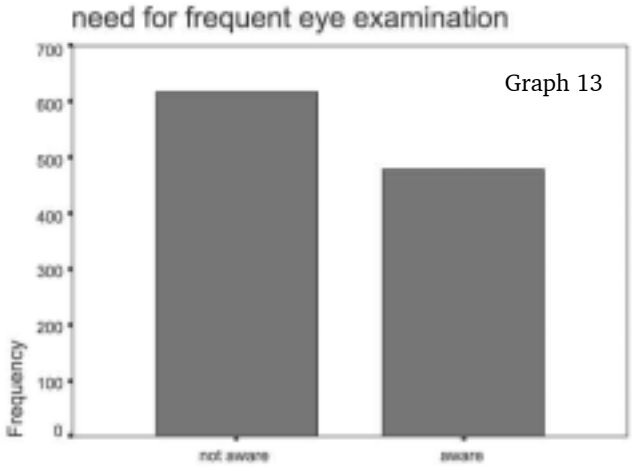
Practice related question was asked to know the frequency of eye check ups. (Graph14)

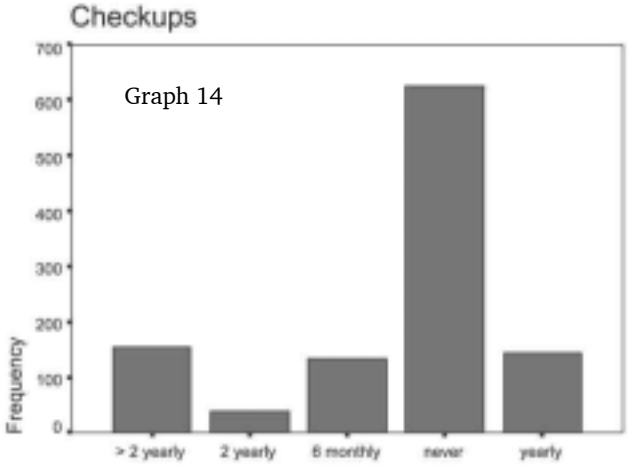
Checkups

	Frequency	Percent
> 2 yearly	154	14.1
2 yearly	39	3.6
6 monthly	134	12.2
Never	625	57.0
Yearly	144	13.1
Total	1096	100.0



Preferred practice pattern of diabetic retinopathy suggest eye examination at the time of diagnosis and





at least yearly check ups in type 2 diabetes mellitus. So we clubbed people who do 6 monthly eye check up and yearly eye check up as 'Adherent' and all the other 3 groups as 'Non adherent'. There were 278 adherent and 818 non-adherent patients. This question regarding practice was analyzed against knowledge questions 1) Whether you know diabetes can affect the eyes? (Q7 in table below) 2) Whether you know the duration

Variables in the Equation – Multiple Logistic Regression Analysis SPSS version 11.0

	B	S.E.	Wald	df	Sig.	Exp(B)	95.0% C.I. for EXP(B) Lower	Upper
Q7	3.134	.612	26.238	1	.000	22.958	6.921	76.153
Q10	.197	.205	.918	1	.338	1.217	.814	1.819
Q11	.841	.226	13.896	1	.000	2.319	1.490	3.608
Q14	.696	.238	8.577	1	.003	2.005	1.259	3.193
Constant	-4.854	.579	70.183	1	.000	.008		

Variable(s) entered on step 1: Q7, Q10, Q11, Q14.

of diabetes has anything to do with the retinopathy? (Q10 in table below) 3) Whether you know frequent eye check ups are essential? (Q11in table below) 4) Whether you know diabetic retinopathy is a blinding disease? (Q14 in table below). Multiple logistic regression analysis was done. From this odds ratio and 95% confidence intervals were calculated.

The OR (Odds Ratio) of non adherence in people who are not aware that diabetes can affect the eyes to those who are aware was 22.958 (95% Confidence interval 6.921 – 76.152)

The OR of non-adherence in people who are not aware that duration of diabetes has anything to do with the retinopathy to those who were aware was 1.217 (95% Confidence interval 0.814- 1.819)

The OR of non-adherence in people who did not know that frequent eye check ups are necessary to those who knew was 2.319 (95% confidence interval 1.490-3.608)

The OR of non-adherence in people who did not know that diabetes can result in blindness to those who knew was 2.005 (95% confidence interval 1.259-3.193)

The proportion of patients who were non aware that diabetes can affect the eye and non adherent is given below. This is reflected in the large odd ratio obtained in the multiple logistic regression analysis.

aware of eye complications * Adherence Cross tabulation

		Adherence Non adherent	Adherent	Total
Aware of eye complications	Not aware	380	3	383
	Aware	438	275	713
Total		818	278	1096

Analysis was done to find the proportion of non-adherence in relation to the age, sex, education and income.

The non-adherence in 21-40, 41-60, 61-80 age groups were 79.7%, 74.53%, 71.84% respectively.

Age group * Adherence Cross tabulation

		Adherence Non adherence	Adherence	Total
Age group	0-20	1		1
	21-40	110	28	138
	41-60	439	150	589
	61-80	245	96	341
	81-100	23	4	27
Total		818	278	1096

The percentage of non-adherence in males and females were 72.58% and 76.68%

SEX * Adherence Cross tabulation

		Adherence Non adherence	Adherence	Total
SEX	Male	397	150	547
	Female	421	128	549
Total		818	278	1096

Percentage of non-adherence in patients with school education, college education, professionals, and illiterates were 76.29%, 60.17%, 43.18%, and 89.81% respectively showing the impact of literacy.

Education * Adherence Cross tabulation

		Adherence Non adherence	Adherence	Total
Education	School	634	197	831
College	68	45	113	
Professional	19	25	44	
Illiterate	97	11	108	
Total		818	278	1096

Regarding economic status the proportion of patients who were non adherent in <2500, 2500-5000, >5000 monthly income group were 72.58%, 71.59% and 50% showing the better awareness in upper socio economic class.

Income * Adherence Cross tabulation

		Adherence Non adherence	Adherence	Total
Income	Less than 2500	546	142	688
	2500 - 5000	226	90	316
	More than 5000	46	46	92
Total		818	278	1096

Discussion

India has a large burden of visual impairment, including blindness. It is estimated that of the population of about a billion 1-1.5% are blind. [13-16] To deal with such a large burden of blindness, the priorities have to be based on reliable population based data. It is felt by some in India that DR is becoming a significant cause of visual impairment. The prevalence of diabetes is reaching epidemic proportions and it is the developing countries that will be affected most in the 21st century. 80% of all new cases of diabetes are likely to appear in developing countries. The number of people with

diabetes in the developing countries is expected to rise to 228 million in 2025. This represents 76% of total umber of people with diabetes in the world. India is likely to experience the highest percentage of increase of diabetic population, i.e. 195% in the next 20 years. In the developing countries less than half of people with diabetes are diagnosed. In India 70% of population live in villages while 80% of which is even worse in the villages. With the current health standards the life span of diabetic patients are also increasing. The complications of diabetes is directly related to the duration of the disease, thereby the morbidity due to diabetic eye disease will reach enormous levels which will be difficult to manage by the available eye health care resources. It is well known that timely diagnosis and treatment of diabetic retinopathy can prevent severe visual loss in a good majority of patients (ETDRS report). Therefore early detection is the key to the prevention of blindness in diabetic retinopathy. This is possible by improving health care facility and awareness of the disease. Kerala state in India is often quoted as a model for developing world. The health statistics in Kerala is superior to Indian national average and close to that of developed countries. The literacy rate in Kerala has contributed to the high health indices. Still the utilization of primary health centers and sub centers have been lower than the expected level. We come across people in our tertiary referral center, even from the high socio-economic class, presenting in the late stages of diabetic retinopathy with vitreous haemorrhage due to lack of awareness. Our in-house pilot study revealed lack of awareness in diabetic patients. This prompted us to undertake a population-based study.

It is very important to differentiate between *awareness and knowledge*. Having just heard about a disease is awareness and having understood the disease is knowledge.

The date clearly indicates the impact of lack of awareness in the non-adherence of diabetic patients doing regular eye examination. Multiple logistic regression analysis shows high odds ratio of non-adherence to lack of awareness in this population. But there is a significant relationship in literacy rate and non-adherence. 698 out of 1096 diabetic patients felt they were not educated about the complications of diabetic retinopathy. 674 patients felt they were not told about eye related problems by treating physician. 713 patients had heard that diabetes can affect the eyes *(Awareness)* But only 479 patients were aware that frequent eye check up are necessary and 674 patients were not aware that the duration of the disease has anything to do with diabetic retinopathy *(Knowledge)*. (Graphs 7,11 and 13). Lack of knowledge with little awareness is seen in this pattern. This data points to possible lacunae in our health care system. The treatment of diabetic retinopathy is out of reach of majority of patients in the rural settings because of lack of facilities in government hospitals. Here lies the importance of nongovernmental organizations and private sector in the management of diabetic retinopathy patients. Our study has the limitation of being conducted among self-confessed diabetes patients. There is a likely hood of missing nearly 5-10% of undetected diabetic patients among the population. The percentage of diabetic patients in our population was 4.47%. So this data is underestimating the impact of lack of awareness in the population. Our study points to some of the future needs in the management of diabetic retinopathy.

Conclusion

The basic necessity in preventing blindness due to diabetic retinopathy is its early detection and one of the methods by which it can be achieved is by improving the awareness and knowledge about the disease in patients with diabetes mellitus. The physicians and the ophthalmologists require further awareness training so that they can make the patient well educated about the disease.

To our best of knowledge this is the only *population based awareness study in non-urban* area regarding diabetic retinopathy.

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ORIGINAL ARTICLE

Endothelial Cell Study in Normal Population

Dr. Sarath R., Dr. Ramya R., Dr. Reena A., Dr. P.S. Girija Devi

Regional institute of Ophthalmology, Trivandrum

Specular microscopy enables a direct view of the endothelial cells.A complete qualitative and quantitative analysis of the endothelium is undertaken to define the cell conformation ,boundaries and the intersections along with definition of mean cell area and cell density using the fixed frame analysis or a variable frame analysis .Individual cell analysis is also possible using a digitizer.So specular microscopy helps in predicting corneas which are more likely to decompensate when subjected to stress like trauma or surgery.

This study is an attempt to understand the age related changes in the corneal endothelium in a normal population both in terms of endothelial cell density and average cell area

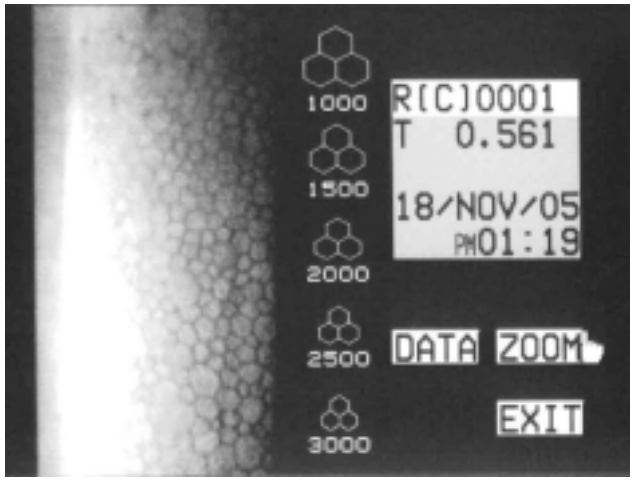
Materials and methods

408 eyes of 204 patients who attended the outpatient department in Regional Institute of Ophthalmology, Trivandrum during the period from May 2005 to September 2005 were included in the study. The age group of patients included for the study ranged from 10-70 years. Patients were grouped into various decades like 10-20, 20-30 and so on until 60-70yrs. So we had 34 patients included in each decade. The participants were enquired about detailed medical history and subjected to a careful slit lamp examination and fundus examination and IOP recording. Patients with history of Diabetes mellitus or Hypertension or evidence of anterior segment pathology like uveitis or glaucoma were excluded from the study. Also patients with past history of intraocular surgery like cataract surgery were excluded from the study.

Procedure

Corneal endothelium was studied using the TOPCON Specular microscope SP2000P.The recordings were



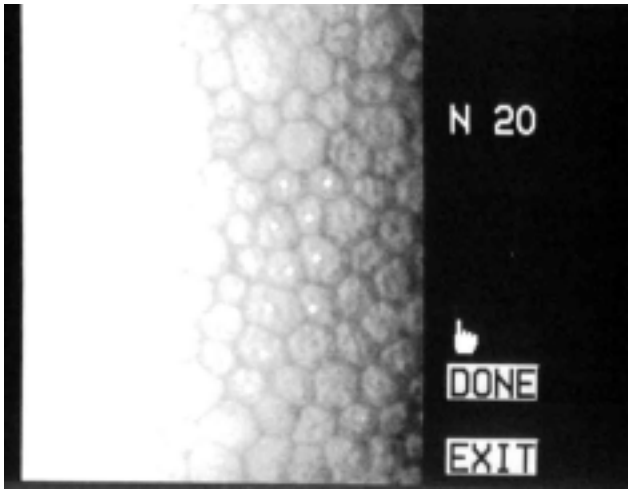


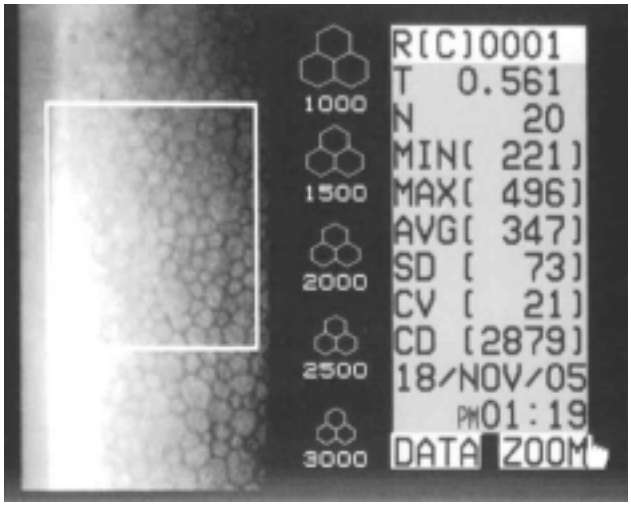
taken by a single examiner. Working distance was set at 25 mm and the field was 0.2x0.5mm.

The patient was seated at the instrument with the chin on the chin rest and the forehead against the forehead band.By moving the joystick the proper focus is obtained.When the endothelium is in proper focus the instrument automatically took a picture of the endothelium.

The above diagram is a picture of the endothelium the specular microscope took.The examiner then selected a field in the endothelium that was zoomed or magnified.In the zoomed in field the examiner was required to select a minimum of 20 individual cells that were adjacent to each other and that had well defined cell borders.The selections were done by clicking on the individual cells.

Once this was done the specular microscope then computed the corneal thickness, maximum cell area,minimum cell area and average cell area and endothelial cell density.Standard deviation and coefficient of variation were also noted.





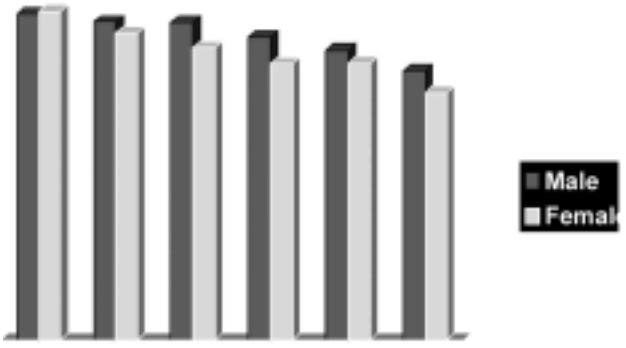
Data analysis

Average cell area and cell density for both eyes were calculated for each decade and the results were compared.In each decade the average cell area and cell density were calculated separately for male and the female sexes and results plotted in line diagram and bar diagram.Results were then statistically analysed.

Cell Density variation with Age

Age	20-Oct yrs	20-30 yrs	30-40 yrs	40-50 yrs	50-60 yrs	60-70 Yrs
Male	3280	3196	3192	3043	2909	2705
Female	3303	3084	2938	2778	2782	2488

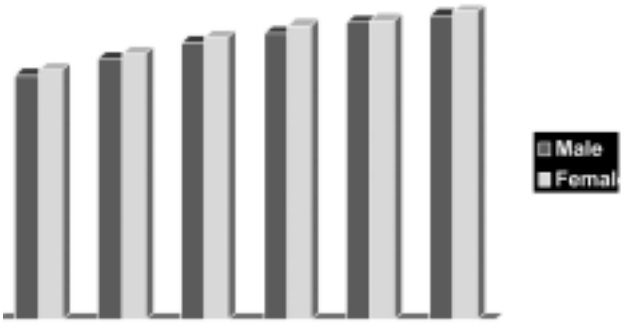
Bar Diagram to show cell density changes with age



Average cell area changes with age

Age	20-Oct yrs	20-30 yrs	30-40 yrs	40-50 yrs	50-60 yrs	60-70 yrs
Male	323.9	345.71	366.4	379.35	394.23	403.03
Female	331.06	352.92	374.7	389.79	396	408.74

Bar Diagram to show cell area changes with age



Results

Statistical analysis showed that there was a decrease in cell density with advancing age and this decline was found to be statistically significant.The average decline in cell density with advancing age was computed to be 0.29%. on an average per year.Though the cell density of females was slightly decreased as compared to males this difference was not statistically significant.

Average cell area also showed a corresponding increase in cell area with advancing age.

No patient in our study group showed polymegathism or any other endothelial cell damage.

Conclusion

We concluded that in a normal population with advancing age there was a decline in the cell density with advancing age ,this decline was found to be on an average 0.29% per year.Similar studies were conducted by Laule and Cable and by Faragher.Faragher et al found the decline in cell density with advancing age to be 0.6%.

We also concluded that there was a corresponding increase in average cell area with advancing age.

Discussion

Specular microscopy enables a direct view of endothelial cells.The instrument was described by Maurice in 1968,modified for clinical application by Laing, Sandstrom, Bourne and Kaufman. Other tests of endothelial function include measuring the corneal thickness by pachymetry.

Corneal endothelium is a monolayer of flattened hexagonal cells, whose lateral borders have extensive interdigitations and junctional complexes.The endothelium is a metabolically very active structure with a large number of tubular mitochondria.The functional importance of the endothelium is that it helps to keep the corneal stroma in a dehydrated state through its physical barrier effect and by action of metabolically active pump.The physical barrier comprises of the junctional complexes .The pump consists of a Na-K ATPase enzyme that catalyses the movement of ions along an osmotic gradient from the stroma to aqueous.Disruption of the barrier and or the pump function of the endothelium results in hydration of cornea and corneal edema.

The corneal endothelial cell density is highest at birth as high as 7500 cells/mm^2. At 5years it ranges from 3000 to 4000 cells/mm^2. Decrease in cell density of 13% between ages of 5 and 7 years and 12% between 7 and 10 years of age have been recorded. The rate of loss slows into the mid twenties and even lower rates of approx 0.52% loss per year continue into older age. In childhood normal endothelial cells are of uniform size and hexagonal shape. As cell density decreases with age individual cells enlarge and lose their hexagonal shape. Polymegathism, irregularity in the normal regular mosaic pattern may be quantified as a coefficient of variation in cell size. Variation in cell size remains most useful as a tool for assessing endothelial damage when populations subjected to different surgical techniques or variables are compared.

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Comparative Results of Various Treatment Modalities for Recalcitrant Diabetic Macular Oedema

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

Chakrabarti Eye Care Centre, Kochulloor, Trivandrum, E-mail: tvm_meenarup@sancharnet.in

Recalcitrant macular oedema is one of the most important causes for significant visual loss in diabetic patients. In these patients the diabetic status is complicated by the presence of other systemic conditions such as hypertension, hyper lipidemias, and nephropathy. Eyes with gross retinal thickening and presence of plaques of hard exudates will respond poorly to laser photocoagulation (Fig :1). Presence of a thickened taut posterior hyaloid membrane exerting traction on the macula resulting in shallow macular detachment could be another cause for recalcitrant macular oedema in a diabetic. Treatment modalities include laser photocoagulation , Intravitreal steroid injection,parsplana vitrectomy or a combination of these.

This prospective randomized study was conducted over a period of 36 months to compare the efficacy of various treatment modalities for recalcitrant diabetic macular oedema.

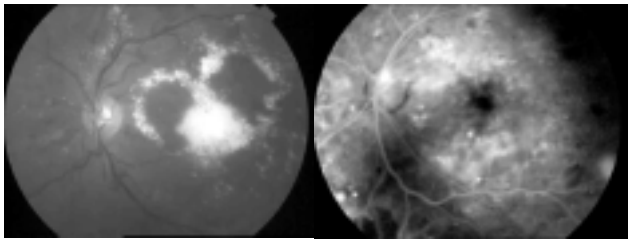


Fig. 1. Fundus photograph and fluorescein angiogram of a chronic diabetic with associated hypertension, hyperlipidemia, and chronic renal failure.

Materials & Methods

A prospective randomized study was carried out on 80 eyes of 40 patients with chronic diabetes and recalcitrant DME both clinically and by angiography to compare the efficacy of the various available treatment modalities.

Inclusion criteria were 1) Presence of clear media 2) Absence of significant lens opacity 3) post laser status– with no history of laser within the preceeding 6 months 4) Stable renal parameters 5) Stable lipid profile 6) Absence of macular ischaemia on FFA. Patients with poor diabetic control, nephropathy, significant cataracts and dyslipidemia were excluded from the study. Table 1 gives the demographic profile of our patients.

Table 1. Demography of Group I & II

• Duration of Diabetes	> 10 years (Type II) / Good Control
• Prior Laser Rx	Minimum 2 sittings
• Last Laser Rx	6 months prior to study
• Visual Acuity	6/24 to 5/60
• M:F ratio	2:1
• Renal Parameters	Stable
• Dyslipidemia	S. Cholesterol ≤ 200 mg %

One eye of each patients enrolled in the study was randomized to undergo either IVT injection of TA(Triamcinolone Acetonide) or TA assisted pars plana vitrectomy with ERM (Epiretinal Membrane) peel and

ILM (Internal limiting Membrane) peeling. The 1st group received focal laser photo coagulation 2 weeks after the injection while the PPV-TA (+) Group received laser to identifiable microaneurysms within circinate rings intra operatively. The other eye of all 40 patients were subjected to standard laser photocoagulation.

Procedure – (I) For IVT injection of Triamcinolone Acetonide.

Intravitreal injection of Triamcinolone Acetonide was performed under topical anesthesia, under sterile aseptic precautions in the operation theatre. 4 mg/ 0.1ml of preservative free Triamcinolone Acetonide (Aurocort; Aurolab, Madurai) was injected into the mid vitreous cavity using 30 g needle slowly and steadily. The needle was withdrawn carefully and indirect ophthalmoscopy performed to assess perfusion of Optic Nerve Head. The patients were specifically informed about visual blurring and presence of floaters after the injection. The risk of developing Endophthalmitis and Retinal Detachment was also explained. Post injection topical antibiotic treatment was given for 1 week and IOP monitored weekly of 8 weeks

II) For the **PPV.TA (+)** group:- Preservative free TA (Aurocort 1ml of 4mg /0.1ml) was injected into the vitreous cavity during pars plana vitrectomy as an aid to visualize the vitreous and posterior hyaloid. The posterior hyaloid was well visualized as a thin white surface because of the contrast between the waving posterior vitreous cortex covered and stained by entrapped TA particles and the fluid filled posteior hyaloid space. Post operative IOP monitoring was performed weekly for 8 weeks . (Fig 2 a & b)

The two groups received the standard care for the fellow eye. The follow up protocol is detailed in TABLE : 2.

Table 2. Follow up Protocol

	CRITERIA	DURATION
1.	Duration of F/U	36 Months
2.	Frequency	Weekly x 8 weeks Monthly x 3 months 3 Monthly remaining period
3.	Visual Acuity, IOP (Applanation)	Every Visit
4.	FFA	Monthly for 3 months 6 Monthly for 1 year
5.	OCT	Monthly (10%)

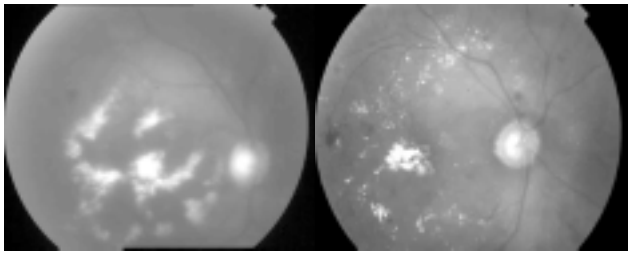


Fig. 2. a&b -(a) Fundus picture of a chronic diabetic with recalcitrant DME . (b)Fundus picture of same eye one year after TA assisted vitrectomy showing good resolution of DME.

Results

40 eyes of forty patients were enrolled in the study. The study eyes were divided into 2 groups

Groups I: IVT TA Injection group : 20 eyes

Group II: PPV TA (+) 20 eyes

The data that was analyzed and compared included improvement in vision, resolution of macular oedema, rate of recurrence of macular oedema and development of complications

During follow up we looked for resolution of macular oedema, improvement in vision,post treatment IOP spike,development of cataract, and recurrence of macular oedema on long term folowup. In 50% of patients who under went TA assisted PPV there wasimprovement in the vision Resolution of CME was noticed in the majority of vitrectomised eyes.. Long term follow up for 3 years (36 months) showed that recurrence of DME occurred in only one eye (5%). 84.6% patient who underwent TA assisted vitrectomy had good resolution of CSME. In patients with hard exudative plaques under the macula, although there was no significant visual improvement, with in 4 weeks of the surgery the hard exudates fragmented into smaller particles and slow resorbtion was noticed during follow up.

TA injection into the vitreous cavity has been well known to be associated with increase in intraocuclar pressure. Patients who underwent TA assisted vitrectomy in our series did not have significant IOP rise. Post op IOP> 21mm even with standard glaucoma medications was seen in only 1 patient (5%) compared to the 19.5% raised IOP in eyes which underwent IVT. This could be because most of the TA is removed from the vitreous cavity at the end of surgery. The small

amounts of TA (few granules of TA were seen in the lower half of the fundus but disappeared with in the third post operative day), that is left behind may not be sufficient enough to cause an IOP rise but may be sufficient to reduce post operative inflammation.

In comparison patients in the IVT group experienced less dramatic visual improvement (40% Vs 50%) and less resolution of DME (40% Vs 84.6%). More number of patients experienced IOP rise post operatively (19.5% Vs 5%) pointing towards need for judicious use of IVT Triamcinolone. In all these patients IOP was controlled medically and none required trabeculectomy. Progression of cataract was less (30% Vs 54.8%) in this group. There were no cases of reinjection and additional laser therapy.

Follow up of the standard care group showed that significant improvement in vision as well as resolution of CSME could be achieved in only 30% of patients. 70% of the patients had recurrence (56 eyes) of CSME which necessitated additional laser. These results clearly indicate that IVT or PPV TA (+) were better treatment options than standard of care in diabetic patients with recalcitrant diabetic macular edema with better resolution achieved with vitrectomy. Table 3 gives the comparative data in our series.

Table 3. IVT Vs PPV TA (+) Vs Standard Care

Criteria	IVT	PPV TA(+)	Standard Care
1. Improvement in vision	40%	50%	30%
2. Resolution of CSME	40%	84.6%	60%
3. IOP	19%	5%	Nil
4. Cataract Progression	30%	54.8%	10%
5. Endophthalmitis	Nil	Nil	Nil
6. RD	Nil	5%	Nil
7. Recurrence of DME	40%	5%	70%

Conclusion

The result of this study shows that TA assisted PPV with peeling of epiretinal membrance when present and denuding the retinal surface of ILM, results in good resolution of recalcitrant DME and significant visual improvement. Intra vitreal injection of commercially available, preservative free TA is non toxic to intraocular tissues, enables surgeons to perform a complete and fast removal of posteior hyaloid, vitreous base gel and ERM thus becoming an additional tool in the management of recalcitrant DME. Postoperative IOP spike was not significant in our series probably because most of TA is removed from the vitreous cavity and the end of surgery. The small amount of TA that is left behind in the vitreous cavity may not be enough to cause an IOP increase but may be sufficient to reduce the inflammatory component of DME

Intravitreal steroid injection or implants act by decreasing the growth factors, by stabilizing endothelial tight junctions, and by decreasing the permeability to water and solutes. Clearance of intravitreally injected TA in normal phakic, nonvitrectomized eyes is 41 days. IVT of Triamcinolone Acetonide is associated with certain severe complications like (1) Endophthalmitis: 1.4% per eye per injection (2) Glaucoma 25.30% (3) Development of Cataract (54.8%) (4) Uveitis / Iritis (5) Retinal detachment (6) Intra Ocular Haemorrhage (7) Hypotony. Results of various studies have shown that Intravitreal injection of Triamcinolone Acetonide gives a mean improvement in visual acuity of 2.4 lines. Follow up showed that this effect of reduction in macular oedema is transient and at 6 months post injection the visual acuity improvement was reduced to 1.3 lines and the reduction of central macular thickening was only 38%. These result clearly show the transient nature of the beneficial effect.

The role of pars plana vitrectomy in reducing recalcitrant diabetic macular oedema and improving the vision has been studied extensively since 1990. Lewis at al demonstrated the beneficial effect of Pars plana vitrectomy in eyes with chronic diffuse diabetic macular oedema associated with thick taut glistening posterior hyaloid. FFA performed in these patients showed good capillary perfusion at macular associated with diffuse late retinal leakage explained by pooling of dye in the underlying serous macular detachment. Beneficial effects of vitrectomy in these cases could be due to the macular flattening achieved by relieving the vitreo macular traction. Even when definite demonstrable vitreomacular traction is not present the beneficial effect in causing resolution of diabetic macular oedema could be due to the removal of the inflammatory sump in the vitreous. Vitrectomy may be combined with removal of epiretinal membrane, peeling

of ILM, removal of sub retinal exudates or various combinations of the above. The concept of Deroofing the inner retinal surface by ILM peeling, was put forward by Gandorfer et al Deroofing of the inner retinal by ILM peeling decompresses what may be regarded as a compression syndrome abnormality. It also promotes migration of cells, and egress of oedema fluid. Reduction in retinal thickness and increased oxygenation could theoretically improve visual acuity.

Triamcinolone assisted vitrectomy for a variety of conditions including recalcitrant DME has been described. From the results of these studies it is possible to infer that intra vitreal injection of commercially available TA is not toxic to intra ocular tissues, enables surgeons to perform a complete and fast removal of posterior hyaloid, vitreous base gel, ERM & ILM thus becoming an additional useful tool in surgical treatment. The IOP rise postoperatively is not significant as only minimal amounts of Triamcinolone is left behind in the vitreous cavity vitreous cavity at the end of surgery. The small amount of TA that is left behind may not be enough to cause an IOP increase but may be sufficient to reduce post operative inflammation.

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ORIGINAL ARTICLE

Outcome of vigilant management of early Steven Johnsons Syndrome

Dr. Meenakshi Dhar, Dr. Sujithra H, Dr. Anuradha Rao, Dr. Lilan Bhat

Address for Correspondence: Dr. Meenakshi Dhar, Consultant, Professor and Head, Ophthalmology Dept., Amrita Institute of Medical Sciences, Amrita Lane, Elamakkara, Cochin, Kerala. 682013

Abstract

Steven Johnsons Syndrome [SJS] is an important cause of ocular morbidity. We present a series of 14 SJS patients who presented to our institution in the acute phase. All patients with the diagnosis of SJS over last 2 years were included. Possible etiologic factors, various ocular manifestations in acute phase is discussed. Early aggressive management included separating the lids frequently, cleaning the discharge, glass rod sweeping along the fornices and voluntarily moving the eyes. This prevented formation of symblepharon, ankyloblepharon and other sequlae. All patients recovered without any sight threatening sequlae. Mild sequlae included - one patient had ankyloblepharon and punctual stenosis, another had inferior corneal opacity, mild dry eye was seen in 11 cases. This study lays emphasis on the importance of early aggressive management in preventing and reducing the severity of sight threatening sequlae of SJS.

Steven – Johnsons Syndrome [SJS] is a symptom complex characterized by symmetrically distributed erythematous bullous lesions of the skin and mucous membranes. It is accompanied by severe constitutional symptoms and also called erythema multiforme major. It predominantly involves the oral mucosa and conjunctiva [ocular involvement 69[1]- 91% in adults]. It is essentially drug induced[1]. Anticonvulsants, antibacterial, some non-steroidal anti-inflammatory drugs have been identified as probable causative factors. Other factors like viral infection, mycoplasma have been suggested to precipitate SJS.

Ocular manifestation can be classified as *mild* with lid oedema, conjunctivitis, chemosis; *moderate* with conjunctival membranes, corneal epithelial loss and corneal ulceration; and *severe* with cicatricial changes and perforation. Prompt diagnosis, identification and early withdrawal of all suspect drugs are the most important.[2] . The management of the patients must be undertaken in the specialized intensive care units. Ocular sequlae can be minimized by prompt, early and regular ophthalmic care.

We present a series of 14 patients [Aged 10-58 years with male: female ratio of 5:9] who presented to our institution in the last 2 years. All presented in the acute phase of SJS within 1 week of onset of symptoms. Eleven of these patients had ocular involvement. 3 of the patients first consulted an ophthalmologist, with only ocular complaints. All patients had a history of drug intake. Use of any systemic drug within 2-3 weeks of onset of prodromal symptoms was considered as possible etiological factor [Table-I]. In our series anticonvulsants were the most common etiologic factor especially - Carbamazepine [n=5] and Phenytoin [n=2].

Ocular involvement was mild to moderate in 11 cases [Table II], and 3 had no ocular involvement. Lid complications were seen in 5 patients, lid edema with blisters [n=1], madrosis [n=1], meibomitis and blepharitiis [n=5] with thick discharge on lid margin. None of the patients had dystrichiasis, trichiasis, entropion or ectropion. Conjunctival involvement was in the form of congestion, membrane formation [n=3] and symblepharon [n=5]. Only one patient had corneal epithelial defect.

Table 1. History of drug intake

	Name of Medicine	No of patients
1.	Anticonvulsant	7
	Carbamazepine	5
	Phenytoin	2
2.	Antibiotic	5
	Amoxycilline	2
	Augmentin + Ofloxacin	1
	Azithromycin + Paracetamol + Homeopathy medicine	1
	Gatifloxacin	1
3.	Non-steroidal anti-inflammatory	
	Nimesulide + Ibuprofen	1
4.	Ayurvedic medicine	1

Table 2. Ocular complications in Steven-Johnsons Syndrome (n=14)

Ocular structure	Clinical findings	No of Patients
Lids (n-5)	Oedema/ thickening / Bullous lesion	2
	Meibomitis / Blepharitis	1
	Thick discharge	5
	Trichiasis / Dystrichiasis	-
	Entropion / Ectropion	-
	Madarosis	1
Conjunctiva (n-6)	Congestion	7
	Dry eye	11
	Symblepharon	1
	Membrane in the fornix	4
Cornea (n-2)	SPK	1
	Epithelial defect	1
	Scarring	-
	Vascularisation	-
	Keratinisation	-
	Thinning	-
No Ocular involvement		3

Treatment was started soon after diagnosis. Suspected drug causing SJS was withdrawn. Ocular management included frequent topical lubricants (Carboxymethyl cellulose with biodegradable preservative) in all patients. A topical antibiotic [usually tobramycin], was added where we suspected secondary infection, although the culture was negative. Flourometholone was added for those with excessive ocular surface inflammation. Membranolysis with glass rod passing was done 2-3 times daily for those who had symblepharon or pseudomembrane formation. The ordinary thermometer serves well as the blunt glass rod. All patients were encouraged to move the eyes voluntarily and to separate the lids frequently in order to prevent symblepharon formation. Good ocular hygiene was ensured. Bandage contact lens was used in the patient with corneal epithelial defect. All patients were put in isolation wards to prevent the chance of secondary infection and those requiring greater supportive measures were put in intensive care units. All were put on systemic steroids; fluid and electrolytes were maintained. Ophthalmic followup was done once or twice daily for each patient till they were discharged. All patients were explained about the chronicity, severity and sequelae of the disease, possibility of dry eye and need of regular and long term ophthalmic follow up. There was no mortality and all were discharged within 2 weeks to 2months. All patients were followed at 1 and 3 months after recovery of the acute phase. 11 patients recovered without any ocular sequelae. One patient had punctual stenosis, and minimal ankyloblepharon [Fig. 1]. One patient had inferior corneal opacity, which was not visually significant. Superficial punctate keratitis was seen in one patient at one month followup. All patients were advised to use lubricants for a long period.

Discussion

SJS is an important cause of ocular morbidity. This analysis aimed to study the presenting features, possible etiologic factors and the outcome of intensive ocular management in reducing the sequelae. Acute phase includes lid oedema, blepharitis, conjunctivitis, pseudomembrane or membranous conjunctivitis. Later complications include from lid scarring – entropion, ectropion, trichiasis, lagophthalmos, conjunctival scarring – symblepharon or ankyloblepharon.[3]. Tear film deficiency leads to conjunctival and corneal xerosis. Late

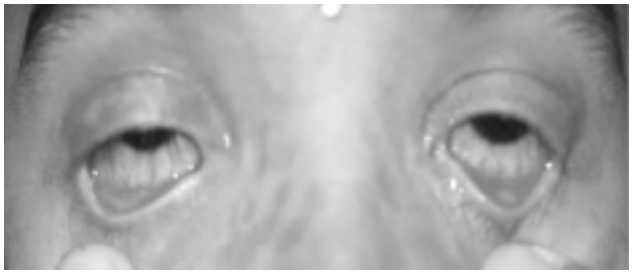


Fig. 1. Child with ankyloblepharon, dry eye and conjunctival congestion

phase corneal complications develop due to corneal exposure leading to recurrent epithelial defect, corneal neovascularisation, conjunctivalisation of cornea, and corneal opacity. Ocular cicatricial pemphigoid has been reported 31 years after SJS in five patients.[4]

Our analysis reconfirms that the most sight threatening sequelae can be prevented by aggressive medical management in the acute phase. Only one patient had a small corneal opacity and 5 patients had mild ocular surface disease. Our 14- year old patient had the most severe ocular and systemic involvement. SJS in children is less common with ocular involvement in 39% cases in a 10-year series. [5]

Corneal transplantation has poor visual prognosis with ocular surface disorders. It is also difficult to correct the structural abnormalities of lid & conjunctiva in the late phase as the tissues are fibrotic and friable. Most patients might require extensive surgical procedures like, amniotic membrane transplantation, skin graft etc. Osteoodontokeratoprosthesis[6] is a recent innovative method to restore the vision of patients with endstage severe ocular surface disorder using autologous canine tooth and buccal mucosa as the artificial cornea or keratoprosthesis. Role of systemic corticosteroids in modulating ocular manifestations is not estabilished.[1]

In contrast to many reports where Sulphonamides was the most common etiology, our patients had history of intake of anticonvulsants and other antibiotics. Carbamezepine was found to be the commonest cause of SJS in another report as well from Kerala .[7] There is an immunologic susceptibility to the development of SJS. It has been reported that patients with HLA-Bw44 are more susceptible to SJS [8]. Genetic factors are suspected, the suspected drug should not be used in the blood relatives of the patient.

At this time the best result come from early diagnosis, immediate discontinuation of any suspected drug, supportive therapy often in intensive care unit and paying close attention to ocular complications. Daily examination by an ophthalmologist is a must to prevent sight threatening sequlae.

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Prostaglandins in Glaucoma

Dr Meena Nair
Consultant, Chaithanya Eye Hospital, Kesavadasapuram, Trivandrum 695 004 Phone: 0471-2447183

Prostaglandins comprise the latest class of drugs added to the list of glaucoma medications. They are ubiquitous local hormones that produce ocular inflammation and hypertension in high levels but in smaller amounts reduce 10P[1-4]. 'Ambache' [5-6] made the first observation of prostaglandin effects in the eye by isolating a substance which was a mixture of PGE_2 and $F_2\alpha$ [7-9] in iris extracts that could produce miosis in cats. Four agents are included in the category of prostaglandins also called hypotensive lipids. These are

- Latanoprost (approved by USFDA in 1996)
- Travoprost (approved in 2001)
- Unoprostone (approved in 2000)
- Bimatoprost (approved in 2001)

Pharmacology

Prostaglandins are eicosanoids which are metabolic products of arachidonic acid, a 20C structure[10] (refer Fig 3).

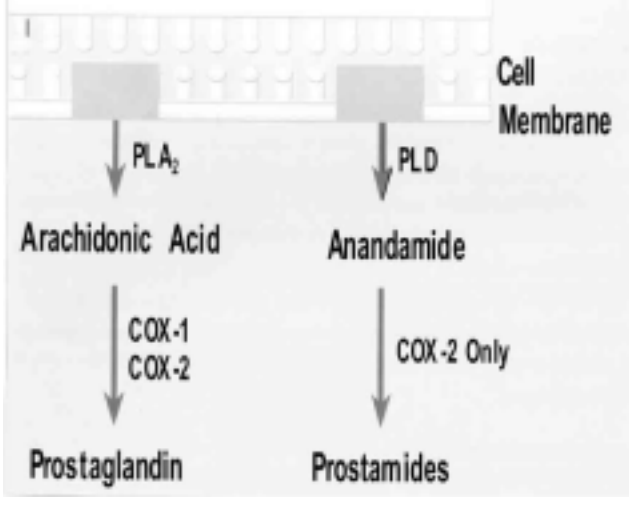


Fig 1

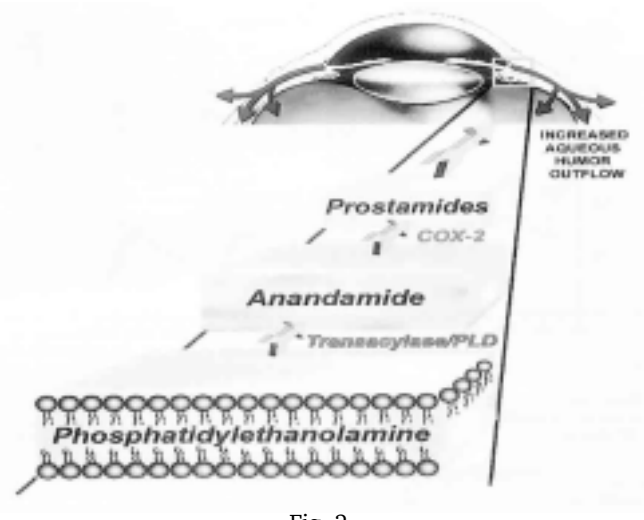


Fig. 2.

For the prostaglandins, the last letter refers to specific chemical modifications of the ring structure. The subscripted number refers to the number of double bonds in the molecule. The Greek letter subscript refers to the orientation of the hydroxyl group in relation to the ring structure e.g. PGF_2.

Prostaglandins are fatty acids which structurally carry a negatively charged – COOH group while prostamides are fatty acid amides that do not carry the negative charge. Both classes are derived from membrane lipids and are formed by different biosynthetic pathways (Fig 1).

Prostanoid Receptors

Endogenous Prostaglandins have 4 subtypes of receptors [11] and prostaglandins have affinity for more than one receptor.

4 subtypes

- EP receptor → PGD_2
- FP receptor → PGE_2

- ❖ IP receptor → $PGF_2\alpha$
- ❖ TP receptor → PGI_2/TxA_2

PGE_2 and $F_2\alpha$ binding sites are seen in the ciliary muscle and iris sphincter muscle. FP and EP_2 receptors are seen in human trabecular meshwork, EP_4,IP and TP receptors to a lesser amount in the trabecular meshwork and EP_1, EP_2 and FP receptors are seen in human scleral fibroblasts.

Structure

Chemical modifications made to the lipid molecule -

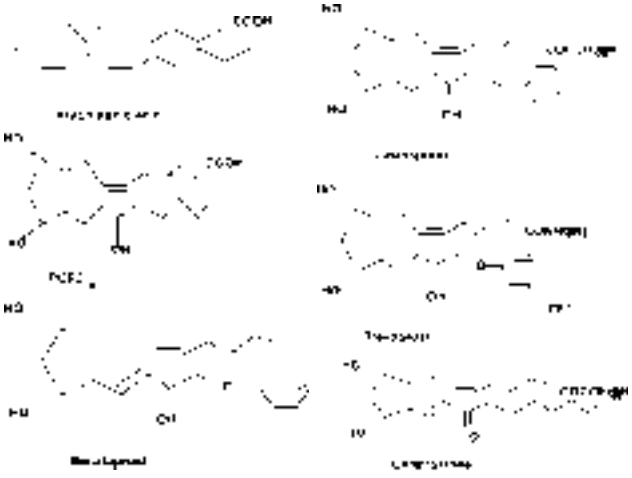


Fig. 3.

1. To improve the molecule solubility, C_1 carboxyl group modified with -
   - an ethyl amide - Bimatoprost
   - an isopropyl ester - Latanoprost, Travoprost, Unoprostone
2. Phenyl ring addition to the omega chain improves FP receptor selectivity and I0P reduction (i.e Latanoprost,Travoprost and Bimatoprost)
3. Saturation of $C_{13\text{-}14}$ doublebond on omega chain reduces hyperemia

Mechanism of Action

Prostaglandins have an unusual effect on aqueous humor dynamics – it increases the non-conventional outflow through ciliary body face and iris root to suprachoroidal space.

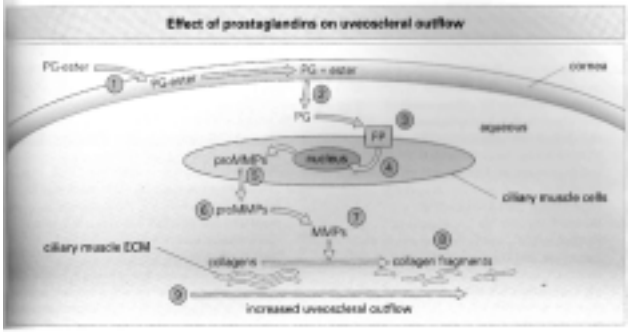


Fig. 4.

Topical PG ester is absorbed into cornea (ref Fig:4)

1. It is converted to free PG. Free PG passes into aqueous
2. It is carried into ciliary muscle and binds to the FP receptor on the ciliary muscle cell surface
3. Binding initiates a single cascade inducing transcription of MMP genes
4. Gene products are translated to proMMP's
5. Pro MMP's are secreted into the extracellular space around ciliary muscle fibers
6. Proteolytic truncation induces activation of the MMP's
7. This initiates collagen degradation in ECM
8. Decreases hydraulic resistance and facilitates uveoscleral outflow

To summarize, possible mechanisms by which prostaglandins improve uveoscleral outflow are by a reduction of ECM collagen I, III, IV, V, VI laminins, fibronectins in the interstitial spaces of the ciliary muscle and induction of ciliary muscle to produce MMP 1,2,3 and 9.

Storage

- ❖ Unopened bottles of Latanoprost should be refrigerated.Once opened they can be stored at room temperature (25°c or 77°F) for six weeks. Other Prostaglandin analogues can be stored at room temperature (15°c to 25°C/59°F to 77°F for Bimatoprost, 2°c to 25°C/36°F to 77°F for Travoprost and Unoprostone).

Drug efficacy and interactions (Table 1)

Latanoprost (0.005%) applied once daily produces a mean IOP reduction of 27% compared to 20% with

Table 1

S. No	Topical Prostaglandin	Preparation	Mechanism of Action	Dose Schedule	Duration of Action	Salient Features	Side Effects
1	LATANOPROST ($PGF_{2\alpha}$ analogue)	0.005% (50 µg/ml) 1 drop contains 1.5 µg the drug	Increases uveoscleral outflow	OD	24-40 hrs	❖ Prodrug ❖ Peak concn. In aqueous 2 hrs after administration ❖ Liver metabolism ❖ Urine elimination	**Proved side effects** ❖Iris color darkening (11-23 %) ❖Eyelash changes ❖Mildconjunctival hyperemia (5-15%) **Unproven casual relationship** ❖Cystoid macular oedema ❖Iritis ❖Herpes simplex keratitis **Minimal systemic side effects** ❖No effect on resting HR,BP, urine or blood parameters ❖Relatively safe in asthma and coronary artery disease ❖Headache, joint pain, muscle pain, upper respiratory tract syndrome
2	BIMATOPROST (a synthetic structural analogue of $PGF_{2\alpha}$)	0.03% (0.3 mg per ml)	❖ 50% increase in uveoscleral outflow ❖ 35% increase in trabecular outflow ❖ minimal FP receptor agonist activity ❖ alternate signal pathway based on intact molecule responsible for its effects	OD	24-40 hrs	❖ not a PRODRUG so less corneal hydrolysis ❖ scleral and corneal drug penetration ❖ blood concn peak in 10 mts, 10P decreases in 4 hrs and max effect in 8-12 hrs ❖ Renal elimination	❖ Conjunctival hyperemia (15%-45%) [13] ❖ Increased pigmentation and growth of eyelashes ❖ Increased iris pigmentation and periorbital tissue pigmentation (only 1.5%/year) ❖Others – ocular pruritus, burning, foreign body sensation, SPK's, blurring vision, lid retraction ❖ Systemic effects – very less, rarely raised LFT ❖ Cautious use in : aphakes, pseudo phakes with PC rent, uveitis, macular edema ❖ Not studied in CCF, Heart block, respiratory failure
3	TRAVOPROST ($PGF_{2\alpha}$ analogue)	0.004%	❖ Preferential affinity for FP receptor	OD	24-40 hrs	❖ PRODRUG produces a lower mean 10P in black patient compared to latanoprost	❖Conjunctival hyperemia (35-50%) ❖ Similar to latanoprost
4	ISOPROPYL unoprostone (a docosanoid)	0.15%	❖ Increased uveoscleral outflow ❖ Poor binding with FP receptor	Twice daily	12 hrs	❖ A 22 carbon structure ❖ Prodrug	❖ Hyperemia conjunctiva (10-25%) ❖ Mild ocular surface irritation ❖ Increased iris pigmentation reports ❖ Others similar to latanoprost

Table 1: Comparison of prostaglandin analogues

	Latono prost OD	Bimato prost OD	Travo prost OD	Unopro stone OD
Mean % 10P	27%	33%	28%	15%
Hyperemia %	5-15%	15-45%	35% -50%	10-25%
Iris pigmentation (6 months)	6.7%	1.2%	3.1%	<1%
Iris pigmentation (12 months)	16.1%	1.5%		

Timolol 0.5% twice daily[14] and 21% with Brimonidine Tartarate 0.2% twice daily[15]. Latanoprost when combined with Timolol twice daily produces an additional IOP reduction of 13% - 37%[16-18].When oral Acetazolamide 250mg twice daily or Dipivefrine 0.1% is combined with Latanoprost an additional 15% IOP reduction[19]and 16-19%[20]IOP reduction has been observed respectively. In clinical trials it was apparent that Pilocarpine did not impair the IOP lowering effect of Latanoprost.

Bimatoprost (0.03%) applied once daily produces IOP reduction of 33-36%. It successfully lowers IOP in patients unresponsive to Latanoprost[21]. Mean IOP reductions were 8mm Hg (32.4%) and 5.5mm Hg (22.7%) with Bimatoprost 0.03% and Timolol 0.5% twice daily respectively[22] . Comparing Bimatoprost efficacy with Travoprost 0.004%, studies have shown 7.4 -8.8mm Hg (34-36%), 4.6 -7.2mm Hg (19-29%[23]), a three month prospective study by Parrish and colleagues [24] showed almost similar IOP lowering efficacy between Latanoprost, Bimatoprost and Travoprost but the Bimatoprost/Latanoprost study group compared the percentages of treated patients achieving IOP reduction after six months of treatment[25] which showed that significantly fewer patients receiving Latanoprost achieved a 15% or 20% decrease in IOP at each time point. On patients uncontrolled on topical Beta blockers alone, Bimatoprost lowered IOP more than combination with Dorzolamide[26]. Bimatoprost and the combination of Latanoprost plus Timolol were equally effective in lowering IOP in glaucomatous patients[27]. There is no additive role in adding other prostaglandin analogues to patients who are on Latanoprost or Bimatoprost.

Uses

- Prostaglandins are potent ocular hypotensives as first line therapy for ocular hypertension and POAG. In addition, Latanoprost has been found to be useful in Juvenile OAG, Primary Angle closure glaucoma following YAG PI if I0P elevation persists [28].Prostaglandins are however a relative contraindication in inflammatory glaucomas and Posner Schlossman syndrome due to their association with CME and uveitis. The role of prostaglandin analogues in glaucoma associated with penetrating keratoplasty is uncertain. In chemical burns, they have to be cautiously used.

Summary

Prostglandin analogues are highly efficient ocular hypotensives, well tolerated and systemically safe and have a promising future as first line drugs, adjunctive drugs and as substitutes in the medical management of glaucoma.

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Comprehensive strategy for management of posterior capsular rent (with or without vitreous disturbance) by the anterior segment surgeon

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

Chakrabarti Eye Care Centre, Kochulloor, Trivandrum 695 011, E-mail: tvm_meenarup@sancharnet.in

Introduction

The outcome of uncomplicated cataract surgery performed by phacoemulsification in the present scenario is excellent. Posterior capsular rent, reported to occur in 0.5 to 7.5% of cases is a significant potential intraoperative complication of phacoemulsification. An improperly managed posterior capsular rent, with or without vitreous disturbance can mar this excellent outcome. Vitreous loss appears to be the crucial factor determining eventual clinical outcome. When the posterior capsule is broken without accompanying vitreous disturbance there is an increased risk of CME, vitreous prolapse into the anterior chamber and pseudophakic retinal detachment. Once vitreous is lost, the postoperative course is complicated in 30% of patients with an increased incidence of hyphaema, retained cortex, corneal edema, blurred vision and long term retinal problems including chronic CME, macular holes and retinal detachment. However, today, the control rendered through closed chamber modern surgical techniques may allow for a final outcome that is not much different from an uncomplicated case.

While each patient with a ruptured posterior capsule is unique, several basic surgical principles apply universally. Every cataract surgeon must understand these fundamental principles of how to diagnose and manage this complication in order to avoid long-term sequelae.

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.

General Predisposing factors for the Torn Posterior Capsule

- Poor visibility secondary to physical problems: hand position, brow, fluid pooling, speculum, which does not provide adequate exposure.
- Poor visibility secondary to pathology; dense arcus, pterygium, band keratopathy, corneal scars. Long and short axial length
- Pseudoexfoliation
- Dense asteroid hyalosis
- Small pupil
- Previous Trauma
- Certain Types of Cataract

Inadequate Visibility

An important predisposing factor leading to posterior capsular rupture is the relationship of the surgeon's hand position to the patients' brow with ensuing visibility problems, irrigation fluid pooling, and torsion of the globe. If the brow is prominent (i.e., a deep set eye) a temporal approach will minimize the inferior torsion of the globe. If there is pooling of fluid, turning the head temporally will allow the fluid to drain. A specially designed speculum allowing easy access to the globe without getting in the way during phacoemulsification should be used. Poor microscope illumination or alignment is another cause of poor visualisation, which should be simple to remedy once recognised. Other factors, which impair visualisation, include dense arcus senilis, dense nasal or temporal pterygia, band keratopathy and old corneal scars. Faced with these challenges to visualisation, the surgeon should slow down, pay more attention to details and be more careful about focusing through the corneal problem to allow visualization of the anterior segment. Use of trypan blue dye to stain the anterior capsule helps to improve visualization in many of these cases.

Long and Short Eyes

In high myopia and eyes that have undergone previous vitrectomy, the anterior chamber is deeper with more trampolining of the posterior capsule, due to the thinner, more elastic tissues. Lowering the infusion bottle and machine vacuum and flow settings lessen this tendency. In contrast, in high hyperopia the anterior chamber is crowded, making the posterior capsule closer to the phaco needle and increasing the risk of posterior capsule rupture. In these situations, early use of pulsed phaco will assist in deepening the anterior chamber. Iris prolapse is more common as the infusion from the phacotip will flow behind the iris, forcing it out of the infusion. A slightly more anterior incision and careful attention to incision size are required in these eyes.

Pseudoexfoliation

This condition is known to be associated with both weak zonules and poor pupillary dilatation. These twin problems lead to an increased incidence of torn posterior capsules or dehiscence of the zonules.

Asteroid Hyalosis

Dense asteroid hyalosis may make it difficult to visualize the posterior capsule during phaco. The surgeon must be careful and stay away form the posterior capsule, thus avoiding inadvertent tears.

Miosis

Intraoperative miosis or preexisting small pupil is an important predisposing factor for rupture of the posterior capsule. Attempt should be made to dilate pupil maximally by pharmacologic means and failing which surgical method (s) should be employed.

Additional Factors

Additional risk factors for posterior capsular rent could include 1) Inexperienced surgeons; (2) demented, disoriented or anxious patients with subsequent inadvertent patient movement; (3) equipment malfunction; and (4) preexisting trauma with unseen capsular rupture or zonular damage.

Prevention

The incidence of posterior capsular rent decreases with the increasing experience of the surgeon. The most severe tears occur during attempted emulsification of the nucleus. An intact capsulorhexis can greatly reduce this complication. The use of low-vacuum, low-aspiration phacoemulsification will also reduce the incidence of this complication by minimizing surge. Low-power phacoemulsification also adds to the safety by reducing the chance of piercing through the nucleus and rupturing the posterior capsule. However, with the current new generation phaco machines available safe phaco can still be performed with high vacuum parameters. During phacoemulsification, a second instrument of an appropriate design may be placed behind the remaining nucleus to hold the posterior capsule back and physically prevent it from contacting the phaco needle. Recently a silicone I/A tip has become available which may provide superior capsular protection compared to traditional metallic tip designs.

When does the posterior capsule tear?

The highest incidence of posterior capsular tear during phacoemulsification occurs 1) toward the end of phacoemulsification when the last pieces of nucleus are to be emulsified, 2) during polishing of the posterior capsule, and 3) during I/A. The next highest incidence of capsular rent is during early to mid phaco when the phaco needle is inadvertently passed through the nucleus and tears the posterior capsule or capsular equator. The least common times to tear the capsule are during hydrodissection and IOL insertion.

Early recognition of zonular or posterior capsular rupture

If a posterior capsular tear is not recognized in time 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 will prevent expansion of the tear size.

Signs of early posterior capsular tear or zonular dehiscence 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.

Some of these signs are transient. However if the surgeon is alert an early diagnosis of posterior capsular rent may be suspected even though the rent as such is not immediately visualized due to the overlying nucleus. If posterior capsule or zonular rupture is suspected the surgeon must decide whether to continue with phacoemulsification or convert to a safer and standard non phaco technique. The management decision is based upon the amount of nucleus remaining, the density of the nucleus, other accompanying risk factors (e. g; small pupil, loose zonules, etc.) and the individual surgeon's level of confidence and experience.

Most tears in the posterior capsule are small when they first occur. The surgeon should try to keep them from enlarging or tearing anteriorly destroying the integrity of the anterior capsular rim.

Posterior capsule rupture - avoiding (or delaying) extension of the rent and vitreous loss

As soon as a problem is sensed one must exercise the discipline to stop working. This doesn't mean abrupt removal of the instruments from within the eye though there is a reflex to suddenly withdraw the phaco or I/A tip. Sudden unplugging of the incision will result in emptying and collapse of the anterior chamber. If a sufficient portion of the nucleus has been removed the posterior capsule bulges forwards, the capsular tear enlarges and the anterior hyaloid face ruptures which will allow vitreous to prolapse through the defect towards the wound. Instead, the anterior chamber should be filled with viscoelastic through the side port incision to block vitreous prolapse and stabilize any remaining lens material prior to removal of the phaco or I/A hand piece. The surgeon should stay in foot pedal position 1, and as the viscoelastic is injected he should change to foot position (0) and the handpiece can then be safely removed after the anterior chamber is filled. A low viscosity, less cohesive and highly dispersive viscoelastic helps to form a better plug in a capsular break and tamponade the anterior hyaloid face. Subsequently the pathology should be carefully assessed which will then determine the subsequent surgical strategy.

Posterior capsular defect and retained Nuclear material without vitreous prolapse

When confronted with this situation, whether to continue with phacoemulsification or convert to a safe non phaco technique (manual small incision cataract surgery or standard extra capsular cataract extraction) depends on the bulk of the residual nucleus material, the degree of nuclear sclerosis, the size of the rent and

the surgeon's experience. The immediate goal is to prevent vitreous prolapse into the anterior chamber and loosing the remaining nucleus into the vitreous. If the nucleus is soft, and particularly if only a small residual amount remains, continuing with phacoemulsification may be a reasonable option. The surgeon should use the second instrument to move the remaining nucleus away from the tear to complete the emulsification. The nucleus should not be rotated using the phaco tip. The procedure should be slowed down by reducing the aspiration flow rate, decreasing the vacuum (thereby reducing post occlusion surge) and by lowering the infusion bottle (to prevent increasing the pressure in the anterior segment and driving the nucleus back into the vitreous cavity). Short bursts of low energy ultrasound with low aspiration, effective vacuum, and reduced irrigation will decrease the risk of nuclear loss, chamber shallowing and vitreous prolapse. Another option developed by Mark Michelson is to introduce a trimmed Sheets glide between the nucleus and the capsular tear by enlarging the phaco incision by 0.5 mm. This maneuver will prevent small nuclear fragments from descending through the capsular defect. Once the nucleus has been emulsified, the phaco handpiece should be removed only after the anterior chamber has been stabilized by injecting viscoelastic through the side port.

Residual cortex and epinucleus removal can be safely accomplished without extending the rent by following several surgical principles. One method is to employ low flow, low vacuum, bimanual I/A through clear corneal incisions. The lack of incisional fluid leak will reduce fluctuation in chamber depth. The bimanual technique offers safer and better access to the subincisional area and allows the aspirating port to be positioned peripherally and aimed away from the rent or dehiscence. Lowering I/A flow and vacuum settings will reduce speed and post occlusion surge respectively. The cortex remote from the tear should be removed initially and should be stripped towards the rent because any force generated away from it will cause its extension. Heroic efforts to remove all cortex should be avoided since such attempts might extend the tear and further compromise the integrity of the capsular bag. An alternative method of cortical removal is manual aspiration using both a bent cannula and a J-shaped cannula under the protection of a viscoelastic material. This manual technique of "dry" aspiration of cortex is more time consuming but decreases the risk of extending the tear and vitreous loss.

Conversion To ECCE

If a posterior capsular rent is suspected or discovered during hydrodissection or early phacoemulsification and if there is a significant amount of residual nucleus, and particularly if it is brunescent, or if other surgical risk factors are present, it is advisable to convert to a large-incision ECCE. The first step would be to prevent the loss of the nuclear fragment(s) into the vitreous. The nucleus should be secured by injecting a dispersive viscoelastic underneath it. A hook, passed through a fresh paracentesis opposite the incision may be used to loosen and manipulate nuclear material into the anterior chamber. If a temporal clear corneal incision is used it may be sutured and abandoned. A fresh sclerocorneal tunnel incision may be constructed temporally or superiorly. If a sclero corneal incision had been utilized it can be extended after appropriate conjunctival peritomy and cautery. The size of this incision is dependent on the size of the residual nuclear fragment. Once an adequate wound has been created an irrigating vectis and / or secondary lens manipulator should be used to extrude the lens nucleus with the help of a generous amount of viscoelastic. While expelling the nucleus the vectis should apply pressure against the posterior lip of the wound, rather than lifting and dragging the nucleus against the cornea. Bimanual pressure counter pressure technique should never be employed. Once the lens nucleus has been removed, anterior segment surgery should proceed as per the guidelines suggested in the succeeding sections. The wound is sutured with interrupted or running 10-0 nylon suture.

Extra capsular Cataract Extraction Over the Sheets Glide

The nucleus is secured with dispersive viscoelastic, which will also create space between the nucleus and vitreous. A Sheets' glide is positioned under the nucleus. Sometimes a hook may have to be used to guide the advancing glide into proper position. Once in position

the nucleus is extruded over the glide. The glide acts like a pseudo-posterior capsule and prevents the nucleus form dropping into the vitreous. At the same time the glide acts as a relative barrier to prevent the forward displacement of the vitreous into the anterior chamber. When using the Sheets glide technique, several precautions should be taken. The viscoelastic should be injected under the superior pole of the residual nucleus to create a cleavage plane so the glide can be safely inserted under the nucleus. The glide should be inserted under the posterior surface of the nucleus gently so that the capsular bag is not damaged. Caution should be exercised to avoid pushing the glide too far to prevent further damage to the residual capsule or even the ciliary body.

Posterior capsular defect with residual nucleus and vitreous prolapse

The goal is to remove the remaining lens matter (nucleus, epinucleus and as much cortex as possible) without causing vitreoretinal traction. The strategies which help the surgeon to achieve the goal are 1) rescuing a partially descended nucleus, 2) an appropriate anterior vitrectomy technique and 3) removal of the residual lens matter.

Rescuing a Partially Descended Nucleus

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. 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). 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" 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.

Managing residual nucleus with vitreous loss

The rescued nucleus, the residual epinucleus and as much cortex as possible should be removed without causing vitreoretinal traction. If the nucleus has been levitated largely intact into the anterior chamber, converting to an ECCE (as described in the previous section) is called for. Smaller fragments may be removed by phacoemulsification. It is advisable to use Sheets glide as a vitreous barrier and a safety support. If there is not much of vitreous in the anterior chamber and if it can be accomplished safely, an attempt can be made to remove cortex and epinucleus by employing

some bimanual I/A or 'dry technique' prior to vitrectomy. This maneuver may decrease the chance of lens matter loss into the vitreous, as the supporting vitreous is surgically excised. However once vitreous gets ensnared in the phacotip or aspiration port, it must be suitably addressed (described in the subsequent section).

Phacoemulsification over the Sheets Glide

The rescued nuclear fragments may be safely emulsified in the presence of a Sheets glide. A Sheets glide (either preordered at 3 mm or cut to size)) is carefully inserted through the wound into the cleavage plane between the superior pole of the nucleus and the posterior capsule and advanced towards the opposite side. A second instrument may be used to guide the glide to the proper position under the nucleus. Occasionally the large bulk of the nucleus and adherent cortex may obscure the view of the glide as it is advanced under the nucleus. The Sheets glide may be inadvertently advanced through the posterior capsular rent into the vitreous thereby severely compromising the integrity of the capsular bag. Careful guidance of the glide beneath the nucleus with the assistance of adequate viscoelastic and a second instrument through the paracentesis should prevent this complication.

Once the glide is in proper place the phacotip can be reintroduced into the anterior chamber and the remaining nucleus emulsified over the glide. The glide acts as a barrier to discourage further advance of vitreous into the anterior chamber with subsequent aspiration by the phaco tip. The barrier function of the glide also prevents the nucleus or nucleus fragments from dropping into the vitreous cavity. Anterior vitrectomy should be performed as and when the vitreous presents into the anterior chamber.

After the nucleus is emulsified, I/A and vitrectomy can be performed over the glide. The glide is removed, final vitrectomy, if necessary, is completed and the IOL is implanted into the capsular bag or ciliary sulcus. Alternatively, after removing the glide, dispersive viscoelastic is injected into the residual capsular bag and the IOL is implanted at an appropriate location. I/A and vitrectomy can than be performed using the IOL as a barrier to further vitreous movement.

Vitrectomy for anterior segment surgeons

All surgeons perform cataract surgery employing methods to prevent vitreous loss from occurring. Nevertheless despite our vastly improved technical expertise, our sophisticated equipments and our heightened awareness that vitreous loss can strike at any moment, this complication continues to haunt us on a regular basis

Every anterior segment micro surgeon must have vitrectomy techniques and equipment at his or her fingertips and should be aware of

1. Vitrectomy Instrumentation
2. Infusion Options
3. Basic Principles and Technique of Anterior Vitrectomy.

Vitrectomy Instrumentation:

Microsurgical (20G) advanced vitrectomy cutter with high performance proportional linear suction control is a necessity for anterior segment surgery.

Using the phacoemulsifier to remove the vitreous is dangerous as the phaco probe liquefies hyaluronic acid alone but does not cut the collagen fibres. Similarly use of a large bore needle to aspirate the fluid vitreous pockets should not be done because it will aggravate vitreoretinal traction. The theoretical pockets of liquid vitreous are more difficult to locate than the fountains of youth.

Cellulose sponge vitrectomy developed by Kasner has been an obsolete and dangerous method for 2 decades in spite of the important role it played before the advent of the vitrectomy machines.

A cellulose sponge causes significant traction on the retina as the sponge is lifted to transect the adherent vitreous. Marked inflammation is the rule after sponge vitrectomy due to the mechanical damage to the iris caused by contact with the sponge as it swells and is lifted up. Retained cellulose material on the anterior vitreous cortex after sponge vitrectomy may add on to the inflammation in addition to that caused by iris trauma.

Settings for Anterior vitrectomy: 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 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 for vitrectomy 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)



Fig. 1,2,3. DISADVANTAGES OF USING COAXIAL INFUSION includes enlargement of posterior capsular rent, hydration of vitreous, and increased vitreous prolapse.

1) Bimanual Technique with Separate Infusion Line

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 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)

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. (Fig: It is advisable to begin the vitrectomy dry and then infuse BSS gently, if the chamber tends to collapse, thought the side port. More vitreous will prolapse if the pressure in the anterior chamber is low when the aspiration continues.

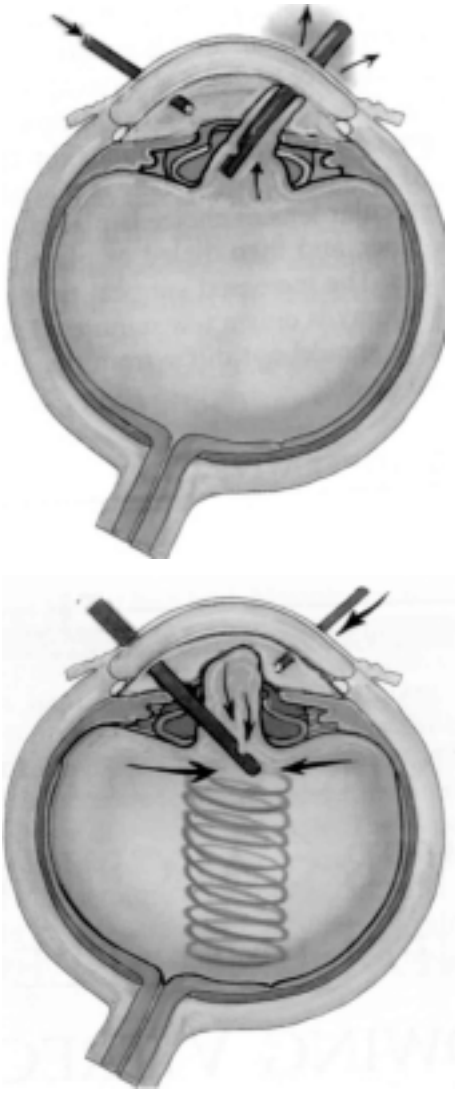


Fig 4 &5. TECHNIQUE OF PERFORMING ANTERIOR VITRECTOMY. Place the cutter through the PC Rent with the cutting port facing upwards. The strategy is to pull the vitreous from the anterior chamber down to the cutter.

The vitrectomy tip should not be placed through the primary phaco incision because the incision is the wrong size for it. 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 phaco tip 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 ealing 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 in to 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.

Intraocular Lens Placement

Prior to IOL implantation the exact anatomy of the tear should be determined and the capsulozonular anatomy should be clearly understood. To understand the integrity of the capsular support the iris is gently retracted under viscoelastic cover at multiple locations. This will provide guidelines as to the most desirable location and orientation of the posterior chamber IOL, its design and the optimal insertion technique.

When the posterior capsular rent is small with well-defined borders the tear can be converted into a posterior continuous curvilinear capsulorhexis. The tear is then less likely to extend if the bag is stretched during IOL implantation. It is important to remember that IOL implantation using a dialling technique may exert more force in the capsular bag than a superior haptic compression maneuver or a slowly unfolding IOL in the capsular bag.

If the tear is large with peripheral extensions and poorly defined borders posterior capsulorhexis is not possible and the IOL should be implanted in the ciliary sulcus after appropriate power adjustment from the capsular bag calculation. The IOL optic may be captured in the capsulorhexis opening.

Regardless of where the IOL is implanted, within the bag or into the ciliary sulcus it should be positioned 90^0 away from the axis of the tear. After the IOL is centred, its fixation and stability should be evaluated. If the IOL shows signs of poor fixation it can be repositioned from the capsular bag into the ciliary sulcus, sutured into the ciliary sulcus or exchanged for an anterior chamber IOL if there are no contraindications to it. It is recommended to suture fixate a PC IOL or not to implant a PC IOL if the surgeon is unsure of the anatomy of the posterior capsular rent rather than to rely on chance alone for proper fixation and enduring centration.

Once the IOL is well centred the pupil should be constricted by injecting acetylcholine into the anterior chamber since miosis will both retard late vitreous prolapse and make any residual vitreous easy to visualize. If the posterior capsule was torn and no vitrectomy was performed a prophylactic peripheral iridectomy should be considered.

At the conclusion of the procedure residual viscoelastic material can be removed manually or with an irrigation-aspiration tip. Anterior chamber should not be allowed to collapse to prevent further vitreous loss.

Conclusion

The incidence of posterior capsular rent can be significantly decreased by identifying the presence of predisposing factors and appropriate modification of the surgical plan. Early recognition and treatment of capsular tear and vitreous loss should help prevent serious complications and improve postoperative outcomes. The surgeon should have a proper game plan ready to face all kinds of posterior capsular rent scenarios.

Suggested Reading

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Innovations in Anterior Segment Imaging :

Slit lamp Photography made very simple

Author : **Dr. Rajiv Sukumaran** MS DO FRCS Ed
Co-author : **Dr. Jayasree Rajiv** MBBS DO

Priyanka Eye Hospital, Vavvakkavu P O, Kollam 690528
E mail: rajivsukumaran@gmail.com

Abstract

Digital photography is an expensive affair with sophisticated equipments for slitlamp and fundus photography. Anterior segment imaging using a WEB CAMERA attached to a slitlamp is described. A PC, a web camera, and a slit lamp are required for this purpose. Web cameras cost less than Rs 2000. If you already have a software for clinical data, these pictures can be added to the case sheet which will be of great help in documenting your findings and for Medico-legal purposes.

Material & Method

A web cam with a reasonably good resolution, a slit lamp and personal computer is all that is needed for this method.The details of models used for this purpose are

1. Intel PC Camera CS330 for Windows 98 (fig 1)
2. Intel Create and Share Software for CS330 for Windows 98
2. INAMI Slitlamp (or any other model)
4. A Standard PC with Windows 98

The Camera is attached to the Slit lamp as show in fig 2 and 3. No special attachment is needed. As we



Fig. 1. The web cam



Fig. 2. The technique of attaching the web cam to the slitlamp



Fig. 3. The way the web cam is attached



Fig. 4. When the Camera is not in use, it is swung away from the eyepiece of the slit lamp

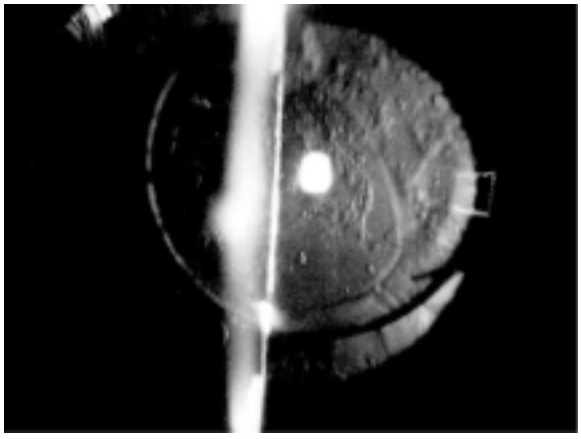


Fig. 5. Posterior Capsular Opacification

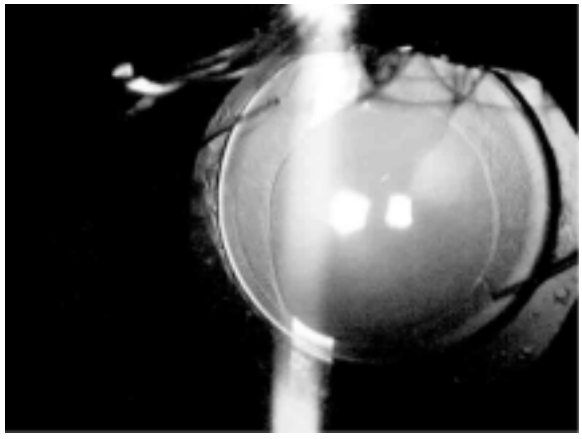


Fig. 6. Capsulorrhexis margin visualised clearly

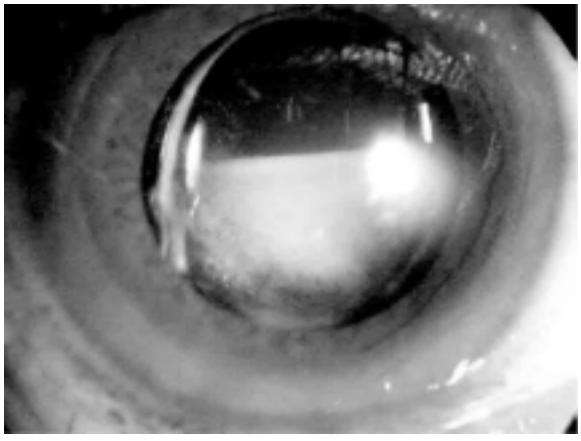


Fig. 7. Amyloid material in capsular bag



Fig. 8. Iris Pigment on lens

keep the eyes in front of one of the eye pieces in the slit lamp, the camera is placed in front of the other eyepiece. The camera sees what our eye sees and transfers the image to the computer.

When the camera is not being used, it can be swung away from the eyepiece (fig. 4).

Conclusion

This simple and cost effective technique is an innovative method of imaging the anterior segment of the eye and can be adopted easily by a practicing ophthalmologist.

Current practice patterns in the management of Diabetic Macular Edema

Dr. Gopal S Pillai, MD, DNB, FRCS

Chief of Vitreo Retinal Services, Amrita Institute of Medical Sciences, Amritha lane, Elamakkara, Cochin, Kerala 682 013

Diabetic retinopathy has been earmarked as one large pandemic which will have gross implications in India and all over the world by 2020. It has already grown into one of the biggest ocular problems in the developed nations. It is projected that India will overtake China as the single largest population of diabetics by 2025. We can rest assured that diabetic retinopathy will be on the rise in the years to come

Diabetic macular edema is the most common cause of diminution of vision among diabetics. In the Wisconsin epidemiologic study of diabetic retinopathy (WESDR), which is the largest epidemiologic study on diabetic retinopathy till date, it was documented that about 20 % of IDDM patients and 25 % of NIDDM patients on insulin will have macular edema after 10 years of diabetes.

In this short review we will go into the methods of diagnosis, evaluation and management of diabetic macular edema and chalk down the currently accepted practice patterns in the management of diabetic macular edema.

Macular Edema

1. Retinal thickening or hard exudates at or near the macula
2. Can be clinically significant or not
3. If clinically significant, has to be treated with photocoagulation
4. Can present with any grade of DR

Clinically Significant Macular Edema (CSME) (any one of the following):

1. Thickening of retina at or within 500 micrometer of the centre of the macula
2. Hard exudates at or within 500 micrometer of the centre of the macula if associated with retinal thickening of the adjacent retina
3. A zone or zones of retinal thickening of more than or equal to 1 disc area, any part of which is within 1 disc diameter of the centre of macula.

Evaluation of Diabetic Macular Edema

Direct ophthalmoscopy

Indirect Ophthalmoscopy

Hruby lens

Slit lamp aspheric indirect fundus biomicroscopy

Contact biomicroscopy

Fundus camera

Fluorescein Angiography

Optical Coherence tomography

Direct ophthalmoscopy

The direct ophthalmoscope gives a magnification of approximately 15 x and a field of view of 6.5 to 10 degrees, therefore, if we want to see more than just the very posterior pole the patient will have to look in 6 to 8 different directions. This 15 x magnification makes

the 1.5 mm disc appear much larger than it really is. The formula M= 60D/4 holds well for up to + or - 10D's of refractive error. The loss of stereopsis makes it a less benefitial tool in the diagnosis and management of diabetic retinopathy.

Indirect ophthalmoscopy

To view the fundus from the posterior pole to the periphery . With experience, can view the entire retina. Provides a minified, high resolution, stereoscopic view of the fundus. The magnification depends on the hand held lens used and in a trained hand it is a good method for the diagnosis, characterisation and management of diabetic retinopathy.

Hruby lens

The Hruby lens is available in both a contact and non-contact version. The non-contact Hruby lens is a high powered plano concave minus 55D which is available for most slit lamp biomicroscopes as an attachment from either above or below that can be rotated into the line of sight.

Slit Lamp Aspheric Biomicroscopy (Indirect Fundus Lenses)

These lenses have an advantage over the non-contact Hruby allowing a better view around cataracts.

These lenses are double aspheric and come in +90D, +78D and +60D powers. The magnification increases inverse to the power of the lens. The +60 D lens gives greater magnification and is preferred by some for examination of the optic nerve and macula. The +90 D lens produces less magnification and larger field of view (30-40 degrees)

However, the slit lamp biomicroscope permits variable magnification which neutralizes this magnification problem.

The +78 D lens obviously falls in between the +60 D and +90 D lenses in terms of magnification and field view. It is slightly smaller in overall size than the +60 D and noticeably larger than the +90 D lens. The +78 D lens is usually preferred by the novice who feels it is easier to hold and manipulate

Magnification of the lens = power of the eye / power of the condensing lens

In a 90 D lens

Mag. = 60d / 90d

Mag. = .666 **x** magnification of slit lamp

Now a days, slitlamp biomicroscopy has become the gold standard in the diagnosis of diabetic macular edema.

Contact biomicroscopy

Contact biomicroscopy has superior resolution and stereopsis as compared with indirect lenses, but needs contact with the cornea and a coupling solution,. It is done with a macular or posterior pole lens. As they are invasive and diabetic corneal epithelium is more amenable to damage, we do not usually retort to contact biomicroscopy routinely.

Fundus photography

Recently, fundus photography has been used for screening purposes using nonmydriatic cameras, which are designed to allow photography of the fundus through an undilated pupil. This has the potential benefit of permitting retinal evaluation without the need for pupillary dilatation, while providing a permanent record for later evaluation. However, these benefits are limited by the frequent difficulties of achieving adequate pictures through small pupils with cataracts. Obtaining useful photographs can be difficult, even for the experienced ophthalmic photographer, though the results can be improved by dilating the pupils. The pictures provided are two-dimensional, which makes evaluation of macular edema difficult.. Because fundus photographs frequently depict extremely subtle pathologic change, these pictures must be analyzed by an ophthalmologist who is experienced at evaluating diabetic retinopathy. This technique should be used only as a screening method to help identify patients who may need more extensive evaluation using one or more of the techniques described.

Fluorescein angiography

Fluorescein angiography with color fundus photography is another useful method for evaluating diabetic

retinopathy. Stereoscopic color fundus photographs are first made of the macula and posterior peripheral retina of each eye. Then, the patient is given an injection of 5 to 10 ml of fluorescein dye intravenously. Rapid sequence photographs using filters matched to the spectral response of fluorescein in vivo are then taken immediately after injection of the dye and are continued periodically for 15 to 20 minutes. This technique can help determine the presence of capillary closure (which frequently cannot be determined by other clinical techniques), the presence of macular edema, the location of leaking microaneurysms and capillaries, and the presence of neovascularization. Fluorescein angiography provides a permanent record of the appearance of the fundus for later evaluation as necessary. The disadvantages of this technique include the need for dilation of the pupil, the need for intravenous injection of dye with its associated (though minimal) risk of an allergic reaction, and the additional cost.

Indications of Fluorescein Angiography in Diabetic Macular edema

1. To determine if retinal thickening in CSME is due to focal or diffuse leaks and to plan the photocoagulation treatment appropriately.
2. To identify ischemic maculopathy
3. To differentiate cystoid macular edema from diabetic macular edema in aphakes and pseudophakes.
4. To monitor the progression or regression of macular edema following macular photocoagulation

Optical Coherence tomography

OCT is a very convenient and accurate quantitative tool in the initial and sequential evaluation of diabetic macular edema. Its closest analogy in ophthalmology is like a visual field in glaucoma. We can have a very accurate anatomical reconstruction of the vitreomacular interface and that of the macula itself. The presence or absence of CME and vitreomacular traction can be easily picked up with this technique only. With the advent of intravitreal triamcinolone and other anti VEGF agents, OCT has become a very necessary tool. Other methods like the Retinal thickness analyzer (RTA) and HRT II has not become as popular as the OCT.

Current practice patterns of evaluation

Best corrected visual acuity

Dilated slitlamp biomicroscopy for diagnosis of CSME

If CSME present, then FFA is done

If CSME involves central fovea, suspicion of CME, suspicion of vitreomacular traction, then proceed for OCT.

Post laser residual CSME needs FFA and OCT

Unexplained visual loss requires OCT and FFA.

MANAGEMENT OF CSME

Systemic factors

Systemic factors like glucose levels, lipid levels and renal status play a role in the management of diabetic macular edema. The DCCT and UKPDS has demonstrated that glucose control and hypertension control will reduce the progression of diabetic macular edema. Other factors like stopping cigarette smoking and antioxidants have a marginal role in the control of progression.

Laser photocoagulation

The Early Treatment Diabetic Retinopathy Study (ETDRS) was a multicenter trial that showed a 50% reduction in visual loss in eyes with clinically significant macular edema treated with focal argon laser photocoagulation when compared with similar severity of edema in eyes not treated with laser photocoagulation.

Photocoagulation for macular edema requires a recent (48-72 hours old) retinal fluorescein angiogram. Laser photocoagulation is directed specifically to the leaking macular microaneurysms noted angiographically. Multiple treatment sessions spread over many months are frequently necessary. Photocoagulation applied in a grid pattern surrounding the fovea may be useful for the management of zones of diffuse leakage or nonperfusion.

The ETDRS showed that focal photocoagulation should be considered for all eyes with clinically significant macular edema. In this study, focal photocoagulation treatment reduced the risk of moderate visual loss and increased the chance of visual improvement. The best timing for initiating treatment remains debatable for patients who have clinically significant macular edema

but are asymptomatic and have normal visual acuity. When considering the initiation of focal photocoagulation, the ophthalmologist must take into account the degree of central macular involvement, as well as the potential risk from photocoagulation. Potential side effects and complications of focal photocoagulation are unusual and include paracentral scotomata that frequently fade with time, development of abnormal neovascularization under the retina (subretinal neovascular membrane) that can lead to visual loss, and transient blurring of vision.

Micropulsed diode laser

The side effects of the focal macular photocoagulation are all caused by collateral and deep burns, and this is precisely what micropulse laser prevents. In this technique, laser is delivered in small trains or measures with sufficient time between these pulses so that the heat can be conducted. The results in diabetic macular edema has been encouraging with resolution in about 60 % of eyes.

Intravitreal steroids (IVTA)

Intravitreal triamcinolone has been a very talked about treatment of diabetic maculopathy in recent times. It is an inhibitor of vascular endothelial growth factor and so it helps reduce the macular edema. It has proven its efficacy in residual macular edemas following one or two sittings of focal macular photocoagulation. Research is directed now to find if it is more effective in the primary management of diabetic macular edema even before laser. Though the results have been good , the selection of cases is of utmost importance. The eyes with a vitreomacular traction will worsen with an IVTA. Thus, it becomes imparative to study the vitreomacular interface before giving an IVTA.

IVTA is usally performed under topical anesthesia and with a 27 guage needle. 4 mg of presevative free triamcinolone is injected through the pars plana and tension controlled with paracentesis. This is done under aseptic precautions in the operating room.

Rise in IOP has been a limiting factor in intravitreal steroid administration, Though many cases are amenable to antiglaucoma drugs, as optic nerve in diabetes in very labile, this has to be managed aggressively. Other Complications associated with IVT injection of steroids include cataract progression, endophthalmitis, RD, hypotony, entrocular bleed etc.

Pars plana vitrectomy

It was found that an attached posterior hyaloid predisposed to the progression of diabetic macular edema, hence pars plana vitrectomy was tried for CSME. Early studies show good response to PVD induction and posterior hyaloid stripping in cases of CSME. Recent studies are concentrating on the effect of ILM peeling on diabetic macular edema. But as of now, PPV is done only in those cases with a documented vitreo macular traction on OCT with low vision. Since the complications of vitrectomy like cataract retinal tears and subsequent retinal detachments cannot be completely excluded, it is better avoided in eyes with vision better than 6/18.

Futuristic trends

Injection of anti VEGF agents (Intra Vitreal Injection of Pegaptanib (Macugen)

Chemical PVD

Protein kinase Inhibitors

COX 2 inhibitors

Extended release implants through parsplana (Flucinolone Implant)

Conclusion

All patients of diabetic macular edema should be encouraged to optimize treatment of systemic risk factors, be it glycemic control, lipid control or renal stabilization. Blood pressure control should be emphasized upon and patient is asked to quit smoking. In presence of renal dysfunction, it is important to initiate the patient on ACE inhibitor also.

After this is done, in majority of patients focal macular photocoagulation direct to microaneurysms picked up on fluorescein angiography should be performed during the first sitting. Do not go closer than 350 microns of the center of the fovea.

Wait for 3 to 4 months before any other intervention. After that, the patients are assessed regarding any

residual macular edema with fluorescein angiography and OCT in select cases. If there are still microaneurysmal leakage, a second sitting of macular laser, this time going more closer to the fovea if needed is done.

In select patients in whom, there is recalcitrant macular edema even after 2 sittings of focal macular laser, 4 mg of triamcinolone intravitreally is advised, provided, the patient completely understands the efficiency and consequences of the procedure. After IVTA, a fluorescein angiography is repeated at 2 months to look for any possible microaneurysms which are amenable to treatment.

In those patients who have failing vision over 6 months and in whom a vitreomacular traction has been demonstrated on OCT, pars plana vitrectomy can be advised. However it is undertaken only if the patient understands the nature of treatment and its efficacy and side effects.

With this approach, we are able to satisfactorily treat the majority of patients with CSME. When ever possible, the least aggressive approach is utilized. The newer approaches to diabetic macular edema looks promising but need large clinical trials & long term followup studies to assess their efficacy.

Management of a "Floating Angioma" with Sequential barrage and feeder vessel photocoagulation followed by Transpupillary Thermotherapy

Dr. Biju Raju MS, FNB, **Dr. NSD Raju** MS, DOMS, **Dr. Anju S Raju** DOMS, **Dr. Sudhakaran CP** DOMS
Smt. Meenaksiamma Center for Vitreoretinal diseases, Ranjini Eye Care, Cochin

Address for Correspondence: Dr. Biju Raju, Smt. Meenakshiamma Center for Vitreoretinal Diseases, Ranjini Eye Care Near Powerhouse, Vyttila, Kochi – 682019, Email: drbijuraju@ranjinieyecare.com

Introduction

Von Hippel angiomatosis is a congenital hereditary capillary angiomatous hamartoma of the retina and optic nerve. Visual loss in this condition occurs due to lipid-rich exudation at the macula, exudative retinal detachment, vitreous hemorrhage, epiretinal membrane formation and rhegmatogenous retinal detachement. [1] Though cryotherapy and photocoagulation are the most common treatment modalities they are associated with various complications like increased macular exudation, macular scar, macular hole and combined traction and rhegmatogenous retinal detachment. [2] This case report describes the management of an angioma dragged forward due to vitreous traction (floating angioma) in an eye with good visual potential.

Case report

18 year old male presented to us in 2001 with complaints of sudden loss of vision in the right eye. The fundus examination showed vitreous hemorrhage. He was managed conservatively and improved from the initial vision of hand motions to 6/6. On examination, the anterior segment was normal (OU). The fundus (OS) was also normal. Fundus (OD) showed resolving vitreous hemorrhage inferiorly. There was a retinal angioma which was dragged forward into the posterior 1/3rd of the vitreous due to traction (Figure 1). The feeder vessels were dilated and tortuous. There was lipid exudation close to the posterior pole (Figure 2 A) and a serous retinal detachment surrounding the angioma. Surface neovascularization and vitreous traction over the angioma were evident on clinical evaulation.

The goals of treatment in this case were to prevent further increase in serous detachment, macular

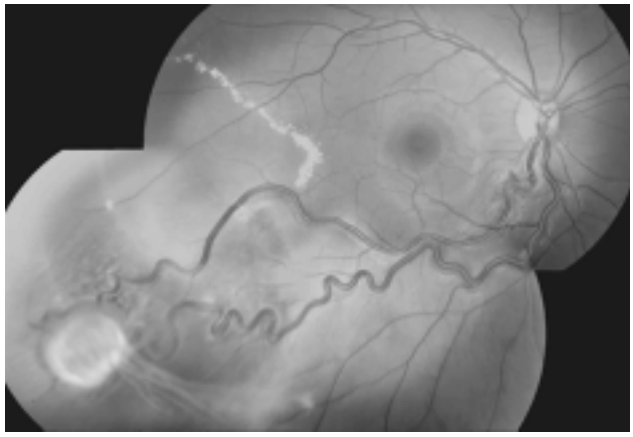


Fig. 1. Floating angioma

exudation and vitreous traction. The initial step was to barrage the area of serous detachement with confluent grade 3 to 4 photocoagulation burns using a laser indirect ophthalmoscope (LIO). The feeder vessels were carefully avoided so as to prevent any increase in exudative detachment (Figure 3). A reduction in the amount of macular exudation was seen at 2 weeks after LIO (Figure 2 B). The 2nd step in the management was feeder vessel treatment to shrink the tumor. It was done in 2 sittings – 1st sitting was directed towards obliterating the feeder artery and then the vein was treated directly after 1 week. One month later the patient was reevaluated and a modified transpupillary thermotherapy using frequency-doubled Nd:YAG (532 nm) (1000 m spots, 300 mW, 1000 ms, 10 burns) was done. The burns were placed directly on the tumor.

The only complication encountered during the management was a hemorrhage from the artery during the feeder vessel treatment (Figure 4). It was managed by applying pressure on the contact lens alone. After the resolution of serous detachment following feeder vessel treatment, further barrage LIO photocoagulation was done till the tumor base. At final visit, the tumor size had markedly reduced. There was a reduction in the macular exudation and the serous detachment.

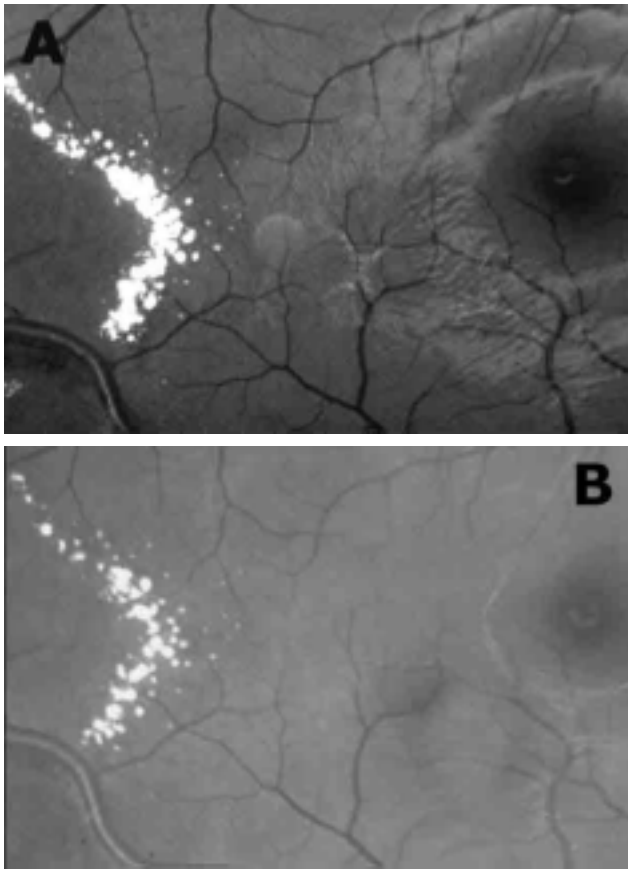


Fig. 2. A. Lipid exudation close to macula B. Decrease in the lipid exudation after the barrage laser

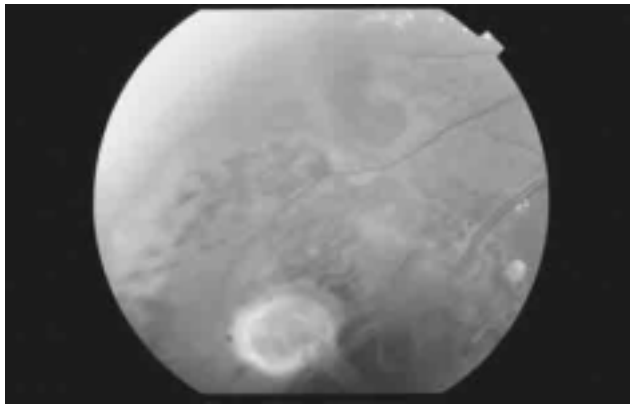


Fig. 3. 4 weeks after the barrage LIO. Note that the feeder vessels were avoided during the treatment

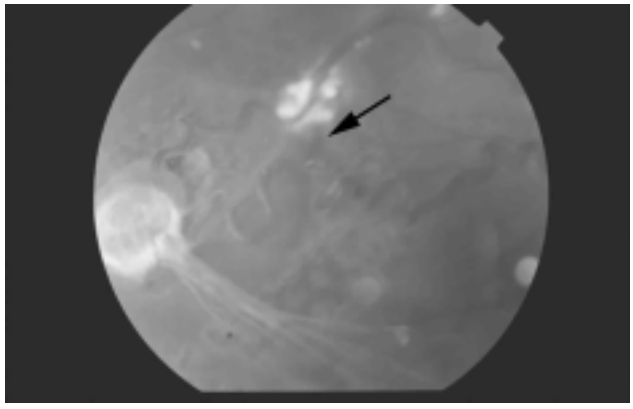


Fig. 4. Hemorrhage from the feeder artery during feeder vessel treatment

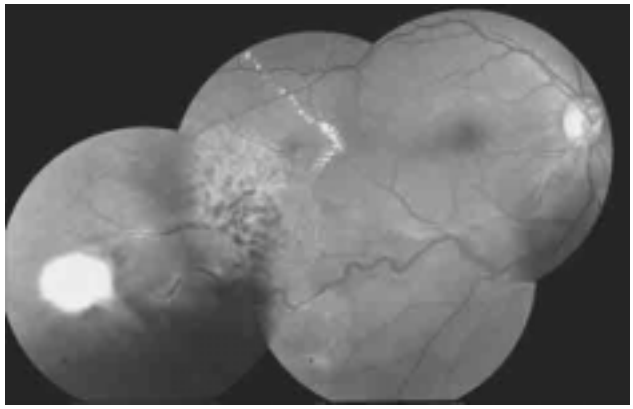


Fig. 5. Fundus at final follow up. Note that the angioma has markedly shrunk and replaced with gliosis. The feeder vessels have regressed with normal diameter and there is a reduction in its tortuosity

The dilatation and tortuosity of the feeder vessels also reduced (Figure 5). The visual acuity was maintained at 6/6 and the intraocular pressure by applanation was 12mm Hg.

Discussion

Mangement of "floating angiomas" is difficult as there is a risk in causing visual loss due to increasing traction, macular exudation and exudative retinal detachment.[1,2] Sequential barrage and feeder vessel photocoagulation followed by transpupillary thermotherapy is a better approach in managing these difficult angiomas. The feeder vessel treatment controlled the exudation and the modified TTT markedly reduced the tumor size. The barrage laser photocoagulation to prevent worsening of serous detachement and vitreous traction is a new concept in the management of these difficult tumors.

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Does cataract surgery induce dry eye?

Dr. Merine Paul, MS,DNB

Vijayashree Eye Hospital, Thrissur

Introduction

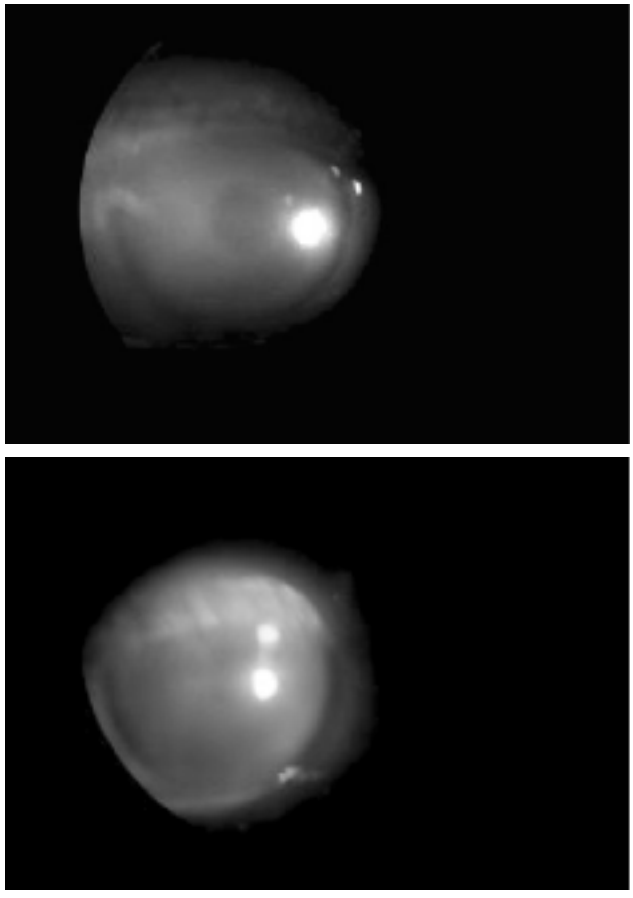
After a good cataract surgery with excellent postoperative visual rehabilitation, we are often nagged by some patients with persistent vague complaints. We put them down as psychosomatic in origin. However a body of scientific evidence is emerging that these may be due to induced dry eye. I am reporting a case which went into severe dry eye following uneventful phacoemulsification surgery.

Case

A 65 year old woman presented with dimness of vision in both her eyes. Immature cataract was diagnosed in both eyes and patient was taken up for surgery in the RE. Pre op evaluation including Shirmmers test and TBUT were normal. She was prescribed glasses after I week, acheiveing a BCVA of 6/6 and post operative medications were tapered gradually. One month into the post operative period patient came back with complaints of itching, foreign body sensation and stringy discharge from the eye. Examination showed vision of 6/18 with glasses, a congested eye and punctate staining of the cornea with a ground glass appearance. Anterior segment and fundus was within normal limits. Differential diagnosis at this stage was epithelial toxicity to the drugs or severe dry eye Shirmmer test showed O mm wetting and TBUT was 3 sec.

Treatment

As post operative steroid and NSAID were down to twice daily dosage by then, it was decided to taper them on schedule and treat the dry eye. Intensive therapy with hourly, preservative free carboxymethyl cellulose drops and twice daily carboxymethyl cellulose gel were initiated along with twice daily cyclosporine 0.05% drops. Patient responded well to the therapy and after a month the cornea was clear and vision improved to 6/6. The lubricants were reduced to thrice daily but it was decided to continue the cyclosporine drops for 6 months.



Clinical photographs
Postoperative dry eye on day 30 & day 45 respectively

Discussion

Cataract patients are often already predisposed to dry eye because of factors like age, surgical trauma and medications.

However the surgery itself compounds the problem in a number of ways, starting from the preoperative investigations like tonometry, scanning etc where local anaesthetics are used liberally along with dilating agents. During the surgery, once block is given care has to be taken to ensure that the eye is properly closed till the patient reaches the OT table. Intraoperative corneal drying and over stretching of lids with speculum should be avoided at all costs. Conscientious surgery goes a long way in preventing post operative dry eye as do a smaller incision length (lesser corneal denervation). Watch out for epithelial toxicity with post operative medications. This is an entity which seems to enjoy a symbiotic relationship with dry eye with the margins between the two often blurring. Routine use of postoperative preservative free ocular lubricants should be actively considered in patients with predisposing factors.

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Pituitary Macroadenoma A Case Presentation

Dr. Raju.K.V., Professor
Dr. Ruby.M.V., P.G.Trainee, Department of Ophthalmology, Medical College, Calicut

Key words: Pituitary Macroadenoma, Prolactin, Proptosis

A 30-year-old male presented to Department of Ophthalmology, Medical College Hospital, Calicut with chronic headache and proptosis of left eye since 1 month. On detailed general examination, we noticed bilateral gynecomastia. There was no thyroid swelling, and all other systems were found within normal limits. Ocular examination showed axial proptosis of 3 mm left eye (Fig. 1 & 2), extra ocular movements were full, a relative afferent pupillary defect and defective colour vision. Best corrected visual acuity of 6/36 LE, vision in right eye being 6/6. Fundus examination was within normal limits. There was no mass palpable in the orbit, no resistance to retropulsion, and no bruit, Lister's perimetry showed a 10° constriction in superotemporal field.

We investigated the patient. CT showed (Fig. 3 & 4) a 31 x 42 x 47 cms well defined moderately contrast enahcing iso-hyperdense lesion with irregular margins and lobulated contour in the sellae, parasellar region with asymmetric growth with greater volume on left

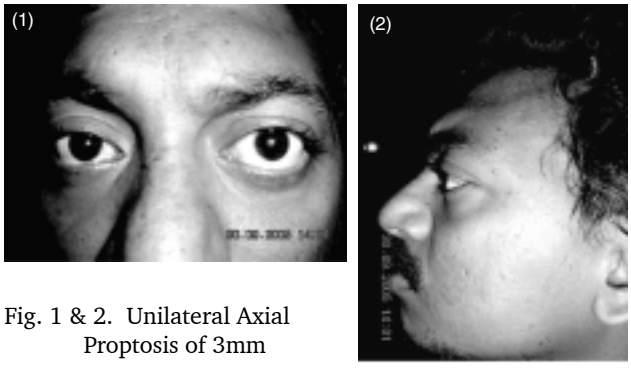


Fig. 1 & 2. Unilateral Axial Proptosis of 3mm

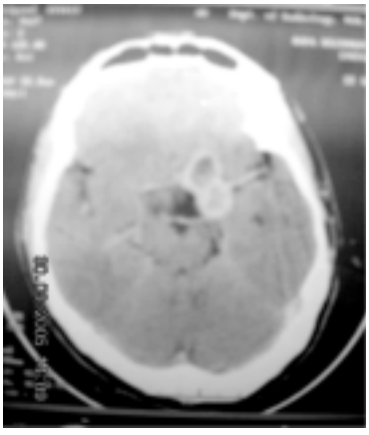


Fig. 3. C.T.scan showing hyperdense lesion in the sellar-parasellar region with asymmetric growth

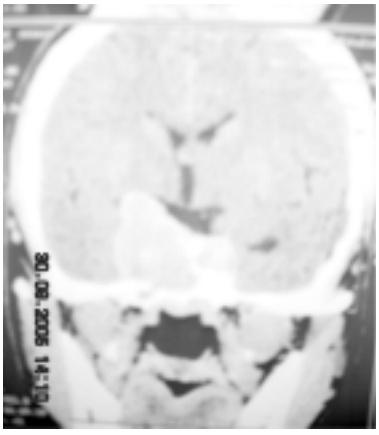


Fig. 4. C.T.scan showing suprasellar extension

side. Posterior aspect of left superior orbital fissure and posterior optic foramen was indented. There was no evidence of significant extension into anterior aspect of optic foramen or into the orbit. Laterally the lesion was extending to the cavernous sinus on left side more than right. CT impression was sellar-parasellar mass lesion. According to the Radiologist, this could be an aggressive pituitary neoplasm. The second and third possibilities were large aneurysm with thrombosis or meningioma.

MRI scan showed (Fig. 5 & 6) large sellar, supra sellar mass lesion with lateral, superior and inferior extension suggestive of pituitary macroadenoma.

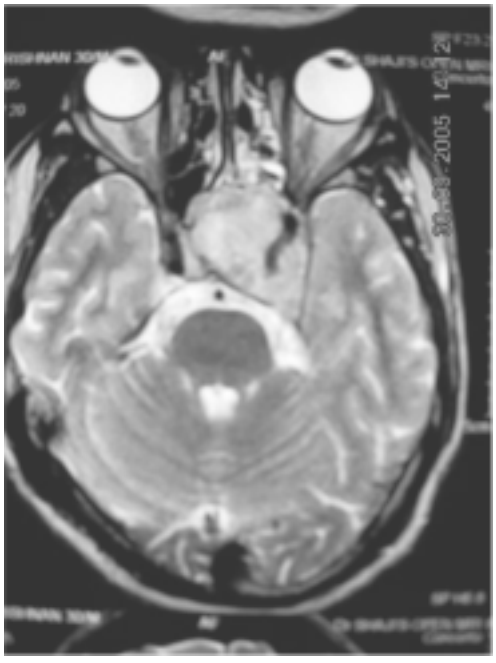


Fig. 5. MRI showing mass in the sellar-parasellar region

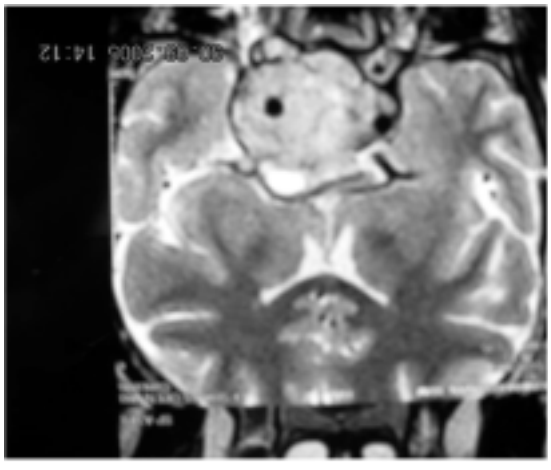


Fig. 6. MRI showing extension of the lesion into the cavernous sinus; Lt.internal carotid artery engulfed by the lesion

Hormonal assay was done. Prolactin level was found to be >470 ng/ml (normal range : Male 4.6 – 21.4ng/ml). Thyroid function test was found to normal.

From the clinical examination and investigation, we came to a clinical diagnosis of pituitary macroadenoma – Prolactinoma.

We treated the patient with bromocriptine, 2.5mg OD along with systemic steroids continued in tapering doses. At present he is on T.Bromocrptine 2.5mg OD and T.Prednisolone 10mg OD.

On follow up, his visual acuity has improved to 6/9. Proptosis and visual field defect remain the same. Since proptosis is not increasing and the vision has improved, the patient is advised to continue medical treatment.

Discussion

Pituitary tumours constitute 10-15% of intracranial neoplasm. Symptoms depend on presence of pituitary hypersecretion, absence or reduced hormone levels caused by destruction of normal pituitary gland or direction of local expansion and invasion of adjacent structures. Tumours can be classified on the basis of their appearance after standard histologic staining – chromophobic, acidophilic and basophilic adenoma. Tumour size also can be used to classify tumors. Microadenomas are tumours measuring less than 10mm in diameter and those of more than 10mm are termed macroadenomas. Physiological classification by immunohistochemical staining or by serum hormone measurement divide tumours into non secreting and secreting types. The secreting (functional tumours) constitute 75% of pituitary adenoma. They include:

- Growth hormone (GH) cell adenoma
- Prolactin PRL cell adenoma (Prolactinoma)
- Mixed GH & PRL adenoma
- Corticotroph (ACTH) cell adenoma
- Thyrotroph (TRH) cell adenoma
- Gonadotroph (LSH) and FSH cell adenoma

40-50% of pituitary adenomas are constituted by prolactinoma. In females, this presents with amenorrhoea, galactorrhea and in males with testicular atrophy, gynaecomastia, reduced body hair and

impotence.

Tumours which are secreting are detected early due to the various syndromes they produce. Non-secreting tumours are large at the time of diagnosis and present with various structural problems like head ache, loss of visual fields, typically bitemporal field loss, cranial nerve palsies due to the invasion into cavernous sinus or with epistaxis due to downward extension through floor of sella.[2] The mass can extend to orbit resulting in proptosis.[3] They can present with sudden onset of head ache / loss of vision due to haemorrhage or necrosis of tumour as pituitary apoplexy.[4]

Diagnosis and treatment planning is greatly facilitated by CT and MRI. Pituitary adenoma are isodense or slightly hyperdense compared with adjacent brain and shows homogenous contrast enhancement with IV contrast material.[5] MRI allows evaluation of the degree of suprasellar extension and involvement of chiasma and cavernous sinus. MRI of pituitary macroadenoma reveals prolonged T_1T_2 signal compared with normal brain tissues.[6] Patients with sellar abnormalities whether clinically symptomatic or not should have lab investigation for anterior pituitary dysfunction.

The newest most specific form of therapy for hypersecreting pituitary adenoma is medical. The drug used is bromocriptine. It is a dopamine D_2 agonist, thereby inhibits prolactin secretion. Bromocriptine effectively reduces high prolactin levels, result in shrinkage of tumour and relief of compressive symptoms.[7] Withdrawal may lead to re expansion of tumour[8]. In 80% of patients with prolactinoma medical therapy is sufficient to control symptoms and reduce prolactin levels to normal levels. For the rest 20%, it provides an useful adjunct to subsequent surgery or radiation therapy. Management of pituitary adenoma often requires the combined or sequential use of multiple forms of therapy. We should follow up the patient with hormonal assay, neuroimaging studies and neuro- ophthalmic evaluation at 6 month intervals.[9].

Conclusion

Usual presentation of pituitary tumours are with visual field defect or head ache and this is one of the rare presentation with axial proptosis.

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The Single Radial Incision (Sinrad) for the small incision cataract surgery

Dr. Samuel P. John MBBS., DOMS.

Medical Superintendent, G.M. Eye Hospital, Annur Road, Mettupalayam – 641 301
Tamil Nadu. Phone: 04254 – 222674, 223127 E-mail: gmeh@sify.com

Abstract

Aim: To perform the Small Incision Cataract Surgery through a single radial incision.

Methodology: 181 patients underwent cataract surgery through a single radial incision, 3.5mm in length, beginning at the limbus and 300 micron in depth. A partial thickness scleral dissection was made on either side and carried on into the cornea

Results: The post operative astigmatism was significantly reduced. 76% had astigmatism of = 0.5D and 41% of these had no astigmatism.

Conclusion: The single radial incision is an excellent alternative incision for the small incision cataract surgery.

Key words: SICS, SINRAD, POST OP ASTIGMATISM

Introduction

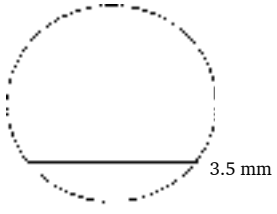
Incisions of a variety of sizes and shapes have been used for the small incision cataract surgery. These incisions have all adhered to the basic principle of incision for this surgery – namely that a) incisions within the astigmatic tunnel do not produce significant astigmatism.

b) The inner corneal incision is equally or more responsible for the post op astigmatism as the external or scleral incision.

Over the past few years, I have been performing the small incision cataract surgery using the 'V' incision. This incision is made without lifting a conjuctival flap and has its apex at the limbus and is in effect 2 tangential incision. A rigid lens of 5 mm is implanted with excellent results.

The Single Radial or SINRAD incision has been a natural progression from the 'V' incision.

This Single Radial Incision though radical in concept, should expand the horizons of the small incision cataract surgery.



A Single Radial Incision of 3.5 mm

Methods

A fornix based conjuctival flap is raised and bleeders cauterized.

A single radial incision of 3.5mm is made using a 300 micron controlled depth blade beginning at the limbus.

A partial thickness scleral dissection for 3-4 mm is made on one side (the left half) using the bevel up crescent blade and the dissection is carried on into the cornea at the extreme end.

The corneal dissection is enabled using a 'spoon' blade so as not to tear the edges of the scleral wound. This dissection is done for 1.5mm into the cornea. The other half of the scleral dissection (right half) is done using the crescent blade.

The incision therefore is shaped like the letter 'T', the vertical limb of the 'T' being the scleral end and the horizontal limb being the corneal end of the incision.

The entry into the anterior chamber is made using a 1.2mm microkeratome and this may be extended on either side for 2-3mm

The capsulorhexis is followed by a gentle hydro dissection and the inner corneal valvular incision extended using the crescent blade.

The nucleus is rotated into the anterior chamber using Sinsky hooks and fractured into 2 pieces using a 2mm serrated vectis and Sinsky hook of ½ mm tip.

The pieces are then extracted using the sandwich technique under abundant viscoelastic cover.

The cortex is cleared and a foldable lens inserted either using an injector or the folder and holder. The conjuntiva is then reapposed.

Results

The post operative astigmatism in these patients is only less than the routine SICS. The results assessed on the first post op day are shown below: Out of 181 cases performed 138 had post op astigmatism of 0.5D or less (76%) and 56 of these had no astigmatism (41%).

Discussion

The single Radial Incision causes lesser amount of post op astigmatism than the routine SICS.

- Foldable lenses can be used.
- Reproducible and conversion to ECCE is not difficult
- Hard brown nucleus above grade III are still a problem, which has to be overcome. The main complication here being the striate keratitis which disappeared in a short time.
- It is of course difficult to implant rigid lenses.

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4. J Cataract Refract Surg. 1989 Jan; 15 (1) : 70-7 Keratorefractive aspects of the scleral pocket incision and closure method for cataract surgery

Doctor-Patient Communications

Dr. Alex Joseph
Professor of Ophthalmology, Jubilee Mission Medical College, Trichur

The main goal of the practice of Ophthalmology is to deliver quality eye care to the patients with eye ailments. All Ophthalmologists have an obligation to prevent impairment of vision by proper control of eye disease and restore sight to the blind. They often fulfill this obligation through private practice, working in Government and private hospitals or in Ophthalmic institutes.

If we look into the previous generation of clinicians, we find that majority of them had scanty communication with their patients. They never bothered to explain to them regarding their ailment, the prognosis of the disease or the treatment schedule. Those days even the dose and mode of administration of medicines were explained by the chemists. That generation of medical practitioners could afford this as he was their living saint and healer. The public often cites the example of ancient Physicians who worked day and night to relieve the sufferings of their patients. Indeed there is no comparison between the physician of that era and the present day physicians. Most physicians of the past could not make the ends meet willingly or unwillingly. They lived on community's support. On the contrary todays physicians are amongst the most affluent in our society. Modernisation of medicine with high technology resulted in monetarisation of medicine which has adversely affected the loyalty of Physicians to their patients. The altered Doctor – Patient relationship to a great extent has brought this noble profession to disrepute. This changed attitude and situations resulted in a sea of change in Doctor – Patients relationship. Now we are in a different society – A consumer protection era – where the benefit of doubt, protection and advantage goes in favour of the patient.

In this present scenario, there is lot of importance for a proper dialogue of the doctor with the patient. Unlike olden days, especially in our part of the country, because of the high literacy and the exposure to media, the public is aware of many medical conditions and its complications. So even though they don't demand, they expect a detailed and useful communication from the physician. A good percentage of consumer protection litigations against doctors is due to lack of communication between the doctor and patient – failure on the part of clinician in explaining the clinical condition, mentioning the prognosis and detailing the treatment schedule. Many of the consumer protection litigations could have been avoided if there was a proper dialogue of the physician with the patient.

Here I would like to narrate one simple example. All of us see Primary Open Angle Glaucoma patients in our routine practice. The disease is detected either during the routine change of glass or when the patient visits with complaints of defective vision, as in this age group they may be having early cataract also. We make the correct diagnosis and put the patient on anti glaucoma drops. The patient on his part regularly uses the drops for few months without any improvement in the vision – his main complaint – and he stops the medicine. In between he may consult a country doctor who will make a diagnosis of cataract. He waits for the cataract to progress for surgery. He will come back after 3 or 4 years for cataract surgery with PL vision and advanced glaucomatous optic atrophy where we can't help him – leaving him blind for the rest of his life. If the consultant in the initial visit has taken the time to explain to the patient that the drops will not, and are not meant for bringing

about visual improvement and also emphasized the important fact that failure to comply can lead to progression of disease, poorly controlled pressures and blindness, I am sure that the patient would have used the medicine and prevented the catastrophe. So by proper dialogue with the patient we can prevent the untoward effects or irregular and improper treatment and can bring down the number of consumer protection litigation.

Communications can be Verbal or Nonverbal. Verbal communications means using words and language to communicate. Non verbal communication refers to all aspects of message which are not conveyed by the literal meaning of words. Both Verbal and Non verbal interaction is necessary in Doctor – Patient communication. The Verbal communication includes the explanation regarding the clinical condition, its prognosis and treatment details. Non verbal communication includes PARALINGUISTIC (BODY LANGUAGE)- facial expressions, gesters, postures and eye contact and KINESIS – sound element of non verbal communication. Doctors facial expression can convey the seriousness of the clinical condition, importance of starting the treatment at the earliest, sympathy etc. The eye contact of the clinician with his patient is a very important aspect of non verbal communication. The other important aspect of non verbal communication is the modulation of sound when communicating with the patient. The tone of the doctors voice conveys the state of his mind like anger, frustration, sympathy, kindness etc. It is very important for the present day clinicians to understand and develop good communication skills which takes care of half of the patients' need.

Birdshot Retinochoroidopathy

Dr. Meena Chakrabarti MS, DO, DNB
Senior Consultant, Chakrabarti Eye Care Centre, Kochulloor, Trivandrum 695011

History

32 years old male presented with increasing floaters and defective vision of acute onset in both his eyes. Ocular examination revealed a best corrected vision of 6/6 (OD) and 6/9 (OS). There was no anterior segment inflammation, the lens was clear and the intraocular pressures normal. Vitreous cavity showed moderate vitritis. Fundus examination revealed (Fig (1) (a) &(b)} - a hyperemia optic disc and multiple deep elevated cream colored lesions in the inferior fundus of both eyes and a small area of vitreous haemorrhage left eye. FFA revealed retinal vascular staining, leakage from the optic disc , absence of cystoid macular oedema and mild hyperfluoresence over the cream colored lesions

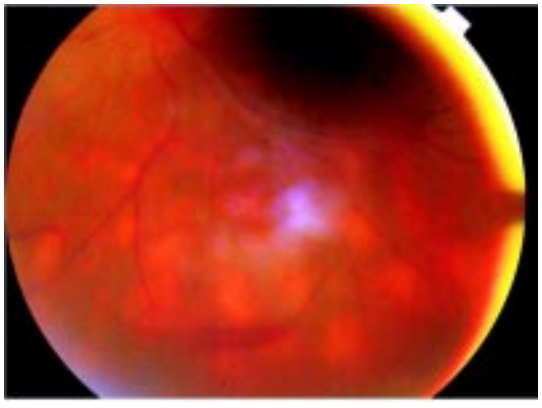


Fig 1(a) Showing multiple deep elevated cream coloured lesions in inferior retina

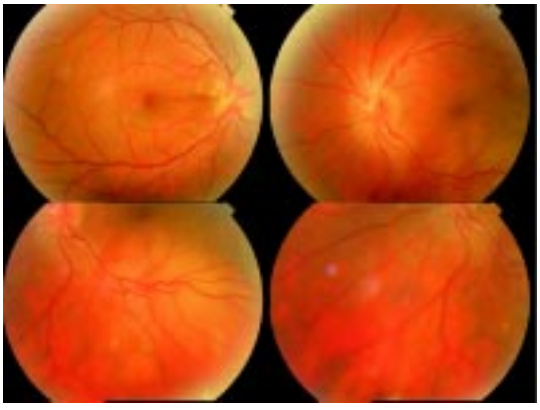


Fig 1(b) Showing hyperaemia of optic discs

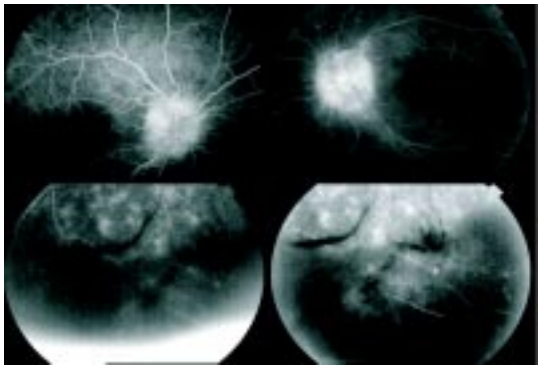


Fig. 2. Fluorescein fundus angiography revealed vascular staining, optic disc leakage & mild hyperflurescence over the cream coloured lesions

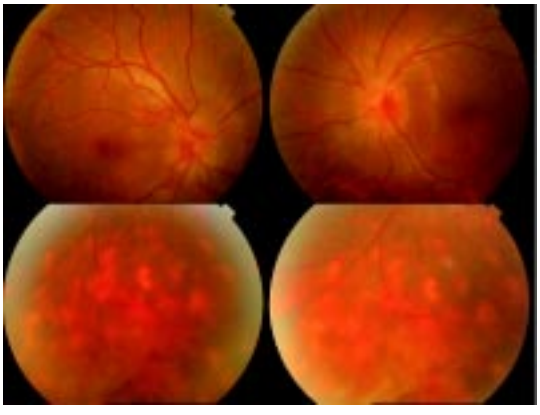


Fig. (3) Follow up fundus photograph showing good resolution with areas of RPE alterations

(Fig (2) }. Good therapeutic response was obtained with systemic steroids in slow tapered doses starting with 1 mg/kg bodyweight . Follow-up showed good resolution with areas of RPE alterations.

CLASSIC FINDINGS OF BIRDSHOT RETINOCHOROIDOPATHY

- **HISTORY**
  - Healthy Women, 3rd -6th decade, Whites (Rare in Non Whites)
  - Presents with floaters, difficulties with night vision and color discrimination.
- **OCULAR EXAMINATION FINDINGS**
  - Quiet Anterior Chamber
  - Vitreal Inflammation; No Snow Banking
  - Retinal Vascular Leakages and CME (41%)
  - Deep Circular Cream Colored lesions mostly in posterior pole and surrounding areas
  - Bilateral
  - Low to moderate risk of subretinal neovascularisation
- **ADDITIONAL TESTS:**
  - Abnormal ERG and EOG
  - Evidence of Retinal Autoimmunity
  - Association with HLA A-29
  - FFA: Delineates degree of Retinal Vascular Leakage & Severity of CME
  - OCT : Delineates presence of Cystoid Macular Oedema
- **THERAPY:**
  - Systemic Steroid in slow tapered doses
  - Intravitreal Triamcinolone Acetonide if CME is present
  - Immunosuppressant: Cyclosporine / Azathioprine
- **SEQUALE**
  - Long term visual prognosis guarded (52% Worsening ; 30% Stabilization; 18% Improved vision)
  - Disconcerting continued decrease in retinal function, retinal thinning and vascular attenuation may be seen even after ocular inflammatory activity subsides

Dr. A. Giridhar MS (Giridhar Eye Institute, Cochin)
Dr. Gopal S. Pillai MS, DNB, FRCS (Amritha Institute of Medical Sciences, Cochin)
Dr. T.P. Ittyerah MS (Little Flower Hospital, Angamaly)
Dr. Roy Mathew Zachariah MS (Medical Trust Hospital, Cochin)
Dr. Thomas Cherian MS (Little Flower Hospital, Angamaly)

Case History

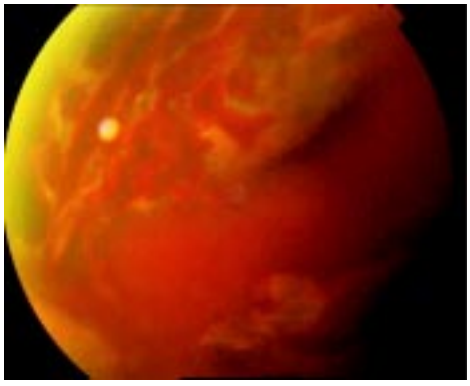
A 60 year old female, a chronic diabetic and hypertensive of more than 15 yrs under reasonable good control presented with a vision of hand movements (HM) in both her eyes. Visual loss was of sudden onset in her left eye and of 4 months duration Right eye had poor vision due to long standing posterior pole traction retinal detachment which on evaluation proved to be inoperable.

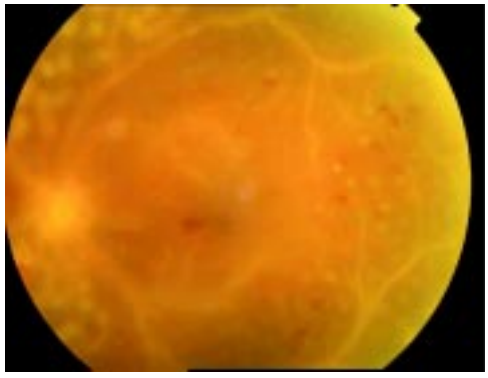
Ocular examination revealed a normal anterior segment with incipient cataract. Fundus examination of left eye showed large subhyaloid haemorrhage with vitreous haemorrhage (Fig 1)

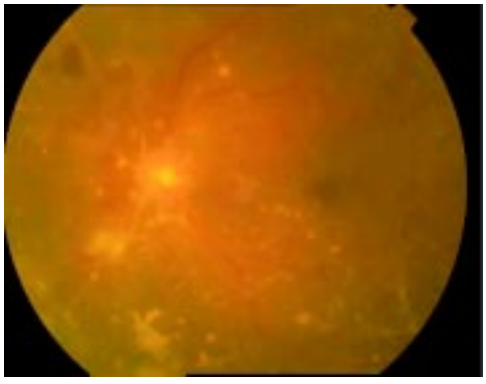
Her diabetic status was brought under control and she underwent Parsplana Vitrectomy with membrane peeling and endolaser PRP under local anaesthesia. The surgical procedure was uncomplicated and intra operative haemostasis and adequate laser ablation was possible.

On the first post operative day she was asymptomatic and felt improvement in vision. However fundus examination revealed a clear media, frosting of all retinal vessels, and exudates on the disc. (Fig:1). Lid oedema, pain or discharge were absent.

There was gross deterioration in the clarity of the ocular media on the second postoperative day. B Scan USG showed plenty of moderately reflective point echoes in the vitreous cavity. Slitlamp biomicroscopy revealed moderate flare and cells.Vitreous biopsy as well as AC tap was performed. Gram stain and KOH mount as well as bacterial and fungal cultures were negative.


Fig. 1. Preoperative


Fig. 2. Postoperative: Day One


Fig. 3. Postoperative: Day Fifteen

The patient was started on parenteral steroids under supervision of her physician The fundus appearance after 15 days showed remarkable improvement

Dr. Giridhar

A 60 year old female with Diabetes Mellitus underwent an uncomplicated Vitrectomy for non-resolving vitreous haemorrhage in the left eye. No intra ocular tamponade was used.

Post operatively she had a retinal vasculitis resembling frosted angitis within 24 hours. This progressed to an inflammatory reaction involving both the anterior and posterior segment within the next 24 hours. There was no hypopyon as per the case history. Patient responded to a course of systemic steroid with improvement in media clarity. However, I notice few patches of inflammation inferior to the optic disc and I also notice that the retinal vasculature is not very clearly noticed in the final photograph after steroid therapy meaning thereby that the recovery is not complete. The case summary does not mention the final visual acuity after steroid therapy and also it does not mention whether there was any worsening after cessation of steroid therapy.

Comments

Immediate fibrinous reaction following vitreous surgery could be either a sterile inflammation or an infective endophthalmitis. Retinal vasculitis as a presenting sign of postoperative inflammation is quite uncommon. Packer[1] et al have reported 3 cases of postoperative bacterial endophthalmitis presenting as retinal periphlebitis. One case was following vitrectomy for dense vitreous haemorrhage. Staphylococcus organism was isolated from the vitreous samples.

There are also reports of retinal periphlebitis as the presenting sign of postoperative infective endophthalmitis in animal studies. In an experimental primate model of bacterial endophthalmitis retinal vasculitis developed early in the disease.

In this particular case since there was response to oral steroids it could be a sterile reaction. Patnaik[2] et al have reported severe sterile vitritis due to bacterial endotoxins from irrigating solutions. However he had a series of cases with a similar clinical picture. We have had severe sterile endophthalmitis following vitrectomy couple of years ago. During that period we used formalin vapour as the method of sterilisation of many instruments like Endoilluminator which cannot be autoclaved. We felt that the sterile reaction could be due to formalin vapour inside the eye. We therefore discontinued the procedure and resorted to EO sterilisation and since then we had no instances of sterile postoperative reaction.

The case summary does not give the complete details following systemic steroid therapy. However, I feel, a vitreous tap is worthwhile to look for the presence of any low virulent pathogens and treat according to sensitivity with antibiotics similar to post cataract intra ocular infection.

Summarising postoperative intra ocular inflammation following vitreous surgery is rare. The incidence of postoperative infective endophthalmitis following vitreous surgery is around 0.039%. Sterile inflammation could be due to toxins from the irrigating fluid and/or excess EO gas or formalin from intra ocular instruments. Presence of retinal vascular involvement as an initial sign need not necessarily mean that it is sterile and there are reports of infective etiology in such cases. In any such situation therefore it may be safer to remove an intra ocular sample for microbiological investigation before initiating therapy.

Dr. Gopal S. Pillai

To start with, the patient is a diabetic and a hypertensive for the last 15 years and has an unoperable TRD in one eye. Usually for this much of change to occur, the patient has to progress through no DR through mild, moderate, severe, very severe NPDR and then PDR. This usually takes about 25 years in the patient's diabetic life. Since she developed an unoperable TRD in one eye within 15 years of diabetes, I presume that her control was not proper. That means that she has gone through most of her diabetic life with unchecked diabetes and hypertension.

Since she was one eyed at the time of presentation having only HM vision in both eyes, and the subhyaloid hemorrhage was more than 4 months in duration, the decision to do a vitrectomy is absolutely justified. We should not be looking for alternate treatments like YAG hyaloidotomy just for the simple reason that she needs

vision and the surgeon need to put in laser photocoagulation for her, both of which would not be possible immediately following a YAG hyaloidotomy as there will be dispersed vitreous hemorrhage immediately.

Post surgery, the picture shows a clean macula and rest of retina, the optic nerve head and the vessels show frosted appearance which is suggestive of two differential diagnosis, either a spasm or closure of the vascular arcade or development of vasculitis. The presence of moderate flare and cells during initial post operative phases is normal, because of dispersed RBCs and WBCs which will be there even following a cataract surgery, let alone a vitrectomy. Media haze can increase in day 2 or 3 in a vitrectomy when the patient becomes more ambulant and the remaining blood along the periphery gets dispersed into the aqueous that now fills the vitreous chamber.

Spasm and or closure of vessels have been described in post operative cases in diabetic vitrectomy before, but most of them proceed towards gross ischemia, and neovascular glaucoma. This may be a prominent feature in some cases where there is a carotid blockage associated with diabetes and hypertension. How ever this may also be occuring as a spectrum where some cases develop reperfusion and others go in for capillary closure and neovascular glaucoma. So this one may have been the favourable case which reperfused.

When we think of the causes of vasculitis in a 60 year old lady with diabetes and hypoertension who never had vascuitis before, our differentials will finally corner around infectious endophthalmitis only. We might not be justified in adding another primary vasculitis into our primary diagnosis because of the setting and the age. However another problem exist in diagnosing infectious post operative endophthalmitis, ie, even without antibiotics, only with steroids it improved.

More over, after we gave systemic steroids, the patient responded and within 15 days, the picture shows no trace of vascular cuffing in figure 3. An infectious endophthalmitis with a frosted vascular arcade would improve in media clarity in 15 days, but it will take much more time for the vascular restoration to occur and for the cuffings to disappear.

Another factor is that for an endophthalmitis to occur after a vitrectomy, the organism should be very virulent and most cases of post vitrectomy endophthalmitis do poorly. In our case, the scene showed complete reversal in 15 days, even without antibiotics, and so my diagnosis moves farther and farther away from endophthalmitis.

A fluorescein angiography at that stage would have differentiated between the two diagnosis, becasue in a vasculitis,there would be active leakage leakage from the vessls, in a spasm or blockade there will be paucity of circulation with delayed arm retinal and AV circulation time. But I understand that the first post operative day is not a good time to suggest a fluorescein angiography to any patient.

Thus I feel that the diagnosis in this case was a reversible circulatory spasm which got relieved with time.

There are a few surgical factors which I would like to consider in doing a diabetic vitrectomy. Diabetic optic nerves are very labile and can develop AION, CRVO or arterial spasm or a combination of the three following any procedure to it. I have had cases of AION developing after a sitting of panretinal photocoagulation or after a diabetic vitrectomy. So that much extra care should be taken to prevent these complications. The factors which we should always consider during a diabetic vitrectomy to prevent such circulatory disturbances are

Bottle height The bottle height determines how much pressure you are exerting on the optic nerve head. This pressure should be kept at less than 40 cms if we are using hydrolic systems and less than 30 mm of Hg if we are using the VGFI systems.

Tamponade Even if we increase the bottle height or pressure in between surgery for tamponade, we should immediately bring it down to normal after hemostasis is achieved.

Air pressure Some times we do air fluid exchange to end the surgery. Again care has to be taken to ensure that air pressure does not rise above 40 mm of Hg.

Duration of surgery Longer duration of surgery may induce circulatiory disturbances in a diabetic vitrectomy. So preferably doit within 1 to 2 hours. Otherwise we may achieve good anatomical results, but poor functional results.

Diathermy Never diathermise very close to the optic nerve even though there is a long frond attached to the disc .Never pull on the frond attached to the optic nerve head. Chances of no PL following surgery increase with these highly risky manuevers.

Laser photocoagulation Should be light and not close to the posterior pole. It may induce AIONs or CME which may undergo macular scarring. A grade 3 burn in the endophotocoagulation system will actually become grade 5 post operatively. So aim for light grade 2 burns away from the posterior pole and grade 1 burns near the posterior pole.

Closing pressure Should not be more than 20 to 25 mm of Hg. It should not be too low, lest there be rebleed.

Postoperative IOP spikes Should be recognised and treated early as post operative glaucoma also decreases optic nerve head perfusion.

Thus in this case, it may be a combination of any of these factors which caused the initial closure of vessels which was reversible. We might have to scrutinise each of these factors before we reach a final conclusion of the cause of this frosted vasculature. The lady's history of having hypertension also gives leverage to this postulation.

Dr. T.P. Ittyerah

The case presented is unique & I don't have a definite answer to it. It looks like early endophthalmitis & even if it is not it is better to treat as endophthalmitis. Vitreous tap, culture, intravitreal antibiotics, etc. should be given. Use of steroid may be postponed till the culture report is obtained & can be given if there is no fungus.

Dr. Roy Mathew Zachariah

A frosted picture of the retinal blood vessels is a sign of acute intraocular inflammation. Since this happened immediately following a vitreoretinal surgical procedure, the first possibility is an intraoperative factor. Samples of fluid from the vitreous cavity as well as from the anterior chamber have been evaluated, Gram's stain and KOH mount, and cultures have come negative.

Dr. Thomas Cherian

I would suggest that the left over irrigating fluid, if any (if not available, a sample from the same batch of fluid), could also be subjected to lab testing, both for cultures as well as pH analysis. Alterations in pH of the irrigating solutions are known to cause corneal oedema following phacoemulsification procedures.

Parenteral corticosteroids seem to be the only feasible treatment, this patient has responded to this. However, a workup of the patient, for Viral antibodies, including HSV 1 & 2 and VZV would complete the evaluation.

Editors Note

Post operative intra ocular inflammation following vitreous surgery is rare. Endophthalmitis following vitrectomy has a reported incidence of 0.40%. Sterile intraocular inflammation following vitrectomy is however uncommon, occurring in 1 in 1000 cases and usually exhibiting a clustering of cases.

This 60 year old one eyed, poorly controlled diabetic presented on the first post operative day following an uneventful pars plana vitrectomy with frosted retinal vaculitis. Although the ocular media was clear initially, it deteriorated rapidly and the vitreous cavity became filled with inflammatory exudates percluding all fundus view from the third post operative day onwards. A suspicion of infective endophthalmitis was entertained, Vitreous biopsy, and AC tap were performed. Gram staining, KOH mount, bacterial and fungal cultures were negative.

Intensive Parenteral steroids were started under the supervision of her attending physician. Although there was not much response in the initial 2 days of starting steroid therapy, dramatic improvement occurred in the 2^{nd} week with clearing of ocular media and improvement in visual acuity to 6/18 the 15^{th} post operative day.

Retinal Vaculitis as the presenting sign of post operative inflammation is quiet uncommon. Review of literature gives few reports on this uncommon presentation. Packer et al have reported a series of three cases of infective culture proven endophthalmitis presenting initially as retinal vaculitis. Retinal vaculitis as the initial presentation of endophthalmitis has been demonstrated in animal models.

In this patient, dramatic response to steroid therapy and a negative smear and culture results (Vitreous biopsy results) could point towards sterile post operative inflammation.

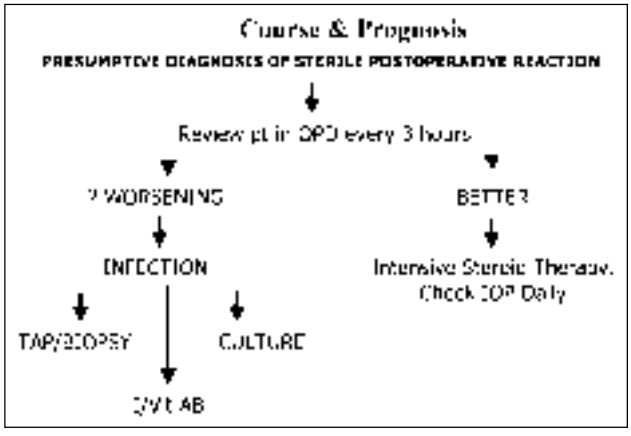
Sterile Inflammation following surgery could be due to a myriad of causes, some of which are listed in Table 1.

Table 1: Proposed Aetiological Factors for Sterile Post Operative Inflammation.

- Intra Ocular Irrigation Fluids
  (pH; Osmolarity, Particle Contamination)
- Intra Ocular Antibiotics
  (Concentration, Preservatives)
- Endotoxins
  (G-; Resistant to Autoclaving)
- Residence
  (Gas, Formalin, VE; Detergent)

There are certain definite characteristics which helps to distinguish a sterile post operative reaction from infective endophthalmitis. Early onset, characteristic limbus to limbus corneal oedema, severity of intraocular inflammation with excessive fibrin reaction in the anterior segment and vitreous cavity, formation of hypopyon, fixed dilated pupil and early high rise of intra ocular pressure are the main differentiating features. However a suspicion of infective aetiology should be borne in mind and appropriate investigative modalities should be carried out. (Table 2)

Table 2



The prognosis depends on severity of the inflammation. The recovery is delayed in cases leaving behind sequlae like a corneal decompensation, cystoid macular oedema, permanent trabecular damage, retinal detachment and phthisis bulbi.

Prevention of sterile reaction in the post operative period can be accomplished by the following measures.

- Periodic Review of Sterilisation Process
- Clean & Dry before Autoclaving
- Use Disposable Tubings
- Check Phaco for Backflush
- Use Preservative Free Agents
- Use BSS at Room Temperature
- Check pH & Ionic Concentrations
- Avoid Denatured VE

It is to be borne in mind that when ever unusually severe post operative inflammation is encountered or when there are clustering of cases with inflammation a suspicion of either infective or sterile reaction should be considered. Distinct differentiating features and results of investigative modalities like vitreous biopsy and culture as well as response to treatment will help differentiate these two entities. In the presence of unexplained post operative inflammation it is mandatory to take samples of all materials used in the OT and subject them to culture. Performing this exercise alone will help identify the source of contamination.

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Compiled by **Dr. Meena Chakrabarti,** Editor, KJO

Step by step small incision cataract surgery

Editors: Anita Panda & Tanuj Dada. Jaypee Brothers, New Delhi

There are a number of books in the step by step series published by the Jaypee Brothers. The volume on small incision cataract surgery is a compilation of articles dealing with each step of the entire process of small incision cataract surgery in a concise and well-prepared manner. Each topic is covered from various angles and finally the author's preferences are clearly stated.

The book starts with a well-argued case for the need for SICS in the present day scenario. Special emphasis on the low cost of SICS in high volume set up is highlighted with a number of references. Instrumentation for SICS has been covered in detail along with plenty of good quality photographs of relevant instruments.

The procedure per se has been covered in detail by allocating chapters for patient preparation and anaesthesia, incisions, anterior capsulotomy, hydro procedures, nucleus delivery, cortical aspiration and IOLs. All the current techniques in vogue for each of the steps mentioned above are discussed in detail. Prevention and management of complications and postoperative infections are also covered in separate chapters. The book is accompanied by 2 very informative interactive CDs. Each step of the SICS procedure is demonstrated clearly and in a simple manner. This book is extremely useful for surgeons starting to do SICS and also for practitioners already doing SICS but looking to widen then repertoire of techniques.

Step by step Neuro ophthalmology-clinical examination and diagnosis

Editors: Satya Karna. Jaypee Brothers, New Delhi

Neuro ophthalmology is a confusing subject to say the least. When you read the textbooks with total concentration, the subject appears extremely logical and hence, like unraveling a ball of string, if you follow the thought through from beginning to end, i.e. from the eye to the occipital cortex everything is crystal clear for a moment. The difficulty is in holding on to the thought… because once your attention wanders the thought blurs around the edges. Soon it becomes nebulous and in no time vanishes into nothingness! This in effect sums up my concept of Neuro ophthalmology. This is precisely where this book comes in handy. Most of the books on Neuro ophthalmology are voluminous tomes with lot of unnecessary details as far as clinical practice is concerned. If you have this book handy in your clinic the moment a Neuro ophthalmic case walks into your OP, you can work up the case as if by a set protocol. Undoubtedly a lot of hard work has gone into making this book, which makes the Neuro ophthalmology workup look ridiculously easy. If you

are looking for in-depth analysis this is the wrong place to look for it.

All aspects of a neurophthalmology work up from history taking; basic examinations, special examinations and investigations are covered in separate sections with separate chapters, denoted for further sub divisions. All common disorders seen in such cases are dealt with comprehensively in a separate section. The last section is denoted to a self-evaluation through MCQs. The questions are based on the text and are easy once you've read the book through.

Overall a book which is handy for the post graduate students especially for their practical exams and for the average ophthalmologist who is not dealing with neurophthalmology cases on a day to day basis but would like to work up the case adequately instead of hurriedly referring the patient to a neurophysician or neurosurgeon. This book is accompanied by 2 interactive CD ROMS with excellent videos demonstrating simple and complex clinical tests used in the assessment of a neurophthalmology case. Definitely value for money for both students and the practicing clinician.

Contributed by **Dr. Merine Paul,** Consultant Opthalmologist, Vijayashree Eye Hospital, Thrissur

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Trabeculectomy with internal Tube Shunt - A novel Glaucoma surgery

Michael E Yablonski. Journal of Glaucoma 2005; Volume 14, Number 2:91-97.

This is a pilot study evaluating the efficacy and mechanism of action of a new glaucoma operation, trabeculectomy with internal tube shunt. Here in this article the author evaluates twenty-three patients who underwent this new operation in one eye for open angle glaucoma.

Under a scleral flap, a deep sclerectomy was performed, resulting in an intrascleral lake. Laterally, on both sides a small silicone tube was placed between the Intrascleral lake and the suprachoroidal space. A trabeculectomy stoma and a peripheral iridectomy permitted easy access of aqueous to the tube. Post operative and preoperative results were analyzed and also compared with results of 45 eyes that underwent a conventional trabeculectomy. After a mean follow up time of 324 days, the mean postoperative IOP was 13.8 mm of Hg compared with a preoperative value of 25.4 mm of Hg. The mean number of postoperative medications was only 1.1 compared with a preoperative value of 3.0. No significant change in outflow facility was seen.

This study concludes that trabeculectomy with internal tube shunt is very effective in lowering IOP. It is postulated to work, to a large extent by allowing access of the aqueous humor to the suprachoroidal space where the protein colloid osmotic pressure of uveal blood causes its absorption.

Diode Laser Transscleral Cyclophotocoagulation as Primary Surgical Treatment for Medically Uncontrolled Chronic Angle Closure Glaucoma, Long-term clinical outcomes

Jimmy C.H. et al. Journal of Glaucoma 2005; Volume14, Number 2:114-119.

This study reports the long term efficacy and safety of Diode Laser Transscleral Cyclophotocoagulation as primary surgical treatment of medically uncontrolled chronic angle closure glaucoma. Thirteen eyes of 13 Chinese patients with medically uncontrolled chronic angle closure glaucoma were treated with Diode Laser Transscleral Cyclophotocoagulation between February 2000 and May 2001, and followed up for over 18 months. Post treatment anti-glaucoma medications were adjusted according to intraocular pressure. If intraocular pressure remained above 21 mm of Hg despite medications for more than 4 weeks after Cyclophotocoagulation, the procedure was repeated.

The mean follow up of the study was +/-SD was 26.5 +/- 4.2 months. Two eyes required repeat Cyclophotocoagulation at 6 weeks. Rate of relative success, defined as maintaining an intraocular pressure of 21 mm of Hg or below with or without medications,

was 92.3%. Rate of absolute success, defined as maintaining an intraocular pressure of 21 mm of Hg or below without medications, was 0%. Mean +/- SD Intraocular pressure was reduced from 36.4 +/- 12.6 mm of Hg pre-operatively, to 18.7 +/- 12.2 mm of Hg at final follow up. The visual acuity improved after treatment in 2 of 13 eyes, remained unchanged in 6 of 13 eyes and deteriorated in 5 of 13 eyes. No major complications were encountered.

Diode laser photocoagulation appeared to be an effective and safe primary surgical treatment of medically uncontrolled chronic angle closure glaucoma, with IOP lowering effect persisting up to two years.

Intravitreal Triamcinolone Acetonide for Diabetic Macular edema

Janet J Chieh et al. Retina 2005; Volume 25, Number 7:828-834.

This paper evaluates the clinical outcome of an Intravitreal injection of Triamcinolone Acetonide as treatment for diffuse diabetic macular edema. This study was conducted as a retrospective, interventional, clinical case series examining 210 eyes of 174 patients who received an Intravitreal injection of 1 or 4 mg of Triamcinolone Acetonide for treatment of diffuse diabetic macular edema. Inclusion criteria were clinically significant macular edema, visual acuity loss, and leakage shown by flourescein angiography. Main outcome measures were visual acuity and Intraocular pressure. Mean follow up time +/-SD was 6.6 +/- 3.1 months.

In the study group, visual acuity improved significantly from a median of 20/200 at baseline to 20/80 at 6 months. Mean IOP +/- SD increased from 15.4+/- 3.4 mm of Hg to a maximal value of 20.4 +/- 6.2 mm of Hg during the follow up period. Complications included culture negative sterile endophthalmitis in six cases and cataract extraction in 5 cases.

This study concludes by saying that Intravitreal injection of 1-4 mg of Triamcinolone Acetonide may benefit patients by improving visual acuity in eyes with clinically significant diabetic macular edema. This study did not provide significant evidence to justify its routine use in clinical practice for all patients with macular edema. A randomized clinical trial on this issue would provide more conclusive evidence and help identify those patients most likely to benefit from Intravitreal Triamcinolone Acetonide.

Effect of Cataract Extraction on the Visual Fields of Patients With Glaucoma

Monica M. Carrillo et al. Archives of Ophthalmology. July 2005;'Volume 123, Number 7:929-932.

This study was to investigate the effect of cataract extraction on the visual fields of patients with open angle Glaucoma. Patients in this prospective cohort study in a tertiary center underwent standard automated perimetry every 6 months. The authors compared the mean results of 2 examinations immediately before and 2 examinations immediately after Phacoemulsification cataract extraction and IOL implant and the mean results of the first 2 and last 2 examinations from 4 consecutive examinations obtained more than 1 year after the cataract surgery.

The study included 34 eyes of 26 patients. While the mean log-MAR best corrected visual acuity improved significantly by approximately 2 Snellen lines after

surgery, the average change in mean deviation in both the effect and control analysis was less than 0.1 dB and not statistically significant. There was a strong correlation between change in foveal sensitivity and change in mean deviation in the effect analysis but not in the control analysis. There was no relationship between change in visual acuity or initial mean deviation and change in mean deviation in either analysis. This study finds that, while there was an improvement in best-corrected visual acuity after cataract surgery, the changes in the visual field as a group were negligible.

Contributed by **Dr. Radha Ramanan,** Little Flower Hospital, Angamaly

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Contact: Dr. Anup Chirayath

P.B. No. 120, Palakkad, Kerala

Phone: 91-4923-235999

E-mail: marketing@afeh.org

April 9th 2006

CME on Glaucoma in association with Glaucoma Society of India

Organized by Al Salama Eye Hospital, Perinthalmanna, Malappuram

Venue :- Fortune Hotel Calicut

Contact :- Dr.Muhammed Swadique

Phone No 04933 – 225524

April 9th 2006

C M E on Updates in Ophthalmic Surgery
under the aegis of T O C & K S O S

Organized by Chaithanya Eye Hospital, Trivandrum

Contact :- Dr K G R Nair

Phone No. 0471 – 2447183

April 30th 2006

C M E on Phaco Today - 2006 under the aegis of K S O S

Organized by L. F Hospital Angamaly

Contact : Dr. Freddy T Simon

Phone :- 0484-2452546

7th May May 2006

Ananthanayanam – 2006 CME-'Ophthalmology Today & Tomorrow' Under the aegis of TOC & KSOS

Organized by : RIO, Trivandrum

Contact : Dr. Anand H

Phone : 0471-2473307

11th June 2006

CME-Update on corneal diseases

(Chaithanya Academix –Kochi 2006)

Under the aegis of KSOS

Organized by Chaithanya Eye Hospital, Kochi

Contact: Dr.Sasikumar S

Phone: 0484-2357098

18th June 2006

CME 2006 Current Concepts in Ophthalmology
(Under the aegis of KSOS &
Trivandrum Ophthalmic Club)

Organized by Chakrabarti Eye Care Centre, Trivandrum

Contact: Dr. Arup Chakrabarti

Phone: 0471-2555530, 98461 10540

E-mail: tvm_meenarup@sancharnet.in

Management of DME (Diabetic Macular Oedema)

ASSESS SYSTEMIC STATUS

- Diabetic Control
- Serum Lipid Profile
- Renal Function
- Diastolic BP

ASSESS MACULA (by FFA)

- FAZ
- Perifoveal Capillary Net
- Leaking Ma's
- CNP / NV

GOLD STANDARD OF Rx

- LASER PHC (ETDRS)
- Direct Rx to Mas
- (50-100mm, 100ms, blanch Microaneurysms)
- Macular Scatter (GRID)

{Areas of diffuse leak, 50-100µ, 100ms, 1-2 burn apart}

PERSISTENT OEDEMA (Post Laser)

- ? Other Causes of oedema
  {VMT, BRVO, post surgical CME, chronic CSR}
- Repeat FFA & OCT
- Confirm -Rx complete
- Confirm > 4m post Rx
- Rule Out ? Macular Ischemia/DME threatening fovea

PPV for DME

Indication
- VMT with / without TRD
- Diffuse DME

Mechanism
- Releives VMT
- Removes Inflammatory Sump in Vitreous

Technique
- ILM Deroofing
- ERM peeling
- S/R Exudates removal
- LASER

POOR PROGNOSTIC INDICATORS

- Waxy Hard Exudates
- Ischaemic Maculopathy
- Cystoid Changes
- Foveal Atrophy
- Chronic foveal detachment

ASSESS OCULAR STATUS

- BCVA, TnA, S/L Biomicroscopy
- FFA
- OCT
- RTA / HRT

ASSESS MACULA (by OCT)

Grade DME —
- mild 200 - 300 µ
- moderate 301- 400 µ
- severe > 400 µ

• Diffuse thickening	(88%)
• CME	47 – 57%
• Serous RD	11-15%
• Post Hyaloidal Traction	14%

POST LASER FOLLOW UP

- Every 4 months

Additional Rx if DME persistent / threatens fovea

- Peripheral Scatter (HR –PDR)
- Benefits :-
  Risk of MVL 15-30% in 3 yrs
  Chance of Moderate Visual gain 5-17%

NEW TREATMENT APPROACHES

- PARS PLANA VITRECTOMY
- PHARMACOLOGICAL THERAPY
  - CORTICOSTEROIDS (IVT / Peribulbar / Vitreous Insert
  - Oral Protein Kinase Inhibitor
  - IVT inj of VEGF Aptamer (Pegaptanib / Macugen)
  - Growth Hormone Therapy

INTRAVITREAL (IVT) Injection
Triamcinolone Acetonide

- Preservative free (4mg/ml 0.1ml)
- Sterile Precautions / in OT
  Informed Consent Visual blurring / Floaters
- Inj related Complications
  (RD, Endo, VH)
  Drug Related Complication
  (Cataract, Glaucoma & Pseudoendophthalmitis)

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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 I" 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.

1. **Title**: The title of the work must be brief and precise. It should not exceed two lines and 40 characters (including comma, period) Author (s) full name (s) must be given along with his (their) degree and the affiliations. Corresponding author's name, correct address (including e-mail and Fax, if available) and phone number must be mentioned at the bottom left hand corner of the first page.

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

4. **Method**: Give in detail the materials used and the methods employed. Describe the type of study. Pharmacological names only must be mentioned for the drugs used and, if proprietary name is used, then the manufacturers name must be given in parentheses. Except for standard, well-accepted abbreviations (Including SI Units), all others must be introduced in parentheses when the full term is used for the first time in the article.

5. **Results**: Give only the results obtained by the study under discussion. State the statistics in the correct scientific form (P value, mean etc). Results based on assumptions must not be given. Indicate in the text the place where the tables have to be inserted

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.

7. **Acknowledgement**: This is to be made only to those who were directly and scientifically involved with the preparation of the paper. Permitting authorities, technicians, photographers who assisted in the work need not be mentioned.

8. **References**: The references should be given in numerical order in which they first appear in text and not in alphabetical order (Citation Order System). It should be numbered consecutively in the text. The references will not be checked by the Editor or by the Peer reviewer and hence the author is solely responsible for its completeness and the accuracy. Period should not be employed anywhere in the references. Personal communication, unpublished data and poster references, if mentioned, should be in the text itself and the source mentioned in parentheses. References should be in the following form:-

Journal reference: - Author(s) full title, Journal name (as abbreviated in Index Medicus), volume number, pages and year. If there are more than three authors, then mention the first three authors and then 'et al'.

Book reference: Authors(s) (& Editor, if any), title of book (and chapter), publisher, place of publication, page number (s) of the cited portion and year.

9. **The Legend**: The legend for the illustrations (and tables, if necessary) must be given in a separate sheet of paper and should be typed double-spaced.

Illustrations: The photos and figures should be prepared in glossy prints with good contrast and of the size 6" x 4". Only salient details should be included. On the back of the illustration, the figure number in text, title of the paper, the first author's name and the top side (marked with an arrow) must be specified. Except for arrows, no text is to be on the photos. It is the duty of the author(s) to get the patient's written permission when the subject is identifiable in the photo. Submit two sets of illustrations. Illustrations from other Journals and books are usually not accepted. If used, it rests with the author(s) to get the copy right permission from the original author / publisher and this permission letter must be sent to the Editor at the time of submitting the manuscript. For Histological figures the stain and magnification used should be noted e.g.: - H & E Stain x 70.

10. **Table**: It should be in double space. Each table must have an Arabic numeral (except for single table) and a title both in a single line. Each column in the table must have a short heading. If a table is large, then it must be continued in a second page, which also must have the table number and the title. Avoid vertical lines in the tables. Two sets must be submitted.

The manuscripts are to be sent to The Editor by Courier Mail or by Registered post. The corresponding author will receive communication from the Editor within two weeks of receiving the manuscript.

11. All manuscripts are subjected to editorial board review.

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b) Review Articles: can be on topics of relevance to clinical practice, research methodology, community ophthalmology or investigative work, of relevance to visual science. These articles should include up to date review of existing literature, and summarize the current status / preferred practice for that particular topic.

c) Brief reports are short communication of new instruments, new laboratory techniques or surgical techniques as well as interesting case reports with unique findings. These should not exceed 1000 words with a maximum of 2 illustrations. They should follow the format - introduction, case, and discussion. No more than 8 references should be cited. Each brief report must begin with a 75-100 word summary that highlights the significance of the articles.

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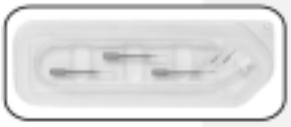


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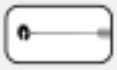
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1281ADO6



1281-BT06



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1281-BTD06



23 GAUGE MEMBRANE INSTRUMENT

1292-E06

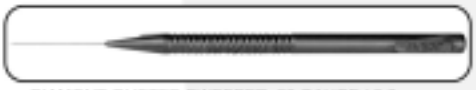


ECKARDT MEMBRANE PIC,23 GAUGE / 0.6 mm.



23 GAUGE DIAMOND DUSTED SWEEPER

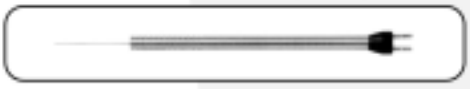
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DIAMOND DUSTED SWEEPER, 23 GAUGE / 0.6 mm.



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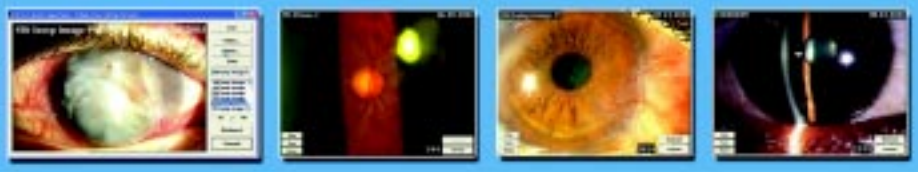
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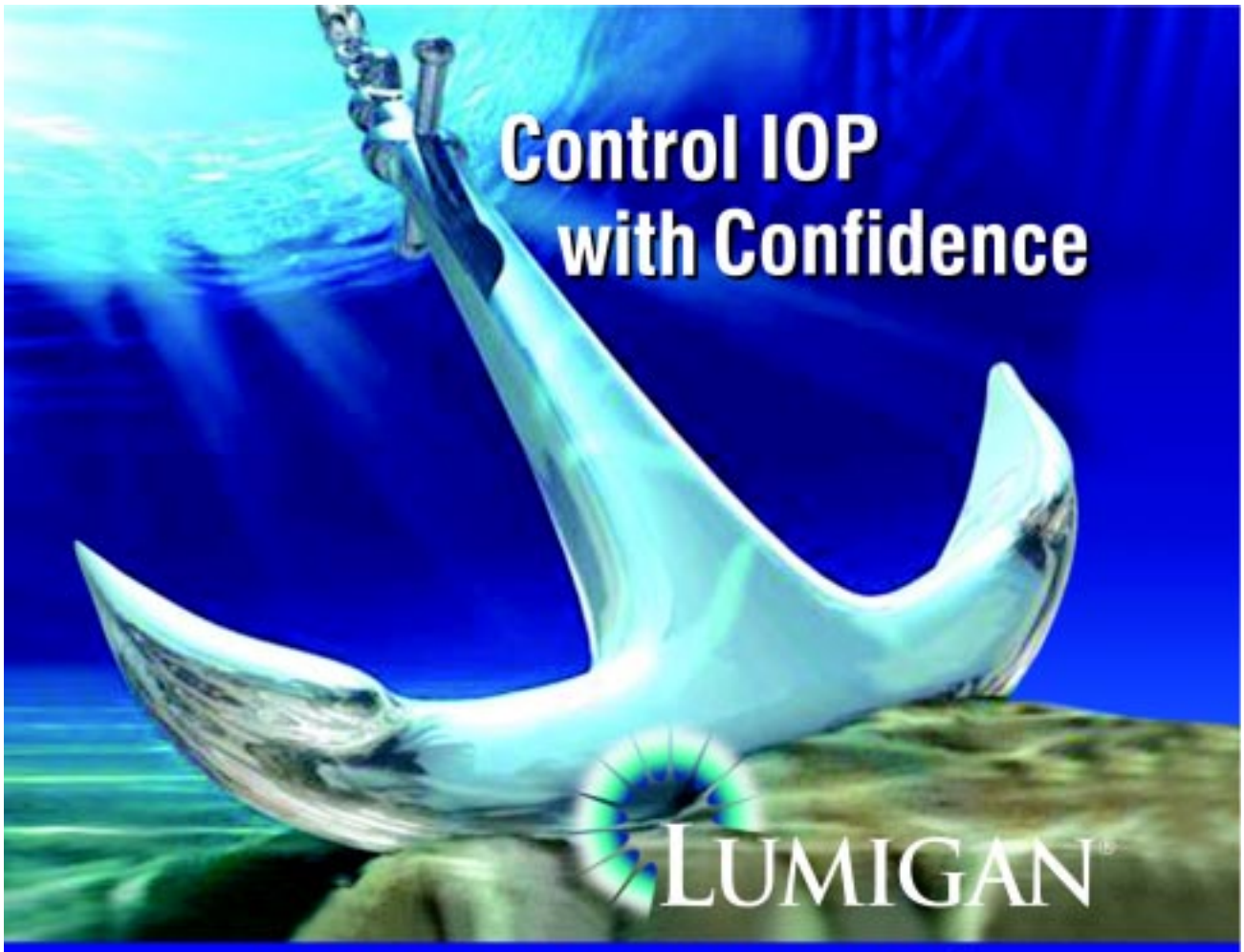
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Noecker: over 6 months, 12 noon time point (N = 289)

vs travoprost[2,3,5,9] **16% to 29%** greater mean IOP reduction than travoprost[3,9]

Cantor: over 6 months, 8 AM time point (N = 20)

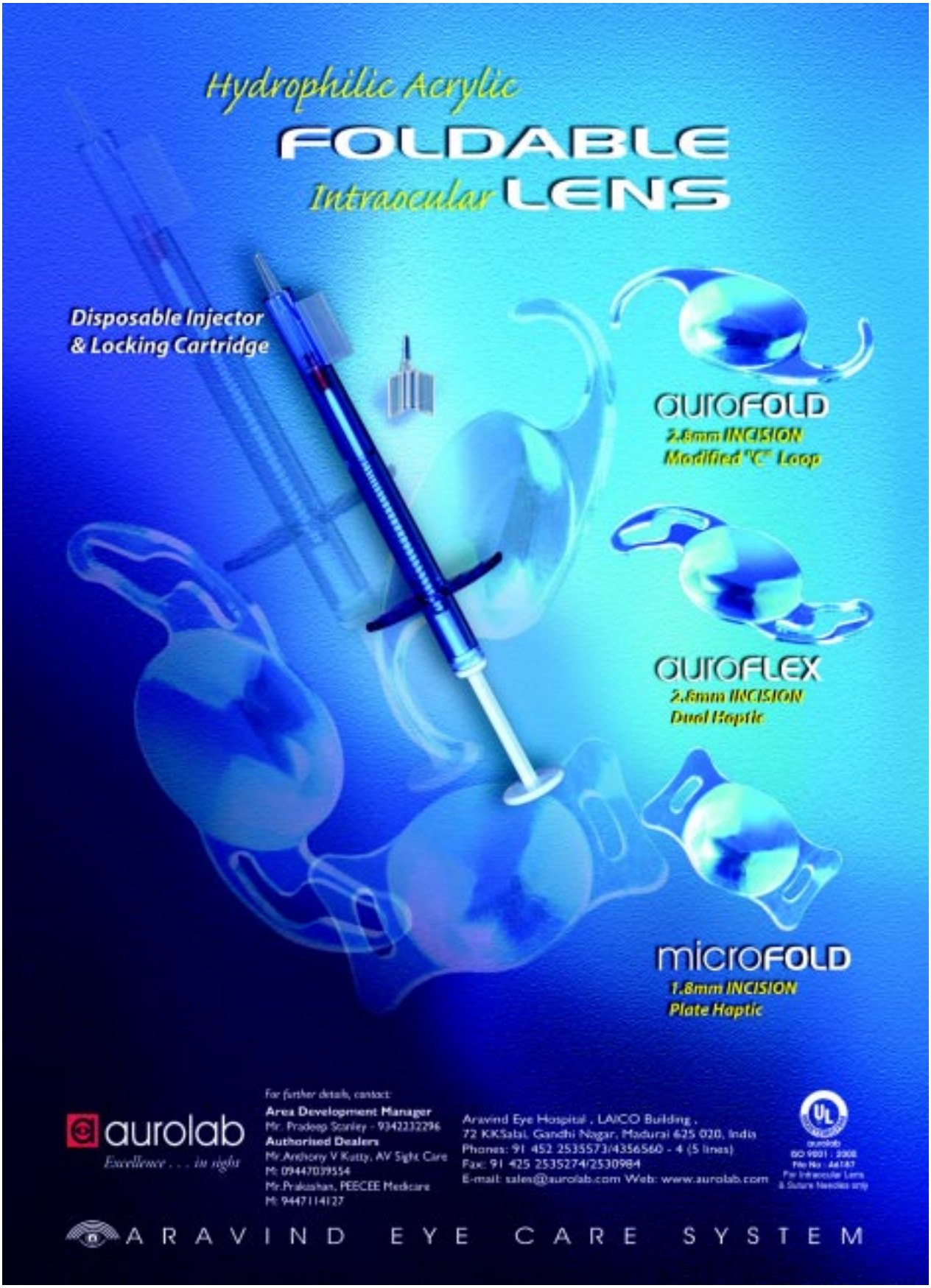
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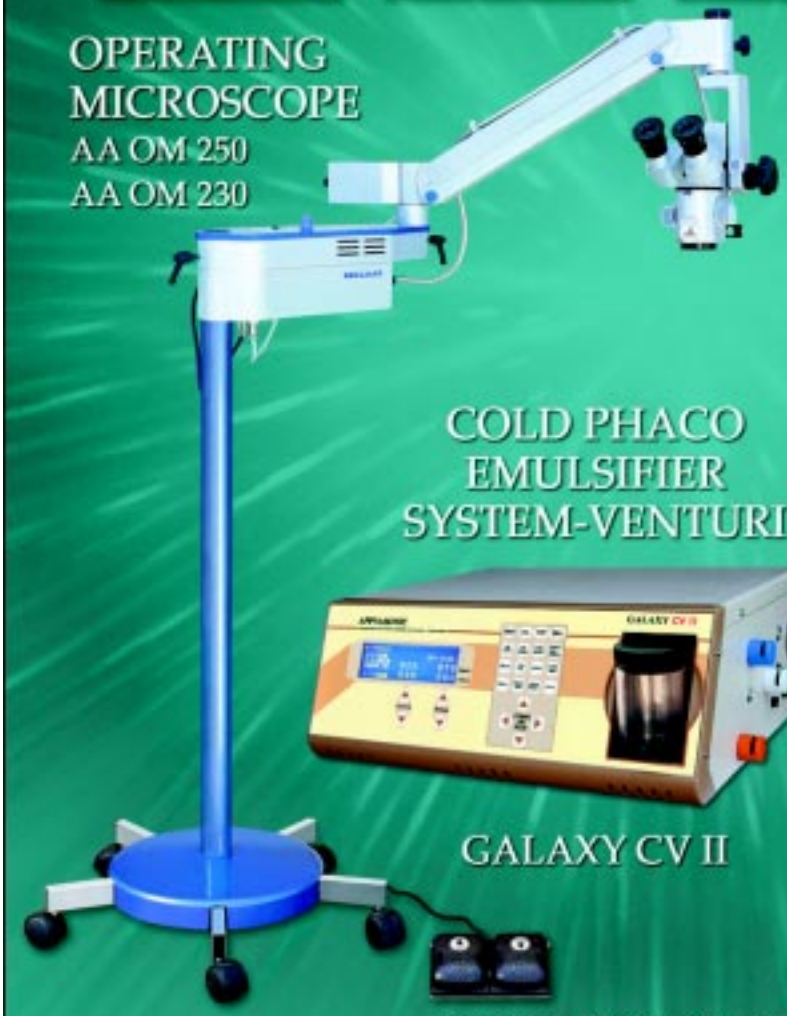
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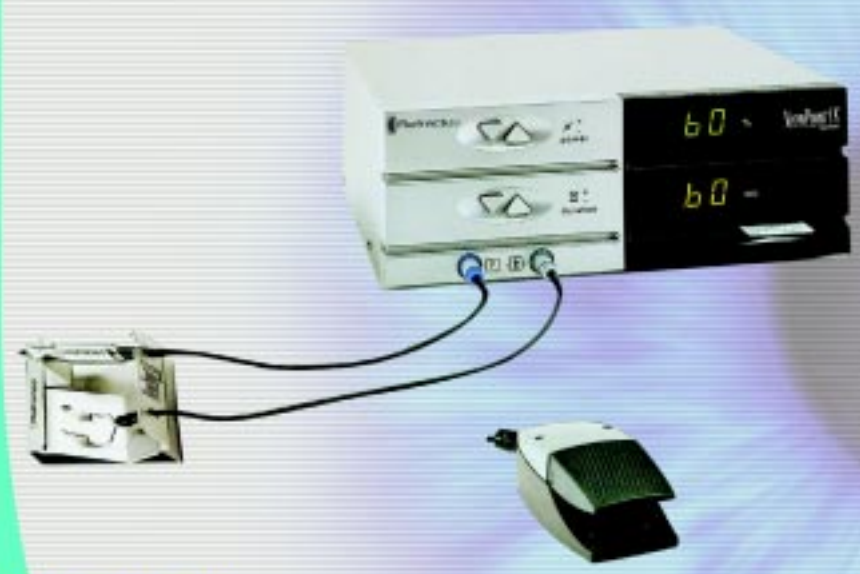
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