The Ocular Communique

The Journal of the Maharashtra Ophthalmological Society

Editor : Dr. B. K. Nayak

Volume 7, Issue 1. Sept - Dec, 2010

JOURNAL OF MAHARASHTRA OPHTHALMOLOGICAL SOCIETY

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Vol. 7 No. 1, Sept - Dec 2010

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For books: Mandel Wanger etal,Atlas of corneal diseases, W.B. Sanders, First edition, 1989, 80-2.

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This journal is edited, printed and published by Dr. B. K. Nayak on behalf of Maharashtra Ophthalmological Society.

Cover designing by Mr. Rajesh Dave from Surgicon

Printing by : Balaji Printers, 6 / 4, Khimji Nagji Chawl, S. B. Marg, Opp. Phoenix Tower, Lower Parel (W), Mumbai - 400 013. Tel.: 2431 1963, Mbl.: 98201 48846, E-mail : lokarts@gmail.com

JOURNAL OF MAHARASHTRA OPHTHALMOLOGICAL SOCIETY

Vol. 7 No. 1, Sept - Dec, 2010

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Editorial

Dear Colleagues,

Greetings from the Editorial Board of JOMOS.

Our journal is entering into the 7th year of its existence. Infancy is over and now it is high time to take a quantum leap in future. The editorial board is committed to take it to a new height. You will see a complete make over of this journal in the coming year. The name, appearance and above all the quality of content will change. All these steps are essential to get it indexed so that it becomes a global journal and economically viable. It is challenging but an achievable Goal. We are following the motto “Think global, act local” I seek your blessings and support in this endeavour.

Any constructive suggestion or feedback will be highly appreciated.

Thanking you,

Dr.B.K.Nayak
Editor-in-Chief
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Original Article

Estimation of the Anterior Chamber Depth in Indian Adults by Smith's Technique Using Slit-Lamp Bio Microscopy

Dr. Amit J. Shinde, Dr. Parikshit M. Gogate

Abstract

Anterior chamber depth (ACD) estimation is needed in clinical practice to help find shallow chambers before cataract surgery and to diagnose angle closure glaucoma. The photographic, ultrasonic and optical methods to estimate ACD rely on instrumentation. Using the Smith's method we can easily measure the ACD using a simple slit-lamp. The method involves increasing the length of a horizontal slit focused on the cornea until the corneal and iris / lens images appear to just touch (JTSL), the illumination system angle is kept at 60º. 77 eyes had their anterior chamber depth estimated using the Smith's technique and then correlated with ultrasonic anterior chamber depth. There was a positive correlation (r=0.97) and JTSL multiplied by 1.4 gave the ACD. This is an easy and inexpensive method to estimate ACD in an eye clinic and can be followed during a routine ocular examination.

Introduction

Anterior Chamber Depth (ACD) positively correlated with anterior chamber angle.[1] Eyes with shallow ACD and narrow anterior chamber angles are more prone to develop primary angle closure glaucoma and drug-induced acute glaucoma. There are various methods of measuring the ACD, e.g. photographic, ultrasonography and optical. [2-4] Angle closure glaucoma is common in Indian population and is often under-diagnosed. [5,6]Ideally gonioscopy needs to be performed by optometrists and ophthalmologists on all patients to identify a significant narrow angle.[7] The procedure however demands skill and patience on part of the eye care practitioner and is not very comfortable for the patient. Hence it is not routinely used in all office practices.

The Smith's technique for the estimation of anterior chamber depth relies on only the slit-lamp and is non-invasive.[8] It was first propounded in 1978 and is fairly easy to perform in an eye clinic to identify low ACD value, narrow angle of anterior chamber and angle closure glaucoma.[8]

There is few published data on estimation on anterior chamber depth in Indian population. We set out to find out whether Smith's technique gives correct estimation about anterior chamber depth in Indian adults. The study aimed to find out the correlation between “Just Touching Slit Length” (JTSL), ACD as estimated by ultrasonography and the conversion factor between them.

Method:

The study was done at a tertiary community eye care centre and an private ophthalmology practice in Pune. Inclusion criteria was all patients coming in out-patient department (OPD), but patients with corneal opacity and those optical media in anterior chamber were not clear were excluded.

In 1978-79 Smith propounded a technique which uses the slit lamp without any attachment and in which the corneal thickness doesn't affect ACD estimation.[8] The refraction of light take place through the cornea, but this is affected by the angle of illumination. The method involves increasing the length of a horizontal slit focused on the cornea until the corneal and iris / lens images appear to just touch. For clinical application the required illumination system angle is 60º.

The subject was given a target of green light of Snellen's drum with other eye while seated on slit lamp (Takagi SM-70, Japan) mounted on a chair unit (Appasamy, India). This was to completely relax the accommodation. The microscope illumination angle was set at 60º and the slit lamp beam was adjusted horizontally.[9]

Objective magnification 6.3 X with12.5 X eyepiece. Slit length was increased till corneal image and iris or lens image 'just touch each other' (JTSL). An average of five measurements was taken.

Measurement of Anterior chamber depth was done with A-Scan (average of 10 measurements) and both values were taken for correlation. All the observations were made by a single observer to prevent intra-observer variation.

Results

Seventy seven eyes were examined for ACD – with A-Scan and by Smith's technique, 39 (50.6%) were males. They were aged 48-80 year. The average ACD was 2.62 mm by ultrasonography and the average JTSL was 1.87 mm. Correlation coefficient between ultrasound ACD and 'just touching slit length (JTSL) was found by using regression analysis with Microsoft excel and presented graphically (figure 3). The Scatter diagram showed a direct (positive) strong correlation. As ultrasonic ACD increases, JTSL increases showing a direct (positive) correlation. Values were close to the “line of best fit” with r = 0.97, suggesting a strong correlation between USACD and JTSL

Smith's Technique

The average ACD was 2.62 mm and the average JTSL

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was 1.87 mm. The conversion factor was: Average ACD / Average JTSL =1.39 (≈1.40)

There was a statistically significant association between USACD and JTSL. So if we know JTSL on slit lamp, by multiplying it by 1.4 we know ACD value.

We did a comparative study of bi-variate distribution by Correlation Analysis method

Conversion factor was =1.40

ACD = JTSL X 1.40

OR ACD = 1.333(JTSL) +0.124

Correlation tells us about the relation between variables and regression tells about the equation (relation) between the variables; so if we put JTSL values this equation gives ACD values.

Discussion:-

The anterior chamber is shallow in the very young and in old age and estimation of ACD would help make more informed clinical judgment. As the mean anterior chamber depth (2.5mm) of Indian adults is shallower than mean anterior chamber depth (3.0mm) of the European adults. The incidence of angle closure glaucoma is more in India. [6,7]

The technique was reliable in cataract patients where ACD is needed to identify potential complications. Distortion of slit of the slit lamp may affect JTSL value. The reliability of the technique would depend on optical media (anterior chamber) clarity. So it is not useful in uveitis, hyphaema and increased flare. This is reliable, simple technique which does not require any extra attachment to the equipment available in a general eye clinic. It is a reliable, simple technique to identify significantly shallow anterior chamber which predisposes to narrow angle glaucoma.

The limitation of this study was that relation with angle of anterior chamber and reliability towards periphery was not tested. But the estimates of the anterior chamber depth were only minimally deviated from the gold standard of A-scan ultrasonography. At a slit lamp angle of 60º, the JTSL can be converted into an anterior chamber depth by multiplying it by 1.40.

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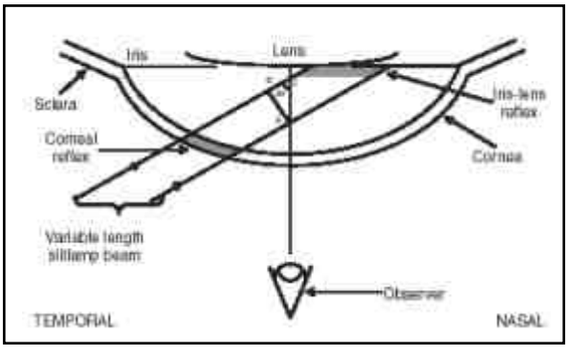
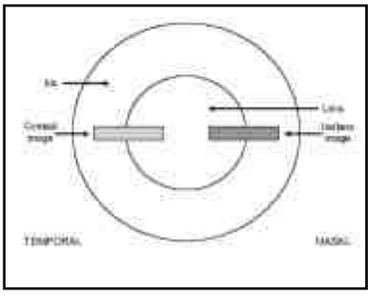
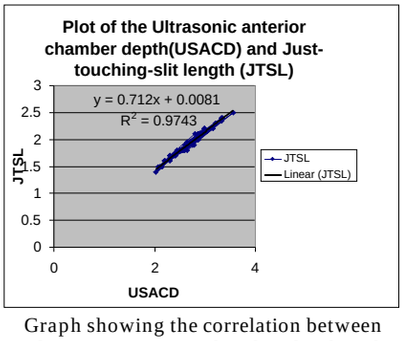


Diagrammatic representation of the corneal and lens images on the slit-lamp with ray diagram



Diagrammatic representation of corneal and lens images on slit-lamp



Graph showing the correlation between ultra-sonic anterior chamber depth and just touch slit length.

Amniotic Membrane Transplantation---A Complete Overview

Dr. Vishnukant Ghonsikar, Dr. Nayana Potdar , Dr. Chhaya Shinde

The human AM is the innermost layer of the placenta. Histologically the amnion is a 0.02 mm to 0.5 mm five-layered membrane, composed of three basic layers.

- Epithelial monolayer
- Thick basement membrane
- Avascular, hypocellular stromal matrix

The epithelium consists of a single layer of cuboidal cells with a large number of microvilli on the apical surface. The basement membrane is a thin layer composed of a network of reticular fibers. Histochemically the basement membrane closely resembles that of the conjunctiva. The compact layer contributes to the tensile strength of the membrane. The fibroblast layer is the thickest layer of the AM made up of a loose fibroblast network. The outermost layer of the amnion is the spongy layer.

The basement membrane is one of the thickest membranes found in human tissue. This layer is resistant to current cryopreservation techniques. The structural integrity, transparency and elasticity of the amniotic basement membrane makes it currently the most widely accepted tissue replacement for ocular surface reconstruction. It is known to promote epithelial cell migration, adhesion and differentiation. It is an ideal substrate for supporting the growth of epithelial progenitor cells by prolonging their lifespan, maintaining their clonigenicity and preventing epithelial cell apoptosis. This action explains why AMT facilitates epithelialization for PED with stromal ulceration. In tissue cultures AM supports epithelial cells grown from explant cultures and maintains their normal morphology and differentiation. The resultant cultured epithelium can be transplanted with the AM to reconstruct damaged corneas.The AM can be used to promote non-goblet cell differentiation of the conjunctival epithelium.

The basement membrane of the AM, cornea and conjunctiva contain collagen types IV, V and VII, in addition to fibronectin and laminin. Though the laminins are very effective in facilitating corneal epithelial cell adhesion Type V collagen helps in the epithelial cell anchorage to the stroma.

AM produces basic fibroblast, hepatocyte and transforming growth factor (TGF). These growth factors can stimulate epithelialization and modulate proliferation and differentiation of stromal fibroblasts.

The AM stromal matrix, rich in fetal hyaluronic acid suppresses TGF B signaling, proliferation and myofibroblastic differentiation of normal corneal and limbal fibroblasts as well as normal conjunctival and pterygium fibroblasts. This action explains why AMT helps reduce scars during conjunctival surface reconstruction, prevents recurrent scarring after pterygium removal and reduces corneal haze following photorefractive keratectomy. The stromal matrix also suppresses expression of certain inflammatory cytokines that originate from the ocular surface epithelia, including interleukin 1a, IL -2, IL-8, interferon ã, tumor necrosis factor-â, basic fibroblast growth factor and platelet derived growth factorThe AM attracts and sequesters inflammatory cells infiltrating the ocular surface and contains various forms of protease inhibitors. This may explain some of its anti-inflammatory properties.

Aminotic membrane graft (AMG) procurement, processing and preservation

Amniotic membrane is obtained from prospective donors undergoing Caesarean section, who are negative for communicable diseases including HIV, hepatitis and syphilis. Different protocols exist for the processing and storage. According to Kim et al the placenta is cleaned with balanced salt solution containing a cocktail of antibiotics (50 mg/ml penicillin, 50 µg/ml streptomycin, 100 mg/ml of neomycin as well as 2.5 mg/ml of amphotericin B) under sterile conditions. The amnion is separated from the chorion by blunt dissection. The separated membranes are cut in different sizes placed on nitrocellulose paper strips with the epithelial side up. Dulbecco Modified Eagles Medium/glycerol (1:1) is used for cryopreservation and the tissues are frozen at -80 degrees until further use. Amnion stored in 50-85% glycerol is reliable and effective for over a year, with the added advantage of antibacterial properties Human AM deprived of amniotic epithelial cells by incubation with EDTA when freeze dried, vacuum packed and sterilized with gamma-irradiation at 25kGy retained most of the physical, biological and morphologic characteristics of cryopreserved AM.

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Lyophilized AMs were found to be impermeable to different strains of bacteria - Bacillus, Escherichia coli, Pseudomonas, Citrobacter, Flavimonas and Staphylococcus . The results indicate that AMs processed by air-drying are stable and can be stored under different environmental conditions without compromising their clinical performance

Principles of surgery

The main objectives of AMT are ocular surface reconstruction, promotion of epithelialization, providing symptomatic relief and reducing inflammation. There are three basic principles upon which the final technique is individualized.

Inlay or graft technique :

When the AMG is tailored to the size of the defect and is meant to act as a scaffold for the epithelial cells, which then merges with the host tissue, it is referred to as a graft. The AM is secured with its basement membrane or epithelial side up to allow migration of the surrounding epithelial cells on the membrane.
Overlay or patch technique :

When the AM is used akin to a biological contact lens in order to protect the healing surface defect beneath, it is referred to as a patch. A patch also reduces inflammation by its barrier effect against the chemical mediators from the tear film. When used as patch the membrane is secured with its epithelial side up and it either falls off or is removed.
Filling-in or layered technique :

In this technique the entire depth of an ulcer crater is filled with small pieces of AM trimmed to the size of the defect. A larger graft is sutured to the edges of the defect in an inlay fashion and an additional patch may help in preserving the deeper layers for a longer duration.

AMG orientation

The preferred surgical orientation of the AM on the ocular surface is with the epithelial side up. The stromal surface can be identified by the presence of vitreous-like strands that can be raised with a sponge. Some people suggest that AM stains with indocyanine green, rose bengal, trypan blue and lissamine green B. The dyes stain both the epithelial and stromal surfaces and may be useful in identifying the edges and wrinkles in the graft. Intraoperative staining with lissamine green B may be a simple and effective way to assist surgeons in the proper handling of AM, while fluorescein staining has no role.

Corneal surface reconstruction

Non-absorbable sutures are used to anchor AMGs to the cornea. A single sheet of AM may be applied as an inlay graft or overlay patch and anchored to the superficial cornea with multiple interrupted 10-0 nylon monofilament sutures. A Weckcel sponge or blade is used to remove all cellular debris or exudates from the base of the defect in case of PEDs, shield ulcers and ulcerative keratitis. Loose epithelium surrounding an epithelial defect or over an area of bullous keratopathy is debrided using a fine forceps and a straight crescent blade. The size of the graft should be at least 1 mm larger than the defect. The sutures must be placed circumferentially or parallel to the cut edge of the graft in an interrupted or continuous manner. The suture knots must be cut short and knots buried in corneal tissue.

If AM is used to fill in deep corneal ulcers, descemetoceles or perforations, a multilayered approach is preferred. Small pieces of AM may be layered into the defect or a single sheet may be folded on itself twice (blanket fold). In either case a larger patch is anchored over the entire defect in an overlay fashion as shown in.

Conjunctival surface reconstruction

Vicryl sutures are used to anchor AM to the conjunctiva. Given the rapid healing ability of the conjunctiva, 8-0 or, 9-0 or 10-0 vicryl may be used for this purpose. The essence of the surgical technique in each of the indications is adequate dissection and removal of pathological subconjunctival tissue.

In order to anchor a sheet of AM to the fornix two sets of double armed 4-0 chromic gut sutures on a cutting needle may be used. The needles are passed from the AM surface through the inferior fornix, via the full-thickness of the eyelid and exit through the eyelid skin. The two needles of each of the two sets of sutures are passed through two segments of an encircling band and then tied.

surface reconstruction

Extensive ocular surface damage seen in severe grades of chemical injury, Stevens Johnson syndrome (SJS) and ocular cicatricial pemphigoid warrants sequential surface reconstruction. It is important to ensure that all fibrotic tissue is meticulously dissected and removed from the corneal and conjunctival surfaces. The AMG must be a continuous sheet devoid of buttonholes. The lower lid is everted with a large

chalazion clamp. A large sheet of AM is placed on the ocular surface and it is first anchored to the inner surface of the everted lower lid close to the lid margin using multiple interrupted 10-0 vicryl sutures. The anchorage to the inferior fornix is as described above. A continuous encircling 10-0 nylon suture is used to anchor the membrane at the limbus or the peripheral 360 ° cornea. In addition, multiple interrupted vicryl sutures are placed to attach the membrane to the inner lid surface, beyond the inferior fornix and onto the bulbar conjunctiva.

Indications of AMT in ocular surgery:

- Conjunctival surface reconstruction
- Pterygium surgery
- Chemical burns
- Cicatrizing conjunctivitis
- Ocular surface squamous neoplasia (OSSN)
- Leaking blebs
- Filtering surgery
- Symblepharon release
- Fornix formation
- Socket reconstruction
- Conjunctivochalasis
- Entropion correction
- Corneal surface reconstruction
- PEDs
- Non-healing stromal ulcers
- Partial LSCD
- Total LSCD
- Bullous keratopathy
- Band keratopathy
- Scleral melt
- Substrate for ex vivo expansion of limbal stem cells

Chemical and thermal injury

Joseph et al. reported that AMT was not found to be useful in the restoration of the ocular surface in Grade IV burns. In very severe ocular burns involving 360° of the limbus and entire conjunctiva there is probably a total loss of epithelial stem cells, leaving little resource for the amnion to allow regeneration. Although AMT in eyes with acute ocular burns has advantages in terms of pain relief and rapid epithelialization in moderate grade burns, no definite benefit of AMT over medical therapy alone has been reported with respect to severe ocular burns. This contrasts with the series in which Meller et al . performed AMT within two weeks after injury in 13 eyes and concluded that AMT is effective in promoting re-epithelialization and reducing inflammation in the acute stage of chemical injury, thereby preventing scarring sequelae in the late stages. Performing AMT during the first 7-10 days following acute burns maximizes the effects of the treatment. Associated lid deformities, symblephara and conjunctival foreshortening complicate management of chemical injury in the late stages. AMT alone gives satisfactory results in partial LSCD. In total LSCD it may be used as an adjunct to limbal stem cell transplantation.

Bullous keratopathy

Results of AMT for bullous keratopathy have been rather conflicting. Its efficacy has been studied in the palliative management of symptomatic bullous keratopathy with poor visual potential. AMT may also be performed as a temporary measure in patients waiting for corneal transplantation and intolerant to bandage contact lens (BCL). However, long-term relief from AMT needs to be studied and compared with other modalities

Conjunctival tumors and OSSN'S

Amniotic membrane transplantation has been reported to be successful in conjunctival surface reconstruction after excision of benign as well as malignant tumors such as conjunctival melanomas, lymphomas and OSSN. When used as a graft to cover the conjunctival wound it provides a substrate for the migration of conjunctival epithelial cells. Surface lesions are particularly challenging when they arise multifocally or extend over large areas and warrant an extensive conjunctivectomy .The advantages of AMT over conjunctival autografts and mucous membrane grafts in this scenario, include superior postoperative cosmesis, absence of donor site morbidity complicating the harvest of mucosal and conjunctival autografts (CAG) and the ability to clinically monitor local recurrence of tumor beneath the transparent AMG. Combined therapeutic approaches consisting of extensive tumor removal, cryotherapy, topical mitomycin C and AM allograft can be effective in the management of diffuse conjunctival melanomas arising from primary acquired melanosis (PAM).

PED

PED signify varying degrees of LSCD and chronic inflammation. AM serves to provide a basement membrane substrate for the migration and adhesion of epithelial cells when used as an inlay graft. When used as an overlay patch it facilitates epithelialization in a

fashion akin to a BCL and by providing a barrier against inflammatory cells and mediators. The AM, being continuously moistened by tears, provides adequate hydration to the regenerating epithelium and protects it from the abrasive effect of an abnormal palpebral conjunctiva.

Amniotic membrane transplantation may be considered an alternative method for treating PEDs that are refractory to conventional treatment such as lubrication, elimination of toxic drugs, BCL and punctal occlusion. Although results have been promising in the epithelialization of PEDs from various causes, early detachment of the patch remains a major problem despite the use of multiple sutures or a protective BCL.

Pterygium surgery

Pterygium excision with a CAG has gained worldwide acceptance as the most favorable technique as it has proven to be both safe and effective in reducing pterygium recurrence. They concluded that AM could serve as a useful alternative to conjunctival grafts when there exists a very large conjunctival defect to cover in primary double-headed pterygium, in previous multiple failed surgeries or in the context of preserving superior bulbar conjunctiva for future glaucoma surgeries.

Shield ulcers of vernal keratoconjunctivitis

Severe shield ulcers that do not respond to surgical debridement and BCL may be eradicated with superficial keratectomy or excimer photo therapeutic keratectomy (PTK).

Amniotic membrane transplantation combined with surgical debridement is an effective alternative modality in the management of these ulcers. The renewed basement membrane promotes epithelialization, reinforces cellular adhesion and prevents epithelial apoptosis.

The surgical procedure involves complete debridement of the mucous plaque and cellular debris from the ulcer base and edge. The surrounding loose epithelium is gently peeled off until normal adherent epithelium is reached. The AMG is tailored to be a millimeter larger than the defect and sutured with 10-0 nylon interrupted sutures.

Ulcerative keratitis:

Although performed in an uncontrolled and non-randomized series of patients, studies indicate that AMT shows promise in selected cases for the restoration of the ocular surface and reduction of stromal inflammation in ulcerative keratitis. Amniotic membrane transplantation can be considered an effective alternative for treating persistent neurotrophic ulcers, non-traumatic corneal perforations and descemetoceles. It can serve as a permanent therapy or as a temporizing measure until the inflammation has subsided and a definitive reconstructive procedure can be performed. Being a relatively simple procedure without risks of allograft rejection it could be particularly useful when faced with shortage of donor corneas.

Lid and orbital surgery

There are limited reports on the application of AMT in oculoplastic procedures. It has also been applied as a punctal patch for punctal occlusion in the treatment of dry eyesAmniotic membrane has been used as a cover for orbital prostheses and successfully used for the closure of a conjunctival defect following hydroxyapatite orbital implant exposure. Stewart et al . successfully used AM for reconstruction of the upper lid and the fornix in cryptropthalmos. Due to its beneficial effect in facilitating rapid epithelialization it appears to be a promising substitute to conventional grafts like mucous membrane grafts.

Postoperative management

A broad-spectrum topical antibiotic is used for one to two weeks initially, until the epithelium heals. Topical steroids are used for six to eight weeks in tapering doses to reduce surface inflammation. Systemic immunosuppression is not required.

Complications of AMT

In the immediate postoperative period one may come across hematoma formation under the membrane.The blood usually absorbs or may need drainage, by making a small opening in the graft, if excessive. Premature degradation of the membrane and cheese wiring may need frequent repeat transplantations. Occasionally, a residual subepithelial membrane may persist in some cases and inadvertently opacify the visual axis.

Calcification occurs in about 12.8% of cases. White plaques have been attributed to ciprofloxacin therapy. The key to reducing postoperative complications is meticulous selection of both donor and recipient and maintaining high standards of quality assurance.

The success of AMT is dependent on the underlying

condition and given the sub-optimal results in some indications, stringent case selection is recommended. The spectrum of clinical indications continues to expand and encompass a varying range of ocular surface pathology.

The future of AMT looks promising and seems like it is here to stay in the management of ocular surface disorders. With continued technological advancements in tissue processing, newer preserved forms such as the low-heat dehydrated AM are being made commercially available. Sutureless applications with fibrin glue have been aimed at making the procedure easier and more comfortable for the patient.

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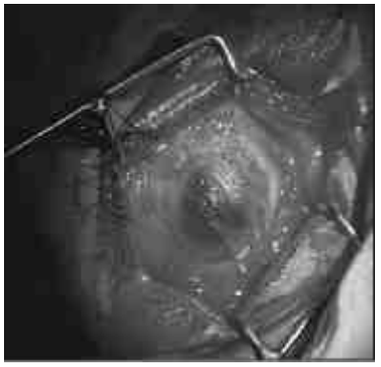
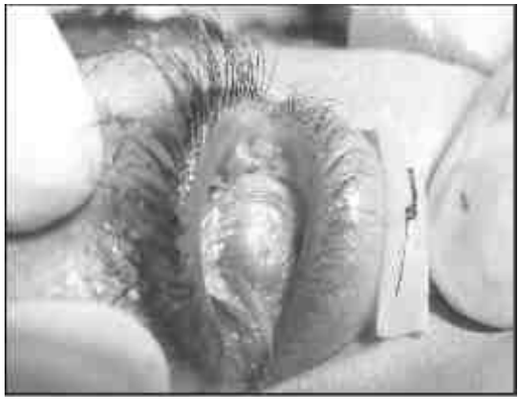
Amt for Pterygium



Amt for symbhlepharon



Age Related Macular Degeneration - Current Concepts

Dr. Vishnukant Ghonsikar, Dr. Nayana Potdar, Dr. Chhaya Shinde

Age-related macular degeneration

Age-related macular degeneration begins with characteristic yellow deposits in the macula (central area of the retina, which provides detailed central vision, called the fovea) called drusen between the retinal pigment epithelium and the underlying choroid. Most people with these early changes (referred to as age-related maculopathy) have good vision. People with drusen can go on to develop advanced AMD. The risk is considerably higher when the drusen are large and numerous and associated with disturbance in the pigmented cell layer under the macula. Recent research suggests that large and soft drusen are related to elevated cholesterol deposits and may respond to cholesterol-lowering agents.

Researchers from the University of Southampton reported October 7, 2008 that they had discovered six mutations of the gene SERPING1 that are associated with AMD. Mutations in this gene can also cause hereditary angioedema.

Advanced AMD, which is responsible for profound vision loss but never total blindness, has two forms: dry and wet.

Dry AMD

Central geographic atrophy, the “dry” form of advanced AMD, results from atrophy to the retinal pigment epithelial layer below the retina, which causes vision loss through loss of photoreceptors (rods and cones) in the central part of the eye.

While no treatment is available for this condition, vitamin supplements with high doses of antioxidants, lutein and zeaxanthin, to slow the progression of dry macular degeneration and, in some patients, improve visual acuity.

Drusen

Clinically, drusen are small, round, yellow lesions located at the level of the RPE within the macula. Histologically, this material corresponds to the abnormal thickening of the inner aspect of Bruch's membrane. Ultrastructurally, the material includesw basal laminar deposits (granular lipid-rich material and widely spaced collagen fibres) and basal linera deposits (phospholipid vesicles and electron-dense granules within the inner aspect of Bruch's membrane.

Drusen have been categorized as:

- small(usually <64 um in diameter)
- intermediate(usually 64-124 um in diameter)
- large (usually > 125 um in diameter)

In addition, the boundaries of drusen have been described as

- hard(discrete and well demarcated)
- soft(amorphous and poorly demarcated)
- confluent(contagious boundaries between drusen)

Wet AMD

Neovascular or exudative AMD, the “wet” form of advanced AMD, causes vision loss due to abnormal blood vessel growth (choroidal neovascularization) in the choriocapillaris, through Bruch's membrane, ultimately leading to blood and protein leakage below the macula. Bleeding, leaking, and scarring from these blood vessels eventually cause irreversible damage to the photoreceptors and rapid vision loss if left untreated.

Pathogenesis in wet ARMD

Neovascular AMD:

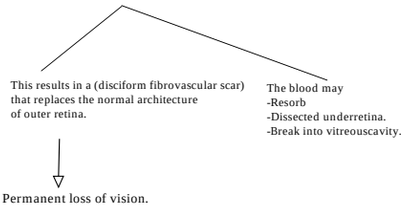
Changes of Non-neovascular AMD

Break in the brusch membrane

Allowing buds of neovasucalr tissue from the choriocapillaries to perforate through the gap or create mew gape.

These new vessels are accompanied by fibroblasts, resulting in a fibrovascular complex that proliferates within the inner aspect of Brusch's membrane, into the sub-pigment epithelial space.

CNV can leak fluid and blood and accompanied by a serous or hemorrhagic detachment of RPE..



Symptoms of cnv:

- Sudden onset of decreased vision.
- Metamorphopsia
- Paracentral scotoma
- Central scotomaRelative
- Positive scotoma.

Signs of cnv:

- Presence of subretinal fluid.
- Subretinal or sub-pigment epithelial blood.
- Subretinal or Intraretinal lipid.
- Subretinal pigment ring.
- Irregular elevation of the pigment epithelium.
- Subretinal grey-white

Until recently, no effective treatments were known for wet macular degeneration. However, new drugs, called anti-angiogenics or anti-VEGF (anti-Vascular Endothelial Growth Factor) agents, can cause regression of the abnormal blood vessels and improvement of vision when injected directly into the vitreous humor of the eye. The injections can be painful and frequently have to be repeated on a monthly or bi-monthly basis.

Examples of these agents include

ranibizumab (trade name Lucentis),

bevacizumab (trade name Avastin, a close chemical relative of ranibizumab) and pegaptanib (trade name Macugen).

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Only ranibizumab and pegaptanib are approved by the FDA for AMD as of April 2007. Bevacizumab is approved, but for other indications. Pegaptanib (Macugen) has been found to have only minimal benefits in neovascular AMD and is no longer used. Worldwide, bevacizumab has been used extensively despite its "off label" status.

The cost of ranibizumab (Lucentis) is approximately US$2000 per treatment while the cost of bevacizumab (Avastin) is approximately US$150 per treatment. Both drugs are made by Genentech.

Photodynamic therapy has also been used to treat wet AMD.

Risk factors

- Aging: Approximately 10% of patients 66 to 74 years of age will have findings of macular degeneration, and 30% in patients 75 to 85 years of age.
- Family history: The lifetime risk of developing late-stage macular degeneration is 50% for people that have a relative with macular degeneration, versus 12% for people that do not have relatives with macular degeneration, a fourfold higher risk.
- Macular degeneration gene: The genes for the

- Complement system proteins factor H (CFH) and
- factor B (CFB) and
- factor 3 (C3)
- There are specific diseases associated with mutations in some of these genes. Below is one of the affected genes and the disease that arises from its mutation.
- Mutation of the ATP synthase gene: Retinitis Pigmentosa (RP) is a genetically linked dysfunction of the retina and is related to mutation of the Adenosine Tri-Phosphate (ATP) Synthase Gene 615.1617
- Stargardt's disease (STGD, also known as Juvenile Macular Degeneration) is an autosomal recessive retinal disorder characterized by a juvenile-onset macular dystrophy, alterations of the peripheral retina, and subretinal deposition of lipofuscin-like material. A gene encoding an ATP-binding cassette (ABC) transporter was mapped to the 2-cM (centiMorgan) interval at 1p13-p21 previously shown by linkage analysis to harbor the STGD gene. This gene, ABCR, is expressed exclusively and at high levels in the retina, in rod but not cone photoreceptors, as detected by in situ hybridization. Mutational analysis of ABCR in STGD families revealed a total of 19 different mutations including homozygous mutations in two families with consanguineous parentage. These data indicate that ABCR is the causal gene of STGD / FFM.
- Drusen CMSD studies indicate that drusen are similar in molecular composition to plaques and deposits in other age-related diseases such as Alzheimer's disease and atherosclerosis. While there is a tendency for drusen to be blamed for the progressive loss of vision, drusen deposits can, however, be present in the retina without vision loss. Some patients with large deposits of drusen have normal visual acuity. If normal retinal reception and image transmission are sometimes possible in a retina when high concentrations of drusen are present, then, even if drusen can be implicated in the loss of visual function, there must be at least one other factor that accounts for the loss of vision. Retinitis Pigmentosa (RP) is a genetically linked dysfunction of the retina and is related to mutation of the ATP Synthase Gene 63.
- Arg80Gly variant of the Two independent genetic studies from two groups published in the New England Journal of Medicine and Nature Genetics in 2007 showed that a certain, common mutation in the C3 gene which is a central protein of the complement system is strongly associated with the occurrence of Age-related Macular Degeneration. The authors of both papers consider their study to underscore the influence of the complement pathway in the pathogenesis of this disease.
- Hypertension: Also known as high blood pressure.
- Cardiovascular status — high cholesterol, obesity.
- High fat intake is associated with an increased risk of macular degeneration in both women and men. Fat provides about 42% of the food energy in the average American diet. A diet that derives closer to 20-25% of total food energy from fat is probably healthier. Reducing fat intake to this level means cutting down greatly on consumption of red meats and high-fat dairy products such as whole milk, cheese, and butter. Eating more cold-water fish (at least twice weekly), rather than red meats, and eating any type of nuts may help macular degeneration patients.
- Oxidative stress: It has been proposed that age-related accumulation of low-molecular-weight, phototoxic, pro-oxidant melanin oligomers within lysosomes in the retinal pigment epithelium may be partly responsible for decreasing the digestive rate of photoreceptor outer rod segments (POS) by the RPE. A decrease in the digestive rate of POS has been shown to be associated with lipofuscin formation - a classic sign associated with macular degeneration.
- Fibulin-5 mutation Rare forms of the disease are caused by geneic defects in fibulin-5, in an autosomal dominant manner. In 2004, Stone et al. performed a screen on 402 AMD patients and revealed a statistically significant correlation between mutations in Fibulin-5 and incidence of the disease. Furthermore, the point mutants were found in the Calcium binding sites of the cbEGF domains of the protein. There is no structural basis for the effects of the mutations.
- Race Macular degeneration is more likely to be found in Caucasians than in people of African descent.
- Exposure to sunlight especially blue light. There is conflicting evidence as to whether exposure to sunlight contributes to the development of macular degeneration. A recent study in the British Journal of Ophthalmology on 446 subjects found that it does not. Other research, however, has shown that High-energy visible light (HEV) may contribute to age-related macular degeneration.
- Smoking Smoking tobacco increases the risk of macular degeneration by two to three times that of

someone who has never smoked, and may be the most important modifiable factor in its prevention. A review of previous studies found that "the literature review confirmed a strong association between current smoking and AMD. ... Cigarette smoking is likely to have toxic effects on the retina."

Fluorescein angiogram pattern of AMD

- Hyperfluorescent lesions
- hard and soft drusen
- RPE atrophy
- RPE tear
- CNV
- serous PED
- subretinal fibrosis
- laser scars
- Hypofluorescent lesions
- Haemorrhage at any level
- lipid
- Pigment proliferations

Fluorescein angiogram patterns of CNV

1. classic CNV
2. occult CNV----fibrovascular PED
   ----late leakage from an undermined source

Studies in Age Arelated Macular Degeneration

1) Age related eye disease study (AREDS)
2) Treatment of Age-Related Macular Degeneration with Photodynamic Therapy (TAP) Study
3) Verteporfin in Photodynamic Therapy (VIP) Trial : AMD and Pathological Myopia
4) VEGF Inhibition Study in Ocular Neovascularization (VISION)
5) Minimally Classic / Occult Trial of the Anti-VEGF Antibody Ranibizumab in the Neovascular AMD (MARINA)
6) Anti- VEGF Antibody for the Treatment of Predominantly Clssic Choroidal Neovascularization in AMD (ANCHOR) Study

1) Age related eye disease study (AREDS)

OBJECTIVES : To evaluate whether antioxidants or zinc supplements can reduce development or progression of cataract or AMD

PARTICIPANTS: 4757 men and women, age 55-80,----------1992-1995 dose of supplements:

antioxidants
vitamins-500mg vitamin C
400IU vitamin E
15mg beta carotine
zinc- 80mg zinc oxide
cupric oxide-2mgs

Conclusions:

Patients with bilateral, intermediate, dry AMD or unilateral advanced AMD benefited from antioxidant and zinc supplementation with respect to reduced rates of progression to advanced AMD and loss of vision from AMD compared with the placebo group.

Whom to Give These Supplementation

patients with high risk AMD

- extensive intermediate drusen
- at least 1 large drusen
- non central geographic atrophy
- advanced AMD in 1 eye

2) Treatment of Age-Related Macular Degeneration with Photodynamic Therapy (TAP) Study

Objectives :

To determine if PDT with verteporfin can reduce the risk of visual loss in patients with subfoveal CNV when compared to placebo-controlled sham treatment.

Outcome:

Vision loss of <15 letters at 1 and 2 years. Secondary outcomes include vision loss of <30 letters, contrast threshold function, quality of life, and morphologic outcomes.

3) Verteporfin in Photodynamic Therapy (VIP) Trial : AMD and Pathological Myopia

Objectives:

To determine if photodynamic therapy with verteporfin can reduce the risk of visual loss in patients with subfoveal CNV when compared with placebo-controlled sham treatment.

Outcome:

Vision loss of <5 letters at 1 and 2 years. For AMD eyes, vision loss of <8 letters at 1 or 2 years for myopic eyes. Secondary outcomes include vision loss of <30 letters, contrast threshold function, quality of life, and morphologic outcomes.

4) VEGF Inhibition Study in Ocular Neovascularization (VISION)

OBJECTIVES:

To determine if pegaptanib can reduce the risk of visual loss in patients with subfoveal CNV when compared with placebo-controlled sham treatment.

Outcome:

Vision loss of <15 letters at 1 year. Secondary outcomes include vision loss of <30 letters and morphologic outcomes.

5) Minimally Classic/Occult Trial of the Anti-VEGF Antibody Ranibizumab in the Neovascular AMD (MARINA)

Objectives:

To determine if monthly ranibizumab can reduce the risk of visual loss in patients with subfoveal, minimally classic or occult with no classic CNV when compared with placebo-controlled sham treatment.

Outcome:

Vision loss of <15 letters at 1 year. Secondary outcomes include vision loss of <30 letters, quality of life, and morphologic outcomes.

6) Anti- VEGF Antibody for the Treatment of Predominantly Clssic Choroidal Neovascularization in AMD (ANCHOR) Study

Objectives:

To determine if monthly intravitreal ranibizumab can reduce the risk of visual loss in patients with predominantly classic subfoveal CNV when compared with photodynamic therapy with verteporfin treatment.

Outcome:

Vision loss of <15 letters at 1 year. Secondary outcomes include vision loss of <30 letters, contrast threshold function, quality of life, and morphologic outcomes.

Management of Wet Armd:

1. Photodynamic therapy:

Agent: Verteporfin of photosensitizer / light activated compound.

Principle: This compound is taken up by dividing cells / neovascular tissue so when given intravenously and activated locally by illumination with a light from diode laser(689nm) has the ability to selectively damage the target tissue.

Indications: Subfoveal CNV
Predominantly classic CNV
CNV not larger than 5400nm.

Probable indications: Small, pure occult lesions with recent decrease in vision.
Lesions more than 5400nm, juxtapapillary CNV with subfoveal extension.
Dose: 6mg / kg body weight is infused intravenously over 10 minutes.
Duration of laser: Applied to CNV with spot size 100nm larger than the greatest linear dimension of the lesion for 83 seconds.
Retreatment: To the same areas at 3 monthly intervals until entire CNV is obliterated.

2. Anti-angiogenic therapy
- Intravitreal steroids
- Anti-VEGF agents

3. Surgery
- Submacular surgery involves: Vitrectomy / posterior retinotomy / subfoveal CNV removal.
- Macular translocation: Surgically moving the fovea away from CNV.
- Pneumatic displacement of submacular hemorrhage involves: Injection of gas into the vitreous cavity followed by face down posturing in order to displace the blood from the fovea.

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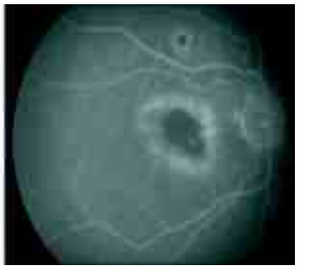
Classic Juxtafoveal CNVM – Pre



Regressed CNVM – Post Laser

Classic CNV on FFA

Laser and Its Various Application in Ophthalmology

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An acronym for Light Amplification by Stimulation Emission of Radiation.

The first laser was produced in 1960. Light produced by a laser is coherent (waves in place and nondivergent), monochromatic,intense, and unidirectional. The exact wavelength of the laser emission depends on the material used to generate the light. It may be delivered by a series of flashes of light (pulsed) or as a continuous beam of light (continuous wave)

All lasers in use require 3 basic elements:

1) An active medium that emits coherent radiation,
2) A means of energy input known as pumping, and
3) The opportunity for oscillation and amplification through optical feedback.

Radiation emitted by spontaneous emission occurs randomly in time, but radiation emitted by stimulated emission is in phase with the stimulating wave and is therefore coherent.

The active medium can be a gas (argon, krypton, carbon dioxide, or helium with neon), a liquid (dye), or a solid (an active element supported by a crystal, such as neodymium supported by yttrium-aluminum garnet (Nd-YAG).

Effects of Lasers

Lasers have three general effects on biologic tissues: photochemical, thermal and ionizing.

Photochemical effects

For exposures longer than one microsec(one millionth of a second) and low to moderate irradiances, low radiation below 320 rm produces primarily photochemical reactions.

Photo-radiation:

Hematoporphyrin derivative is selectively taken up and retained by metabolically active tumour tissue that predisposes this tissue to photochemical damage if the tumour is irradiated by light 64w and 635 rm. This is used to treat some choroidal melanomas.

Eximer lasers producing ultraviolet light below 300 rm can provide precise experimental corneal incisions to predetermined depth. Adjacent tissue is not affected. This procedure is undergoing experimental testing for radial keratotomy.

Thermal effects

Photocoagulation: In peripheral thermal tissue damage requires a 10' to 20' C increase in corneal or retinal temperature.The extent of thermal injury is proportional to the magnitude and duration of a temperature increase.

Temperature rise in an irradiated tissue is proportional to light absorption in that tissue, which in tum is determined by how effectively its constituent molecules absorb incident photons of aparticular wavelength.

For example,the excellent absorption of visible light by melanin in the trabecular meshwork or retinal pigment epithelium make these tissues excellent targets for argon ion laser photocoagulation. The poor absorption of visible light by corneal collagen, however, helps make the cornea an effective window for vision and for photocoagulation or photodisruption.

Closure of blood vessels by photocoagulation is caused by light absorption in the blood column, which heats the hemoglobin to a temperature high enough to produce thrombus formation and collagen shrinkage of the wall of the blood vessel and its surrounding fibrous tissue. Argon green light is well absorbed in small intraocular blood vessels and is therefore very useful in closing them.

Photovaporization:

If very high laser irradiances are used, tissue temperature can quickly reach the boiling point of water. Rapidly expanding water vapour will cause tissue disruption (photovaporization) before denaturation can cauterize the tissue.

In most situations, photovaporization (cutting) is accompanied by photocoagulation (cautery); in CO2 laser surgery, for example, cautery during incision produces a virtually bloodless operating field.

Ionizing effects

Short-pulse Nd-YAG lasers disrupt even transparent tissues by delivering enormous near-infrared (1064 rm) irradiances to tissue targets. These irradiances are obtained by using small spot sizes and extremely brief pulses, ranging from 30 nanoseconds down to 20 picoseconds (ipsec = 10-12 sec).The high irradiance ionizes material in small volume of space at the laser beam focus, disintegrating it into a collection of ions and electrons

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called a plasma. The plasma expands rapidly producing shock and acoustic (pressure) waves that mechanically disrupt tissue adjacent to the region of disintegration.

Types of Lasers

The first type of laser used in clinical ophthalmology was a ruby.

The only general requirement for a laser material is that it provide an upper energy state into which molecules or atoms can be placed and a lower energy state to which they will return with a spontaneous emission of photons. By choosing between the large array of substances that can be made to laser, one can obtain almost any desired wavelength. Most substances have several different excited energy states into which electrons can go. Therefore they can emit light at several different wavelengths and can create a laser beam which is a mixture of these. These wavelengths can be separated in total or in part by using a prism (e.g., argon gas creates laser light in more than nine different wavelengths).

Most common commercial systems use just two of these wavelengths, giving a beam which is a mixture of blue and green light.

Lasers can deliver energy in different ways. Some deliver the energyas a continuous stream of photons.

Others deliver the energy in pulses. These have pulse durations of a few tens of microseconds to a few milliseconds.

Pulsed laser can be operated to produce repetitive pulses. Laser pulses can be delivered in much shorter periods of time by changing the nature of the optical cavity in which

the laser light is created. Laser systems can be made with pulse durations of a few nanoseconds (10-9 sec).

Lasers in Glaucoma

Angle-closure glaucoma is usually due to pupillary block between the lens and iris. This prevents aqueous from following its normal course through the pupil to the angle and Schlemm's canal and out of the eye. Pupillary block results in the peripheral iris being pushed against the cornea, thereby blocking off the angle. Therapy is directed towards creating an internal bypass with an opening in the iris between anterior and posterior chambers. Before lasers, this was achieved by a surgical iridectomy, but now the laser has all but eliminated this procedure. An iridotomy is created by the absorption of irradiated light by the melanin in the iris, resulting in a thermal effect with disruption and hole formation. This is achieved with the patient sitting at a slit lamp, and is carried out in approximately 5-10 minutes on an outpatient basis.

Lasers in Diabetes

Diabetic retinopathy is broadly divided into non-proliferative and proliferative types. In the non-proliferative type, VAHEX is an acronym for its features (venous dilation, aneurysms, hemorrhage, edema, exudates). These five stages can then be classified on a 1-4 severity rating. Proliferative retinopathy includes the production or proliferation of new tissue, supportive or neovascular in nature in the chorio-retinal area secondary to injury by the diabetic process.

These include neovascularization at the disc and / or retina, glial proliferation and vitreoretinal traction. The basic rationale of laser photocoagulation is to destroy neovascular complexes, to obliterate areas of micro infarction or capillary closure, to destroy leaking vessels in the macular and paramacular region and to produce a chorio-retinal adhesion that will resist the later ravages of increasing vitreoretinal traction. The proliferation of neovascular tissues is probably the result of localized hypoxia in the region of the retinal vessels near the internal limiting membrane. It would seem that these blood vessels are proliferating in response to some biochemical substance. Intravenous fluorescein angiography consists of injecting sodium fluorescein into the antecubital vein and recording the results photographically at intervals of 0.6 to 0. 8 seconds.

All abnormalities of the retinal circulation can be seen and treated accordingly.

Panretinal photocoagulation appears to successfully obliterate or cause the regression of neovascularization by one of four mechanisms: the reduction or destruction of areas of hypoxic retina that are producing the vasoformative factor that causes neovascularization from healthier areas of the retina; adherence of retina to choreocapillaris (choroid), allowing more oxygen from choroid to retina; destruction of infarcted areas of retina, allowing more blood to the healthier retina; destruction of leaking vessels and abnormal vascular complexes, which normalizes blood flow to the macular area.

The panretinal photocoagulation(PRP) iS conducted in three to six stages in approximately two to seven days.

Coagulation of 100 to 200 microns indiameter with power intensities from 100 to 400 mw and exposures of 0.05 to 0.2 seconds with sites increasing as one treats the more peripheral retina.

Focal photocoagulation of neovascularization attacks the results, not the cause, of the process and carries a higher risk of hemorrhage.

Posterior Capsulotomy

One of the most common uses of the Nd-YAG laser is to perform a posteriorcapsulotomy. There may be less incidence of edema of the macula (cystoid maculopathy) and decreased retinal detachments with the extracapsular method, which also allows for the insertion of a posterior chamber intraocular lens vs.anterior chamber lens. This again is not clear cut, but it may be more physiological in the posterior chamber and there may be less long-term comeal complications. One of the problems with the extracapsular technique is that

the capsule sometimes becomes opaque. Before the YAG laser this had to be dealt with surgically, but now can be done with the ionizing effect of the YAG.

Retinal Tears

The symptoms that may occur with retinal tears are variable, and include floaters, sudden showering of spots and opacities, lightning flashes, and blurring of vision. The problem with retinal tears is akin to a tear in the vinyl lining of a swimming pool. Water will eventually seep under the lining and lift the lining off. If the tear is diagnosed early before the retina has lifted, detachment can be prevented by scarring down the retina surrounding the tear. This may be accomplished by using a laser usually the argon laser. If detachment has occurred, then the retina has to be

drained and a buckle placed around the sclera. Most floaters are indicators of vitreous degeneration, but a sudden onset probably should be assessed for retinal problems.

Macular Disease

Serous retinal pigment epithelial detachments are round or oval domed elevations of the retinal pigment epithelium. Fluorescein dye will readily collect there. These serous detachments can occur with or without subretinal neovascularization. The treatment of the serous detachments without neovascularization is controversial.

One prospective study found that argon laser treatment was of no benefit and perhaps harmful.

Clinical signs of a subretinal neovascular membrane are a greyish or green pigmentation deep to the retina, subretinal or retinal hemorrhage, hard exudates, and subretinal fluid. The membrane is then outlined by fluorescein and its distance from the fovea is measured. There is a foveal avascular zone measuring 400 microns in diameter which cannot be treated with laser as it may obliterate central acuity.

Therefore, if the membrane is 200 microns or more from the center of the fovea, the entire membrane should be treated with laser photocoagulation. The most common scenario is that the patient comes to see the ophthalmologist after losing central acuity in one eye, and there is no treatment due to scarring following hemorrhage, serous detachment with neovascularization or atrophic degeneration. It is important to advise the patient to monitor the fellow eye daily (by Amsler grid testing) with a view that if subretinal neovascularization should develop,

it may be amenable to laser therapy .

Cataract surgery

Cataract surgery has been performed for over two millennia, but advances in technology have transformed the fundamental procedure only over the past 40 years. The use of ultrasound vibration to remove cataracts through a small incision was pioneered by Charles Kelman in the 1960s, and the technique has been developed to become the standard procedure for most cataract extractions in developed countries. The Kelman phacoemulsification procedure has also become the main framework upon which innovations in cataract surgery are built. Such innovations are driven by the need for less trauma during surgery and faster visual recovery after surgery. Surgeons have strived to reduce incision size, heat, intraocular turbulence and fluid level in order to achieve these objectives. Ultrasonic phacoemulsification probe tips tend to create relatively high levels of heat within the eye, resulting in the possibility of injury to the cornea such as corneal burns and endothelial damage. Technological advances over the past decade have reduced the effective energy liberated by the probe, chiefly through using ultrasound energy more efficiently.

Perhaps the most promising front in atraumatic phacoemulsification surgery is the application of the yttrium-aluminum-garnet (YAG) laser towards emulsifying the cataract. The first laser procedure for cataract surgery was reported in 1975 by Krasnov, who used a technique called “laser phacopuncture” to make microperforations on the anterior capsule. These pores then allowed the release of lens material into the anterior chamber, which would be theoretically resorbed over time. Krasnov's Q-switched ruby laser technique had limited application, since the micropores would only allow the release of very soft cataracts. Furthermore, patients had to be maintained on dilator drops for extended periods to prevent the puncture sites from

closing, and steroid drops to reduce anterior uveitis that inevitably occurred from the released cataract in the anterior chamber. Additional experiments with laser cataract surgery occurred with excimer lasers, most notably the 308 nm laser. The xenon chloride 308 nm laser was introduced in the late 1980s, but was abandoned for cataract surgery due to concerns over retinal toxicity. The neodymium:yttrium-aluminum-garnet (Nd:YAG) laser was used successfully to perform posterior capsulotomy in 1980. The YAG laser gained wide acceptance among surgeons as an excellent method of treating posterior capsular opacification after cataract surgery. The popularity of the Nd:YAG laser in posterior capsulotomies motivated researchers to explore how the YAG laser could be used to treat cataracts. A technique called laser photofragmentation, which uses the Nd:YAG laser to soften the nucleus before phacoemulsification, was explored in the mid-1980s. This procedure did reduce phacoemulsification power and time, but also increased the risk of capsular perforations.

The YAG laser has the potential to dramatically reduce the energy required to perform cataract surgery. Two types of YAG lasers are being developed for cataract surgery: the neodymium:YAG (Nd:YAG) and Erbium:YAG (Er:YAG) laser. The pulsed Q-switched Nd:YAG laser, which emits at 1064 nm, does not produce direct laser light at the tip; instead, it generates shock waves through a titanium block at the tip to photolyse the cataract. This technology produces negligible heat at the tip, and therefore does not require a cooling sleeve to avoid corneal burns. Consequently, incisions as small as 1.25 mm can be used to perform the procedure. Laser emulsification is relatively short for most cataracts, but can take over 10 minutes for nuclear sclerosis over 3+. Another Nd:YAG laser, which uses photoacoustic ablation under aspiration, delivers energy through a ski-shaped distal tip to create a “photon trap”. This technology is most useful for softer nuclear sclerosis. The Er:YAG laser, which emits at 2940 nm, relies on its infrared spectrum wavelength in cataract surgery. At this wavelength, the laser produces cavitation bubbles that collapse slowly in the cataract and very quickly in water. This leads to propagated energy within the lens, allowing the laser to emulsify the material efficiently without producing thermal energy. The laser can be used with a prechopper to reduce the operating time.

Each of these YAG laser technologies can be coupled with standard I/A pumps to allow a lenticular emulsification with little or no thermal energy. The technologies open up the possibility of performing cataract surgery through very small (<2 mm) incision sizes, intraocular lenses to fit through such small openings are being developed rapidly.

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Principles & Applications of B Scan

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Principles of ultrasound:
An ultrasound wave has a frequency greater than 20 kHz
.
Wavelength α Depth of penetration of the ultrasound
1 / α Resultant resolution of Echograph
UBM: very high resolution ocular B scan probes of 20-50 MHz are in use that penetrates only 5-10mm of eye .They are mainly used for detailed examination of anterior segment.
Stimulated by an electric oscillator
A piezoelectric transducer Ultrasound Pulse-High frequency soundwaves
are projected into the eye & orbit Each type of tissue conducts sound waves at a different velocities When sound waves reach a boundary bet. two different tissue types, an acoustic interface is noted. Sound waves are reflected back by an acoustic interface.(Echo)
The amount of reflection α differences in sound velocities through the tissues.i,e greater is the differences in sound velocities of the media,
stronger is the echo.
Aqueous & vitreous =1,532 m / s
Intraocular & orbital tissues = 1,550 m / s
Cornea & Lens =1,641 m / s
Lens (vel =1,641m / s) producesstronger echo,when adjacent toaqueous (vel=1,532 m / s) as
opposed to blood(vel= 1,560 m / s)

Echo Strength	A Scan	B scan
Stronger Echoes	As higher spike s	Bright dots
Weaker Echoes	Low spikes	Less bright dots

Echoes are captured by receiver of transducer which deform the crystal to produce a
voltage .This voltage is proportional to the strength of the echo .The resultant voltage is weak.
Amplifier section of B scans
Unit
Amplified image is sent for display. There are two principal display modes
A scan(A for Amplitude) Echoes appear on thescreen as amplitudespikes
B scan(B for Brightness)
Echoes are displayed as a dot in grayscale. The brightness of dot is a function of amplitude of the received echo. The amplification can be changed by adjusting the gain, measured in dB
When the gain is high, weaker signals can be seen(VH,PVD)
When it is low, only stronger signals are displayed (Retina, IOFB)
Resolution of image is better, when the gain is low
Indications of ocular B scan
A opaque ocular media
Anterior Segment – CornealOpacity, hyphema, hypopyon.Miosis ,papillary membrane,Cataract
Posterior Segment-Vitreoushaemorrhage ,inflammation
B clear ocular media
Anterior Segment - Iris Leison,,Cilliary body Leison - Tumours & masses:detection &differentiation
RD:rhegmatogenousvs.exudative
IOFB: Detection &localization
OD abnormalities
Technique
B scan probe contains a marker that contains a transducer that moves rapidly back & forth near the tip of the probe. The probe tip is often oval in shape with the back and forth motion of the transducer corresponding to the longest diameter of the probe tip. Each probe has a marker (dot / line) that indicates the side of the probe that is represented on the upper portion of the B scan screen. It is important to know how the probe or marker is oriented during the examination
Initial line on the left side of the echograph : probe tip
Right side f the echograph: Fundus opposite the probe position
Upper part of the echograph : portion of the glove where probe marker is directed
We apply methylcellulose to the probe face as a coupling
Medium & do closed lid examination which minimizes the discomfort &
allows a better visualization of peripheral fundus.
Three basic probe positions are
1 Transverse
2 Longitudinal
3 Axial
The transverse and longitudinal scans are most commonly used
because the probe is placed on the conjunctiva peripheral to the cornea. Thus sound beam
bypasses the crystalline lens, allowing better sound penetration. These scans are
performed with patients gaze directed away from the probe , toward the meridian being
examined.(there by providing wider surface of the globe to place & shift the probe.
In axial scan, the patient fixates in primary gaze and the probe is
placed on the centre of the cornea, thus displaying lens & ON in the centre
Transverse Scans: Shows lateral extent of lesion (2 to 4 O'clock) Designation of transverse scan is determined by the meridian that lies in the middle of the scanning section. For example- If probe is held horizonatally with its face centered on the 6 O'clock meridian, the middle of the echogram will display the 12 O'clock meridian of fundus, this probe position is called a transverse scan of 12 O'clock meridian. Probe placed on the globe with marker parallel to the limbus in two direction either nasally or superiorly By Conventions, there are three different varieties of transverse scans ---
HorizontalTransverseScan - Marker orientedtoward the nose

VerticalTransverse Scan - Marker directedsuperiorly
ObliqueTransverse Scan - Marker towardsupper portion of the globe
Longitudinal Scans : It produces an anteroposterior slice of the globe along one meridian.
Resultant echograph shows,
ON + post fundus appearing on the inferior portion &
Peripheral fundus on the upper portion of the echograph
The designation of longitudinal scan is simply that meridian which is being examined.
For ex, if probe is placed on the 6 O'clock meridian, the sound beam sweeps along the 12 O'clock meridian, this is designated as a longitudinal scan of 12 O'clock position.
Axial Scan : Patient is asked to direct his / her gaze in primary position & probe tip is centered on the cornea.
Easy to interprete because of presence of evident landmarks (lens & ON) but offers less resolution & more distortion because of attenuation & refraction of the sound beam caused by the lens
Baum's Bump: Not only does the lens refract light, it also refracts sound waves, which results in distortion of the echography, particularly if some of the sound wave passes Probe placed peripheral to the limbus.
Marker is perpendicular to the limbus (directed toward the centre of the cornea, regardless of which meridian is being examined)
There are mainly two varieties of Axial scans
1 Horizontal Axial Scan: Marker is oriented towards the nose (3 O'clock), which places macular region just below the OD
2 Vertical Axial Scan: Marker is oriented toward the superior position of the globe. (12 O'clock),Macula is not seen in these scans through lens & some not. Resultant demarcation produces bulges known as Baum's Bump In axial view of a phacic eye, post lens capsule is the first intraocular structure to appear
in the screen, followed by vitreous gel (clear, sonoluscent),by the posterior eyeball(i.e.
retina, choroids & sclera).In an axial scan, the ON is shown at the centre of the screen as a sonoluscent structure with a few echo inside surrounded by highly reflective intra orbital fat tissues..
Point Structures:
4 common point echoes can be detected in the vitreous cavity :
1 Degenerative Changes: A few low reflective high mobile point echoes can be seen
2 Asteroid Hyalosis: Calcium particles are highly reflective & appear on USG as strong

3 Vitreous Haemorrhages:
A)Fresh+ mild VH= dot & short lines displayed in USG. More dense the haemorrhages, more opacities seen in B-scan Because of the gravity, blood may layer inferiorly resulting in highly reflective pseudo membranes that may be confused with RD AH is highly ecogenic,they are still visible when the gain setting is reduced upto 60dBunlike VH which usually disappears by 60 dB
Gain should be increased to visualize vitreous echo in a patient suspected to have a fresh VH Retrohyaloid Haemorrhages:Blood in Retrohyaloid space tends to remain in a fluid state and does not clot, so it has a low reflectivity, therefore a high gain (90dB) is necessary to image retrohyaloid haemorrhage.
4 Vitreous Inflammation: Clumps of vitreal inflammatory cells appear as scattered particle or large aggregates.Usg is helpful in suspected endophthalmitis., in absence of external inflammatory signs, to differentiate between endophthalmitis & VH .VH is generally associated with PVD and layering of blood in inferior portion of the eye to produce sheet-like echoes
Membranous Structure:
1 PVD: it can be partial or total. The post hyaloid may completely detach from or remain attached to OD, producing a funnel shaped configuration On B-scan, detached post. vitreous face is usually smooth and may be thick posteriorly if blood is layered along its post.surface. In PVD with normal eye, the reflectivity is very low, high

gain(90dB) setting is required the reflectivity disappears lowering the sensitivity,under 70 dB.It should be kept in mind that PVD with haemorrhage shows extremely high reflectivity.
Kinetic echography typically shows a very undulating movement that continue
after the eye movements stops,which differentiates PVD from less mobile retinal and choroidal detachments.fig RD with PVD
2 RD: In presence of opaque media, the differentiation between PVD and RD is challenging. RD is usually uniformly high reflective and of even thickness whereas tilting of probe in different direction may reveal uneven thickness & reflectivity of membranes in PVD The image of PVD will disappear from the screen at higher gain setting(70dB) than a RD(40-50dB). PVD may appear as a line with multiple discontinuities or may be completely detached from O kinetic Echography,a PVD has much more after movements when compared to RD.The mobility of RD depends on duration of the detachment.Recent bullous RD may be highly mobile,whereas chronic RD
with PVR appear stiff.
Rhegmatogenous RD:appear as a mobile membrane attached anterior to the ora serrata and posterior to the ON head

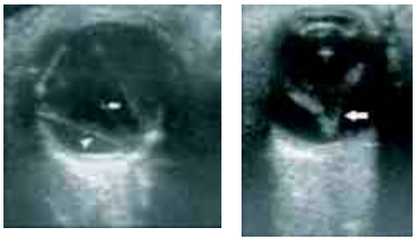


figure : bscan with RD

3 Choroidal Detachment: CD appear as smooth, convex elevations from the posterior eye wall. In massive CD, choroids from opposite fundus areas may touch in the middle of the vitreous cavity-"Kissing Choroid"
Fig bscan with choroidal detachment
Differentiation from RD:
CD is usually thicker & more reflective than a RD.
It usually extends anteriorly to involve cilliary body unlike RD which stop at ora serrata.
With ocular movements,CD are not particularly
mobile & donot show after movements
Differentiation between Exudative & Haemorrhagic CD:
Exudative CD = more sonoluscent
Haemorrhagic CD = presents multiple echoes in the suprachoroidal space
Mass Like Leison:
Most important is intraocular tumour.
Bscan is used for Topographic localization,Size Measurement.Shape evaluation,Tissue characteristics of the Tumour Gain setting is reduced to better delineate the different structures of post eye wall
Probe is directed as perpendicular as possible to the tumour surface Once the ideal position is obtained,the image is frozen.Measurement calipers are positioned on the ant. Surface & base of the tumour. Measurement of the lateral & posteroanterior extension of the tumour are then performed in a similar way on
transverse & longitudinal scan
Shape: Melanoma begin as a dome shaped solid thickening of choroids protruding into vitreous,but when Bruch's membrane ruptures,the mass expands
toward the vitreous pushing the retina & RPE infront of it -- Collar button /
mushroom configuration

Intensity of initial echoes (i,e border echoes) of the lesion & number & intensity of internal reflections.Generally intial echoes are very strong in tumour lesions because of abrupt changes in the acoustic impedence from the normal vitreous to tumour tissues.(Choroidal Osteoma= formed by deposition of bony tissues especially around ON & appear as a yellow orange
well defined lesion)
Internal reflections are directly dependent on variation in the acoustic impedence
of the tissues which inturn is a function of histologic type of tumour. Melanomas
are fairly homogenously tissues & have little internal reflection .
Fig bscan with melanoma
Haemangioma have spongy characteristics (being composed of stroma, bloodvessels,
blood) - Highly echogenic interior
Metastatic Leison- Moderate to high internal reflectivity
bscan with retinoblastoma
Ocular Trauma:
Large amount of coupling gel should be used as it allows better contact with less pressure
on the globe Placing the USG probe in a sterile rubber sleeve (surgical gloves) helps to reduce contamination

IOFB:FBs are strong reflectors because of their acoustic impedence is so different from

that of other body tissues.Once a highly reflecting echo is identified (suspected IOFB),the gain is gradually reduced,while the probe is held still in the same direction. If
the echo is caused by an IOFB it will be the last echo to disappear from the screen.
Mainly two acoustic artifacts are caused by IOFB & may help in their localization
Shadowing An area of shadowing appears posterior to the IOFB due to lack of sound
waves travelling through IOFB

Ringing / Trailing : Spherical IOFB (small lead pellates) produce a reverberation of sound waves between the objects surfaces,this appears on the screen as multiple reflective echoes posterior to IOFB It is recognized because a slight movements of the eye / probe causes simultaneous movement of the IOFB echo & of all the associated echoes of ringing

Structures that mimic an IOFB:
1. ON head drusen
2. Choroidal Calcification
3. Small bubbles of air,entered at the time of injury (this is because of increased

speed of sound through air compared to vitreous. The presence of IO air in a

patient with endophthalmitis in absence of an open globe or recent surgical

intervention is sugg
FIG :IOFB
estive of Clostridial / Bacillus infection)

Areas where an IOFB may not be easily seen are –AC,Lens,Cilliary Sulcus,Pars plana
Standoff,immersion scans or high frequency USG can be used
FB may be small & linear and so may be missed if the probe is not perpendicular to
the flat side of the FB
Due to decrease in speed of sound in blood compared to aqueous or vitreous , a small
IOFB in presence of dense vitreous haemorrhage

A Rare Case of Vogt Koyanagi Harada Syndrome in Pregnancy

Dr. Omkar Telang, Dr. Sanika Borkar, Dr. Shanta Motwane, Dr. Ajay Dudani

Case

A 26 yr old female with 2 months of amenorrhea presented with a history of sudden unilateral blurring of vision in the Right eye.

The Best Corrected Visual Acuity in the right eye was 6/60.Vision in the left eye was 6/6.

Fundus examination revealed central serous retinopathy in the right eye with accumulation of sub retinal fluid. The left eye was normal.

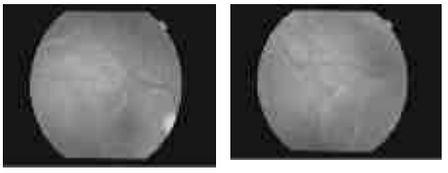


Fig. A and Fig.B show central serous retinopathy with exudative retinal detachment.

Retina specialist was consulted. A differential diagnosis of exudative CSR in pregnancy was made and no steroids were given. The patient was advised to undergo medical termination of pregnancy.

Post Medical Termination of Pregnancy, the patient presented with worsening of the condition in both the eyes. Vision in the right eye deteriorated to hand movements close to face and the Best Corrected Visual Acuity in the left eye was finger counting 2 mts.

Fundus examination revealed CSR with multiple pockets of exudative Retinal Detachment in both the eyes. Mild Optic disc hyperemia was also seen. Mild anterior vitritis with iris pigments

Fig. D shows multiple pockets of exudative retinal detachment.

Fig C FFA shows pinpoint areas of leakage at the level of RPE on anterior capsule of the lens were noted. Fundus Fluorescein Angiography was therefore done which revealed multiple pin-point areas of leakage at the level of RPE.Fig. E and Fig. F shows FFA picture depicting multiple pin-point areas of leakage at the level of RPE.

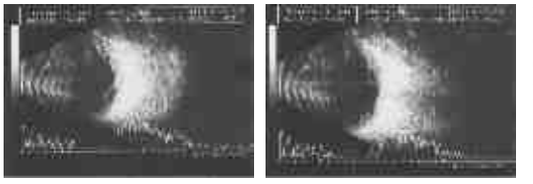
The findings were confirmed by USG which showed both eyes exudative retinal detachment and therefore some other disorder, probably Vogt-Koyanagi-Harada Syndrome was suspected.

Fig. G and H- USG shows both eyes exudative retinal detachment.

The patient's vision in both eyes deteriorated further to Hand movements close to face in the next three weeks. On examination, the blood pressure was normal. HLA typing revealed the presence of HLA-DR4.Other hematological

investigations were normal. X-ray findings were normal. After much deliberation, the patient was started on oral prednisolone 1mg/kg/day.Patient's vision recovered to 6/9 in both eyes over next six weeks.

Fundus examination revealed leopard spotting and sunset glow of the fundus and subretinal



pigmentation on Fig. I and J shows leopord spotting and sunset glow of the fundus Figs. K,L and M shows subretinal peripapillary and macular pigmentary deposits in resolving VKH.Fig N and O shows both eyes exudative retinal detachment.

Patient was put on maintainence dose of prednisolone of 10 mg/day for six months.

Patient did not show any signs of neurological, cutaneous and auditory manifestations.

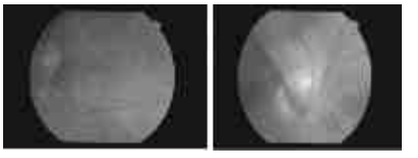


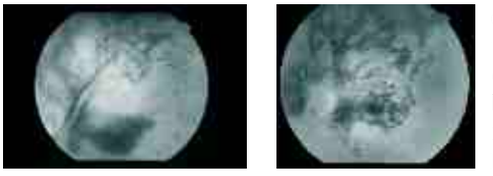
Fig P shows normal retina post steroid treatment for VKH

Discussion

This case is very interesting and the patient would have lost vision if the diagnosis of VKH syndrome would have been missed and timely steroids not given.

Vogt-koyanagi-harada (vkh) syndrome Introduction

Vogt Koyanagi Harada syndrome is a multisystem disorder characterised by bilateral granulomatous panuveitis with serous retinal detachment and signs of meningeal irritation with or without auditory disturbances and cutaneous involvement.



This condition was first described by Vogt in 1906[1] and Koyanagi in 1929[2].In

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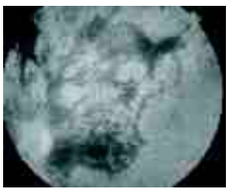
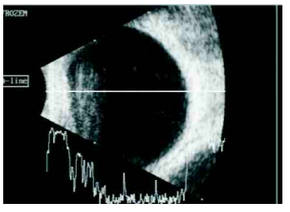


1926, a similar condition was described by Harada[3] in Japan.Originally separately categorized as Vogt-koyanagi syndrome

(poliosis,vitiligo,alopecia and chronic anterior uveitis) and Harada syndrome[3](bilateral posterior uveitis with exudative detachments and CSF abnormalities such as pleocytosis)the 2 are now clubbed together as the distinction in clinical pattern is not always present.[21]



Revised diagnostic criteria
1.COMPLETE VKH : Ophthalmic + neurologic +auditory +cutaneous
2.INCOMPLETE VKH : Ophthalmic + cutaneous or neurologic and auditory
3.PROBABLE VKH : Ophthalmic alone.

Epidemiology
More common in Japan.[4,5]
Races – darkly pigmented,typically Asians.[6,7]
Sex – females more than males[8]
Age – second to fifth decades affected[8]
Etiology

VKH disease currently is considered to be a cell-mediated autoimmune disease[9] directed against melanocytes in the uveal tract,skin,meninges and inner ear.

Genetics
The strong association between VKH disease and certain racial and ethnic groups suggests that the disorder may have an immunogenetic predisposition. HLA-DR4, HLA-DR53, and HLA-DQ4[10,11] are associated strongly with the disease.

Clinical Features
FOUR STAGES:

1. ProdromalStage
   This stage lasts for few days and consists of headache, orbital pain, nausea, stiff neck,fever,vertigo,meningism.
   CSF pleocytosis is present in 84% cases
2. Acute Uveitic Stage
   This stage follows prodroblurring of vision.
   mal stage with acute bilateral Clinically, this is manifested as bilateral posterior uveitis with retinal edema, optic disc hyperemia or edema, and eventually serous retinal detachments[12]. Often, an accompanying anterior uveitis characterized by mutton-fat keratic precipitates and iris nodules are present[22].The intraocular pressure may be elevated because of forward rotation of the lens-iris diaphragm.
3. Chronic or Convalescent Stage
   This stage follows after several weeks of acute phase and is characterized by depigmentation of skin and choroid.perilimbal vitiligo (Sugiura's sign)[13] is a earliest depigmentation(reported only in Japan).Choroidal depigmentation with clumping of pigments (Leopord spotting)[14]occurs 2 to 3 months after acute stage and appears as an orange red discoloration of fundus(sunset glow)[15]
4. Chronic Rrecurrent Stage
   During the recurrent stage, patients may develop chronic panuveitis with recurrent granulomatous anterior uveitis

ExtraocularSsigns

- Cutaneous manifestations

Sensitivity to touch of the hair and skin may be noted during the prodromal stage. Vitiligo, poliosis, and alopecia[23] typically develop during the chronic stage. Vitiligo often is distributed symmetrically over the head, face, and trunk. The sacral region is a common site for the development of vitiligo. Poliosis may involve the scalp hair, eyebrows, and eyelashes

- Neurologic and auditory manifestations

Meningeal signs develop during the prodromal stage and include meningismus, headache, and occasional confusion. CSF pleocytosis is relatively common during the prodrome. Focal neurologic signs include cranial nerve palsies, hemiparesis, transverse myelitis, and ciliary ganglionitis.[24]
Inner ear disorders, including dysacusis, tinnitus, and vertigo[25], occur in as many as 75% of patients. Cochlear hearing loss occurs mainly in high-frequency ranges. Inner ear dysfunction improves several months after onset in most patients

Investigations

1. Fluorescein angiography[16]

Acute VKH disease: Multiple pinpoint areas of leakage at the level of the RPE overlying areas of choroiditis are visible during the arteriovenous phase.The pinpoint areas gradually enlarge and stain the adjacent subretinal and sub-RPE fluid
Chronic VKH disease: This is characterized clinically by depigmentation of the choroid. With angiography, signs of RPE atrophy are visible, such as a moth-eaten appearance, multiple window defects.

2. Optical coherence tomography (OCT): Serous retinal detachments with subretinal septa may be visible, especially early in the disease. OCT may be useful to monitor serous detachments and response to therapy.
3. Ultrasonography[17]: The most characteristic finding is diffuse, low-to-medium reflective thickening of the posterior choroid. Additional findings include serous retinal detachments, mild thickening of the sclera and / or episclera adjacent to areas of choroidal thickening, and mild vitreous opacities. These ultrasonography features may be useful in monitoring the response to therapy.

4. Indocyanine Green (ICG) Angiography[26]: during acute stage revealed abnormal dark background that resolved during recovery.

5. MRI[27]: During the active phase of the disease, the choroid is thickened visibly and enhances following administration of gadolinium

Histologic Findings[13]

Nongranulomatous inflammation with plasma cell infiltrate of the uvea. Lymphocytes, multinucleated giant cells and epithelioid cells may be seen in the uvea of patients with VKH disease.

Lumbar Puncture - CSF Pleocytosis

Treatment

The key to successful therapy for VKH disease is early and aggressive treatment with systemic corticosteroids. [18]

Systemic therapy

For most patients with bilateral serous detachments and severe visual loss, begin therapy with systemic prednisolone (1-2 mg / kg / d).

Severe cases require intravenous methylprednisolone (up to 1 g / d) for several days before beginning oral prednisone (1 mg / kg / d).

Most patients require therapy for 6 months and occasionally up to 1 year before successful tapering of systemic corticosteroids. In general, systemic therapy should not be discontinued during the 3 months following the onset of the disease because of the risk for recurrence.

For those patients who fail to respond to high-dose systemic corticosteroids or develop intolerable adverse effects, immunodulatory therapy, such as cyclosporine[19], tacrolimus, mycophenolate mofetil, azathioprine, cyclophosphamide, or chlorambucil[20], should be instituted.

o In chronic exudative detachment that doesnot respond to medical therapy,drainage of fluid from non rhegmatogenous detachment might prove beneficial.

o Laser therapy is indicated from choroidal neovascular membrane.

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An Analytical Study of Congenital and Developmental Cataracts

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Background / Introduction :

Cataract is responsible for about 10% blindness among children in India. The etiology is not well defined especially for childhood cataracts and epidemiological data for Indian population is not available in details. This study was performed to survey the causes of childhood cataracts and to identify the preventable factors.

Material and Methods :

It is a hospital based analytical study on 30 children. The morphological type of cataract was determined using slit lamp or operating microscope. Parents of patients were interviewed in their native language using a standardized questionnaire. Biochemical and microbiological tests were performed as required.

Results :

Out of 30 children, the commonly affected age group was upto 4 years. The most common type of cataract was lamellar. Male to female ratio was 1.3:1. Bilateral cg. cataract were 70 and unilateral were in 30% cases. 73.1% cases were associated with ocular defects. 20% cases had associated systemic diseases and 23.3% cases had h / o consanguinity.

Conclusion :

Our study shows that majority of non-traumatic cataracts are due to potentially preventable causes. Health education to women of child bearing age and ophthalmological checkup early in life can prevent visual morbidity.

Keywords : Congenital cataracts, Developmental Cataract, Ocular and systemic associations.

Introduction :

The prevalence of blindness in children varies from approximately 0.3 / 1000 in developed countries to 1.2 / 1000 in developing nations.[1]

Vision 2020 : The right to sight, a global initiative to eliminate avoidable blindness, was launched by WHO on 18th February 1999. Its objective is to assist member countries in developing sustainable system which will enable them to eliminate avoidable blindness from major causes which include childhood blindness by year 2020.[2] The high number of blind years resulting from blindness during childhood is one of the reasons why the control of childhood blindness is a priority of the WHO and International Association for Prevention of Blindness (IAPB).[3] Cataract in pediatric age group create a major ophthalmological, socioeconomic and national problem. Around (0.4%), in 250 newborns have some form of congenital cataract.[4] These cataracts often deprive its victim of the vision in the crucial period of life. Hence successful visual rehabilitation is critical.

Since no such study was done in our region, we analysed the congenital and developmental cataracts with a view to study its correlation with ocular and systemic conditions. We also studied the various morphological forms.

Aims and Objectives :

1. To study the relation of various etiological factors with congenital and developmental cataract.
2. To study various morphological types of congenital cataracts.
3. To evaluate relationship of various ocular anomalies and associated systemic diseases with the cataracts.

Material and Methods :

This study was carried out in the Department of Ophthalmology, Government Medical College, Aurangabad. It is an analytical study of congenital and developmental cataracts in children attending the Out Patient Department (OPD).

Study design analytical study done from January 2006 to July 2007.

Setting and study population.

All children less than 18 years of age with congenital or developmental cataract attending Ophthalmology OPD during study period were included.

Approval of Ethical Committee of Government Medical College Aurangabad was taken.

30 Children were included in our study, of which youngest child was of 4 months and eldest was 16 years of age.

Inclusion criteria for case selection :

1. Patients of either sex upto 18 years of age with unilateral / bilateral cataract diagnosed clinically, ophthalmologically and with slit lamp.
2. Congenital cataracts included are present since birth and developmental meant any cataract after birth.
3. Patients with associated ocular or systemic abnormalities.
4. Consanguinity was considered.

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I grade in case of parent's marriage amongst blood relation, II grade in marriage between cousins and III grade in marriage between second cousins.
5. Low birth weight is birth wt.<2500 gm.
6. Systemic abnormalities like mental retardation, Down's Marfan's or neurofibromatosis were considered only after pediatric evaluation.
7. Vision was tested by Snellen's chart (older children >5 years), animal charts in verbal, following light in children <1 year.

Exclusion criteria :
1. Secondary cataracts like traumatic, post uveitis, post ROP were excluded.

Procedure :

Patients satisfying all inclusion and exclusion criterion were enrolled for the study. Those included in the study were required to answer the questionnaire by parent or legal guardian after taking informed consent.

Blood tests and fundus examinations were done.

Statistical Analysis :

The collected data was worked upon Excel spreadsheet. 2 tests was applied to draw the probability to find the significance of parameters.

Probability $P<0.05$ were termed as significant while probabilities $P>0.05$ were termed as non-significant. Probability $P<0.005$ was termed as highly significant.

Calculations were done by using Software Epi Info 2002.

Results :

Out of 30 patients, 12 cases were below 4 years of age, followed by 11 cases between 4–9 years of age. Youngest patient was 4 months of age whereas eldest was 16 years of age.

Table-1 : Age Distribution

Age (Years)	No.of cases	Percentage
00 – 04	12	40.0
05 – 09	11	36.6
10 – 14	06	20.0
15 – 18	01	03.3

Table-2 : Sex Distribution

Sex	No.of cases	Percentage
Male	18	60.0
Female	12	40.0

The incidence of congenital cataracts and developmental cataracts is more in males (60%) than in females (40%).

Table-3 : Incidence of laterality

Sex	Unilateral	Bilateral	Total
Male	05	13	18
Female	04	08	12
Total	9 (30%)	21 (70%)	30

In our present study, bilateral cases were more than unilateral cases. While applying statistical analysis Odds ratio 1.3, relative risk 1.08.

Table-4 : Morphological types of cataract

Type	No.of cases	Percentage
Lamellar	12	40.0
Total congenital	08	26.7
Membranous	04	13.4
Sutural	01	3.3
Developmental - Punctate - Coralluform	 02 01	 6.7 3.3
Embryonic nuclear	02	6.7
Total	30	100.0

The commonest presentation was of lamellar cataract and least was sutural and corraliform.

Table-5 : Relation of cataract with ocular anomalies

Type	Male	Female	Total
Non-associated	17	10	27
Associated	01	03	03
Total	18	12	30

While applying statistical analysis Odds ratio 3.4, Relative risk 1.89.

Though 10% cases were shown to be associated with ocular anomalies, it was not shown to be statistically significant.

Table-6 : Association with ocular defects

Ocular Defect	Male	Female	Total
Nystagmus	04	03	07 (23.1%)
Strabismus	05	04	09 (30%)
Nystagmus + Strabismus	02	01	03 (10%)
Typical coloboma with microphthalmos	01	--	01 (3.4%)
Retinitis Pigmentosa	--	01	01 (3.4%)
Aniridia	--	01	01 (3.4%)
Total	12	10	22 (73%)

The above table shows that strabismus is the commonest associated ocular defect, followed by nystagmus.

· The case of aniridia was part of WAGR syndrome (Wilm's tumour aniridia, genitourinary abnormality, mental retardation).

· The case of R.P. was associated with hypothyroidism.

Table-7 : Relation of cataract with systemic disease

Systemic disease	Male	Female	Total
Cg heart dis with PDA	01	--	01
CRS	01	--	01
MR	01	--	01
Hypothyroidism	--	01	01
Rickets with convulsions	01	--	01
WAGR	--	01	01
Total	04	02	06

Table-8 : Relation of cataract with consanguinity

	Male	Female	Total
Heredity	04	03	07
Consanguinity	05	02	07
Total	09	05	14

Discussion :

We carried out an analysis of 30 cases over a period of 19 months. This, being a hospital based study incidence of congenital and developmental cataract and sample size could not be calculated.

Age and sex distribution :

The youngest patient was a 4 months old whereas eldest was 16 years old. The age of presentation was found to be maximum at birth. 60% and 30% presented between birth to 3 years of age.

We had 60% male children as against 40% female patients. The M : F = 1.3 : 1.

Rahi et al (2000)[5], median age of detection of cataract was 8 weeks with 70% detected by the age of one year.

Narendran R (2007)[6], 21% presented before 3 months of age and 68% before one year.

Martinez et al (2007)[7] 75% diagnosed within 1 month of clinical manifestation. As far as sex incidence is concerned, present study tends to match findings with those of Singh et al and Narendran K.

Laterality in the present study, out of 30 cases, 21 (70%) were bilateral cataracts of which males accounted for 61.9% and female 31.1%. Our findings tend to coincide with those of Rahi et al. Narendran K (2007) series suggests that bilateral cataracts 4 times more common as compared to unilateral.

Martinez et al (2007) found 50% bilateral and 44% unilateral cataract.

Associated ocular anomalies :

In our study, the commonest ocular defect was found to be strabismus (30%) followed by nystagmus (23.1%)

Jain et al (1983) found strabismus and nystagmus to be most common associated ocular anomaly standing at 28.94% and 15.79% respectively.

Rahi et al (2000), associated ocular anomalies were more common in unilateral than bilateral cases. Microphthalmos being most commonly associated.

Systemic Association :

In our study a total of 20% of cases were associated with various systemic disorders including 26% of

cases associated with some delayed milestones with 6% having frank mental retardation.

Jain et al (1983)[8], mental retardation found to be most commonly associated systemic abnormally, 9% associated with metabolic disorders and 5% with various syndromes.

Rahi JS (2000)[5], various systemic associations highlighted in study. Down's syndrome, prenatal rubella infection, cerebral palsy and metabolic disorders.

Bhatti et al (2003)[10], 22% of cases were associated with some or other syndrome and 20% with major birth defects.

Conclusion

1 The most commonly clinical congenital cataract was Lamellar cataract (40%)s
2 The most commonly affected age group was upto 4 years (40 %)
3 The male:female ratio is 1.3:1
4 Bilateral Congenital cataracts were 70% &unllateral were 30%,whereas73.1% cases were associated with ocular defects&26.9% were not associlated with any ocular defects.
5 20% cases were having systemic diseases associated with congenital cataracts.
6 23.3% cases were associated with heredity or consanguinity.

In our study, it was noticed that most of our congenital (non traumatic) cases are due to potentially preventable diseases .Ophthalmological check up s at the earliest & awareness amongst paediatricians for early reference is also necessary to prevent visual morbidity & reduce the socio-economical burden on the family & society at large.More such studies must be undertaken for establishing the etio-pathogenesis to prevent ocular morbidity.

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