ISSN : 2250-1916
2015, Vol 01

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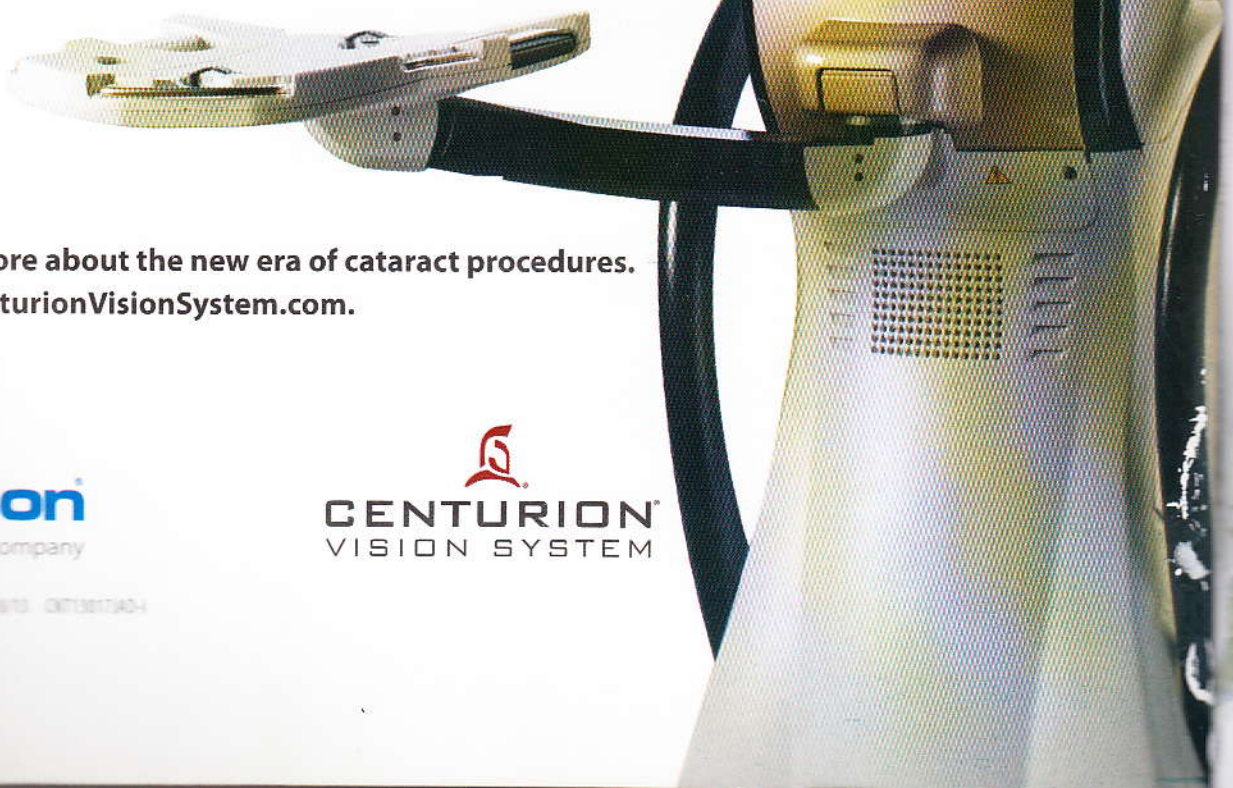

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Guest Editorial

Gone are the days when doctors were worshipped for their noble deeds. Pick any newspaper, you will find more news on violence against doctors than miracles in medical field. Today while the world is struggling with nuclear terrorism, we, the doctors are continuously victimized with medical terrorism. What's medical terrorism? It's any form of verbal abuses, violent act or threats for violent act; by patients or their attendants against health care personnel.

First we need to find out the root cause of this violence. It mainly stems from young aggressive males, drugs and alcohol intoxication, communication gap between doctor and the patient, increased cost of health care, unrealistic expectations of patients, overworked but underpaid health care staff, lack of faith in judiciary, insufficient security in hospitals, no strong laws against people who engage in such violent acts etc. There are certain ways by which we can decrease the incidence of such attacks. Firstly, there is a very famous saying in medical schools that few extra minutes with patient in the examination room can prevent us from lots of trouble after the treatment. There should not be any cutting in communication time. We should clearly explain the patient about prognosis of disease and cost of the treatment. Develop better communication skills so as not to meet the anger with the anger. Training to learn the warning signals of body language that can precede aggressive act. There should not be any cost hike later on without explaining it to the patient. We should try not to charge for the iatrogenic complications. There should be boards in the waiting area displaying information regarding waiting time, hospital charges list, diseases information in layman language, name and contact of local IMA patient grievance redressal cell and most importantly, laws and rules concerned with the medical terrorism. CCTV cameras should be installed with boards clearly mentioning it. There should be a security guard in front of doctor's room. Take written consent with patient that they will not indulge in any violence and doing so should be compensated by them. Identify the trouble makers in the group and be extra cautious with them. Be knowledgeable in your subject and try not to venture out of your area of expertise.

What to do if the violence happens? Inform the police immediately. Take written statements from people who witnessed the incident. Lodge a FIR. Do a press conference. Get it published in newspaper. Contact your local IMA. Insist your state IMA to contact state government and to frame laws for violence against doctors like Punjab Protection of Medicare Service Persons and Medicare Service Institutions (Prevention of Violence and Damage to Property) Act, 2008 and Tamil Nadu Medicare Service Persons and Medicare Service Institutions (Prevention of Violence and Damage or Loss to Property) Act, 2008

Good luck for the elections to be held at Allahabad in November 2015.

Dr. Nisha Chauhan
Dr. Shikha Prakash

JOSEPH NICOLAS BLAISE FORLENZE

Joseph-Nicolas-Blaise Forlenze (3 February 1757 - 22 July 1833), was an Italian ophthalmologist and surgeon, considered one of the most important ophthalmologists between the 18th and the 19th century. He was mostly known in France, during the Napoleonic Empire, for his cataract surgery.

In 1797, he practised an eye surgery at a retirement home in Paris, in the presence of a commission appointed by the Institute, as well as several members of the government, and French and foreign scholars. In 1798, he became surgeon at the Hôtel national des Invalides and the Hôtel-Dieu of Paris, where he made many remarkable interventions.

Forlenze cured the soldiers of Napoleon's army returning from Egypt, affected by serious eye diseases. He healed renowned personalities such as Jean-Étienne-Marie Portalis, minister of Worship, and the poet Ponce Denis Lebrun, removing a cataract which had been present for twelve years from one of his eyes. Lebrun dedicated to him a verse in his ode called Les conquêtes de l'homme sur la nature. Napoleon, with a royal decree, gave him the assignment of "chirurgien oculiste of the lycees, the civil hospices and all the charitable institutions of the departments of the Empire" thus Forlenze was sent into the French provinces to treat eye diseases.

His activity extended to England and Italy, where he performed free surgeries in cities such as Turin and Rome. In Rome, he cured the Cardinal Doria and was publicly honored by Caroline de Bourbon, Duchesse de Berry. His manuscript Considérations sur l'opération de la pupille artificielle (1805) is considered one of the most important medical works of the time. Forlenze died in 22 July 1833, stricken with apoplexy at the "Café de Foy", in Paris, where he spent often his evenings.

Dr. Shephali Jain

This publication has been made possible by grants from
M/s Pharmtak Ophthalmics, Appaswamy, Alcon, Allergan
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Published by Uttar Pradesh State Ophthalmological Society (NOS).

Printed and designed by JADIA GRAPHICS, Agra

Original Article

Epidemiologic and Microbiologic profile of Fungal keratitis in Rohilkhand region

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ABSTRACT

Purpose: To report epidemiologic and microbiologic profile of 106 culture positive cases of fungal keratitis at a teritary centre in Rohilkhand region of Western Uttar Pradesh.

Methods: Clinical and microbiological records of 106 culture positive cases of fungal keratitis reported over a period of 2 years between June 2012 and May 2014 were analyzed. Review of demographic features , clinical course , risk factors and laboratory findings were done retrospectively.

Results: Of the 106 patients, 68 (64%) were males (male:female ratio 1.79:1), 77 (72.6%) were in age group between 20 and 45 (mean age 37.6), 83(78%) were rural based agriculture workers. Ocular trauma was reported in 83 (78.3%) cases. Time of reporting following advent of symptoms ranged from 1.3 to 6.8 weeks (avg. 2.4 weeks). 89 Patients (83.96%) were already on multidrug therapy including steroids and antibiotics at the time of presentation.

Microbiologically, Fusarium species were the predominant isolates in 58 cases (54.7%) followed by Aspergillus in 28(26.4%), Candida in 12(11.3%), 02 cases of Alternaria and Curvularia and solitary cases of Nocardia & Scedosporium .02 strains remained unidentified. Mode of injury had a causal relationship with etiologic agent ; Fusarium with vegetative injuries 36 (62.06%) , Aspergillus with exposure to soil/dust, 17 (60.71%)and candida with prolonged antibiotic and steroid use in 8 (66.6%) eyes. Coexisting ocular surface disorders & systemic diseases were present in 9 (8.4%) & 7 (6.6%) of the fungal keratitis cases , respectively.

Conclusion: Epidemiologic and Microbiologic profile of fungal keratitis has revealed variation inter regionally even in subtropical zone. The study aims at providing epidemiologic and microbiologic data on fungal keratitis in Rohilkhand region of Northern India.

Key Words: Fungal Keratitis, Epidemiology, Etiology

INTRODUCTION

Fungal Keratitis (Keratomycosis or Mycotic Keratitis) refers to a suppurative usually ulcerative mycotic infection of cornea.[2]Accounting for nearly 50% of all cases of suppurative keratitis in the tropical and it poses a major public health challenge. The severity of these infections , late detection, limited treatment options together with a confounding etiological spectrum results in an invariable poor prognosis.

The first reported case of fungal keratitis was by'Leber' in 1879. [10] Since then nearly 105 fungal species classed in 56 genera have been reported to cause keratitis and Oculomycosis.[12] Two basic forms have been recognized due to Filamentous fungi (especially Fusarium and Aspergillus) which commonly occur in tropical and subtropical zone , and due to yeast like and related fungi (particularly Candida) which are the predominant isolates in temperate climates.[25]

Ocular trauma is the key predisposing factor for filamentous fungal infections.[11,13] The usual sufferers are healthy young males engaged in agricultural or outdoor activities. Traumatizing agents of plant or animal origin as even dust particles either directly implant fungal conidia in the corneal stroma or abrade corneal epithelium permitting invasion of exogenous fungi.[25] Environmental factors like temperature, humidity, wind, influence occurrence of fungal keratitis as

also the type and frequency of fungal isolate. Less frequent predisposing factors include immunological incompetence , prior administration of steroids or antibiotics , Ocular surface disorders , systemic diseases like diabetes and use of hydrophilic contact lenses.[11,24] The latter factors are particularly important in keratitis due to Candida albicans and related fungi.

Although specific clinical features are documented for corneal infections due to bacterial, fungal or parasitic agents, accurate diagnosis is commonly elusive due to prolonged use of wrongly administered antimicrobials, steroids or propensity for mixed infections. Delay in accurate diagnosis forestalls a favorable outcome in majority of patients. Information about epidemiological and etiological features of a large series of fungal keratitis from representative geographical region is limited.

This review analyzes epidemiological record and laboratory findings of 106 culture proven cases of fungal keratitis diagnosed at a tertiary care hospital of Rohilkhand region in Northern India over a 2 year period (june2012 to may2014).

MATERIALS AND METHODS

All cases of fungal keratitis identified from the clinical & microbiological records at Rohilkhand Medical College & Hospital between 01 June 2012 and May 31 2014 were retrospectively analyzed. Medical records of patients were reviewed for age, sex, occupational background, mode and month of onset of infection, predisposing risk factors, prior medications, associated systemic illness , clinical course and duration of active disease.

A uniform protocol for laboratory diagnosis comprised of subjecting corneal scrapings taken from the base and advancing edge of the ulcer using a kimura spatula, to Gram stain and 10% potassium hydroxide wet mount (Figures-7). Scraped material was further inoculated on Blood Agar, McConkey's Agar and Sabouraud's Dextrose Agar (SDA) supplemented with 50microgram/ml gentamicin. SDA was kept at ambient temperature and other media were incubated at 370°C. Fungal cultures were followed for 2 weeks before a negative result was declared. Patients with a negative culture from initial specimen or insufficient inoculums underwent repeat scraping/corneal biopsy at a later date if the disease progression so warranted.

A positive culture was defined by (i) a positive smear with fungal elements substantiated by a confirmatory growth of fungus in a culture media (ii) growth of the same fungus on two or more culture media (iii) growth of fungus on at least one medium followed with growth of the same fungus on at least one medium at a subsequent date (Figures-5, 6).

RESULTS

The study included 106 eyes (106 patients). Of these 68(64%) were males and 38(36%) females; Male : female ratio was 1.79:1. Age of patients ranged from 14 to 76 years . Mean Age was 37.6 years. Majority of patients 77(72.6%) were in the productive age group between 20 and 45 years. Occupationally, 83(78%) patients were rural based, agricultural workers of low socio-economic group.

The chief predisposing factor was Trauma, observed in 83(78.3%)patients particularly caused by vegetative matter 42(39.6%), (Table-1). Of particular note was the inadvertent injury caused by sugarcane leaf(12 cases).

The time duration between advent of symptoms / injury and the reporting at our centre varied substantially from 1.3 to 7.4 weeks. Mean reporting time was 2.4 weeks. Majority of patients, i.e. 89 cases (83.96%) were under an empirical multidrug treatment that included broad spectrum antibiotics, steroids, antivirals and unconventional domestic drugs at the time of reporting.

The filamentous fungi dominated the etiological spectrum 92 cases (86.79%)as shown in Table-2.

Of these the majority comprised of Fusarium species, 58 cases (54.72%) followed by Aspergillus, 28 cases (26.41%) and two cases each of Alternaria and Curvularia. Candida infections accounted for 12 cases (11.32%), 9 cases had a history of ocular surface disorders, 7 had coexisting systemic disease including 4 diabetics (Table-1). 13 were on pre-infection long term topical antibiotics and steroids therapy.

Mode of injury had an interesting etiological relationship with vegetative injuries accounting for 36(62.06%) of total 58 Fusarium infections, Soil and dust exposure eyes leading to 17 (60.71%) of 28 Aspergillus infections and long term steroids use in 8 (66.6%) of 12 Candida infections.

Seasonal variation as shown in Figure-1 was more marked with Aspergillus infections, with the majority i.e. 22 cases (78.57%) reported in the warm and humid months of June to September. Infections due to Fusarium and Candida were uniformily distributed throughout the year. This was a statistically significant variation as per chi-square test ($p<0.005$). Diagnostically, the simple test of KOH wet mount was most effective in diagnosing fungal keratitis in the majority, 96(90.56%).

DISCUSSION

Incidence of fungal keratitis may have increased in recent decades as a result of improved clinical awareness, better diagnostic techniques an increased use of topical broad spectrum antibiotics and corticosteroids and cosmetic contact lens wear.Three genera (Fusarium, Aspergillus and Candida) have emerged as important pathogens that cause fungal keratitis world wide.[12]

Successful management of this blinding disorder relates to its early and accurate etiological identification. This is especially so as the prevailing antifungal drugs vary in their clinical efficacy and in- vitro sensitivity depending on the causative fungal strain.

Epidemiological and microbiological data reveals disparagingly dissimilar results not only between temperate and tropical territories but also inter regionally in the same climatic zone.

Isolated case reports even through useful are ineffective in portraying the realistic clinical scenario of a particular region. The present case series of 106 culture proven cases of fungal keratitis reported from Rohilkhand region of western Uttar Pradesh is a pilot effort in reflecting the epidemio-microbiological status of fungal keratitis in this agro-dominated state.

Notable findings of the study include a high risk factor for males ($p<0.0001$); a greater incidence in younger age group of 20-45 years i.e. 77 cases; (72.6%), a greater preponderance for filamentous fungal infections i.e. 92 cases (86.79%) and with vegetative trauma, 36 cases (33.96%) as the commonest mode. A significant association with direct vegetative trauma was noted for Fusarium infections while falling of dust or soil in eyes particularly in hot and humid weather contributed towards corneal infections with aspergillus. Role of pre existing 'Ocular surface disorder', systemic illness and immune suppression was more evident in yeast category of infections.

The largest case series of fungal keratitis are reported from southern India.[13,1]The primacy of Fusarium as the primary etiological agent and trauma as the chief predisposing factor is corroborated by both these studies as well as studies from Ghana, Florida, Paraguay,Nigeria and China.[3,4,5,7,8,9,11,18] A much higher incidence of Aspergillus is reported, however in similar studies in Indian subcontinent, [19], neighboring countries like Srilanka, Bangladesh , Nepal, [15,16,21] and worldover.[2,23] A solitary study in northern India on Pediatric Fungal Keratitis. [14] sites a much higher incidence of Aspergillus (40%) as opposed to Fusarium (10.7%) which shares near equal pathogenecity with Alternaria (10.2%), Curvularia (7.4%) and

Penicillium (7%). A recent study on Fungal Keratitis at Moorfield Hospital, London [22] implicated Candida as the major fungal etiological isolate (60.6%) as compared to the filamentous fungi which accounted for the remaining. Even though Aspergillus is accorded an ubiquitous status with continued detection in cooler climates, unlike Fusarium, [20]. Our study has noted a marked predilection of this isolate for the hot and humid months. The seasonal preponderance of Aspergillus in hot and humid months, while a seemingly constant incidence of Fusarium suggests a variation in etiopathogenesis of these two important genera of filamentous fungi. Equally pertinent is the role of immunosuppressions and ocular surface disorders in Candida infections. A significant number of Candida infections (11.6%) were noted in our case series, is in contra diction to similar studies in South India[13], where the role of Candida as an etiologic agent is reported to be minimal (0.7%). Yeast infections are rarely reported in tropical climates but are common in temperate zones [20,22]. The role of contact lenses as an important cause of fungal keratitis is not substantiated by our study, largely due to a lower socioeconomic group of study population with insignificant contact lens usage. The role of 'trauma' as the primary risk factor for fungal keratitis as reported in many studies,[1,11,13] is well substantiated in our review(Table-1). The high association of Fusarium infections with direct invasive plant trauma is well explained by the fact that Fusarium species constitute important plant pathogens with wide range of plant diseases like 'Crown rot' ,'Head blight' in cereal grains and 'Pokkah boeng' on sugarcane.[24] All 12 cases reported with trauma due to sugarcane leaves , grew Fusarium in culture suggesting high etiological association. The wide spread distribution of Fusarium in tropical regions along with their ability to grow on a wide range of substrates probably explains their high prevalence as causative agents.

The delay in seeking medical help at a tertiary centre(avg. duration 2.4 weeks) and multidrug empirical regime followed by majority of patients(83.9%) at the time of presentation explains poor clinical outcome.

In this report , an attempt has been made to enlist key epidemiological and microbiological features of fungal keratitis in the region to arouse high index of suspicion among treating physicians andenable early initiation of treatment on the basis of established regional epidemio-etiological data. The authors believe that a larger case series encompassing extensive areas of rural and urban India , would be more representative of a factual profile of fungal keratitis.

REFERENCES

1. Srinivasan M, Gonzalis CA, George C et.al. Epidemiology end aetiological diagnosis of corneal ulceration in Madurai , South India. Br J Opthalmol 1997;81 : 965-71.
2. Jones BR, Principles in the management of occulomycosis.xxxi Edward Jackson Memorial Lecture. Am J Opthalmol 1980;90:38-47
3. Leisegang TJ, Forster RK. Spectrum of microbial keratitis in South Florida. Am J Opthalmol 1980; 90: 38-47
4. Cuero RG. Etiological distribution of Fusarium solani and its oppurnistic action related to mycotic keratitis in cali, Colombia. J Clin Microbiol 1980; 12 : 455-461.
5. Houang E, Lam D, Fan D, Seal D. Microbial keratitis in HongKong: relationship to climate , environmental and contact lens disinfection. Trans R Soc Trop Med Hyg 2001;95:361-67.
6. Wilson LA, Ajello L. Agents of oculomycosis: fungal infections of the eye. In: Collior L, Ballows A, Sussman A(eds). Toplay and Wilson's Microbiology and Microbial infections, 9th edn, Vol 4, Medical Mycology (Ajello L , Hay RJ(section edn)). Arnold:London, 1998 ,pp 525-67.
7. Leck AK, Thomas PA, Hagan M,et.al. Aetiology of suppurative corneal ulcers in Ghana and South India, and epidemiology of fungal keratitis. Br J Opthalmol. 2002; 86 : 1211-1215.

8. Mino de, Kaspar H, Zoulek G, Parades ME,et.al. Mycotic Keratitis in Paraguay.Mycoses 1991; 34:251-54.
9. Xie L, Zhong W, shi W et.al. Spectrum of fungal keratitis in North China. Opthalmology. 2006; 113:1943-48.
10. Leber T H. Keratomycosis aspergilliria als ursache von hypopyon keratitis. Graefes Arch Opthalmol 1879;25;285-301.
11. Rosa RH, Miller D, Alfonso EC. The changing spectrum of fungal keratitis in south florida. Opthalmology 1994;101:1005-13.
12. Thomas PA,Mycotic Keratitis. J Madros State Opththalmic Assoc. 1988;24:121-129
13. Gopinath U, Garg P,et.al. The epidemiology features and Laboratory results of fungal keratitis .Cornea.2002 ;21(6):555-559.
14. Panda A, Sharma N, Das G, etal. Mycotic keratitis in a children. Epidemiological and microbiologic evaluation. Cornea 1007;16: 259-9.
15. Dunlop A.A., E.D Wright, S.A. Howlader , I.Nasrul, R. Husain, K.McClellan and F.A.Bilson.1994. Suppurative Corneal ulceration in Bangladesh: a study of 142 Cases, examining the microbiological diagnosis, clinical and epidemiological features of bacterial and fungal keratitis. Aust.N.Z.J. Ophthalmol. 22 :105-110
16. Gonawerdena, S.A.,K.P. Ranasinghe , S.N. Arseculeratne, C.R. Seimon, and L. Ajello.1994. Survey of mycotic and bacterial keratitis in srilanka. Mycopathologia 127:77-81.
17. Gugnani HC , Gupta S, Talwar RS. Role of oppurnistic fungi in ocular infection in Nigeria. Mycopathologia 1978;65 :155-66
18. Hagan m, Wright e, Newman M, etal. Causes of Suppurative keratitis in Ghana. Br J Ophthalmol 1995;79 : 1024-8.
19. Kulshreshtha OP, Bhargava S, Dube MK, Keratomycosis: a report of 23 cases . Indian J Ophthalmol 1973; 21: 51-4
20. Denis m. O'Day . Selection of Appropriate Antifungal thereapy. Cornea ,1987; 6(4) : 238-245
21. Upadhyay MP, Karmacharya PC , Koirala S , Tuladhar N, Bryan LE , Smolin G , etal. Epidemiological characteristics, predisposing factors, and etiologic diagnosis of corneal ulceration in Nepal . Am J Ophthalmol 1991 ;111: 92-9.
22. Gallareta D.J., Stephen j.T., Ramsay A , Dart J.K.G. Fungal keratitis in London . Microbiological and clinical evaluation. Cornea , 2007 ;26.
23. Foster CS. Fungal Keratitis. Infect Dis Clin North Am 1992; 6 : 851-7.
24. Nelson P.e. , Dignani .M. Cecilia , Anaissie E.J. Taxonomy, Biology and clinical aspects of Fusarium Species. Clin. Microbiol. Rev. 1994 , p. 479-504.
25. PA Thomas. Fungal Infection of the Cornea. Eye. 2003; 17 : 852-862.

Table 1: Predisposing Factors of Fungal Keratitis

Risk Factor	No. of Eyes (%)
Trauma	83 (78.3%)
Vegetative matter	42(39.62%)
Industrial	04(3.77%)
Domestic	24(22.64%)
Animal related	13(12.26%)
Severe systemic illness	07(6.60%)
Ocular surgery	02(1.89%)
Dry eye	05(4.71%)
Contact Lens	Nil
Use of Steroids	13(12.26%)
Others	03(2.83%)

Table 2: Organisms Isolated in Fungal Keratitis

Organisms	No of Isolates(%)
Fusarium	58(54.72%)
Aspergillus	28(26.41%)
Candida	12(11.32%)
Alternaria	2(1.88%)
Curvularia	2(1.88%)
Nocardia	01(0.94%)
Scedosporium	01(0.94%)
Others (Not Identified)	02(1.88%)

Figure 1: Seasonal Distribution of fungal isolates

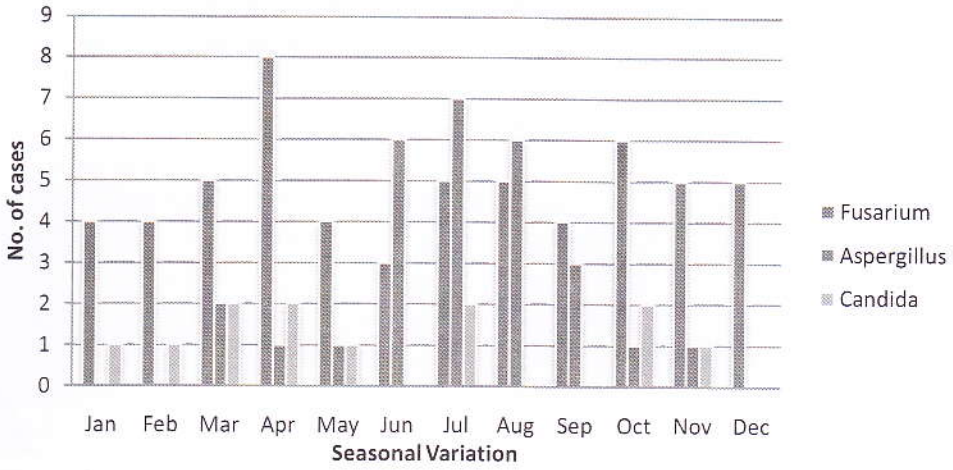


Fig 2 : Aspergillus fungal keratitis

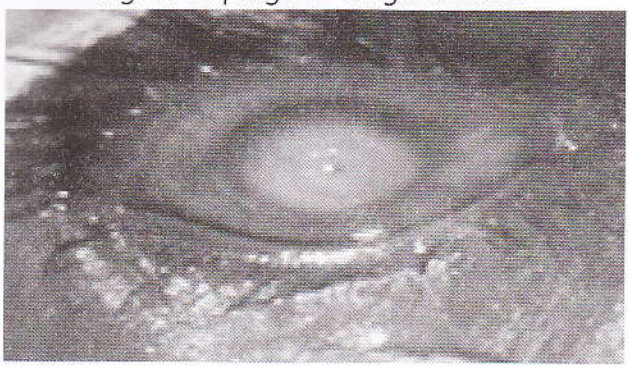


Fig 3: Fungal keratitis due to Fusarium sp.

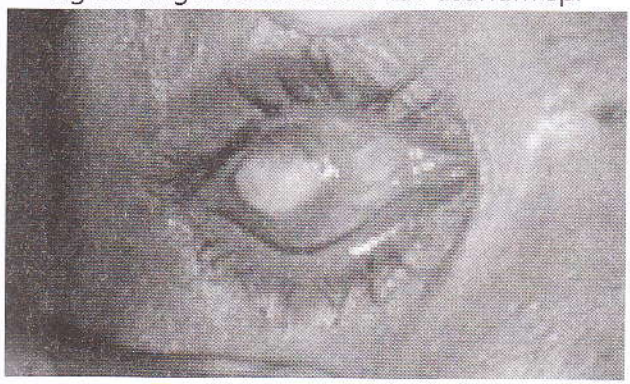


Fig 4:Fungal keratitis by Candida sp.

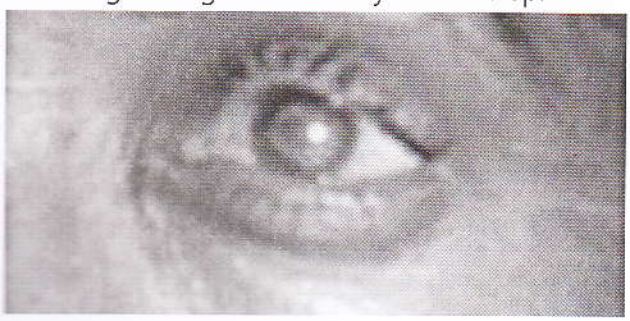


Fig 5: SD Agar showing Aspergillus growth

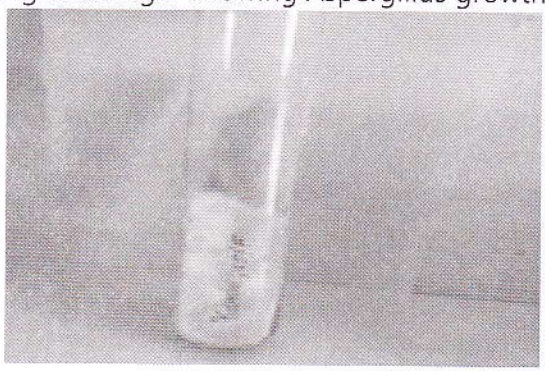


Fig 6: SD Agar showing Fusarium growth

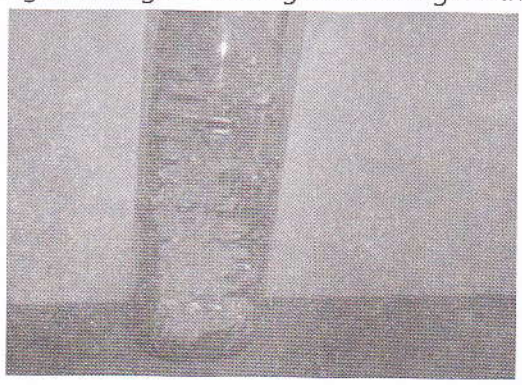
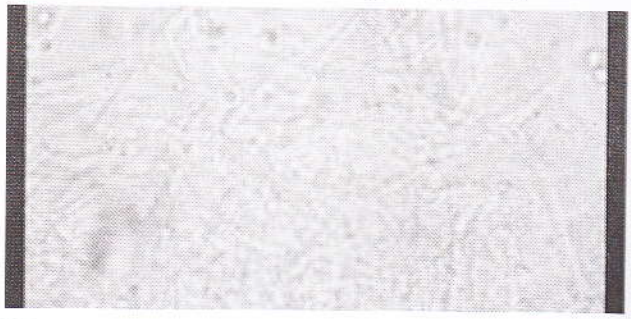


Fig 7: KOH Smear showing Fungal Hyphae



Lipid and Eye

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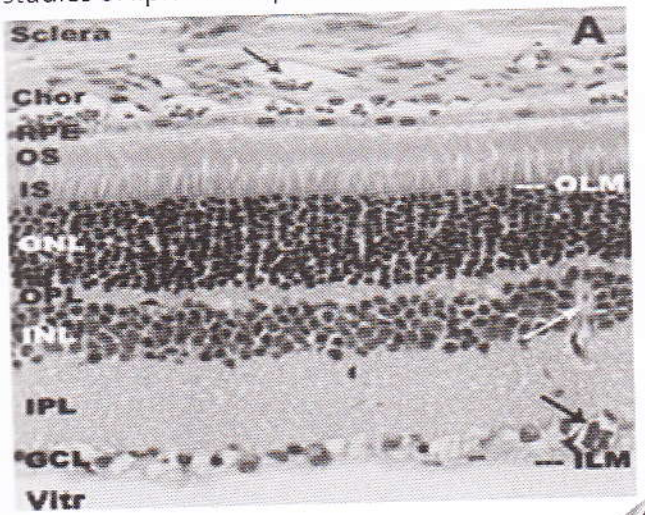
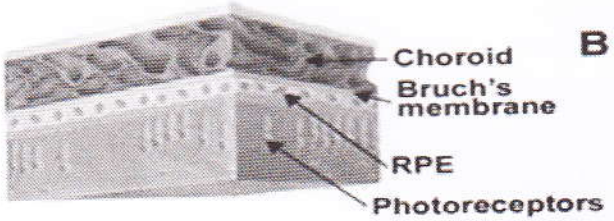
The humaneyeis a complex sensory organ consisting of multiple, distinct tissues, each having its own unique biochemical composition, structure, and physiological function. Key among these are the retina, lens, and cornea, working in concert to bring photons of light into the eye, focus them correctly on the retina, and convert their energy into electrochemical signals that are conveyed to the brain where, ultimately, they are processed into a coherent visual image. Defects in any or all of these tissues, whether inborn or acquired, whether through a disease process or by traumatic injury, can compromise vision and, eventually, may result in complete and irreversible blindness. Lipids and lipid-soluble compounds are essential constituents of the cells and tissues that comprise theeye, and defects in their synthesis, intracellular and extracellular transport, and turnover underlie a variety of significant, common, and often severely debilitatingeyediseases.

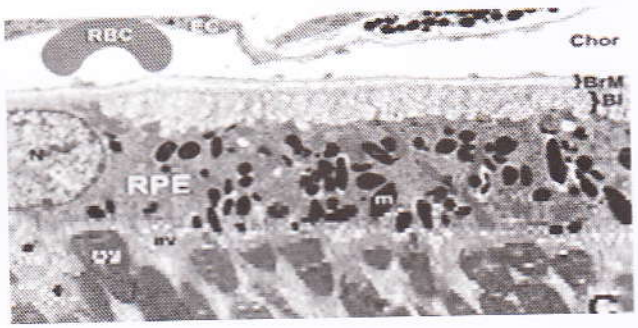
Lipid in retina

The retina is a complex neurosensory tissue comprised of at least six neuronal cell types that are organized into distinct cell layers, in addition to glia (e.g., Müller cells) and astrocytes. Recent studies of lipids and lipid metabolism in the retina



have focused on disease processes caused by either an over-abundance or, in some instances, a deficiency of specific lipid species within retinal cells or their surrounding extracellular environment, often resulting in toxic insult to these cells and ensuing retinal dysfunction, cell death, and progressive retinal





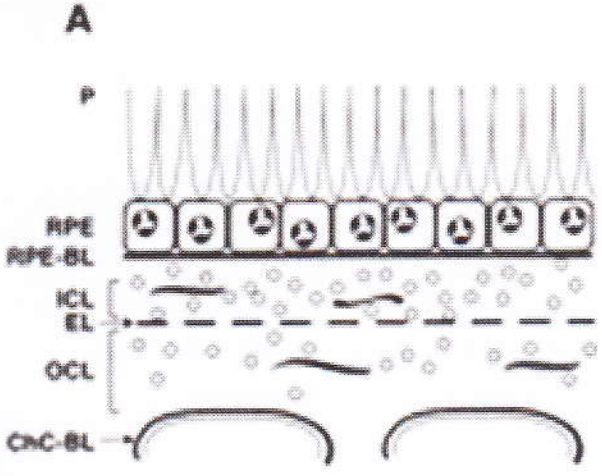
degeneration.Various seminal studies of Lowell and Mabel Hokin in the early 1950s, extended over subsequent decades by the work of many other investigators (e.g., Yasutomi Nishizuka, Michael Berridge, Robert Michell, and others) have shown that the Phosphoinositide 3-Kinase dependent pathway is regulated by light in retinal rod photoreceptors. Their work and that of others has suggested that PI3K inactivation leads to cell death, whereas PI3K activation promotes cell survival (neuroprotection).Diabetic retinopathyhas been linked to dysregulation of PI3K, and there is great potential for development of therapeutic interventions in a variety of ocular diseases that target phosphoinositide metabolism.

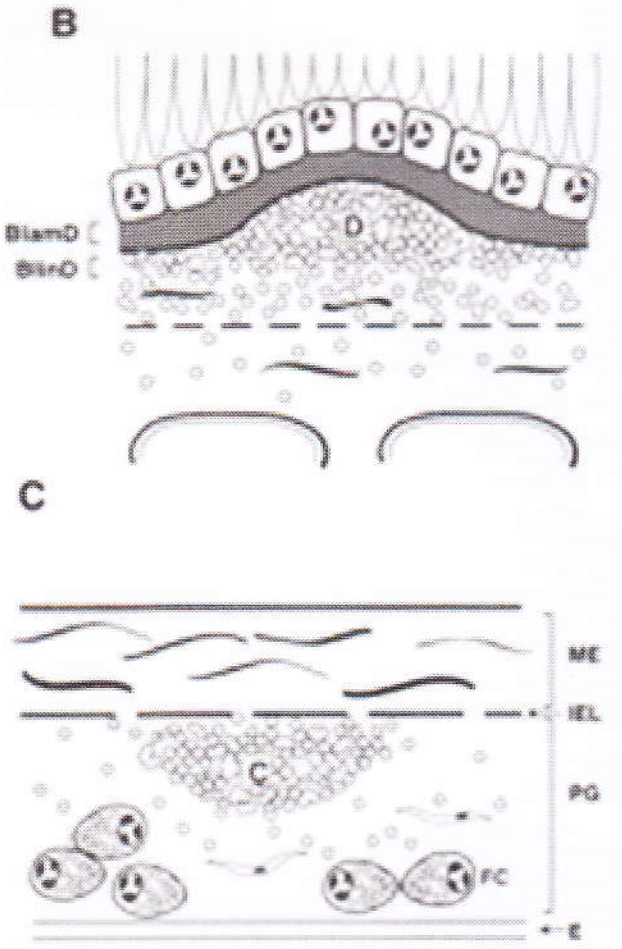
The lipid phase of the photoreceptor outer segment membrane is essential to the photon

capturing and signaling functions of rhodopsin. Rearrangement of phospholipids in the bilayer accompanies the formation of the active intermediates of rhodopsin & condensation product between the photolyzed chromophore all-trans-retinal and phosphatidylethanolamine following photon absorption.[2] The downside of these interactions is the formation of bisretinoid-phosphatidylethanolamine compounds that accumulate in RPE. The propensity of these compounds to negatively impact on the cells has been linked to the pathogenesis of retinal disorders including juvenile onset recessive Stargardt disease and age-related macular degeneration.

With aging, there is a striking accumulation of neutral lipids in Bruch's membrane (BrM) of normal eye that continues through adulthood.[3] This accumulation has the potential to significantly impact the physiology of the retinal pigment epithelium (RPE). It also ultimately leads to the creation of a lipid wall at the same locations where drusen and basal linear deposit, the pathognomonic extracellular, lipid-containing lesions of ARMD, subsequently form. Studies suggest that lipid deposition in BrM is at least partially due to accumulation of esterified cholesterol-rich, apolipoprotein B-containing lipoprotein particles produced by the RPE.[4]

ARMD lesions versus plaque. Schematic cross-sections of RPE/ BrM complex from a normal eye (A) and an eyewith ARMD (B), compared with





atherosclerotic arterial intima (C). Endothelium and vascular lumens (choriocapillary, A, B; artery, C) are at the bottom. Small circles indicate EC-rich lipoproteins, native and modified. P, photoreceptors; RPE, retinal pigment epithelium; RPE-BL, RPE basal lamina; ICL, inner collagenous layer; EL, elastic layer; OCL, outer collagenous layer; ChC-Bl, basal lamina of choriocapillary endothelium. B: BlamD, basal laminar deposit; BlinD, basal linear deposit; D, druse. C: ME, musculo-elastic layer; IEL, internal elastic layer; C, lipid-rich core; PG, proteoglycan layer; FC, foam cells.

Retinal long-chain PUFAs (LC-PUFAs, C12-C22) play important roles in normal human retinal function and visual development, and some epidemiological studies of LC-PUFA intake suggest a protective role against the incidence of advanced age-related macular degeneration (AMD).[5]With ocular aging, some VLC-PUFAs in

retina and retinal pigment epithelium (RPE)/choroid peaked in middle age.Compared with age-matched normal donors, docosahexaenoic acid, adrenic acid, and some VLC-PUFAs in AMD retina and RPE/choroid were significantly decreased, whereas the ratio of n-6/n-3 PUFAs was significantly increased.[6] All these findings suggest that deficiency of LC-PUFAs and VLC-PUFAs, and/or an imbalance of n-6/n-3 PUFAs, may be involved in AMD pathology.

Lipid in cornea

Corneal injury induces an inflammatory reaction and damages the sensory nerves that exert trophic influences in the corneal epithelium.[7]Lipids play important roles in this complex process. Whereas lipid mediators such as platelet activating factor (PAF) and cyclooxygenease-2 metabolites contribute to tissue damage and neovascularization, other mediators, such as the lipoxygenase (LOX) derivatives from arachidonic acid, 12- and 15-hydroxy/hydroperoxyeicosatetraenoic acids, and lipoxin A4, act as second messengers for epidermal growth factor to promote proliferation and repair. Stimulation of the cornea with pigment epithelial derived factor in the presence of docosahexaenoic acid gives rise to the synthesis of neuroprotectin D1, a derivative of LOX activity, and increases regeneration of corneal nerves. More knowledge about the role that lipids play in corneal wound healing can provide insight into the development of new therapeutic approaches for treating corneal injuries. PAF antagonists, lipoxins, and neuroprotectins can be effective therapeutic tools for maintaining the integrity of the cornea.

Lipid in lens

A wide range of membrane morphological changes has been observed with cataract.[8]Phospholipid oxidation and subsequent degradation could account for the dramatic changes observed in human lens lipid composition with age and cataract.[9] A decrease in <1,000th of the total lens lipid could account for all of the membrane changes and protein oxidation/aggregation observed with human cataract.Not only is the degradation of membrane lipids significant to cataractogenesis, but lipid synthesis, necessary for growth and repair, could also be compromised in cataractous lenses. Systemic factors such as fatty acids in the aqueous humor or drugs that decrease cholesterol synthesis could contribute to cataractogenesis. Indeed, age-related changes in human lens lipid composition may serve as markers for systemic stresses.

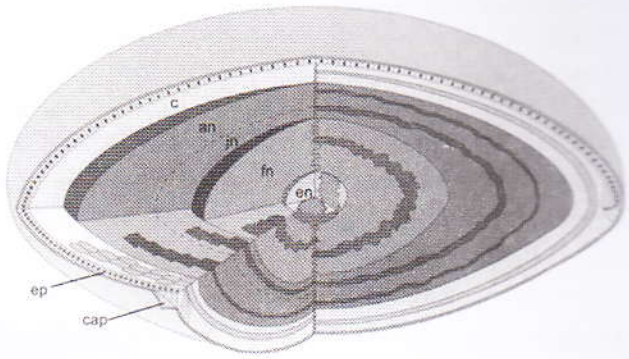


Diagram of an aged normal human lens.The epithelium (ep) and capsule (cap) are enlarged for clarity. c = cortex; an = adult nucleus; jn = juvenile nucleus; fn = fetal nucleus; en = embryonic nucleus.

References

1. Fliesler S. J., Anderson R. E.1983. Chemistry and metabolism of lipids in the vertebrate retina. Prog. Lipid Res. 22: 79131
2. McBee J.K., Palczewski K., Baehr W., Pepperberg D.R.2001. Confronting complexity: the interlink of phototransduction and retinoid metabolism in the vertebrate retina. Prog. Retin. Eye Res. 20: 469529.
3. Marshall J., Hussain A.A., Starita C., Moore D.J., Patmore A.L.1998. Aging and Bruch's membrane. In The Retinal Pigment Epithelium: Function and Disease.Marmor M. F., Wolfensberger T. J., editors. Oxford University Press, New York.66992.
4. Pauleikhoff D., Harper C. A., Marshall J., Bird A. C.1990. Aging changes in Bruch's membrane:

a histochemical and morphological study. Ophthalmology.97: 171:78.
5. Cho E., Hung S., Willett W. C., Spiegelman D., Rimm E. B., Seddon J. M.,Colditz G. A., Hankinson S. E.2001. Prospective study of dietary fat and the risk of age-related macular degeneration. Am. J. Clin. Nutr. 73: 209:18.
6. Sangiovanni J. P., Agron E., Meleth A. D., Reed G. F., Sperduto R. D., Clemons T.E., Chew E.Y.2009. {omega}-3 Long-chain polyunsaturated fatty acid intake and 12-y incidence of neovascular age-related macular degeneration and central geographic atrophy: AREDS report 30, a prospective cohort study from the Age-Related Eye Disease Study. Am. J. Clin. Nutr. 90: 160:17.
7. Imanishi J., Kamiyama K., Iguchi I., Kita M., Sotozono C., Kinoshita S.2000. Growth factors: importance in wound healing and maintenance of transparency of the cornea. Prog. Retin. Eye Res. 19: 1:1329.
8. Taylor V. L., al-Ghoul K. J., Lane C. W., Davis V. A., Kuszak J. R., Costello M. J.1996. Morphology of the normal human lens. Invest. Ophthalmol. Vis. Sci. 37:1:396410.
9. Babizhayev M. A., Deyev A. I., Linberg L. F.1988. Lipid peroxidation as a possible cause of cataract. Mech. Ageing Dev. 44: 69:89.
10. Journal of lipid research.2010

We don't often give our eyes as much thought as we should, that is until something goes wrong and our vision is affected. But when you learn more about eyes, you realize just how amazing they are. Here are a few facts you may enjoy :

1. *Eyes began to develop 550 million years ago. The simplest eyes were patches of photoreceptor protein in single-celled animals.*
2. *Your eyes start to develop two weeks after you are conceived.*
3. *The entire length of all the eyelashes shed by a human in their life is over 98 feet with each eye lash having a life span of about 5 months.*
4. *To protect our eyes they are positioned in a hollowed eye socket, while eyebrows prevent sweat dripping into your eyes and eyelashes keep dirt out of your eyes.*
5. *Your eyeballs stay the same size from birth to death, while your nose and ears continue to grow.*
6. *An eye is composed of more than 2 million working parts.*
7. *Only 1/6 of the human eyeball is exposed.*
8. *Corneas are the only tissues that don't have blood.*
9. *The human eye weights approximately just under an ounce and is about an inch across.*
10. *An eye cannot be transplanted. More than 1 million nerve fibers connect each eye to the brain and currently we're not able to reconstruct those connections.*
11. *80% of our memories are determined by what we see.*
12. *Eyes heal quickly. With proper care, it takes only about 48 hours to repair a minor corneal scratch.*
13. *There are about 39 million people that are blind around the world.*
14. *80% of vision problems worldwide are avoidable or even curable.*
15. *Humans and dogs are the only species known to seek visual cues from another individual's eyes, and dogs only do this when interacting with humans.*
16. *A fingerprint has 40 unique characteristics, but an iris has 256, a reason retina scans are increasingly being used for security purposes.*
17. *People who are blind can see their dreams if they weren't born blind.*
18. *"Red eye" occurs in photos because light from the flash bounces off the back of the eye. The choroid is located behind the retina and is rich in blood vessels, which make it appear red on film.*
19. *80% of what we learn is through our eyes.*
20. *Eyes are the second most complex organ after the brain.*

An Unusual Case Of Retained Foreign Body In Upper Fornix

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College : Subharti Medical College, Meerut

Abstract - Retained foreign bodies account for a small but significant number of ocular trauma cases in ophthalmologic practice. This type of injury may occur in occupational context and can cause various complications in early or late stages of injury. We report an unusual case of urad dal seed (Vigna mungo) trapped in the upper conjunctival fornix for about three months. This article illustrates the importance of careful examination of upper fornix after everting upper lid in patients of refractory chronic conjunctivitis.

Introduction - Retained foreign bodies are frequently seen in ophthalmologic practice. Fairly large foreign bodies may remain hidden in the recess of upper fornix for a considerable period of time. They may produce severe irritation and some discharge. These cases are of particular interest as they may often be overlooked unless the upper lid is everted [1]. There have been several reports on retained foreign bodies like wooden materials [2], contact lenses [3], sutures [4], dust particles [5], hairs [6], vegetative matter [7], insects (beetle [8], leeches [9], caterpillar setae [10], cotton wool ball [11], millet seed [12] etc but a retained urad dal seed (Vigna mungo) in the conjunctival fornix has not, to our knowledge, been reported previously. We report an unusual case of urad dal seed trapped in the upper conjunctival fornix for about three months.

Case Report - A 55 year old farmer presented at the outdoor department of Subharti Medical College, Meerut with diminution of vision in right eye for six months. On examination the unaided visual acuity in right eye was 2/60 improving to 6/60 with +3D sphere. The lids were healthy and there was no conjunctival congestion. Slit lamp examination of right eye revealed grade 3 nuclear sclerosis with posterior subcapsular cataract. Rest of the anterior and posterior segment examination was normal. Regurgitation test was negative and syringing was patent. Intraocular pressure was 14.6mm Hg in both eyes. Examination of left eye was unremarkable. The patient was prescribed topical moxifloxacin four times daily and was posted for right eye cataract surgery after two days.

On the day of surgery, there was conjunctival congestion and mucopurulent discharge in medial canthus. Surgery was postphoned and topical moxifloxacin and tobramycin was prescribed for one week. After one week, conjunctiva cleared and the patient was taken for cataract surgery. Peribulbar injection was given prior to surgery followed by massaging. As soon as the pad was removed, to our surprise, there was a pool of discharge in the eye (Fig1). The discharge was massive and confusing. On eversion of upper lid the palpebral conjunctiva was intensely injected (Fig 2). When pressure was applied on superomedial aspect of globe, suddenly something popped out. It was an urad dal seed (Fig 3). Then on repeated asking patient told that while threshing three months back, probably something entered his eye but as he had no symptoms (pain, foreign body sensation, lid swelling etc) so he ignored. Surgery was postphoned and patient was prescribed topical antibiotics for another one week. Following removal of foreign body, conjunctival congestion and discharge cleared rapidly. Patient underwent uneventful cataract surgery with PCIOL implantation and had good post-operative vision.

Discussion-

It is quite common, especially in rural areas areas to see cases where vegetative foreign bodies have been retained in upper fornix for considerable periods. The upper fornix is a cul-de-sac with relatively low sensitivity. The presence of foreign

Fig 1

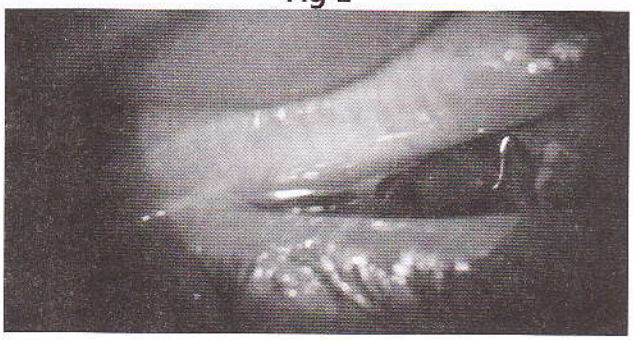

Fig 2

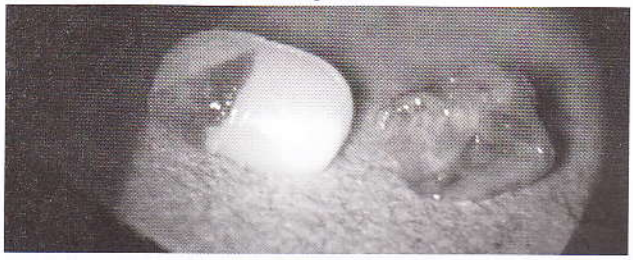

Fig 3

body in this area is often undetected by patients unless a local reaction has been set up. The patient may not recollect a history of foreign body penetration and a quiescent period of days to months may pass before the patient becomes symptomatic.

The reaction to vegetative matter varies[13] and depends largely on concurrent introduction of micro-organisms at the time of injury. Contaminated vegetable matter frequently produces an acute pyogenic panophthalmitis. A common pathogenic reaction of a vegetative foreign body is chronic proliferative granulomatous response. However, in the absence of infection, vegetative matter may behave as a relatively inert foreign body. Several reports in the literature describe eyes remaining quiet for years with retained intraocular foreign body.

Foreign bodies in upper fornix usually present with discharge which is initially watery and later becomes mucopurulent[14]. The lashes stick together with discharge and when the lids are separated the conjunctiva appears healthy. The upper lid is edematous and there is intense reaction in the upper fornix (the intensity depends on the length of time during which the foreign body has been retained). Pain is usually absent. The vegetative matter must be removed as early as possible as they serve as a nidus for infection leading to various complications like granuloma, abscess or chronic discharging sinus.

This article, thus emphasizes the importance of careful examination of upper fornix for retained foreign body after eversion of upper lid in all cases where a mucopurulent discharge is present on one side only and where lacrimal obstruction and dacryocystitis are absent.

References-

1. Sihota R, Tandon R. Parson's Diseases Of The Eye, Publisher Reed Elsevier India Private Limited. 2011; 21/e: 375.
2. Banerjee A, Das A, Agarwal PK, Banerjee AR. Late spontaneous extrusion of a wooden intraocular foreign body. Indian J Ophthalmol 2003; 51: 83-4.
3. Zola E, Van der Meulen IJ, Lapid Gortzak R et al. A conjunctival mass in the deep superior fornix after a long retained hard contact lens in a patient with keloids. Cornea. 2008; 27(10): 1204-6.
4. Chung H. S, Feder R. S, Weston B. C, Bryar P. J. Suture reaction masquerading as a conjunctival malignancy. Can J Ophthalmol. 2006; 41: 207-9.
5. Villaseca A. Dust particles retained in the upper fornix. Am J Ophthalmol. 1977; 83(2): 188-90.
6. Adrian M, Lavina MD, Louise A et al. Unilateral follicular conjunctivitis with retained hair and Pseudomonal infection. Arch Ophthalmol. 2001; 119(6): 901-3.
7. Bhaduri G, Ghosh A. Vegetative intraocular foreign body of 25 years duration. Indian J Ophthalmol 2003; 51: 184-5.
8. Chung R S H, Chew R Y, Au-Eong K G. Coleoptera cincindelidae beetle in the eye. Singapore Med J. 2005; 46(10): 564-5.
9. Alcelik T, Cekic O, Totan Y. Ocular leech infestation in a child. Am J Ophthalmol 1997; 124: 110-2.
10. Horng CT, Chou PI, Liang JB. Catterpillar setae in the deep cornea and anterior chamber. Am J Ophthalmol. 2000; 129: 384-5.
11. Komolafe OO, Omolade CO. A ball of cotton wool masquerading as a conjunctival mass. Niger Med J. 2011; 52: 66
12. Rudolf H. Bock. The upper fornix trap. British J Ophthalmol. 1971; 55: 784.
13. Duke Elder S. System of Ophthalmology. St. Louis: CV Mosby. 1972; 449-554.
14. Colley T. Foreign bodies in the upper conjunctival fornix. British Med J. 1930; 2: 600-1.

RNFL and GLAUCOMA

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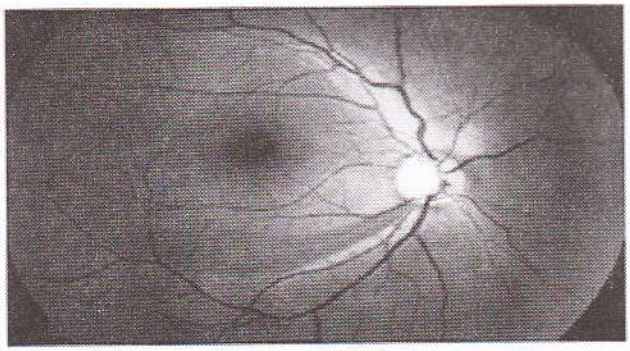
The retinal nerve fiber layer is formed by about 1.2 million ganglion cell axons. The axons of the ganglion cells nasal to the optic disc run directly toward the optic disc, similarly to the axons originated in the macular area that form the papillomacular bundle. The axons coming from ganglion cells situated in the temporal fundus describe an arc around the fovea and run toward the superior or inferior poles of the optic disc.

The nerve fiber layer is thickest at the vertical optic disc poles and thinner at the temporal and nasal optic disc borders.

This pattern can be observed in black and white, red-free, wide-angle fundus photographs, which show the nerve fiber bundles as bright striations in the retinal reflex.



Nerve fiber layer defects: Black-and-white nerve fiber layer photograph showing a typical defect in the inferior bundle compatible with glaucomatous optic nerve damage.

In 1987, Hoyt and Newman first described retinal nerve fiber layer (RNFL) defects as an early sign of glaucoma. Subsequently, several studies confirmed the importance of RNFL assessment in the diagnosis and management of glaucoma.

Sommer and colleagues have shown that RNFL abnormalities are one of the firstclinically detectable changes in patients with glaucoma, and may precede visual field damage by up to 5 years.[1]

Histological studies support these findings and suggest that a 40% loss of nerve fibers is possible in the presence of a normal visual field examination.

RNFL defects may be localized (wedge and slit defects), or diffuse.

Localised defects are visualized as dark areas without striations,by contrast to the adjacent normal RNFL.

Although localized defects are easier todetect, diffuse RNFL loss is more common and more difficult to diagnose. It is characterized by the visualization of second order retinal vessels, which are normally invisible and hidden by the RNFL.

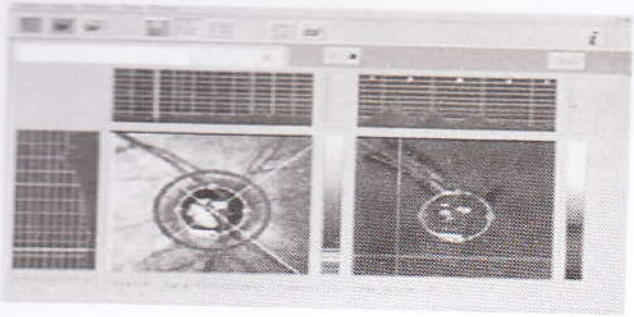
As the glaucomatous damage increases, there is a progressive loss of the RNFL in both the superior andinferior poles, but the RNFL in the papillomacular bundle remains intact.[2]

In end-stage glaucoma, nostriations are found, and a diffuse RNFL loss is observed.

Examination of RNFL

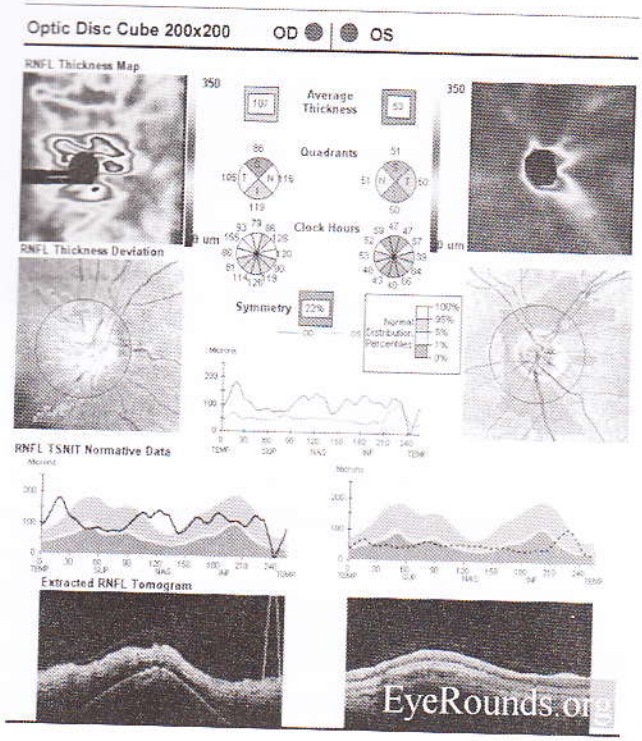
- The RNFL may be examined through a dilated pupil, with a red - free light and a directophthalmoscope. However, a better view can be obtained with a 78D or 90D lens or a contact lens at the slit lamp with a green filter.

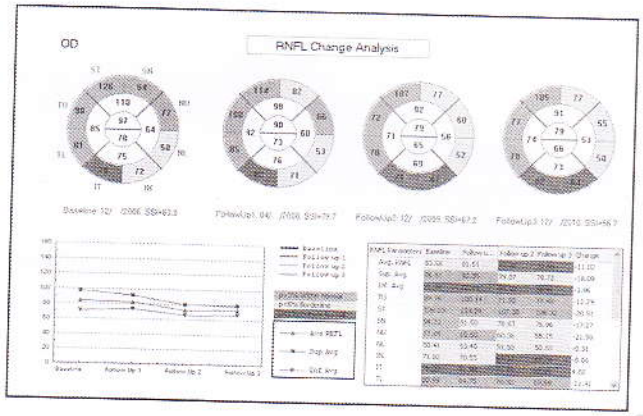
- Scanning Laser Polarimetry (GDx, Laser Diagnostic Technologies, San Diego, USA) measures the RNFL thickness.[3] It is based on the birefringent properties of the RNFL, which has its neurotubules disposed in an organized, parallel fashion.



Scanning laser polarimetry (GDx, Laser Diagnostic Technologies, San Diego, USA). Redsignals show areas with greater retardation and nerve fiber layer thickness.

The RNFL thickness can also be assessed through Optical Coherence Tomography (OCT), an optical analog of B-scan ultrasound that can create high-resolution cross- sectional images of the RNFL.[4]





The RNFL thickness can also be assessed through Optical Coherence Tomography (OCT), an optical analog of B-scan ultrasound that can create high-resolution cross- sectional images of the RNFL.[4]

Irrespective of the instrument, it is important to emphasize that, although these technologies seem promising, optic disc topography and RNFL thickness among the general population are highlyvariable, which limits their use in the detection of early glaucoma.

At present, they cannot replace anexperienced examiner. Longitudinal studies are being done to determine the ability of these systems to detect changes in the optic disc or RNFL, indicating what can be considered a true sign of progressionin glaucomatous patients. The increasing use of image analysers in research and office settings, and the introduction of modifications specifically designed to neutralize their limitations, will increase theirrole in clinical practice.

References

1. Sommer A, Miller NR, Pollack I, et al. The nerve fiber layer in the diagnosis of glaucoma. Arch Ophthalmol. 1977;95:2149-56.

2. Funaki S, Shirakashi M, Abe H. Relation between size of optic disc and thickness of retinal nerve fibre layer in normal subjects. Br J Ophthalmol. 1998;82:1242-45.

3. Hoh ST, Greenfield DS, Mistlberger A, Liebmenn JM, Ishikawa H, Ritch R. Optical coherence tomography and scanning laser polarimetry in normal, and glaucomatous eyes. Am J Ophthalmol 2000;129: 120-33.

Schuman JS, Hee MR, Puliafito CA, et al. Quantification of nerve fiber layer thickness in normal and glaucomatous eyes using optical coherence tomography.Arch Ophthalmol. 1995;113:586-96.

Steroid Induced Glaucoma

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Steroid-induced glaucoma is a form of open-angle glaucoma occurring as an adverse effect of corticosteroid therapy.However, an even higher percentage of normal individuals develop substantially increased IOPs if the glucocorticoid is administered in greater frequency, at higher doses or for a longer period.It is usually associated with topical steroid use, but it may develop with oral,intravenous,inhaled and peri-ocular steroid administrations by causing decrease in aqueous outflow facility.A number of drugs have been implicated in corticosteroid-induced glaucoma like dexamethasone, prednisolone,betamethasone,fluoromethalone, hydrocortisone, cortisone etc.

Apart from glaucoma, corticosteroid administration is associated with posterior sub-capsular cataract development.

INCIDENCE

Steroid induced intraocular pressure(IOP) elevations can occur in all age groups.No gender and racial predilection exists for steroid induced glaucoma.

Incidence of steroid induced IOP elevations in patients on systemic corticosteroids is unknown.These patients may be discovered during routine eye check orelse seeking medical care to an ophthalmologist due to some other reason

However, 5% of the general population is considered to be 'steroid responders',i.e,may develop steroid induced glaucoma when steroid is administered.Approximately one-third of individuals experience moderate increase in IOP after topical steroid use.Following 4-6 weeks of topical corticosteroids,in about 5% of patients IOP will rise by more than 16 mmHg and 30%, by 6-15mmHg.1,2Classic studies by Armaly and Becker indicate that 56% of normal develop marked IOP rises after 46 weeks of topical dexamethasone or betamethasone administration.

TABLE 1: IOP response to topical corticosteroids

	Armaly[3]	**Becker**[4]
Frequency	QID	TDS
Duration	6 weeks	4 weeks
Parameter	Final IOP	IOP change
Type of Responder	IOP(mmHg)	IOP(mmHg)
Low	<20 (58%)	<6 (66%)
Intermediate	20-31 (36%)	6-15 (29%)
High	>31 (6%)	>15 (5%)

RISK FACTORS

1. PATIENT'S SUSCEPTIBILITY

Patients who are steroid responders are likely to develop Primary open angle glaucoma (POAG) following steroid administration and those with pre-existing POAG. 92% of POAG patients are high steroid responders;among their children, 19%.

A higher than average risk is found in patients with:

1. Patients with pre-existing POAG
2. A first degree relative with POAG
3. A history of previous steroid induced IOP elevation
4. Type 1 Diabetes Mellitus
5. Connective tissue diseases
6. Penetrating Keratoplasty,especially in eyes with Fuchs Endothelial dystrophy or keratoconus.
7. High Myopia

2. ROUTES OF ADMINISTRATION:

Steroid induced glaucoma is mostly caused by exogenous steroids.In rare cases, glaucoma is produced by endogenous glucocorticoids associated with adrenal hyperplasia or adenoma. It is mostly seen with topical steroids, however,it is possible with other local (dermal or inhalational), depot (sub-conjunctival, sub-Tenon's,intravitreal), or systemic steroids. Intravitrealtriamcinolone can increase IOP for months.A 20mg dose increased IOP above 21mmHg in 40% of people for upto 9months: 1% required trabeculectomy. In some patients, the IOP rise persists and may require topical medication, laser trabeculoplastyor even trabeculectomy to lower the pressure and preventoptic nerve damage or progression of optic nerve damage.

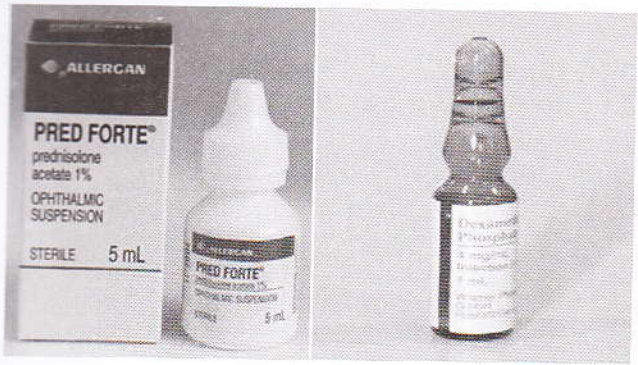
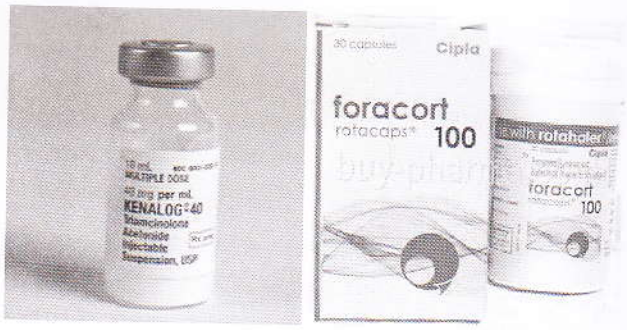


TABLE 2: Route of administration

EXOGENOUS CORTICOSTEROIDS
OCULAR
-Eye-drops
-Ocular ointment
PERIOCULAR/INTRAVIREAL INJECTIONS
SYSTEMIC
-Oral
-Injection
-Topical to skin
ENDOGENOUS CORTICOSTEROIDS
-Adrenal hyperplasia
-Adrenal adenoma or carcinoma
-Ectopic ACTH production



Systemic corticosteroids are least likely to induce glaucoma.IOP elevations may occur weeks to years after treatment. There have been several case reports of increased IOP following use of a corticosteroid inhaler for asthma.Even new inhalational agent with safety profile like Fluticasone propionate has been reported to cause a significant rise in IOP especially in patients predisposed to POAG.

3. STEROID PREPARATIONS:

More potent the steroid more is its pressure inducing effect i.e, directly proportional.Potent steroids like dexamethasone, betamethasone and prednisolone have a higher tendency for IOP rise compared to lesser potent steroids like fluoromethalone and medrysone.Even more the concentration of steroids, more is the likelihood of IOP rise.

4. DURATION OF STEROID-THERAPY:

It takes from weeks,months to years to develop rise in IOP following steroid administration.It

depends upon various factors like steroid formulations, mode of administrations etc.However, topical steroids can cause rise in IOP following 4-6 weeks of therapy whereas systemic steroids takes a longer time for IOP rise.Longer the duration of steroid therapy, higher is the propensity to develop IOP rise and glaucoma.

PATHOPHYSIOLOGY:

Glucocorticoids raise IOP by lowering outflow facility through an unknown mechanism. The most common explanation for this phenomenon has been that glucocorticoids cause an accumulation of glycosaminoglycans in the trabecular meshwork, perhaps by stabilizing lysosomal membranes and inhibiting the release of catabolic enzymes.Thus, they reduce trabecular outflow facility.

Other explanations for corticosteroid glaucoma include an inhibition of the phagocytosis of foreign matter by trabecular endothelial cells and decreased synthesis of prostaglandins that regulate aqueous humor outflow.

Southren and co-workers and Weinstein and co-workers found abnormal glucocorticoid metabolism in trabecular tissue from patients with POAG. This finding may explain the increased susceptibility of patients with POAG to the ocular hypertensive effects of glucocorticoids.

CLINICAL FEATURES:

As stated, corticosteroid glaucoma usually resembles POAG.History of systemic or ocular disease which could require chronic corticosteroid use (asthma, uveitis, collagen vascular disease, asthma, dermatitis) should be elicited in patients having open angle glaucoma.However, the clinical picture may be altered by the age of the patient.Infants treated with corticosteroids may develop a condition thatresembles congenital glaucoma. In contrast; elderly patients who received corticosteroid treatment in the past may have normal tension glaucoma.

Clinical evaluation reveals an elevated IOP, open and normal appearing angles on gonioscopy,white painless eye, optic disc cupping and visual field defects.

There have been several reports of moderate to severe IOP elevation in patients treated with corticosteroid eye-drops following laser-assisted in-situ keratomileusis (LASIK). These cases can be particularly challenging because of the difficulty in obtaining an accurate measurement of the true IOP in the postoperative period (or, in fact, anytime after LASIK because of the subsequent very thin cornea).

DIFFERENTIAL DIAGNOSIS:

1. POAG
2. Uveitic glaucoma
3. Glaucomatocyclitic crisis
4. Normal tension glaucoma
5. Congenital glaucoma

MANAGEMENT:

The first step in managing corticosteroid glaucoma is to discontinue the drug. In most cases, IOP returns to normal over a few days to several weeks. During this period, anti-glaucoma medications may be used to control IOP.[20]

If glucocorticoid treatment is necessary for the patient's life or well-being, therapy should be altered to the weakest possible drug at the lowest possible dose. The residual glaucoma is then treated in the same fashion as is POAG. In the cases that require topical ocular corticosteroid therapy, the patient should be treated if possible with drugs such as medrysone or fluorometholone because these drugs have less of a tendency to raise IOP. Topical NSAIDS are other alternatives with no IOP rising potential but lacks enough anti-inflammatory property compared to steroids.

If medication is unsuccessful in controlling IOP

and the optic nerve is threatened, laser-trabeculoplastyor filtering surgery should be considered. However, if both medical and laser therapy fails, trabeculectomy with or without antimetabolites is the primary procedure. In cases of eyes with active neovascularization or inflammation, a glaucoma drainage device may be used as the primary procedure.

In rare cases, IOP remains elevated months to years after thecorticosteroid has been discontinued. In these situations, it may be impossible to determine whether this is a residual effect of glucocorticoid treatment or whether the patient has had underlying open-angle glaucoma unmasked by the treatment. In either case,the patient is treated in similar fashion.

CONCLUSION:

All patients under corticosteroid therapy especially those with a family history of glaucoma should be routinely examined to rule out glaucoma. Inadvertent use of steroids and self-medication should be discouraged. Long term use of steroid should be avoided or substituted by other alternatives without compromising treatment standard to the best possible way.

REFERENCES:

1) Armaly MF: Statistical attributes of the steroid hypertensive response in the clinically normal eye. Invest Ophthalmol. 4:187-197 1965.

2) Becker B: Intraocular pressure response to topical corticosteroids. Invest Ophthalmol. 4:198-205 1965.

3) ArmalyMF : The heritable nature of dexamethasone hypertension and glaucoma.ArchOphthalmol 1966:75:32.

4) Becker B: Intraocular pressure response to topical corticosteroids. Invest Ophthalmol. 4:198-205 1965.

5) Bayer JM: Ergebnisse und Beurteilund der subtotalenAdrenalektomiebeimhyperfunctions Cushing, Langenbecks Arch Chir 291:531, 1959.

6) Haas JS, Nootens RH: Glaucoma secondary tobenign adrenal adenoma, Am J Ophthalmol 78:497, 1974.

7) Jonas JB, Degenring RF, Kreissig I, et al.: Intraocular pressure elevation after intravitreal triamcinolone acetonide injection. Ophthalmology. 112:593-598 2005.

8) Rhee DJ, et al: Intraocular pressure alterations following intravitreal triamcinolone acetonide, Br J Ophthalmol 90:999, 2006.

9) Armaly MF: Effects of corticosteroids on intraocular pressure and fluid dynamics. I. The effect of dexamethasone in the normal eye, Arch Ophthalmol 70:482, 1963.

10) Francois J: The importance of themucopolysaccharides in intraocular pressureregulation, Invest Ophthalmol Vis Sci 14:173, 1975.

11) Bill A: The drainage of aqueous humor, InvestOphthalmol Vis Sci 14:1, 1975.

12) Weinreb RN, Mitchell ME, Polansky JR:Prostaglandin production by human trabecularcells: in vitro inhibition by dexamethasone, InvestOphthalmol Vis Sci 24:1541, 1983.

13) Southren AL, et al: 5-Beta-dihydrocortisol: possiblemediator of the ocular hypertension in glaucoma,Invest Ophthalmol Vis Sci 26:393, 1985.

14) Southren AL, et al: Altered cortisol metabolism incells cultured from trabecular meshwork specimensobtained from patients with primary open-angleglaucoma, Invest Ophthalmol Vis Sci 24:1413, 1983.

15) Weinstein BI, et al: Defects in cortisol-metabolizingenzymes in primary open-angle

glaucoma, InvestOphthalmol Vis Sci 26:890, 1985.

16)Kass MA, Kolker AE, Becker B: Chronic topicalcorticosteroid use simulating congenital glaucoma,J Pediatr 81.

17)Scheie HG, Rubinstein RA, Albert DM: Congenitalglaucoma and other ocular abnormalities withidiopathic infantile hypoglycemia, J PediatrOphthalmol 1:45, 1964:1175, 1972.

18)Davidson RS, Brandt JD, Mannis MJ: Intraocularpressure-induced interlamellar keratitis after LASIKsurgery, J Glaucoma 12:23, 2003.

19) Hamilton DR, et al: Steroid-induced glaucoma afterlaser in situ keratomileusis associated with interfacefluid, Ophthalmology 109(4):659, 2002.

20) Scherer WJ, Hauber FA: Effect of latanoprost onintraocular pressure in steroid-induced glaucoma, JGlaucoma 9:179, 2000.

21) Sihota R, et al: Prospective, long-term evaluation ofsteroid-induced glaucoma, Eye 22:26, 2008.

22) Thomas R, Jay JL. Raised IOP with topical steroids after trabeculectomy. Graefes Arch ClinExpOphthlmol 1988;226:337.

23) Reiss G,Wilenksy J, Higginbotham E. Laser trabeculoplasty. SurvOphthalmol 1991;35:407.

24) Spaeth GL, Rodrigues MM, Weinreb S: Steroidinducedglaucoma. A. Persistent elevation ofintraocular pressure. B. Histopathologic aspects,Trans Am OphthalmolSoc 75:535, 1977.

You've had your peepers since you were born, so you may think you know them pretty well, but here are some fun facts you may not know about eyes:

- *The average blink lasts for about 1/10th of a second.*
- *While it takes some time for most parts of your body to warm up to their full potential, your eyes are on their "A game" 24/7.*
- *Eyes heal quickly. With proper care, it only takes about 48 hours for the eye to repair a corneal scratch.*
- *Seeing is such a big part of everyday life that it requires about half of the brain to get involved.*
- *Newborns don't produce tears. They make crying sounds, but the tears don't start flowing until they are about 4-13 weeks old.*
- *Around the world, about 39 million people are blind and roughly 6 times that many have some kind of vision impairment.*
- *Doctors have yet to find a way to transplant an eyeball. The optic nerve that connects the eye to the brain is too sensitive to reconstruct successfully.*
- *The cells in your eye come in different shapes. Rod-shaped cells allow you to see shapes, and cone-shaped cells allow you to see color.*
- *You blink about 12 times every minute.*
- *Your eyes are about 1 inch across and weigh about 0.25 ounce.*
- *Some people are born with two differently colored eyes. This condition is heterochromia.*
- *Even if no one in the past few generations of your family had blue or green eyes, these recessive traits can still appear in later generations.*
- *Each of your eyes has a small blind spot in the back of the retina where the optic nerve attaches. You don't notice the hole in your vision because your eyes work together to fill in each other's blind spot.*
- *Out of all the muscles in your body, the muscles that control your eyes are the most active.*
- *80% of vision problems worldwide are avoidable or even curable.*

Craniosynostosis: an ocular perspective

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Premature fusion of one or more cranial sutures leading to abnormal head shape.

Classified asprimary and secondary. Also as simple , complex and syndromic Includes :

a. Crouzon syndrome

b. Apert syndrome

c. Pleiffer syndrome

d. Carpenter syndrome

Ossification of cranial vault starts in the central region of each cranial bones,then extends towards sutures.

- Primary-when one or more suture fuse prematurelyprimary ossification. Defect. e.g. scaphocephaly, brachycephaly, plagiocephaly, trigonocephaly.
- Secondary - primary failure of brain growth, more common, 90% of total

Baller-Gerold Syndrome

Ocular Features

The ocular features are a rather minor part of this syndrome and are found in less than a third of patients. These primarily involve lids and adnexae with telecanthus, downslanting lid fissures, and epicanthal folds. Some also have nystagmus while strabismus, blue sclerae, and ectropion have also been reported.

Systemic Features

The cardinal features of this syndrome are craniosynostosis and radial defects. However, a large number of variable defects such as imperforate or anteriorly placed anus, rectovaginal fistula, absent thumbs, polydactyly, and mental retardation may also be present. The radius may be completely absent or abnormally formed and occasionally the ulnar bone is involved as well. Some patients have a conductive hearing loss.

Treatment Options

No treatment is available.

References

1. Ramos Fuentes FJ, Nicholson L, Scott CI Jr. Phenotypic variability in the Baller-Gerold syndrome: report of a mildly affected patient and review of the literature. Eur J Pediatr. 1994 Jul;153(7):483-7.
2. Temtamy SA, Aglan MS, Nemat A, Eid M. Expanding the phenotypic spectrum of the Baller-Gerold syndrome. Genet Couns. 2003;14(3):299-312.

Carpenter Syndrome

Ocular Features

A variety of ocular anomalies have been reported in Carpenter syndrome with none being constant or characteristic. The inner canthi are often spaced widely apart and many have epicanthal folds and a flat nasal bridge. Other reported abnormalities are nystagmus, fovealhypoplasia, corneal malformations including microcornea, corneal opacity, and mild optic atrophy and features of pseudopapilledema.

Systemic Features

Premature synostosis involves numerous cranial sutures with the sagittal suture commonly involved causing acrocephaly (tower skull). Asymmetry of the skull and a 'cloverleaf' deformity are often present. The polydactyly is preaxial and some degree of syndactyly is common especially in the toes. The digits are often short and may be missing phalanges. Some patients are short in stature. Structural brain defects may be widespread including atrophy of the cortex and cerebellarvermis. Septal defects in

the heart are found in about one-third of patients. The ears can be low-set and preauricular pits may be seen. Some but not all patients have obesity and a degree of mental retardation.

Treatment Options

No treatment of the ocular defects is necessary in most cases. Craniectomy may be required in cases with severe synostosis.

References

1. Hidestrand P, Vasconez H, Cottrill C. Carpenter syndrome. J Craniofac Surg. 2009 Jan;20(1):254-6.
2. Jenkins D, Seelow D, Jehee FS, Perlyn CA, Alonso LG, Bueno DF, Donnai D, Josifova D, Mathijssen IM, Morton JE, Orstavik KH, Sweeney E, Wall SA, Marsh JL, Nurnberg P, Passos-Bueno MR, Wilkie AO. RAB23 mutations in Carpenter syndrome imply an unexpected role for hedgehog signaling in cranial-suture development and obesity. Am J Hum Genet. 2007 Jun;80(6):1162-70.
3. Temtamy SA. Carpenter's syndrome: acrocephalopolysyndactyly. An autosomal recessive syndrome. J Pediatr. 1966 Jul;69(1):111-20.

Pfeiffer Syndrome

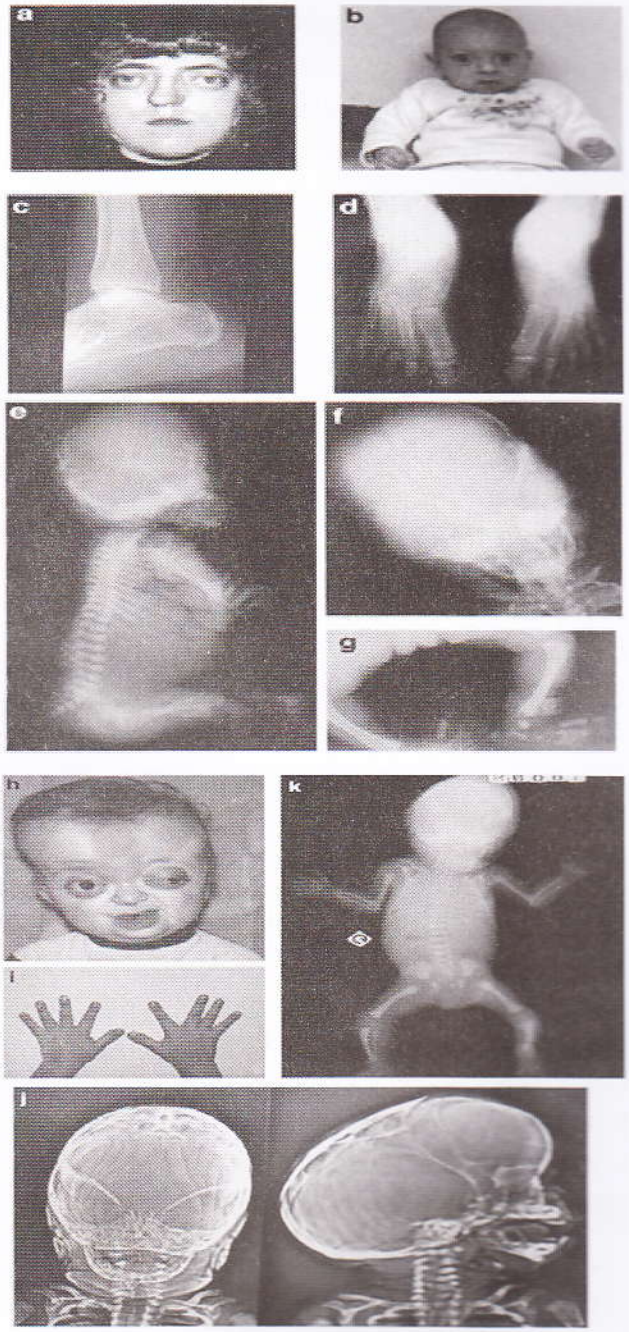
Ocular Features

Patients may have extreme proptosis secondary to shallow orbits and exposure keratitis is a risk. Hypertelorism, strabismus, and antimongoloid lid slants are common. More rare signs include anterior chamber anomalies and optic nerve hypoplasia.

Systemic Features

Pfeiffer syndrome has been divided into 3 types, of which cases with types 2 and 3 often die young. Type 1 has the more typical features with midfacehypoplasia, broad thumbs and toes, craniosynostosis, and often some degree of syndactyly. Adult patients with type 1 may be only mildly affected with some degree of midfacehypoplasia and minor broadening of the first digits. Hearing loss secondary to bony defects is relatively common. Cleft palate is uncommon. Airway malformations especially in the trachea can cause respiratory problems.

Treatment Options



Exposure keratitis requires the usual treatment. Surgery for the midface underdevelopment may be helpful for the proptosis. Airway obstruction may require tracheostomy or surgical correction of the air passages.

References

1. Glaser RL, Jiang W, Boyadjiev SA, Tran AK, Zachary AA, Van Maldergem L, Johnson D, Walsh S, Oldridge M, Wall SA, Wilkie AO, Jabs EW. Paternal origin of FGFR2 mutations in sporadic cases of Crouzon syndrome and Pfeiffer syndrome. Am J Hum Genet. 2000 Mar;66(3):768-77.

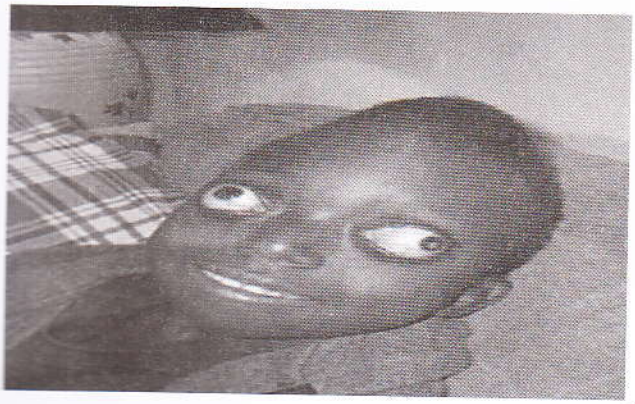
2. Schell U, Hehr A, Feldman GJ, Robin NH, Zackai EH, de Die-Smulders C, Viskochil DH, Stewart JM, Wolff G, Ohashi H, et al. Mutations in FGFR1 and FGFR2 cause familial and sporadic Pfeiffer syndrome. Hum Mol Genet. 1995 Mar;4(3):323-8.

3. Lajeunie E, Ma HW, Bonaventure J, Munnich A, Le Merrer M, Renier D. FGFR2 mutations in Pfeiffer syndrome. Nat Genet. 1995 Feb;9(2):108.

Crouzon Syndrome

Ocular Features

The primary ocular features result from pattern-specific, premature synostoses of cranial sutures. The orbits are often shallow resulting in proptosis, sometimes to such an extent that exposure keratitis or even spontaneous subluxation of the globe results. This is exacerbated by the midfacehypoplasia that is often present. As many as 22% of patients have optic atrophy, most likely secondary to chronic papilledema from elevated intracranial pressure. Strabismus is common, often with a V-pattern exotropia. Overaction of the inferior obliques and underaction of the superior obliques have been described.



Systemic Features

The coronal sutures are the most commonly affected by the premature synostosis and hence the skull is often brachycephalic and the forehead is prominent. Increased intracranial pressure is a risk. The nose is parrot-beaked and the upper lid is short. Maxillary hypoplasia from the midface underdevelopment can cause crowding and displacement of the upper teeth.

Treatment Options

Exposure keratitis must be treated. Cranial surgery has been necessary for some patients to relieve the papilledema but the post operative outcome can be complicated by hydrocephalus.

References

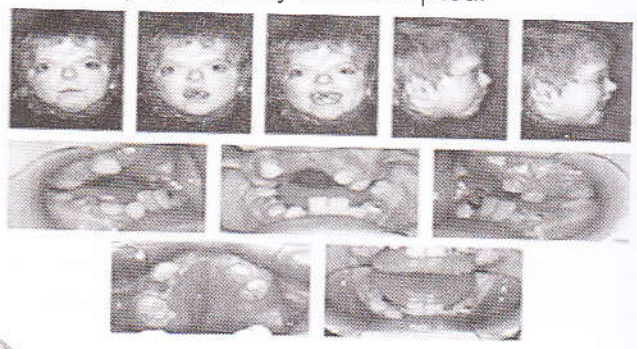
Bertelson TI. The premature synostosis of the cranial sutures.ActaOphthalmol Suppl. 1958;36(Suppl 51) : 1-176.

Kreiborg S. Crouzon Syndrome. A clinical and roentgencephalometric study. Scand J PlastReconstrSurg Suppl. 1981;18:1-198.

Apert Syndrome

Ocular Features

In 10% of patients, keratitis and corneal scarring occur from the sometimes marked proptosis and corneal exposure. Optic atrophy is present in over 20% of patients. Strabismus, primarily exotropia, is found in more than 70% and various extraocular muscle anomalies may be detectable. Usually the exotropia has a V-pattern with overaction of the inferior oblique muscles while the superior oblique is weak. Amblyopia occurs in nearly 20%. The lid fissures often slant downward and the eyebrows may be interrupted.



Systemic Features

This brachysphenocephalic type of acrocephaly is associated with syndactyly in the hands and feet. Pre- and postaxial polydactyly may be present. There is considerably variation in expression with some patients so mildly affected that they appear virtually normal, whereas others have extreme degrees of brachycephaly with high foreheads, midfacehypoplasia, and proptosis secondary to shallow orbits. Imaging often reveals one or more CNA anomalies such as defects of the corpus callosum, partial absence of the septum pellucidum, ventriculomegaly, and sometimes hydrocephalus. A small but significant proportion of patients have some developmental delay and cognitive impairment. Over 39% of patients have a normal IQ.

Treatment Options

No specific treatment is available for this disorder but exposure keratitis may require surveillance and therapy.

References

- Khong JJ, Anderson P, Gray TL, Hammerton M, Selva D, David D. Ophthalmic findings in apert syndrome prior to craniofacial surgery. Am J Ophthalmol. 2006 Aug;142(2):328-30.
- Glaser RL, Broman KW, Schulman RL, Eskenazi B, Wyrobek AJ, Jabs EW. The paternal-age effect in Apert syndrome is due, in part, to the increased frequency of mutations in sperm. Am J Hum Genet. 2003 Oct;73(4):939-47.
- Kreiborg S. Crouzon Syndrome. A clinical and roentgencephalometric study. Scand J PlastReconstrSurg Suppl. 1981;18:1-198.

- *The human eye can distinguish about 10 million different colors.*
- *Some women can have a genetic mutation which causes them to see millions of more colors.*
- *People with blue eyes have a higher alcohol tolerance.*
- *If the human eye was a digital camera it would have 576 megapixels.*
- *All blue eyed people can be traced back to one person who lived near the Black Sea almost 10,000 years ago.*
- *We spend about 10% of our waking hours with our eyes closed, blinking.*
- *Researchers have successfully used the game TETRIS to treat "lazy eye" in adults.*
- *Albert Einstein's eyes remain in a safe box in NYC.*
- *Your eyes can get sunburned.*
- *Black lemurs are thought to be the only primates, besides humans, to have blue eyes.*
- *The space between your eyebrows is called Nasion.*

Management Of Vernal Keratoconjunctivitis

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Vernal keratoconjunctivitis (VKC) is a chronic, bilateral, at times asymmetrical,seasonally exacerbated, allergic inflammation of the ocular surface,involving tarsal and / or bulbar conjunctiva.It is more common in children and young adults having an atopic background.

Different authors, at different times, described it as spring catarrh, phlyctenula pallida, circumcorneal hypertrophy, recurrent vegetative conjunctiva, verrucosa conjunctiva and aestivale conjunctiva, calling attention to the various aspects of this disease. Although the allergic nature of this entity has been accepted for a long time, its exact aetiology and pathogenesis is still unclear.

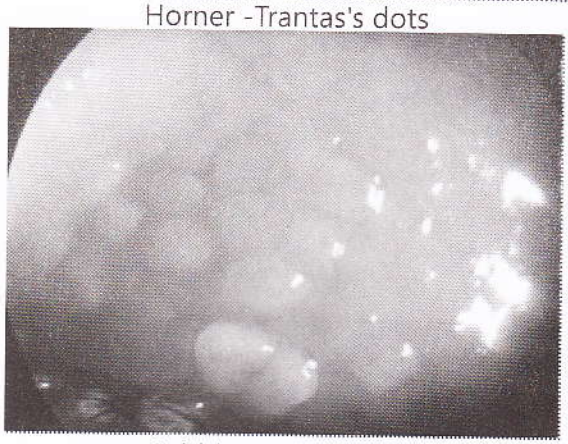
The accumulation of a large amount of immunological data has established that the pathogenesis of VKC is much more complex than a mere type 1 hypersensitivity reaction. To the present day, the precise role played by genetic predisposition and environmental factors in the onset, progression and resolution of this selflimiting, but at times incapacitating, childhood entity is an enigma. Despite the universal acceptance of the nomenclature vernal keratoconjunctivitis, occurrence of this disease is not limited to spring, with episodes of reactivity being quite common in the winter. The initial seasonal attacks turn into perennial disease after a few years. The efficiency of school-aged children decreased profoundly because of the chronic and recurrent course. Although this is not usually a blinding disease, visual impairment may occur if the cornea is involved.

Clinical features and diagnosis

Patients suffering from VKC may have several episodes of active inflammation throughout the year. Initially seasonal disease may become perennial after a few years. In approximately one quarter of VKC patients the disease smolders throughout the year, without any remission, from the onset.

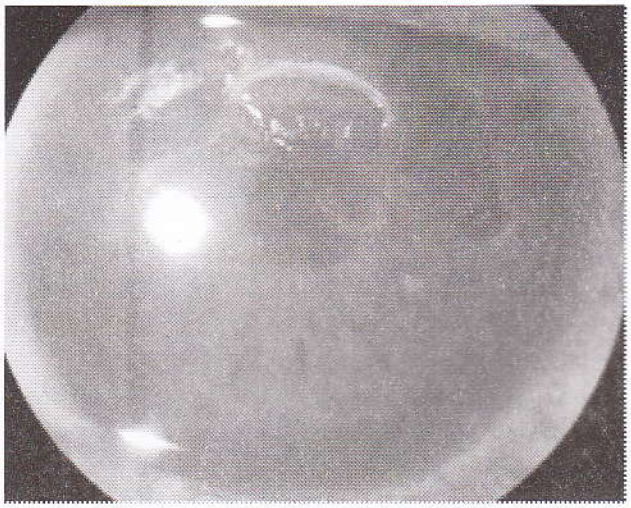
In its typical form, VKC presents with pruritus, hyperaemia, photophobia and watering. Thick mucus hyper-secretion with sticky mucous filaments, called 'ropy discharge', is a characteristic of VKC. Transient limbal or conjunctival yellow-white points or deposits, known as HornerTrantas's dots are degenerating eosinophils and epithelial cell debris. Large (> 1 mm) papillae in VKC occur predominantly at the upper tarsus. Papillae size correlate positively with the persistence or worsening of symptoms over long-term follow-up . These papillae become quite swollen during the active stage but persist even during the quiescent stage.

Horner -Trantas's dots

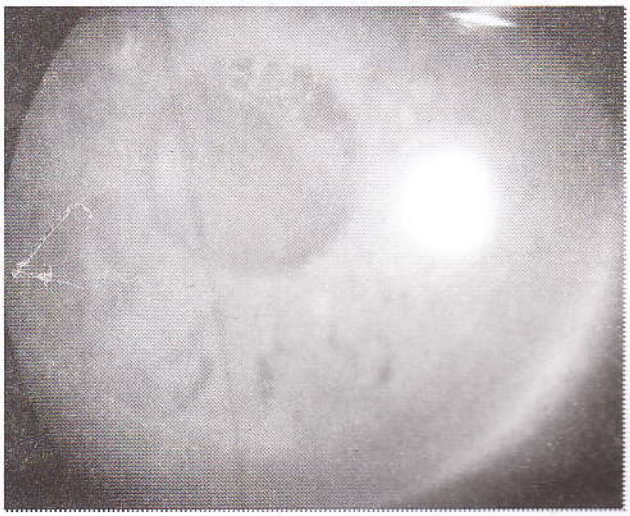


Cobblestone papillae

Photophobia, pain and foreign body sensation are caused by involvement of the cornea. Corneal changes include punctate epithelial keratitis, epithelial macro-erosions, shield ulcer plaque formation and late corneal vascularization. The oval-shaped epithelial defects, known as shield ulcers , usually have their lower border in the upper half of the visual axis. Healed shield ulcers may leave a subepithelial ring-like scararization. Pseudogerontoxon, which resembles arcus senilis, is a waxing and waning grey-white lipid deposition in the superficial stroma of the peripheral cornea.



Shield ulcer



Ring shapedcorneal opacity after healed shield ulcer

The lack of standardized diagnostic criteria and lack of common language among physicians regarding the severity of VKC renders this disease more difficult to diagnose and treat. Despite mounting data suggesting the role of both IgE and non-IgE mediated immune responses in the pathogenesis of VKC, no clinical or laboratory test has evolved to support the diagnosis in atypical cases or predict the course of this disease.

Treatment

- Preventive measures and patient education
- Compliance with instructions is better with a well-informed patient and outcome of treatment is gratifying.
- Patient education and preventive measures to improve the management of vernal keratoconjunctivitis
- VKC is a chronic, recurrent condition that usually improves by adulthood
- Avoid rubbing itchy eyes, as this makes the condition worse
- Avoid provocative nonspecific triggers such as sun, wind, and salt water, that exacerbate the condition, using sunglasses, hats with visors, swimming goggles where necessary
- Avoid contact with commonly known allergens
- Application of cold compresses and preservative-free artificial tears help to provide symptomatic relief
- Hands, face and hair should be washed frequently to reduce exposure to allergens

Pharmacological therapy

The variety of currently available drugs to treat VKC include anti-histamines, mast-cell stabilizers, dual acting agents, corticosteroids and immunomodulators but none is enough to treat all aspects of multifaceted pathophysiology of VKC. Most drugs used are merely palliative and do not eliminate the complex immune response initiating and perpetuating the allergic ocular inflammation, so there is recurrence of disease when the therapy is discontinued.

Common topical ocular allergy medications for the treatment of vernal keratoconjunctivitis

Class	Drug	Indication	Comments
Vasoconstrictor/ antihistamine combinations	Naphazoline/ pheniramine	Rapid onset of action Episodic itching and redness	Short duration of action Tachyphylaxis, Mydriasis, Ocular irritation Hypersensitivity, Hypertension Potential for inappropriate patient use
Antihistamines	Levocabastine Emedastine	Relief of itching Relief of signs and symptoms	Short duration of action Frequently does not provide complete disease control when used alone
Mast cell stabilizers	Sodium cromoglycate Nedocromil Lodoxamide	Relief of signs and symptoms	Long-term usage Slow onset of action Prophylactic dosing Frequently does not provide complete disease control when used alone
Antihistamine/ mast cell stabilizers (dual-acting)	Azelastine Epinastine Ketotifen Olopatadine	Relief of itching Relief of signs and symptoms	Bitter taste (azelastine) No reported serious side effects Frequently does not provide complete disease control when used alone
Corticosteroids	Loteprednol Fluormetholone Desonide Rimexolone Dexamethasone Betamethasone	Treatment of allergic inflammation Use in moderate to severe forms	Risk for long-term side effects No mast cell stabilization Potential for inappropriate patient use Requires close monitoring

Non-steroidal anti-inflammatory drugs

Topical formulations of ketorolac and diclofenac have been shown to diminish ocular pruritus and conjunctival hyperaemia associated with allergic conjunctivitis.

Corticosteroids

Topical corticosteroids are one of the most effective drugs to control thesigns and symptoms of VKC. Because of complications associated with their prolonged use, these should not be prescribed as first-line treatment. Prolonged application of corticosteroids may cause steroid-induced cataract, glaucoma and increase susceptibility to viral and fungal infections.

In comparison to other steroids, loteprednol has a superior safety profile, which has been attributed to its 'soft drug' characteristics. Loteprednol is highly effective in the acute and prophylactic treatment of allergic conjunctivitis.

Supratarsal injection of corticosteroids can be used to treat VKC refractory to conventional treatment.

Although corticosteroids are the most efficacious drugs, steroid-resistant forms of VKC are not unusual and may necessitate an alternative therapy.

Immunomodulators

Topical corticosteroids and artificial tears have

been shown to act synergistically with cyclosporine 0.05% eyedrops and help in the re-epithelialization of corticosteroid-resistant vernal shield ulcers.

Anti-metabolites

Mitomycin-C is an inhibitor of fibroblast proliferation. Mitomycin-C (0.01%) eyedrops were shown to decrease the mucous discharge, conjunctival hyperaemia and limbal oedema in VKC patients refractory to topical steroids and mast-cell stabilizers.

Surgical treatment

Surgical excision of giant papillae is recommended if they cause corneal lesions. Excision or cryocoagulation of large papillae helps in the early resolution of corneal epitheliopathy or ulcer, although papillae regrow in most patients. Cryotherapy of giant papillae promotes inflammation and may cause conjunctival scarring. Intraoperative application of 0.02% mitomycin-C (MMC) to the upper palpebral conjunctiva immediately after papillae resection for 2 min reduces the chances of recurrence of papillae significantly. Free autologus conjunctival graft after resection of giant papillae facilitates the re-epithelializaion of nonhealing shield ulcer.

Debridement of ulcer base, surgical removal of plaque or excimer laser phototherapeutic keratectomy helps in early re-epithelialization of vernal shield ulcer refractory to medical treatment. Amniotic membrane implantation leads to complete re-epithelialization of persistent corneal epithelial defects and vernal plaques recalcitrant to conventional medical treatment. Corneal epithelial cell transplants could be beneficial when amniotic membrane transplant is not sufficient to restore the ocular surface

References

1. Bonini S, Coassin M, Aronni S & Lambiase A Vernal keratoconjunctivitis. Eye (2004) : 18: 345351.

2. Nishiwaki-Dantas MC, Dantas PE, Pezzutti S & Finzi S Surgical resection of giant papillae and autologus conjunctival graft in patients with severe vernal keratoconjunctivitis and giant papillae. Ophthal Plast Reconstr Surg (2000): 16: 438442

3. Sunil Kumar Vernal keratoconjunctivitis:a major review. Acta Ophthalmol (2009):87: 133147

Childhood means simplicity.
Look at the world with the child's eye - it is very beautiful

Review Article

Fugo Plasma Blade and its uses in Ophthalmology

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Fugo blade

Fugo blade is a new operating tool that produces "laser like plasma" on the operating blunt metal wire tip. It is an FDA approved device for capsulotomy, iridectomy and glaucoma (Transciliary Filtration).

How does Fugo blade work?

It focuses electro-magnetic waves to one point, i.e. the tip of the instrument and the energy is tuned to the tissues by the process of resonance. The moment a tissue is touched by the activated tip, the plasma energy gets transferred to the molecules of the tissue. When tissue molecules absorb plasma energy, they go to higher energy levels, thereby becoming unstable. The unstable molecules explode in the same fashion as excimer laser explodes corneal tissue molecules. The exploding molecules carry with them water from the tissue and produce a plume, which gives a peculiar aromatic smell. The molecules/tissue split in the line of incision/ablation, without bleeding, since the blood vessels are also ablated from the path. It is obvious that

Fugo blade provides a new cutting energy, the plasma which makes it different from the electrosurgical devices that we are familiar with. Fugo blade makes it possible to ablate surfaces and to create channels/tracks in one or multiple tissues in one single movement. The plasma is surrounded by orange light, the photonic cloud. The cutting power resides only in the plasma cloud. With this kind of plasma cloud, it is possible to make precisely measured filtration channels. The cutting/ablation by Fugo blade is not accompanied by clinically visible collateral damage. This fact is corroborated by microscopic and electron microscopic studies on the lens capsule, cornea and other tissues. Instrument:

Fugo Plasma Blade comprises of:

- Portable table top model
- Autoclavable hand piece & cable
- Power cord & charger
- Foot pedal & cable
- Individually packaged sterile ablation tips

It works on 4 rechargeable battery cells. Total cut time of one charge is 40 minutes. Numerous glaucoma operations can be done after one charge. Cut power and cut intensity can be adjusted from the console. The width and the power of the plasma are controlled from the controls of "power" and "intensity". The plasma width may be kept at 25, 50 or 75 microns.

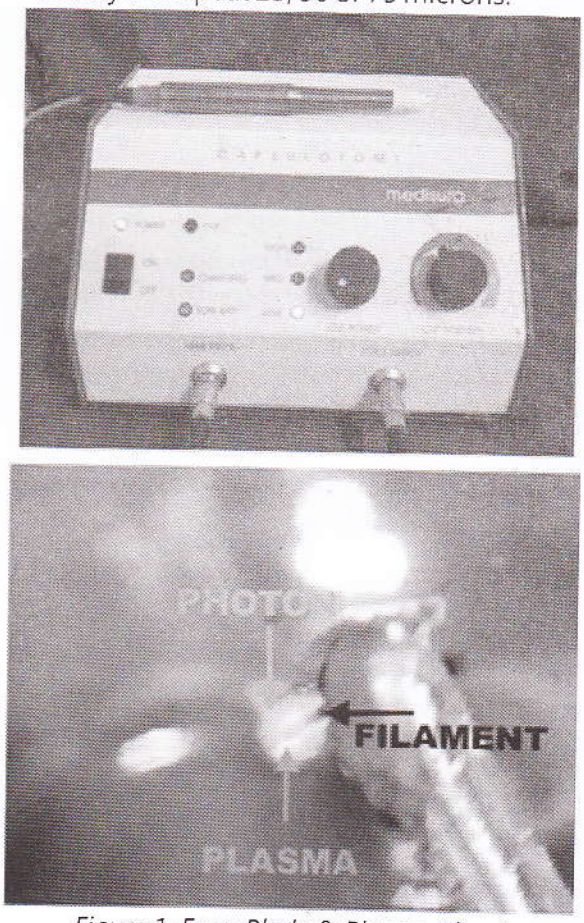

Figure 1: Fugo Blade & Plasma Cloud
Ref: intechopen.com

Uses of Fugo Plasma Blade:

1. **Transciliary filtration (Singh filtration)** with the Fugo plasma blade: The process involves the Fugo Plasma Blade to ablate a small scleral reservoir under a conjunctival flap. Creating a micropore in the posterior chamber allows the aqueous to seep into the reservoir, where it is slowly absorbed. The conjunctival flap is replaced and fastened with a suture. The entire procedure hardly takes a few minutes to perform. It is easy on surgeon and the IOP is lowered with a lower tendency for flat anterior chamber.

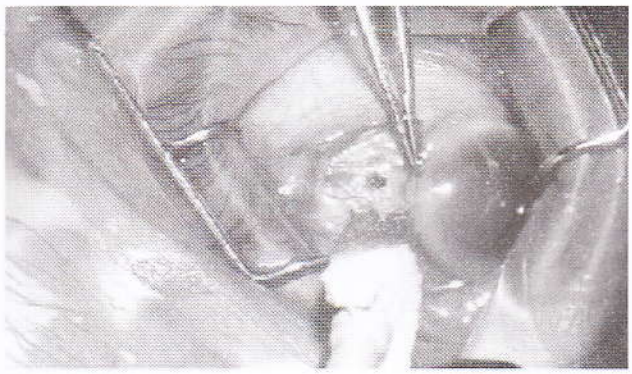

Figure 2: Transciliary Filtration with Fugo Blade
Ref: reviewofophthalmology.com

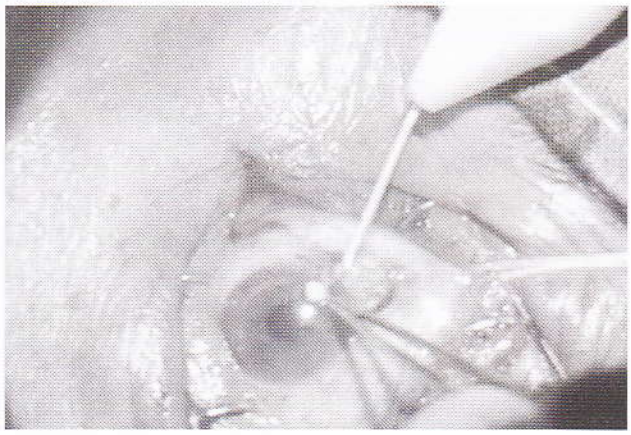

Figure 3: Fugo Plasma Blade Trabeculectomy

2. **Capsulotomy:** Plasma blade capsulotomy2 for cataract surgery was first approved by the FDA in 2000, and it has provided a unique ability to manage difficult cases, as well as an ability to surgically manage capsular tears. The resistance-free ablation is invaluable in cases with weak zonules, dense membranes or small pupils such as in intraoperative floppy iris syndrome. A plasma blade capsulotomy can be performed beneath a penetrating keratoplasty graft, and no postoperative graft decompensation has been reported in more than 10 years with such graft-associated capsulotomies. Fugo Blade can safely and quickly produce resistance-free cuts in corneal tissue in animals, opening additional avenues for use of this device in corneal surgery3. Although performance of an anterior capsulotomy with the Fugo blade was associated with some margin irregularities, the geometry of the centrally directed tags prevented them from becoming the site of radial tear formation.

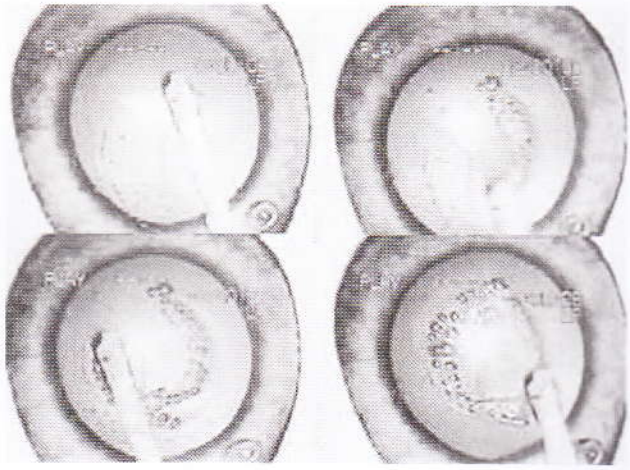

Figure 4: Capsulotomy using Fugo Plasma Blade
Reference: www.ophthalmologymanagement.com

3. Iridotomy and pupilloplasty: Iridotomy is performed by placing the ablation tip at the intended site of iridotomy, and the tip is activated for a second or two, thereby creating a bloodless iridotomy. The size of the iridotomy is under surgeon control. Such an iridotomy can be placed far out in the periphery adjacent to the iris root and can be useful in Visian implantable Collamer lens (STAAR Surgical) surgery. The plasma blade is also useful in performing pupilloplasty.

4. Glaucoma surgeries: Goniotomy ab interno5 using the Fugo Blade was found to be a safe alternative to conventional trabeculectomy,

which safely and effectively reduced intraocular pressure in more than 80% of cases. Besides it has also been found useful in failed trabeculectomy cases to achieve good IOP control. Cases of buphthalmos, resistant forms of glaucoma like neovascular glaucoma has also shown benefits with Fugo Plasma Blade.

Figure 5: Goniotomy ab interno using Fugo Plasma Blade

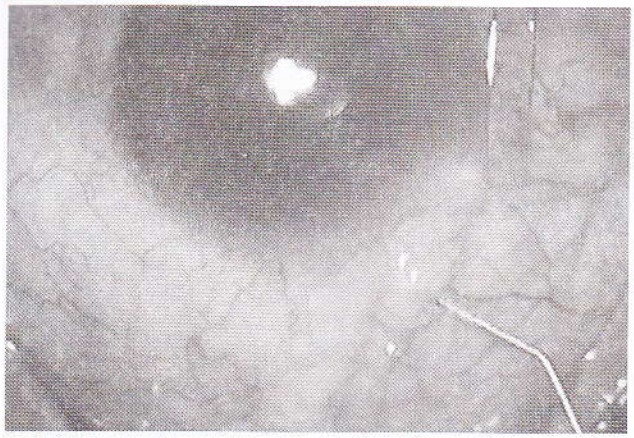

Figure 6: Ab interno Filtration with Fugo Blade
Reference: eyetube.net

5. Excision of skin and ocular masses: Bloodless excision of lid, conjunctival masses can be easily performed with Fugo Blade. Besides limbal dermoid, hemangioma, cysticercus, nevus have also been successfully removed using Fugo Plasma Blade.

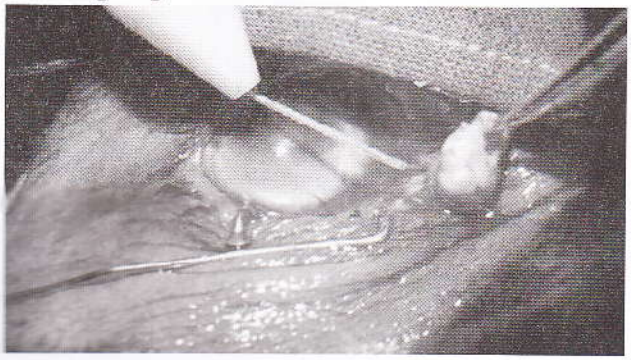

Figure 7: Conjunctival mass excision using Fugo Blade

6. Squint surgery: Distinct advantages for using the Fugo Blade in squint surgery were increased surgeon control, bloodless field and decreased operative time.

7. Entropion surgery, destroying roots of cilia to correct misdirected eyelashes, Pterygium excision, DCR surgery, and vitrectomy with Fugo Plasma Blade have all shown promising results.

Figure 9: Entropion Surgery using Fugo Blade
Reference: youtube.com

References:

1. Singh D, Singh K. Transciliary filtration using the Fugo Blade. Ann Ophthalmol. 2002;34(3):183-187.

2. Fugo RJ, DelCampo DM. The Fugo Blade: the next step after capsulorhexis. Ann Ophthalmol. 2001;33(1):12-20.

3. Peponis V, Rosenberg P, Reddy SV, Herz JB, Kaufman HE. The use of the Fugo Blade in corneal surgery: a preliminary animal study. Cornea. 2006;25(2):206-208.

4. Izak AM, Werner L, Pandey SK, Apple DJ, Izak MG. Analysis of the capsule edge after Fugo plasma blade capsulotomy, continuous curvilinear capsulorhexis, and can-opener capsulotomy. J Cataract Refract Surg. 2004;30(12):2606-11

5. Singh D, Bundela R, Agarwal A, Bist HK, Satsangi SK. Goniotomy ab interno "a glaucoma filtering surgery" using the Fugo Plasma Blade. Ann Ophthalmol . 2006;38(3):213-7.

6. Singh D, Singh RS, Kaur H, Singh K, Singh I, Singh R. Plasma powered squint surgery with the Fugo blade. Annals of Ophthalmology. 2003; 35(1):12-4

Laser Photocoagulation for Diabetic Macular Edema

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Diabetic macular edema (DME) remains a significant cause of central vision loss in patients with diabetes mellitus. DME is defined as retinal thickening involving the macular area. The edema is caused primarily by a breakdown of the inner blood retinal barrier resulting in leakage of fluid and plasma constituents from abnormally permeable micro-aneurysms, intraretinal microvascular anomalies and dilated retinal capillaries. The visual loss may also be worsened by capillary closure involving the foveal capillary arcade, so called ischemic maculopathy.

Retinal photocoagulation basically involves thermally damaging the abnormal tissues in retina to produce their destruction or inducing adhesions. In this article we will basically discuss the practical & basic techniques of photocoagulation and the proper settings of laser parameters to produce the desired effect and minimize the side effects of laser application. Before we discuss these aspects, it is important to know the fundamentals of retinal photocoagulation.

Mechanism of action

Photocoagulation uses light to coagulate tissue. When energy from a strong light source is absorbed by tissue and is converted into thermal energy, coagulation necrosis occurs with denaturation of cellular proteins as temperature rises above 65 degrees C.

Since the Diabetic Retinopathy Study, technology evolved from using a diffuse Xenon arc to using well-focused laser in photocoagulating retinal tissue in high risk proliferative diabetic retinopathy. Presently, laser retinal photocoagulation is a therapeutic option in several retinal and eye conditions.

Effective retinal photocoagulation depends on how well light penetrates the ocular media on its way to the retinal tissue and how well the light is absorbed by pigment in the target tissue. In retinal tissue, light is absorbed by melanin, xanthophyll or haemoglobin.

Melanin absorbs green, yellow, red and infrared wavelengths; xanthophyll (in the macula) absorbs blue but minimally absorbs yellow or red wavelengths; hemoglobin absorbs blue, green and yellow with minimal red wavelength absorption.

In the ETDRS study, laser photocoagulation reduced the risk of moderate visual acuity loss for all eyes with DME and mild to moderate Non-proliferative diabetic retinopathy by approximately 50%.

Various parameters of photocoagulation

i) Power(its unit is milliwatts or mW)- power needed varies with host of other parameters-

a) Spot size- Larger spot size needs larger power to cause similar photocoagulation effect e.g. with 100µ spot size, just 100 mW power might suffice while if spot size is increased to 200µ, power required would be greater, say 200 mW to cause similar photocoagulation effect.

b) Duration of exposure- Larger exposure time means (normally 0.1 sec is used for average setting) less power required.

c) Clarity of media- hazier media e.g. due to cataract or vitreous hemorrhage means more power required.

d) Pigmentation (melanin) of patient's fundus- Pigmented eyes need less power than Caucasian eyes. Moreover within patient's own fundus,

pigmentation varies requiring variable power for different areas. Macular region has maximum melanin pigment and hence absorbs greater energy than surrounding paramacular regions.

ii) Spot size- Already mentioned. Simple rule of thumb in FD-YAG (Frequency Doubled) or Argon green laser with exposure time set at 0.1 sec is to use same number of milli Watts power as the spot size in microns (i.e. for 100ì spot size at 0.1 sec exposure time, 100 mW is the average power required; practically however as the machines get older, it needs higher power to cause similar effect). Larger spot sizes destroy greater retinal thickness.

iv) Wavelength (colour) used- Wavelengths most commonly used are Green as in Frequency-Doubled YAG (FD-YAG with 532 nm) & Argon green (515 nm), Infrared in Diode (810 nm). Now rarely used are krypton red (647 nm), krypton yellow (568 nm). Argon blue (480 nm) is no more used.

When to Treat DME?

The goal of macular laser photocoagulation is to limit vascular leakage through focal laser burns of leaking microaneurysms or grid laser burns in areas of diffuse breakdown of blood retinal barrier. Laser treatment should be considered for patients with clinically significant diabetic macular edema (CSME). Macular edema is considered clinically significant if any oneor any combination of the following is observed :

- Retinal thickening at or within 500 microns from the center of macule.
- Hard exudates at or within 500 microns of the foveal center, if associated with thickening of the adjacent retina.
- A zone or zones of retinal thickening one disc area in size, at least part of which is within one-disc diameter of the foveal center.

How to Evaluate a Patient before Laser Photocoagulation?

CSME is a clinical diagnosis made with slit-lamp biomicroscopy. Macular edema is best evaluated by dilated eye examination using slit-lamp biomicroscopy with a 78 or 90 D lens and/or stereo fundus photography. It is seen as a thickening of the macula with blurring of the underlying choroidal pattern, loss of foveolar light reflex when fovea is involved and presence of cystoid spaces in severe cases.

Fluorescein angiography (FA) prior to laser for CSME is helpful for identifying areas of focal and diffuse leakage and for identifying pathologic enlargement of the foveal avascular zone (ischemic maculopathy), which may be useful in planning treatment.

It is helpful to have the angiogram printout or digital display in the laser room to better direct therapy.

Ocular coherence tomography (OCT) is desirable and should always be done if available. It is helpful to detect subtle edema, quantify it, look for serous macular detachment and to follow the course of edema after treatment.2

Advantages of Laser photocoagulation

Photocoagulation decreases the release of vasoproliferative factors by conversion of hypoxic foci into anoxic areas and leaking vascular anomalies into inert scars.

This relieves the retina of edema and improves its function and also causes the regression of new vessels, inhibiting further hemorrhages.

The procedure does however save the center of the patient's sight. Laser may also slightly reduce colour and night vision.

This treatment slows the growth of new abnormal blood vessels that have developed over a wide area of the retina.

Decision Making

Points that need to be considered before

treatment are

- **Fluorescein Angiography**

Is there discrete or deep diffuse leakage ?

Is there macular ischemia ?

- **Optical Coherence Tomogram**

Are there vitreomacular interface anomalies?

How thick is the macula? Is there a subclinical serous macular detachment?

- **What is the visual acuity**

Laser treatment works best in eyes with discrete areas of leakage on FA

- Mild to moderate thickening on OCT
- No vitreo-macular interface abnormalities on OCT
- Visual acuity good or with only a mild to moderate decrease

Pharmacotherapy works best in eyes with -

- diffuse leakage on FA with moderate to severe thickening,
- cystoid macular oedema (CME) or a serous macular detachment on OCT,
- moderate to severe decrease in visual acuity

Systemic work- up

Control of diabetes (Blood sugar, HbA1c),

- Blood pressure
- cholesterol is essential for the success of this treatment.

The recommended values for HbA1c, B.P. and LDL Lipoproteins are <7%, <130/80 mm Hg and < 100mg/dl respectively.

Treatment considerations

Informed Consent: should be obtained from every patient, stressing that the treatment is designed to stabilize or slow the rate of vision loss rather than to improve vision.. Patients with very good vision (ie 6/6 visual acuity) should also be treated if they have CSME. Treatment is unlikely to restore the visual acuity once it goes down, so treatment prior to visual loss is sometimes appropriate. The patient should be informed about the need for periodic follow up, repeated FA/OCT and the possibility of more laser treatment. Eyes with CSME which need PRP for severe NPDR/PDR should always undergo treatment for the macular edema first followed by panretinal photocoagulation (PRP) after 4-6 weeks.

Choice of lens-Laser photocoagulation requires a contact lens which is placed in the eye under topical anaesthesia with the aid of a viscous solution like goniosol or methylcellulose eye drops.

The Area-centralis lens and Mainster standard (now called Mainster Focal/Grid) are excellent lenses for macular photocoagulation.

Patient fixation is used to locate the foveal center.

Focal vs Grid laser

Local treatment for CSME consists of direct focal treatment, grid treatment or a combination of direct and grid treatments. If the oedema is diffuse, grid laser is applied. If the leakage affects a small part of the macula, as in circinate retinopathy, focal laser is applied.

Focal laser

All focal leaks (mainly microaneurysms) located between 500µ -3000µ from the centre of the macula that contribute to retinal thickening and/or lipid exudates are treated directly with 100µ spots at 0.1 sec duration to produce a very light subtle white burn. Try a test burn in a non-critical paramacular area with increasing power to determine power settings. The usual starting point is 80 to 200 mw. The end point of laser is an immediate blanching of larger microanurysms.

Laser may induce changes in the metabolic activity of the retinal pigment epithelium and cause the release of signals that reduce vascular leakage and facilitate fluid absorption. Although spot sizes as small as 50 µm were used in the ETDRS 5 , a 50-µm spot increases the power density and may increase the risk of breaks in Bruch's membrane and secondary choroidal neovascularization. If macular oedema persists on follow up examination,focal leaks located within 300-500 from the centre may also be treated provided there is a good perifoveal network.

Grid treatment

This type of laser is aimed directly at the affected area or applied in a contained, grid-like pattern to destroy damaged eye tissue and clear away scars that contribute to blind spots and vision loss. This method of laser treatment generally targets specific, individual blood vessels.

All areas of thickened retina within the arcades that show diffuse fluorescein leak or capillary dropout are treated with 100µ -200µ spot size placed one burn width apart at 0.1sec duration. The end point of each laser burn used in a grid pattern is a light intensity barely visible burn. Low power (often 80-100 mw) is needed for pseudophakia and clear media; higher power is needed if there is a cataract or marked retinal thickening. (Usually <180mw). It is difficult to know how heavy to make the laser, but burns that do not show on a fluorescein angiogram later were probably too light, so you can tell retrospectively. Burns that were too heavy cause significant atrophy. Even 'perfect' burns may produce very slight pigmentation, but significant pigmentation indicates excessive power. Usually ideal burns are visible 1 minute after the laser. The laser burns must be at least 500µ from foveal centre and 500µ from disc margins. Treatment within the papillomacular bundle is usually avoided. Any focal leaks within the zone of grid treatment are treated focally. In cases where the oedema is limited to certain sectors of the macula, it is important that the laser is not done all over the macula but only in the thickened areas showing dye leak (Modified Grid).6

Re-Treatment

Revaluate the patient every 4 weeks. If after 8-12 weeks, there are areas of thickening (i.e. edema) still present; give a little touching to all leaking areas (repeat FFA is done before this) upto 250 microns from the foveal centre. Lipid exudates may initially increase as fluid is reabsorbed and precipitation of lipid occurs. Exudates can also persist for many months after absorption of fluid; therefore associated thickening must be present if re-treatment is considered. Most patients require more than one treatment session (one to three on an average), for macular edema to resolve. CSME requiring more than 3 treatment sittings becomes recalcitrant and requires alternative pharmacological agents.

Table 1: Clinical Guidelines

Clinically Significant Macular Edema (CSME) Treatment

First-line therapy

- Focal or modified ETDRS grid photocoagulation for focal or diffuse CSME
- Intravitreal pharmacotherapies ± photocoagulation for more advanced, diffuse CSME

Repeat photocoagulation

For persistent or recurrent CSME (visual acuity <20/40) Intravitreal triamcinolone acetonide or intravitreal antivascular endothelial growth factor (VEGF) agent

For CSME refractory to photocoagulation and intravitreal pharmacotherapies, consider pars plana vitrectomy (PPV)

No traction: PPV with internal limiting membrane (ILM) peeling

Taut posterior hyaloid face or vitreomacular traction syndrome:PPV

Complications

- Photocoagulation of the fovea-This is the most-feared complication of macular laser treatment. Loss of foveal function can occur after direct laser treatment of the fovea or from "creep" of a juxtafoveal laser scar into the fovea.
- Secondary CNVM-A larger spot size decreases the power density and reduces the risk of this complication. We always start at the lowest power setting and gradually increase until the lightest color change is observed.
- Paracental scotomas- Avoided by lower-intensity spots.
- Mydriasis due to sphincteric damage if pupil was not well dilated or due to damage to nerves in uveal tract. It is usually permanent.
- Paralysis of accommodation- usually temporary

References

1. Photocoagulation for diabetic macular edema. Early Treatment Diabetic Retinopathy Study reports number 1. Early Treatment Diabetic Retinopathy Study Research Group. Arch Ophthalmol 1985; 103:1796-1806
2. Rivellese M, George A, Sulkes D, Reichel E, Puliafito C. Optical coherence tomography after laser photocoagulation for clinically significant macular edema. Ophthalmic Surg Lasers. 2000; 31(3):192-7.
3. Jonas JB, Kreissig I, Sofker A, Degenring RF. Intravitreal injection of triamcinolone for diffuse diabetic macular edema. Arch Ophthalmol 2003; 121:57-61.
4. Aroca PR, Salvat M. Risk factors for diffuse and focal macular edema. Diabetes Complicat. 2004; 18(4):211-5.
5. Treatment techniques and clinical guidelines for photocoagulation of diabetic macular edema. Early Treatment Diabetic Retinopathy Study Report Number 2. Early Treatment Diabetic Retinopathy Study Research Group. Ophthalmology 1987; 94(7):761-74.
6. Olk RJ. Modified grid argon (blue-green) laser photocoagulation for diffuse diabetic macular edema. Ophthalmology. 1986; 93(7):938:950.

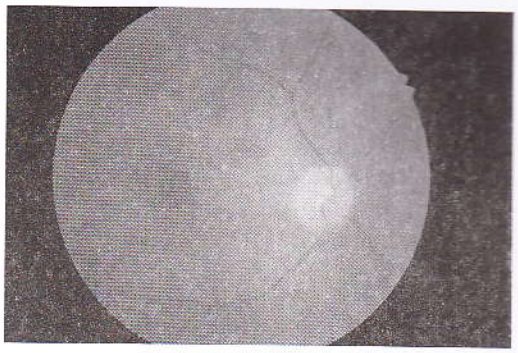

Figure-1 showing CSME

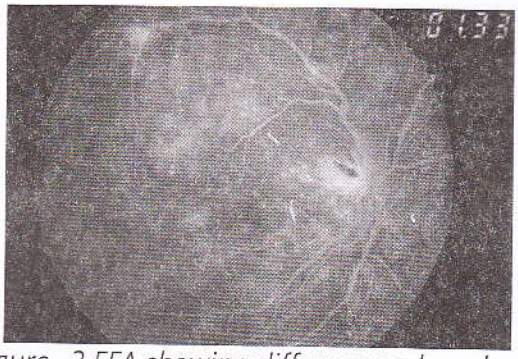

Figure -2 FFA showing diffuse macular edema

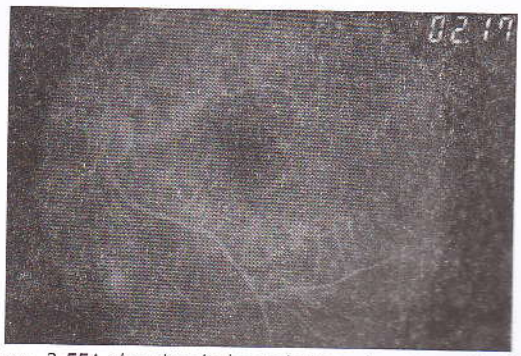

Figure -3 FFA showing ischaemic Maculopathy with IRMA

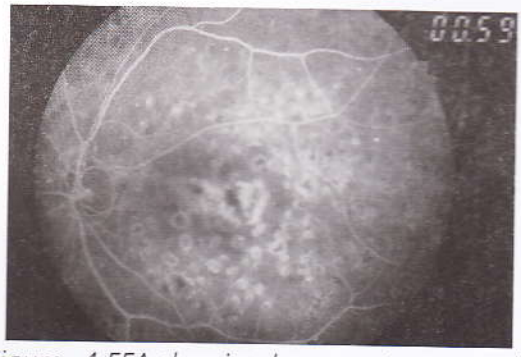

Figure -4 FFA showing laser spots at macula

Laser Iridotomy (LI)

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Laser Iridotomy is the creation of full thickness opening in the iris using photodisruptive property of lasers. It has become the procedure of choice in all forms of angle closure due to relative or absolute pupillary block, which permits equalization of pressures in anterior and posterior chambers.

Indications for laser Iridotomy

Therapeutic

- Acute angle closure glaucoma - after termination of attack by medical therapy
- Subacute angle closure glaucoma
- Chronic angle closure glaucoma
- Combined-mechanism glaucoma
- Pupillary block glaucoma including subluxated lens
- Iris bombe due to secclusio pupillae
- Iridovitreal block after cataract surgery
- Imperforate surgical iridectomy
- Before argon laser trabeculoplasty in eyes with narrow angles

Prophylactic

- Occludable angle
- Fellow eye with either acute or chronic angle closure glaucoma
- Fellow eye after complicated surgical iridectomy
- Patient with silicone oil placed in the eye inferior iridectomy is performed
- Pigment Dispersion Syndrome
- Nanophthalmos

Diagnostic

- Plateau iris
- Aqueous misdirection syndrome

Contraindications

- Uncooperative patient and unable to sit at the slit lamp
- Corneal opacities and edema
- Chronic inflammation
- Hazy and flat anterior chamber
- Widely dilated pupil
- Presence of 360° peripheral anterior synechiae
- An eye with active rubeosis iridis risk of bleeding
- Patient taking systemic anticoagulants, including aspirin

Preoperative considerations

- An informed consent
- Well controlled IOP - Pretreatment with apraclonidine 0.5% or brimonidine 0.2% can help blunt IOP spikes.
- Cornea should be clear - Corneal edema may be improved by pretreatment with topical glycerine.
- Pupil must be constricted to stretch and thin the iris - a drop of pilocarpine 1% is instilled two to three times at 10 minutes interval.
- If the pressure is high despite topical medications and oral acetazolamide, use intravenous mannitol to lower the tension.

Operative technique

Topical anesthesia is applied in the form of 4% Xylocaine. An Abraham's type of contact lens is applied. This lens has a +55 D peripheral button over a routine contact lens.

Advantages of contact lens

- Stabilizes the eye and keeps the lid retracted during the procedure.
- Acts as a heat sink, decreasing the number of epithelial corneal burns.
- Smoothens out the corneal surface.
- Provides highly magnified peripheral view.
- Helps to reduce the axial expansion of plasma which reduces the unnecessary spread of damage.
- Increases the power density of the spot by factor of 4, thus facilitating the production of a full thickness iris hole.

Settings

Argon laser : Long pulses (0.2 seconds) for light-colored irides and short pulses (0.020.05 seconds) for dark brown irides. Power of 1000 mW and a spot size of 50 μm.

Nd : YAG Laser : The Q-switched mode is used. Iris blood vessels are avoided. The iridectomy spot may be placed anywhere between the 11 and 1 o'clock positions. The red laser-aiming beam is brought to a focus when the multiple beams are brought into a single spot, aimed through the center of the contact lens. The energy used is 38 mJ, there are 13 pulses per shot, and one or more shots are used as required for penetration

Argon vs Nd-Yag

Nd-YAG laser has replaced all other lasers as far as iridotomy is concerned. The Nd:YAG laser does not coagulate tissue (like argon laser which has photocoagulation effect) and small hemorrhages occur more frequently with this modality. Therefore, in eyes that have prominent unavoidable vessels or in patients affected by a bleeding diathesis, combined treatment is preferred, first with the argon laser (to ablate vessels in the area) and then with the Nd:YAG laser (to establish a patent peripheral iridectomy). Sometimes this combination may be required in eyes with thick brown iris.

Choice of iridotomy site

- Upper nasal quadrant under lid - to prevent "second pupil" effect
- As close to limbus as possible - less likely to damage the lens
- Aim to hit the spot in a crypt where the thickness is much less.
- One iridotomy, if patent, is usually sufficient. However, in patients with uveitis, multiple iridotomies are suggested because of high failure rate.

End point

Once the iridotomy is complete, one can notice a sudden outflowing of the pigment from the posterior to anterior chamber along with sudden deepening of anterior chamber.

- The presence of retro illumination may not be a sure sign of total penetration.
- The minimum recommended diameter of LI is around 150 to 200 µm.

Postoperative management

- Antiglaucoma medications - should be continued along with an additional anti glaucoma agent for 1 week.
- Steroids three times/Day for 4 days to control inflammation.
- Pilocarpine 1% drop thrice a day to keep the iris stretched and iridotomy patent.
- Check IOP after 24-48 hours and patency is confirmed at slit lamp.
- Gonioscopy at 6 weeks, to see the opening up of angles.

Advantages over Surgical Iridectomy

- Safe, effective, OPD procedure.
- Lesser rate of complications such as serious intraoperative hemorrhage and cataract.
- Avoids complications like endophthalmitis, wound leak, flat anterior chamber and problems related to anesthesia.

Complications

1) Transient increase in IOP antiglaucoma drug must be added.
2) Microhemorrhages -. Not serious, easily controlled by applying pressure with contact lens for a few seconds
3) Uveitis because of pigment dispersal and a result of irritation to the iris rather than a specific iritis. Steroids antibiotic combination should be started three times for atleast 3-4 days.
4) Closure of iridotomy - due to accumulation of pigment granule or debris, give pilocarpine for 4 to 6 weeks.
5) Corneal damage - heal quickly without sequelae.
6) Cataract formation : because of direct damage from laser irradiation. Iridotomy should be performed close to limbus.
7) Retinal burns very rare. Minimized by always aiming beam towards peripheral retina and by using Abraham lens.
8) Monocular blurring - If iridotomy is not fully covered with upper lid, patient may complain of monocular blurring.

Other complications include lens capsule rupture,

Correction of pre-operative astigmatism by on-axis clear corneal incision in Phacoemulsification

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•**Sanjiv Kumar** MS, •**Charu Jain** MS, •**Rishi Jhalani** MS
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The two main determinants of the refractive state of the eye following phacoemulsification and intraocular lens implantation are IOL power and surgically induced astigmatism

(SIA). Factors such as wound location and architecture are important predictors of SIA.1-2 Studies on the use of 3.5-mm to 4-mm corneal incisions in phacoemulsification have led to the conclusion that superior and nasal locations induce greater refractive changes than temporal corneal incision,3-4 and this effect could be intentionally used by the surgeon to reduce or treat preexisting astigmatism.5 Recent evidence indicates that a small 2.8-mm clear corneal incision induces little refractive change, at least in eyes with low preoperative corneal cylinder, regardless of incision site.6 However, a retrospective study describes larger changes induced by superior rather than temporal 2.8-mm incision, which had been considered nearly astigmatism neutral. 7 This study compares the short and intermediate term astigmatic outcomes of temporal versus superior clear corneal on-axis phacoemulsification incisions.

METHODS

This prospective clinical trial included a consecutive series of 60 patients with senile cataracts with 30 patients scheduled for Phacoemulsification with foldable IOL with a superior incision (group A) & 30 patients with temporal incision (Group B). Inclusion criterion was astigmatism of upto 1D . Exclusion criteria consisted of previous ocular surgery, presence of corneal pathology, pseudoexfoliation and astigmatism (WTR or ATR) exceeding 1 diopter (D). Patients with diabetes mellitus, connective tissue disease and those taking systemic steroids were also excluded. All procedures were performed at Subharti Medical College, Meerut, India by a single experienced surgeon using the same technique under topical anesthesia.

Based on the axis of the cylindrical component of refractive error according to keratometric reading, superior (for with-the-rule astigmatism) or temporal (for against-the-rule astigmatism) a triplanar, 2.8 mm horizontal corneal Incision was constructed . First a half-depth perpendicular groove incision was made using a 15° stab knife followed by lamellar dissection 2.0 mm into clear cornea by using a sharp crescent knife, entry is made with the help of a disposable 2.8mm keratome. After phacoemulsification and cortex removal, a 6 mm hydrophobic acrylic foldable IOL was inserted using an injector system, the anterior chamber was formed and the wound was checked for leakage and left unsutured.

Postoperative examinations were conducted 1, 2, 7 and 28 days, and 6 months after the procedure. Uncorrected visual acuity and best corrected visual acuity (BCVA) were measured and keratometry was performed at each followup visit. Main outcome measures included keratometric astigmatism and SIA calculated by the vector analysis formula using the Holladay-Cravy-Koch formula. Data were compared between the two study groups using Mann-Whitney and t-tests with statistical significance set at 5%.

RESULTS

Overall 60 eyes of 60 patients including 34 (56.7%) female and 26 (43.3%) male subjects with mean age of 64.6 ± 9.2 (range 52-85) years underwent clear corneal phacoemulsification and intraocular lens implantation . The two study groups did not differ significantly in terms of age . Consequently 30 eyes underwent temporal phacoemulsification and 30 eyes underwent superior phacoemulsification.

Mean preoperative keratometric astigmatism was 0.80±0.28 D in the superior and 0.68±0.29 D in the temporal group (P=0.08). SIA at 1 week, 4 weeks and 6 months postoperatively was 0.96±0.17 D versus 0.75±0.15 D (P<0.001), 0.83±0.12 D versus 0.69±0.11 D (P<0.001), and 0.73±0.15 D versus 0.51±0.10 D (P<0.001) in the superior versus temporal groups respectively. In both groups the amount of SIA at sixth months was much lower than the initial visit (P<0.001).Mean keratometric

astigmatism at 6 months postoperatively was 0.07±0.03 D versus 0.17±0.12 D. Despite the significant differences in keratometric and surgically induced astigmatism, BCVA was comparable in both the study groups.

DISSCUSION

Patients undergoing cataract surgery expect clear vision and less dependence on spectacles.

To attain this goal, one important consideration is reduction of astigmatism. Different methods have been used to correct pre-existing astigmatism during cataract surgery. The simplicity and usefulness of clear corneal on-axis incisions cannot be assessed without considering the other options of astigmatic correction in cataract surgery. Astigmatic keratotomy, is an alternative which entails drawbacks such as glare sensation, diplopia and fluctuation of refractive error due to proximity of the incisions to the center cornea. In addition, it requires preoperative pachymetry and use of a diamond knife.8 Corneal relaxing incisions are another method for correction of pre-existing corneal astigmatism. However, this method also suffers from limitations such as requiring pachymetry and use of a diamond knife.9 Implantation of toric IOLs is another option, however these lenses are expensive and their implantation requires additional skills; moreover, postoperative rotation remains a major drawback. Excimer laser ablation may also be used to correct residual or induced astigmatism after cataract surgery. Major concerns include the cost of the procedure, limited number of centers equipped with excimer machines, adverse effects specific to excimer laser surgery such as loss of BCVA, flap related complications, night vision disturbances and regression.10Tejedor J,et al reported that Temporal corneal incisions created less surgically induced astigmatism than those placed superiorly ,mean degree of surgically induced astigmatism was <0.25 diopters for temporal incisions & between 0.25 to 0.75 diopters for superiorly placed incisions. The results obtained in the current study are in a marginally higher range than previously reported.11

CONCLUSION

Keeping in mind the expected degree of corneal astigmatism created by 2.8mm keratomes will allow surgeons who use this size incision to estimate the effect of the incision itself on the corneal curvature. When patients have pre-existing astigmatism and it is desired to minimize the degree of postoperative astigmatism, performing surgery in the steep meridian can be beneficial. Knowledge of these principles is also useful in the preoperative assessment and surgical decision-making when using toric intraocular lenses. In conclusion, clear corneal on-axis incisions are useful for correcting mild to moderate pre-existing astigmatism during cataract surgery. Employing this technique during routine phacoemulsification using a 2.8mm mm incision does not require additional instruments and therefore can be performed without altering the surgical setting.

To summarise, in eyes with mild to moderate astigmatism , on-axis CCI phacoemulsification lead to partial to complete correction. Hence it can be utilized as a simple tool to correct mild to moderate astigmatism & also provides excellent UCVA.

REFRENCES

1. Koch DD, Lindstrom RL. Controlling astigmatism in cataract surgery. Semin Ophthalmol 1992;7:224-233.
2. Kohnen T. Corneal shape changes and astigmatic aspects of scleral and corneal tunnel incisions. J Cataract Refract Surg 1997;23:301-302.
3. Mendívil A. Comparative study of astigmatism through superior and lateral small incisions. Eur J Ophthalmol. 1996;6:389 392.
4. Roman S, Ullern M. Astigmatisme induit par les incisions corne´ennes supe´rieures et temporales dans la chirurgie de la cataracte. J Fr Ophthalmol. 1997;20:277283.
5. Tejedor J, Murube J. Choosing the location of corneal incision based on preexisting astigmatism in phacoemulsification. Am J Ophthalmol. 2005;139:767776.
6. Giasanti F, Rapizzi E, Virgili G, et al. Clear corneal incision of 2.75 mm for cataract surgery induces little change of astigmatism in eyes with low preoperative corneal cylinder. Eur J Ophthalmol. 2006;16:385393.
7. Marek R, Klu´s A, Pawlik R. Comparison of surgically induced astigmatism of temporal versus superior clear corneal incisions. Klin Oczna. 2006;108:392396.
8. Lindstrom RL, Lindquist TD. Surgical correction of postoperative astigmatism. Cornea 1988;7:138-148.
9. Nichamin LD. Astigmatism control. Ophthalmol Clin N Am 2006;19:485-493.
10. Stojanovic A, Nitter TA. Excimer laser in the treatment of myopic astigmatism; outcomes of laser in situ keratomileusis and photorefractive keratectomy. J Cataract Refract Surg 2001;27:1263-1277
11. Tejedor J, Murube J. Choosing the location of corneal incision based on preexisting astigmatism in phacoemulsification. Am J Ophthalmol. 2005;139:767776.

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Comparison of Subconjuhnctival and Intrastromal injection of Bevacizumab in the treatment of Corneal Neovascularisation in cases of penetrating Keratoplasty — ***Dr Swati Chawla***

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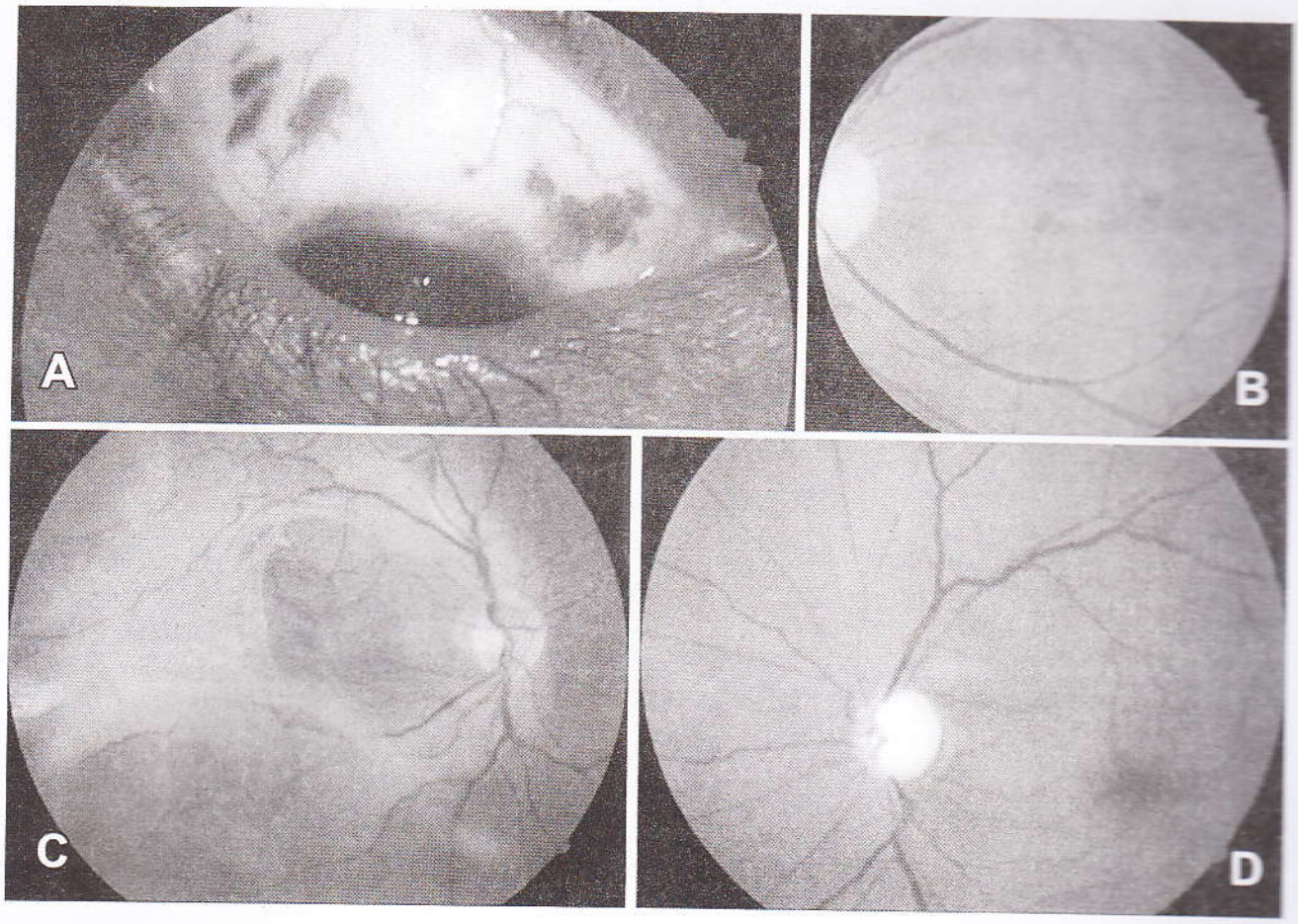
Answer to Ophthalmic Quiz - 3



A Nevus

B Sick RPE

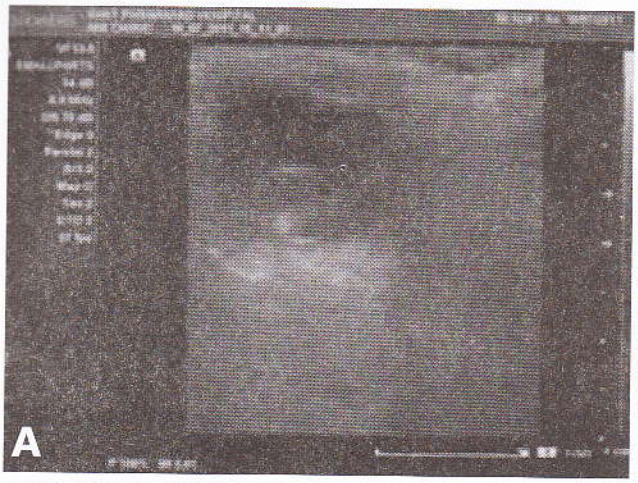
C Epimacular Membrane

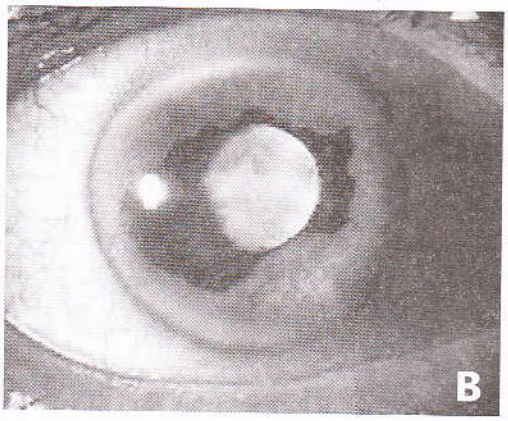
D Glaucomatous Optic Atrophy

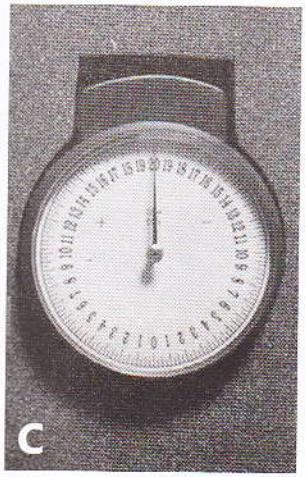
The Lucky Winner of this is **DR. SHUBHAM ROY,** Agra
He would receive a Certificate from our society.

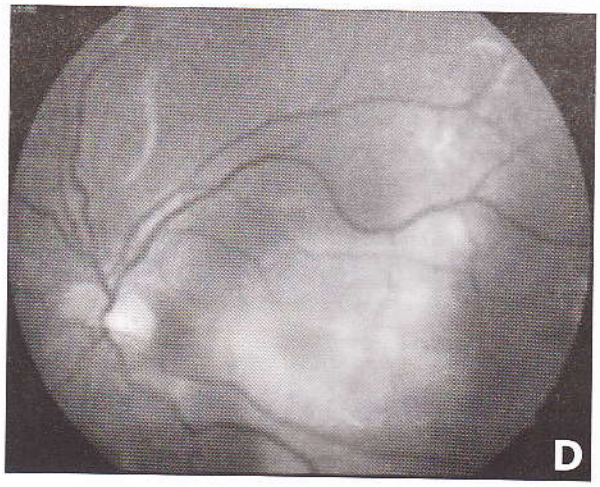
The Ophthalmic Quiz - 4

Send your answers by post to Dr. Gunjan Prakash, Editor, UPJO, Department of Ophthalmology, SNMC, Agra. Mobile 9319204073 or email at **editorupjo@rediffmail.com**. **Do not forget to mention your UPSOS Membership Number.** One of the lucky UPSOS member, contributing all the correct answers would get a **Certificate** from UPSOS , sponsored by **SPG Medicare & Diagnostics, Kamla Nagar, Agra.** Last Date to send your answers is 31st August 2015.









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C ..... D .....

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