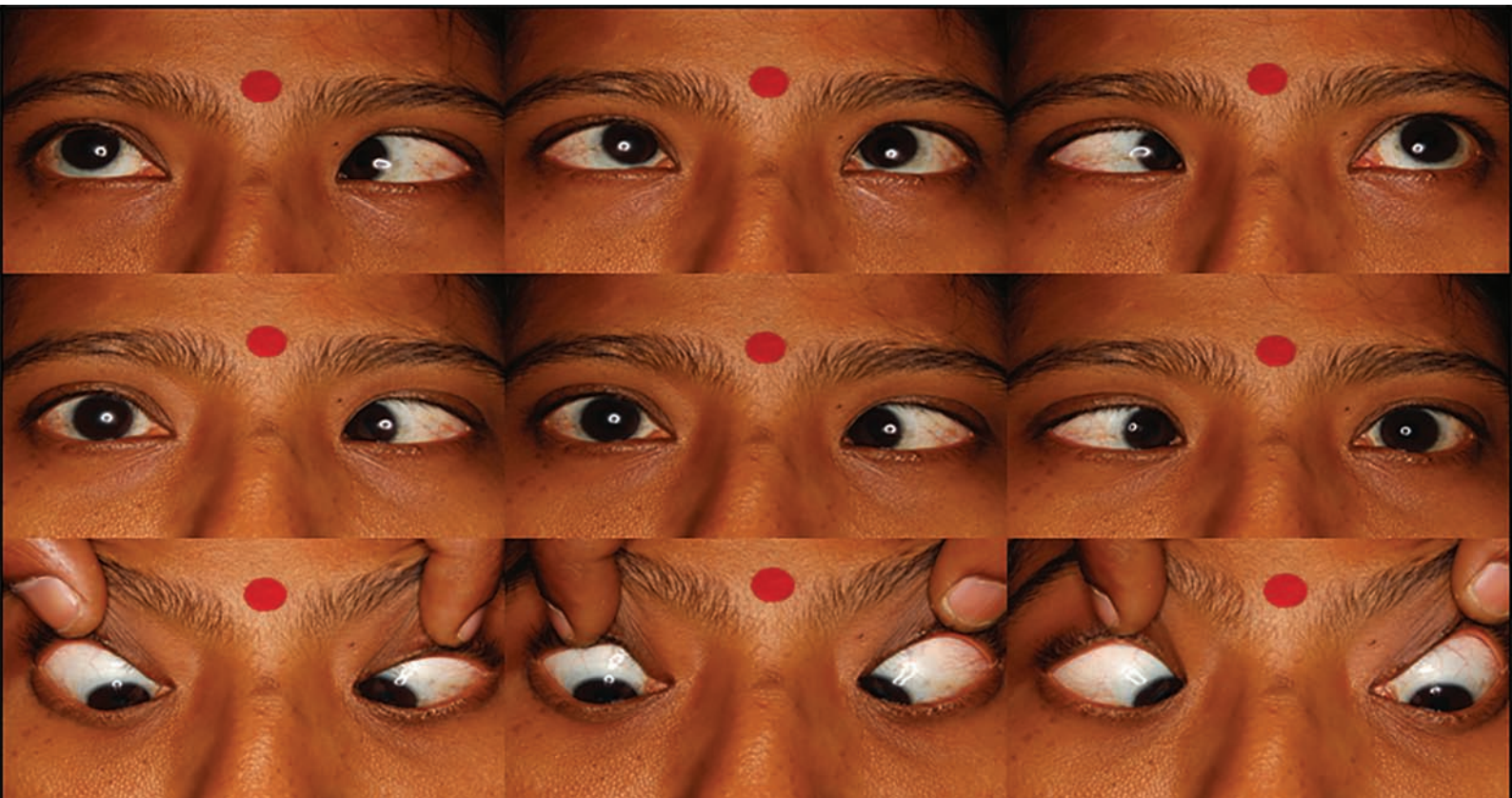


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Sankara Nethralaya – The Temple of the Eye.

It was in 1976 when addressing a group of doctors, His Holiness Sri Jayendra Saraswathi, the Sankaracharya of the Kanchi Kamakoti Peetam spoke of the need to create a hospital with a missionary spirit. His words marked the beginning of a long journey to do God's own work. On the command of His Holiness, **Dr. Sengamedu Srinivasa Badrinath**, along with a group of philanthropists founded a charitable not-for-profit eye hospital.

Sankara Nethralaya today has grown into a super specialty institution for ophthalmic care and receives patients from all over the country and abroad. It has gained international excellence and is acclaimed for its quality care and compassion. The Sankara Nethralaya family today has over 1400 individuals with one vision – to propagate the Nethralaya philosophy; the place of our work is an Alaya and Work will be our worship, which we shall do with sincerity, dedication and utmost love with a missionary spirit.

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Cover Puzzle

Contributors: Dr. Nidhi J Chopra, Dr. Sandeep Mark Thirumalai, Dr. Meenakshi S.

A 26 year old female presented to us with complaints of inability to move the eyes outwards since past 4-5 years. No h/o trauma, previous squint, diurnal variation, thyroid disorder, DM, HTN. Refraction OD -19.0 DS 6/18, N6, OS -20.0 DS 6/18, N6. OU Anterior segment WNL. OU Fundus posterior staphyloma with barraged lattices. OU Axial Length 30.98mm. Ocular motility is as shown in the cover page. What is your diagnosis?

(Answer to cover puzzle on page: 5)

Inquiries or comments may be mailed to the editor at insighteditor@snmail.org

Recent Advances in Paediatric Ophthalmology

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The field of paediatric ophthalmology has witnessed rapid strides in many areas. This has been due to the advancements in basic science and clinical methods in paediatric ophthalmology.

By making childhood blindness as one of the priority areas for vision 2020, WHO has helped refocus a lot of attention to this field.

Screening for childhood vision impairment is the first step towards early detection and treatment and prevention. In the rural areas, screening is done by Anganwadi workers, while there has been a steady increase in school vision screening and training of teachers. The Government too has pitched in through its Sarva Sikshabhyan Programmes. In countries like Bangladesh 'the key informant method' has been hugely successful.

A very popular modality of screening is the photoscreening technique. Several models of photoscreeners are available. These have been used successfully in the USA, Australia, New Zealand, and Taiwan. NGOs such as Lions Club International have been active in this area. This was able to reliably screen even infants in the 6–9 months age group.

In regards to surgical approaches to various types of strabismus, there have been several efforts at augmenting results with various modifications and additions to existing techniques. For infantile esotropia a group from Turkey found that botulinum toxin injection into the medial recti as effective as incisional surgery, while Lueder *et al.* found that botulinum toxin augmented bimedial recessions were good for large angles. A study from Germany suggested adding a posterior fixation suture to the surgical plan.

Approach to intermittent exotropia has always been a hot topic of debate the world over. Clinicians are still trying to answer fundamental questions regarding this condition. A study from the United Kingdom (UK) looked at the question whether children with intermittent exotropia really deteriorate with time and found that this rare. The best alignment in the early postoperative period has been a matter of controversy and continues to be so with studies from USA and UK suggesting that there is no correlation between early post op alignment and long-term motor

outcomes while a study from Japan suggested that early esotropia was associated with long-term successful outcome.

Adjustable suture strabismus surgery in adults is well described in literature and has been around for decades. The same principles have been extended to children with groups describing excellent results. Children as young as 6 months underwent this technique and the adjustment was done anywhere starting from the recovery room to 4 days after the procedure. Studies also varied in using either topical anaesthesia or propfol sedation for the adjustment.

Control of progressive myopia is an area of intense research and speculation for clinicians and researchers alike. The ATOM study which used Atropine for control of myopia showed a clear benefit with a good safety profile long term. Several studies have followed using lesser percentages of atropine to minimize side effects. Contact lenses by themselves have proven ineffective. Recent studies suggest increasing outdoor activities and reducing near work as promising interventions for myopia control.

Paediatric ophthalmology will continue to be a hot area for research, for all eye care professionals, NGOs, and basically for anyone who cares about children and their welfare

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Cortical Visual Impairment in Children

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Cortical visual impairment (CVI) has become a major contribution to childhood blindness due to advanced neonate care, which enables survival of babies with significant brain damage.

Features of CVI in children can be quite different from that in adults in many aspects like; spectrum of severity, pupils may be abnormal, nystagmus can be present, and optic atrophy may be coexistent.

Causes of CVI in children

Perinatal hypoxic ischaemia (asphyxia) is the most common cause in children. It is due to ischaemic damage to the watershed zones in the brain. It may involve different regions of the brain; in premature infants the periventricular area is common to be involved, whereas in mature infants parieto-occipital or the frontal area is involved. These children generally show some improvement in their visual function with time. This improvement is due to mechanisms such as rerouting of axons, reactive synaptogenesis, and interruption of axon necrosis.

Postnatal hypoxic ischaemia caused by immediate postnatal problems like hypotension or hypertensive crisis, cyanotic heart disease, cardiac procedures, thrombo embolism, polycythemia, trauma, infections like meningitis, etc. can also cause CVI in infants.

Other causes could be congenital malformations like parieto occipital encephaloceles, Chiari malformations, Dandy-Walker malformations, porencephalic cysts, etc. Head trauma either accidental or battered baby syndrome can lead to transient or permanent visual impairment.

Metabolic and neurodegenerative conditions like hypoglycaemia, MELAS, Adrenal dystrophy, Leigh's disease, etc. can be rare causes of CVI in children. Meningitis or encephalitis due to neonatal sepsis can also cause CVI. Hydrocephalous can affect both the anterior and the posterior visual pathway to impair vision.

All phases of seizures, pre-ictal, ictal, and post-ictal, can manifest with visual impairment usually transient. Not so uncommonly bilateral episodic vision loss could be the only symptom of occipital epilepsy. So, an Electro encephalogram should be part of a work up of any child with unexplained vision loss.

CVI could also be a component of neurological and systemic conditions.

Visual function

Children with CVI can present with varied vision characteristics. Visual function assessment can

pose challenges due to the other associated conditions like poor coordination, delayed development, poor motor function, effects of medications that the child might be using, and lots of other aspects. But a good assessment is essential to enable designing of intervention for overall rehabilitation of such children. In many instances, vision may be the only sense with which functional improvement is possible. Visual behaviour assessment rather than visual acuity measurement is what is important.

Various vision characteristics can be as follows:

- 1 Can range from blindness to barely detectable defect.
- 2 If unilateral or macular sparing can have normal visual acuity.
- 3 Better vision in familiar surroundings.
- 4 Close viewing.
- 5 Variable visual responses related to state of alertness, physical health, etc.
- 6 Light gazing or photophobia (both can co-exist) can be retinal, thalamic, and cortical.
- 7 Colour perception may be intact. The reasons may be because
 - a bilateral response,
 - b diffuse representation,
 - c extra geniculostriate visual system for colour vision.
- 8 Eccentric viewing.
- 9 Severe construction of fields.
- 10 Better vision for moving objects (can see better while travelling).

Absence of nystagmus

For nystagmus to occur there needs to be an intact geniculostriate pathway and damaged anterior visual pathway. By this theory, presence of nystagmus in the setting of mixed vision loss could mean lesser involvement of the posterior visual pathway. So absence of nystagmus could then mean severe anterior as well as posterior disease.

Other associated ocular abnormalities

In children with CVI associated ocular anomalies are very common. They can have retinal and optic nerve involvement. Optic nerves can be hypoplastic and optic atrophy is common.



Optic atrophy in children with CVI can imply concurrent anterior visual pathway insult or retrograde trans-synaptic degeneration. In humans this is possible only if the cortical insult happens in the early neonatal period and so can be a feature of only congenital cortical vision problem. Adults with only cortical vision impairment will never manifest optic atrophy. There is a disproportionately lower incidence of optic atrophy reported in children with CVI. This could be because of overlooking, cortical damage due to damage to visual association areas, nature, and location of the cortical insult not sufficient to cause retrograde degeneration, and other mechanisms responsible for retrograde degeneration. The differences between optic atrophy which is primary and that caused by retrograde degeneration are documented healthy optic nerve prior to insult (this is not possible in children) and subsequent optic atrophy, presence of nystagmus may suggest primary cause, and optic atrophy with an intact pupillary response.

Investigation and diagnosis

Computed tomography and magnetic resonance imaging (MRI) of the brain are the modalities of choice to localize and determine the extent of damage.

Imaging findings in CVI:

- 1 normal to virtually absent visual cortex;
- 2 diffuse cerebral atrophy;
- 3 occipital;
- 4 periventricular leucomalacia;
- 5 cerebral dysgenesis;
- 6 parieto occipital ischaemic changes.

Anatomic imaging findings may not correlate with the functional aspect of the brain in these situations. Functional imaging methods like PET/SPECT techniques can provide more useful information on the functional aspects especially when MRI is normal.

Visually evoked potential (VEP)

- 1 VEP tests the intactness of posterior cortical visual pathways but not perception.
- 2 Can have technical and interpretational pitfalls.
- 3 Flash VEP may be abnormal in neurological disorders even in patients with normal vision.
- 4 Does not have prognostic benefits.

Electro retino gram is useful to exclude associated retinal disease.

Prognosis and management

Cortical vision problems due to the common non-metabolic causes are caused by a single one time insult and hence are almost always non-progressive. So some improvement is the rule. Management is multidisciplinary involving the paediatric neurologist, ophthalmologist, physiotherapists, occupational therapist, and of course parents. There is a need for repeated assessments to appreciate the improvement in various functions and tailor the therapies

Role of the paediatric ophthalmologists is to provide relevant ophthalmic care like correcting refractive errors when high, accommodation issues, strabismus management, cataract surgeries when necessary and to provide low vision aids.

Delayed visual maturation

Delayed visual maturation (DVM) is a distinct entity and is usually a diagnosis in retrospect. It is a very narrow diagnosis with strict exclusion criteria. By definition, it is a condition where a child with normal neurological examination and no causative factors like perinatal asphyxia, cerebral abnormalities, etc. show subnormal visual behaviour but

have normal pupillary response, normal VEP, etc. and who on follow up develop full visual function.

Pathogenesis for DVM is unknown. The fact that VEP is normal means the visual pathway from the retina to the cortex is unaffected suggesting possible involvement of the visual association areas. The fact that myelination of the cortical association is completed last may support

this assumption. Or DVM could simply be a mild form of CVI. Some children with DVM have developmental problems, autism, etc.

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Answer to the cover puzzle

The patient has **Heavy eye syndrome**, also called Myopic strabismus fixus or progressive esotropia fixus. It is a rare strabismus disorder seen in high myopes > -6.0 DS in which there is acquired progressive esotropia, limited abduction and hypotropia This condition may progress over several years. The enlarged globe in high myopia herniates superotemporally and retroequatorially through the muscle cone. On CT scan Superior rectus is nasally displaced, while Lateral rectus is displaced inferiorly. Radiological evidence of muscle displacement (SR, LR) is essential to establish the diagnosis. FDT revealed limited elevation and abduction of eye. Bilateral simple loop myopexy under GA was performed for this patient.

Glial Cells in Retinal Disorders: An Overview

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The eye has evolved to limit intraocular inflammation so as to protect visual acuity. The normal brain and retina are protected by vascular endothelium at the blood brain barrier and blood retinal barrier, while epithelial cells of the choroid plexus form one more barrier. Additionally, astrocytic end feet and the parenchymal basement membrane form a further barrier, the glia limitans. A range of other mechanisms exist to limit immune responses in the retina. An active anti-inflammatory milieu is maintained as well as suppressing systemic induction of immunosuppressive regulatory T cells by eye-specific mechanisms.

This protection is relative rather than absolute and is partial. Apart from the lack of antigen presenting cells (APCs) and a lymphatic drainage system, a further blockage is in place by production of FAS and TGF- β , implicating soluble factors released either in paracrine or autocrine manner as contributors to the ocular immune privilege. Traditionally, innate immunity has been viewed as the first line of defence discriminating benign from dangerous molecules. Emerging literature suggests that innate immunity actually serves as a system for sensing signals of 'danger', such as pathogenic microbes or host-derived signals of cellular stress, while remaining unresponsive to 'self' motifs, such as normal host molecules, and dietary antigens. Infectious agents may cause neurological disease through a direct lytic effect, by inducing immunopathology directed against CNS tissue, by induction of immune responses that damage CNS tissue in a bystander fashion, or through induction of molecular mimicry.

In the normal CNS, the cells capable of immune response are microglia, astrocytes, and ependymal cells. There are also CD11c+ cells found in the juxtavascular parenchyma, having processes extending into the glia limitans, with characteristics similar to dendritic cells (DCs). Apart from these populations of DCs, the CNS parenchyma is relatively devoid of APCs. The retina has both astrocytes and microglia. These cells have evolved to permit effective immune surveillance while limiting immune pathology. Infection of glial cells inflicts excitotoxic and inflammatory response because there is an increase in glutamate levels and oxidative stress, together with over-expression of proinflammatory cytokines and chemokines, resulting in collateral damage. In the retina the number of astrocytes and microglia are few compared with the predominant glia, the Muller cells. The Muller glia are one of the most

robust cells in the retina and are involved in practically all types of injury that occur. Normally, they function to provide a stable environment in the retina. By mediating trans-cellular ion, water, and bicarbonate transport, Muller cells control the composition of the extracellular space fluid. Additionally, they provide trophic and anti-oxidative support of photoreceptors and neurons and regulate the tightness of the blood-retinal barrier. Recent work has also identified a Muller glial role in innate immune response. It has been established that the retinal Muller glia senses pathogens via TLR2 and contributes directly to retinal innate defence via production of inflammatory mediators and antimicrobial peptides (Shamsuddin & Kumar, 2011). We have also shown that they are a major source of inflammatory factors during infection (Krishnan & Chatterjee, 2012).

Inflammation in the retina need not be a by-product of infection. In age-related macular degeneration (AMD) persistent inflammation may be a reason for pre-disposition. Current reports propose a vital role for innate immunity and complement over-activation for AMD pathogenesis. AMD primarily affect the photoreceptors, RPE, Bruch's membrane, and chorio-capillaries. Early work had shown RPE to possess dysfunctional complement system in AMD; recent reports also suggest the role of microglia. A recent study on age-related change demonstrates recruitment of leukocytes and activation of the complement cascade in mouse RPE and choroid (Chen *et al.*, 2008). This involves the complement system and the innate immune response embodied by cytokines. Chemokines are cytokines with chemo-attractive properties, playing a central role in recruitment of immune cells to inflamed tissues. These chemokines bind to chemokine receptors on inflammatory cells such as macrophages to promote the mobilization of these cells out of circulation and into tissues. One chemokine receptor that has generated much interest in AMD is CX3CR1. The CX3CR1 chemokine receptor is a G-coupled receptor found on a variety of inflammatory cells, including microglia, macrophages, T-cells, and astrocytes. When bound by its ligand, CX3CL1 (also known as fractalkine), CX3CR1 moves leukocytes to inflamed tissues and subsequently causes activation of these inflammatory cells (Fong *et al.*, 1998). CX3CR1 and CX3CL1 are present in the retina and brain (Combadiere *et al.*, 1998). Histological analysis of AMD retinas has shown microglia to be the only cells to express the

receptor and has observed the presence of many activated microglia in the macular lesions (Gupta *et al.*, 2003). Activated microglia can speed up their proliferation, migration to damaged tissue, phagocytize debris, and secrete pro-inflammatory cytokines, and neurotoxins (Langmann, 2007). Photoreceptor injury noticeably increases after administration of activated microglia to healthy photoreceptors. Drusen formation is a notable feature in AMD. The notion that microglia contribute to drusen-like deposits is further strengthened by another study demonstrating that increasing fundus autofluorescence seen in aging wild-type mice, also contain lipofuscin granules (Xu *et al.*, 2008). Accumulation of macrophages has been linked to that of drusen in the Ccl2^{-/-} and Ccr2^{-/-} murine models. Key features in these models also include (Ambati *et al.*, 2003) lipofuscin accumulation, thickening of Bruch membrane, and increased melanosomes in the RPE. Furthermore identification of inflammation-associated SNPs emphasise the inflammatory underpinning that modulate AMD risk. These SNPs encode complement factors, chemokines and chemokine receptors, and toll-like receptors.

A variety of symptoms seen in patients can be classified as being causally related to uveitis. While some symptoms may be part of a generalized systemic syndrome in which the eye is one of several organs affected, others are confined to the posterior eye, such as sympathetic ophthalmia and birdshot retinochoroidopathy. It has also been suggested that an individual whose T cell repertoire contains retinal antigen-specific T cells with higher affinity may have a greater likelihood of developing uveitis. T cells capable of recognizing retinal antigens are primed in the periphery on microbial stimuli by antigenic mimicry. While APCs in the retina are not known, the retina does possess DC and microglia which have MHC class II molecules. While infiltrated T cells are considered as playing a major role in driving the inflammation, it is increasingly thought that retinal glia can also be part of the supporting cast. Glial support to the retina may be achieved in several ways, including inherent provision of neurotrophic support factors and by immunomodulatory mechanisms. Because no cell culture model can reproduce the full complexity of a human disease, it is necessary to develop and use a variety of models to represent the different aspects and diverse clinical/immunological manifestations of any disease. The immune nature of uveitis may be dissected in experimental autoimmune uveitis. Retinal antigens like CFA injected into animals can cause and replicate many of the features seen in patients. Future therapeutic approaches which might take advantage of tolerogenic administration of retinal antigen to correct defects in peripheral tolerance and to regulate T cell numbers

and/or functions are also likely to take into cognisance the glial cells.

Increasingly, stem cells in the retina and from non-retinal ocular tissue such as iris and ciliary body epithelia are being considered as mediators of neuroprotection. In the retina, the glial cells confer trophic support of injured neurons and Muller glia under certain conditions show progenitor potential (Bhattacharya *et al.*, 2008). Glaucoma is a common condition with a neurodegenerative component and pathophysiology in glia where current therapies are often insufficient. Persistently high intraocular pressure leads to retinal ganglion cell (RGC) death and visual impairment. Intraocular increase in pressure is the major diagnostic indicator of glaucoma, and is the only treatment that has been shown to reduce progressive visual loss. Strikingly, intraocular pressure reduction fails to alleviate RGC degeneration in a subset of patients with glaucoma. It is thus critical to think of adjunctive therapy along with reduction of intraocular pressure. In animal models of glaucoma it has been shown that oligodendrocyte precursor cells (OPCs), a type of neural stem cell, can protect RGCs from high glucose damage *in vivo*. As RGCs are the only neuron type in the retina to possess the myelin sheath, it is crucial for their survival to have intact myelin. The success of OPCs differentiating into myelinating oligodendrocytes depended on activation of the OPCs with inflammatory stimuli. They were also more successful in providing long-term protection. Intravitreal transplantation of oligodendrocyte progenitor cell in glaucoma show differentiated OPCs become myelinating that expressed myelin basic protein and other markers of mature oligodendrocytes. It has been suggested that a battery of factors secreted by activated microglia and likely astrocytes, may be responsible for OPC activation communicated by a diffusible signal. Indeed, multiple studies (Bull *et al.*, 2009) support the assumption that OPCs themselves can improve retinal neuronal survival, by secretion of diffusible trophic factors, such as IGF-1 and GDNF. While, OPC role in rescuing glaucomatous RGC neurons has been clearly, shown it is suggested that they can also alleviate neurodegeneration in an assortment of neuro-pathologies *in vivo*.

Many ophthalmic disorders occur at a higher frequency in immune-suppressed individuals. Such an example is AIDS, where much of the ocular manifestations were known beforehand but seen rarely. A genuinely new ophthalmic disorder, however, occurs in immune recovery uveitis (IRU), once the goal of immune system reconstitution, has been reached (Sudharshan *et al.*, 2013). Uncontrolled inflammation is a hallmark of disorders such as IRU. Paradoxically, this is caused in the retina and uvea as the HIV1 infected patient recovers on responding to successful anti-retroviral

therapy. Adjunct therapies include treatment with corticosteroids to inhibit inflammation. Unfortunately, many patients do not achieve a functional benefit, despite objective evidence of improvement (Holland, 2008). In the retina, Muller glia is the largest producers of cytokines and likely candidates that communicate by both diffusible signals and contact-mediated options. In our in-house cell culture model with Muller glia stimulated by the HIV1 coat protein Tat, simulating overwhelming inflammation, produce copious amount of pro-inflammatory factors, including several chemokines known to attract macrophage and monocytes (Chatterjee *et al.*, 2011; Krishnan & Chatterjee, 2012). An *in vitro* model is more easily manipulated and dissection of basic cellular and molecular mechanisms underlying progression of the disorder suggests that immune-modulation of Muller glia may be able to alleviate unrestrained cytokine production. Multiple points in the machinery for cytokine production can be influenced by HIV1 coat proteins to cause inflammation. Indeed slight modifications in the HIV1 Tat structure can affect differing aspects of the innate immune response apparatus. The mechanism of action acts at the level of transmembrane receptors, kinases, phosphatases, transcription factors, and non-coding RNAs.

Additional studies looking at specific glial cell types in the retina would aid our understanding of disease processes, refine treatments, and help in devising long-term strategies for the management of ocular disorders.

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Tips and Tricks in Paediatric Ophthalmology

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According to the World Health Organization in May 2009, childhood blindness remains a significant problem, with an estimated 12 million blind children below age 15.

Of the eye problems in children, the most important are refractive errors, squint, and amblyopia. Children may not complain if they do not see out of one or both eyes. Sometimes the only clue may be a good history from parents, a fine observation by the paediatrician or health care worker, poor performance in school, as well viewing the blackboard at a very close distance. Hence, all children suspected to have subnormal vision needs an eye exam.

Recommended examination frequency for the paediatric patient.

Patient age	Examination interval	
	Asymptomatic /risk free	At risk
Birth to 24 months	At 6 months of age	By 6 months of age or as recommended
2–5 years	At 3 years of age	At 3 years of age or as recommended
6–18 years	Before first grade and every 2 years thereafter	Annually or as recommended

Children considered being at risk for the development of eye and vision problems may need additional testing or more frequent re-evaluation. Factors placing an infant, toddler, or child at significant risk for visual impairment include:

- 1 prematurity, low birth weight, oxygen at birth, grade III or IV intraventricular haemorrhage,
- 2 family history of retinoblastoma, congenital cataracts, or metabolic or genetic disease,
- 3 infection of mother during pregnancy (e.g. rubella, toxoplasmosis, venereal disease, herpes, cytomegalovirus, or AIDS),
- 4 difficult or assisted labour, which may be associated with foetal distress or low Apgar scores,
- 5 high refractive error,
- 6 strabismus,
- 7 anisometropia,
- 8 known or suspected central nervous system dysfunction evidenced by developmental delay, cerebral palsy, dysmorphic features, seizures, or hydrocephalus.

The aim of this article is to provide few general guidelines and to be followed while examining a child with an eye problem.

History

- 1 Document the relationship between the informant and the patient.
- 2 If both the parents are there it is better to get the complaints and other ocular/medical history from the mother.
- 3 Apart from the usual questions, history should include Birth History comprising of the following details:
 - a gestation period,
 - b delivery type (normal/caesarean/forceps delivery),
 - c intrauterine/neonatal complications,
 - d incubation/hypoxia/birth asphyxia history,
 - e TORCH,
 - f H/O consanguineous marriage,
 - g siblings,
 - h vaccination.

Infants (Newborn to 1 year)

Birth to 4 months

At birth eyes and visual system are not fully developed. But significant improvement occurs during the first few months of life. By 8 weeks, babies begin to focus their eyes on the faces of a parent or other person near them. For the first 2 months of life, an infant's eyes are not well coordinated and may appear to wander or to be crossed. This is usually normal. However, if an eye appears to turn in or out constantly, an evaluation is warranted. Babies should begin to follow moving objects with their eyes and reach for things at around three months of age.

Five to eight months

During these months, control of eye movements and eye-body coordination skills continue to improve. Depth perception and colour vision develops by 5 months of age.

Nine to twelve months

By the age of 9–12 months, babies should be using their eyes and hands together. They are able to grasp objects with thumb and forefinger and can now judge distances fairly well and throw things with precision.

It is possible to assess:

- 1 Vision by fixing and following method.
- 2 EOM while following torch light or Doll's Eye Manoeuvre.
- 3 Squint assessment.
- 4 Media clarity and refraction.

One to 5 years (where child can speak but can neither identify letters nor pictures)

Children this age are highly interested in exploring their environment and in looking and listening. They recognize familiar objects and pictures in books and can scribble with crayon or pencil.

Additional tests include:

- 1 Sensory evaluation (just a try) and Vision assessment.
- 2 Media assessment – can do slit lamp from even 4 years onwards.

Greater than 5 years

Most children at this age can co-operate as well as any adult for eye examination including slit lamp and indirect ophthalmoscopy.

All of the above-mentioned tests and a Detailed squint evaluation can be carried out.

Exceptions in this include mentally retarded children – for whom vision assessment could be difficult in even older patients.

Tips and tricks

- 1 Your patient is usually a child; always remember to carry a toy or a sweet.
- 2 Never lose a chance to observe the child in the waiting area, and make note of the squinting eye, facial asymmetry, or abnormal head posture if any.
- 3 In office, *make the child comfortable* in parents lap.
- 4 Before examining the child, build a rapport with the child by asking him/her few favourite questions like, name of school? Favourite colour? Favourite cartoon?, etc.
- 5 Always perform *sensory tests*—WFDT and STEREOPSIS before checking vision or performing a Cover Test (i.e. before breaking FUSION).
- 6 We must evaluate the *extra ocular muscle motility* before doing cover tests both versions and ductions.

For infants to children 5 years of age: Hirschberg's test and prism neutralization using

modified Krimsky's method (that is prism should be placed in front of the better eye).

For children older than or are of 6 years of age: Cover test and Prism base cover test should be done.

Modified Krimsky can be done if the patient has poor vision and cannot fixate well irrespective of age.

- 1 If the child resists occlusion of one eye, suggests poor vision in the unoccluded eye.
- 2 *Never use a torch light* as fixation target while measuring deviations as it is not an accommodative target.
- 3 Angle measurements with Hirschberg's may not coincide with PBCT in eyes with +ve or -ve angle Kappa, in which cases measurements taken by Hirschberg reflex method will be unreliable.
- 4 Measurements of the *Prism Bar Cover Test* should be taken in all nine cardinal gazes. For both Distance and Near.
- 5 Do not stack the same kind of prisms, try and split them between two eyes.
- 6 Always look for patterns.
- 7 For any vertical deviation, whether or not you are measuring the deviation *Parks three step test* should be done.
- 8 Always check for DVD.
- 9 In presence of *nystagmus*, first check vision binocularly, in the adopted head posture, in forced primary gaze and then unocularly by fogging the other eye.
- 10 In case the patient adopts an *abnormal head posture*, try and turn the head away from that posture and look what happens to the eyes, either increased deviation or increased nystagmus.
- 11 After a detailed evaluation of ocular motility and binocular function, perform *cycloplegic refraction* (an indispensable step of paediatric examination).
- 12 Examination of a case of strabismus is incomplete without a dilated *fundus examination* to look for torsions and ruling out sensory causes.

It is important to identify children with eye problems at this young age. Vision development and eye health problems are easier to correct if treatment begins early.

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Guidance for children with special needs

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Introduction

Special children are defined as children with identified or non-identified disability, physical health, or mental health conditions requiring early intervention, special education services, or other specialized services and supports. The special need can arise due to either of the following:

- 1 visual impairment,
- 2 hearing impairment,
- 3 mental retardation,
- 4 locomotor impairment,
- 5 learning disability,
- 6 cerebral palsy,
- 7 multiple disabilities,
- 8 others.

According to Indian legal aspects, a person with disability is defined as a person suffering from not less than 40% of any disability as certified by a medical authority [Persons with Disabilities Act (1995)].

As per survey conducted by the National Sample Survey Organization in 2002, out of 1 billion people in the country, 2.01% (~20 million) suffers from one or the other disability. The population with multiple disabilities in India estimated to be of 10.63% (~2million) of the total population with disabilities¹. The Rapid Rural Appraisal states that in a population of 7454 people in two Panchayats in Tamil Nadu, 4.8% was noted to have disability of one type or the other. Among the disabled, 26.47% were found to be multiple disabled. A marginally higher percentage of female (1.27%) than male (1.24%) suffered from multiple disabilities².

Current educational status of children with special needs

A recent study by the World Bank (2007) noted that children with disability are five times more likely to be out of school than children belonging to scheduled castes or scheduled tribes. Moreover,

Table 1: The number of CWSN in India².

Year	Total numbers identified as CWSN
2002-03	683,554
2007-08	2,621,077

when children with disability do attend school they rarely progress beyond the primary level, leading ultimately to lower employment chances and long-term income poverty². Even though various efforts have been made in the recent past, both the rates of educational participation and outcomes of education, remain very poor for children and young adults with disabilities. Illiteracy rates for this group remain much higher than the general population and school attendance continues to lag behind than that of normal children.

Types of schooling

- 1 Mainstream: School with normal children without any disability.
- 2 Integrated school: A school where both normally sighted and visually impaired students will be studying in the same class.
 - a Resource model: The school is provided with the resource set up, where a teacher will be available all the day. During the free hours, the students with visual impairment go to the resource room for guidance.
 - b Itinerant model: The Resource teacher (Special Educator) goes to different schools and visually impaired children's place to teach them.
- 3 Inclusive school: Mild to moderate form of all types of differently abled children will be included along with the normal children.
- 4 Special school: A school for visually impaired/deaf-mutism/mentally retarded/learning disability/any specific disability.

Table 2: The nature and severity of disability (expressed in percentage) in school going children.

Nature of impairment	Grades		
	I-V STD	VI-VIII STD	I-VIII STD
Visual impairment	20.79	32.87	24.02
Hearing impairment	11.69	11.04	11.52
Speech impairment	13.04	8.28	11.77
Locomotor impairment	27.28	32.09	28.56
Mentally retardation	19.68	8.62	16.73
Others	7.51	7.10	7.40
Percentage to total enrolment	0.79	0.80	0.80

Table 3: 'Niramaya' card—Revised Benefit Chart (on reimbursement basis only).

Section	Subsection	Detail	Sublimit	Over all limit of section
I	Over all limit of hospitalization			100,000/-
	A	Hospitalization	100,000/-	
	B	Corrective surgeries for existing disability including congenital disability	50,000/-	
	C	Non-surgical	15,000/-	
	D	Surgery to prevent further aggravation of disability	15,000/-	
II	Overall limit for out patient department			10,000/-
	A	OPD treatment including the pathology, diagnostic tests	5000/-	
	B	Regular medical checkup for non-ailing disabled	5000/-	
	C	Ongoing therapies to reduce impact of disability, disability and disability related complications	7500/-	
	D	Dental preventive dentistry	7500/-	
III	Alternative medicine (to be considered with in limit of IPD or OPD)			2000/-
IV	Transportation costs (to be considered within limit of IPD or OPD)		150 per visit (for three visits)	450/-

Overall limit of the coverage for a person: Rs. 100,000/- per year.

The National Trust (Central Government)

The National Trust is a statutory body under Ministry of Social Justice and Empowerment, Government of India set up under the National Trust for the welfare of persons with Autism Cerebral Palsy, Mentally Challenged and Multiple Disabilities Act (Act 44 of 1999)³. The National Trust carries out various schemes of capacity building, training and care and shelter through its registered organizations. Some of the major schemes are given below.

Gharaunda

Group Home and Rehabilitation Activities Under National Trust Act for Disabled Adults is a new scheme for providing Life Long Shelter and Care to Persons with Disabilities in Group Homes.

Sahyogi

A new and revamped training module has been designed and a system of training and deployment of Caregivers has been provided for under this scheme.

Samarth

Its a Centre-Based Scheme which was introduced in July 2005 for residential services—both short term (respite care) and long term (prolonged care). Activities in a Samarth Centre should include early intervention, special education or integrated school, open school, pre-vocational and vocation training, employment-oriented training, recreation sports, etc.

Aspiration

This is an early intervention programme for school readiness. The scheme is to work with children of 0–6 years with developmental disabilities, to make them ready for mainstream and special schools.

Niramaya

This is a Health Insurance Scheme to provide affordable Health Insurance of up to 1 Lakh per year to persons with Autism, Cerebral Palsy, Mental Retardation and Multiple Disabilities. The scheme is implemented in all the districts of the country (except J & K).

Niramaya scheme and its coverage

The scheme envisages delivering comprehensive cover which will

- 1 Have a single minimum premium across age band.
- 2 Provide same coverage irrespective of the type of disability covered under the National Trust Act.
- 3 Insurance cover up to Rs. 1.0 Lakh.
- 4 All persons with developmental disabilities will be eligible and included and there will be no 'selection'.

Remote area funding

The objective of this scheme is to stimulate National Trust activities in unrepresented districts. Under the scheme, fund is provided to set up an NGO, including parents association and then to

carry out activities for the welfare of persons with National Trust disabilities.

Uddyam Prabha

It is an Interest Subsidy Scheme for self-employment. A person with multiple disabilities who takes a loan from any bank or NHFDC can get interest subsidy of 5% for BPL or 3% for APL on loan amount upto Rs. 1 Lakh.

Gyan Prabha

Scholarship Scheme for doing, post schooling, any employment oriented course. Under the Scheme, a monthly scholarship of Rs. 1000 shall be paid for upto 1 year.

Arunim

Association for Rehabilitation Under National Trust Initiative of Marketing has been launched to help person with multiple disabilities in product designing, production processes, packaging and marketing enabling them to live a life with dignity and independence.

Concessions and benefits for children with special needs (State Government)

The Government of India as well as the State governments offers wide variety of concessions and benefits to persons with multiple disabilities⁴. For each category of concessions apart from eligibility requirements, magnitude of the assistance, definition of the handicap, clear guidelines regarding application form, procedure of availing the benefit, etc. are clearly enunciated and elaborated by Government orders issued from time to time.

Railways

- 1 75% concession in the basic fare in the first and second class is allowed to persons with multiple disabilities accompanied by an escort.
- 2 50% concession in the first and second class monthly/quarterly season fares both for the individual with disability and his/her escort over suburban and non-suburban section of Indian railways is allowed.

Roadways

- 1 Most State Governments having state owned and operated transport undertakings or corporations allow subsidized/free bus travel in the city and rural routes, and an escort is charged 50% of the fare.

Preferential allotment of public telephone booths

- 1 Persons with multiple disabilities are being given preference in allotment of telephone

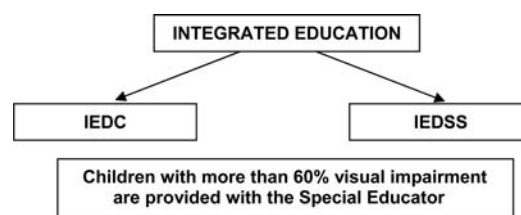
booths as means of sustenance, vocational rehabilitation, and income generation.

Scholarship

- 1 The Union Ministry of Welfare since 1955 has been operating through the state Governments and Union territories a scheme of scholarships awarded to challenged person for pursuing education in special schools being run by non-government organizations.
- 2 The Rate of scholarships is Rs. 1000/- per annum for cases hailing from the lower socio-economic status and is renewable from year to year.
- 3 In case of severely challenged persons who require special arrangements for transportation, an additional monthly allowance of Rs. 50/- is sanctioned.

Integrated education for children with special needs

This centrally sponsored scheme was launched by the Department of Social Welfare in 1974 and has been transferred to the Department of Education since 1982. The handicapped children have the benefit of receiving education in the regular school system.



IEDC

- 1 Integrated Education for the Disabled Children (STD I to STD VIII).
- 2 Undertaken by Sarva Siksha Abyan (SSA) since 2008.

IEDSS

- 1 Integrated Education for Disabled at Secondary Stage (STD IX to STD XII).
- 2 Resource training teacher from National Association for the Blind goes to schools with visually impaired children.
- 3 One teacher for eight visually impaired children.

Sarva Siksha Abyan

From the year 2006, according to SSA–Central Government organization

All Government schools (regular) follow the Active learning methodology (ALM) and Activity-Based Learning (ABL) system of education

STD I to STD V ABL method,
STD V to STD VIII ALM method.

Primary motive: Education for all.

Method explanation

- 1 No dictation/black board works (except for mathematics)/regular teaching methodology.
- 2 Teaching with the help of Flash cards.
- 3 Worksheet will be given to the students.
- 4 Hand works/assignments/chart works are part of the evaluation.
- 5 Student's performance will be calculated using 'Mile map'.
- 6 Each student will be given a file to follow up their academic status.
- 7 Every student will be passed on to the next academic year irrespective of their performance in the examinations (up to V STD).
- 8 Advantage: Children who are slow learners and students with poor academic performance will be involved actively in the studying process.

The following allowances and facilities are provided under this scheme

- 1 Books and Stationary allowance of Rs. 400/- per annum.
- 2 Uniform allowance of Rs. 50/- per annum.
- 3 Transport allowance of Rs. 50/- per month (if a challenged child admitted under the scheme resides in a hostel of the school within the school premises, no transportation charges would be admissible).
- 4 Reader allowance of Rs. 50/- per month in case of blind children after class V.
- 5 Escort allowance for severely handicapped children with lower extremity disabilities at the rate of Rs. 75/- per month.
- 6 Annual cost of equipment subject to a maximum of Rs. 2000/- per student for a period of 5 years.

Children educational allowance

- 1 The tuition payable and actually paid by the Government servant is reimbursable subject to

Rs. 50/- per month per child in the case of multiple disabled children.

Assistance to challenged persons for purchase/fitting of aids and appliances

- 1 This scheme is available to all employed, self-employed and pensioner whose average monthly income from all sources does not exceed Rs. 2500/-. The quantum of assistance ranges from Rs. 25/- to Rs. 3600/-. The full cost of the aid is reimbursed if the income of the challenged persons is up to Rs. 1200/- per month while 50% of the cost of the aid is reimbursed if the income is between Rs. 1200/- and Rs. 2500/-.

Preferential allotment of house sites

- 1 Most housing boards and urban development authorities have schemes of preferential allotment of plots and housing sites to individuals with disability.

Regarding employment

- 1 Identification of posts, which can be reserved for persons with disabilities.
- 2 To reserve posts through special employment exchange.
- 3 Gives power to inspect records or documents in possession of any establishment.
- 4 Employers to maintain records of challenged employees.
- 5 To encourage schemes which ensure employment of challenged such as training requirements, upper age limit, etc.
- 6 All educational institutions to reserve the seats for disabled.
- 7 All poverty alleviation schemes will also reserve seats for disabled.
- 8 Will give incentives to employers who have 5% of the work force as disabled persons.

Conclusion

The children with special needs (CWSN) are rarely identified and even if identified are not under care of appropriate organizations from where they can get the concessions and benefits available for them from the Central and State Governments. The need for awareness on guidance and referral of CWSN keeps on increasing in this Millennium due to exponential increase in their number.

Appropriate guidance and referral can change the life of the CWSN.

Important links

- 1 For Central Government concessions, refer to National Trust:
http://www.thenationaltrust.co.in/nt/index.php?option=com_content&task=view&id=217&Itemid=280

OR

The Spastics Society of Tamil Nadu (SPASTN)—Main Branch

Opposite. T.T.T.I. Taramani Road,
Chennai 600 113, India.
Phone: 91-44-22541651, 22541542
Fax: 91-44-22541047
Email: spastn@md2.vsnl.net.in
Contact person: Mr. Arun for National Trust scheme details.

- 1 For State Government concessions, refer to

State Resource cum Training Center (SRTC)

Girm (Annexe),
Jawaharlal Nehru road,
K.K. Nagar,
Chennai 600078, India.
Contact person: Mrs. Sheeba,
Phone: 044-24745233/044-24744737.

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4. Government and Law; Government concessions and Benefits: http://www.fourstepsindia.org/govt/govt_concessions.html.

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Candida infections in ophthalmology

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Fungal infections of the eye are the leading causes of ocular morbidity and blindness in the tropical and subtropical zones and are in the upward trend due to the increasing number of immunocompromised patients with modern therapeutic management facilities. The common ocular fungal infections are keratitis/corneal ulcer and endophthalmitis though infections in the orbit, lids, lacrimal apparatus, sclera, conjunctiva may occur rarely. The causative fungal agents in the eye are mainly the filamentous fungi, though yeasts, particularly *Candida* species may also be responsible in a small percentage of cases¹. The infections caused by yeasts are associated with the risk factors such as chronic ocular surface disease, contact lens wear, and use of topical steroids^{2,3}. Endophthalmitis due to *Candida* species is well documented, both as a consequence of fungemia and as a result of dissemination from endogenous source in an immunocompromised individual. In addition, infection due to *C. albicans* may result in choroidal revascularization, which is a potential cause of late visual loss in patients who have had sepsis and endogenous chorioretinitis due this organism.

Clinical features

The onset is usually insidious, often following corneal injury. The ulcer often runs on indolent course. Although the overall incidence of fungal infection following penetrating keratoplasty is low (0.16%) infection with *Candida albicans* and other *Candida* species is higher due to contaminated donor corneoscleral rim⁴. The epithelium may show defect at the site of infiltration or epithelial defect would have healed with deep stromal infiltrates. Intra-ocular signs and symptoms such as diminished visual acuity, severe vitreous inflammation with persistent iritis, whitish puff balls and strands are more commonly seen in endophthalmitis due to *Candida* and *Aspergillus* species^{5,6}.

Laboratory diagnosis

Once there is clinical suspicion of a fungal infection, every effort should be made to recover the causative fungus so that appropriate antifungal therapy may be instituted timely. The various clinical samples, for laboratory diagnosis, include (a) corneal scraping, (b) corneal biopsy, and (c) anterior chamber aspirate or vitreous aspirate.

Processing of ocular specimens for demonstration and isolation of fungi

As a routine, the scraped out corneal tissue or the biopsied material after homogenization is divided into three portions, one for Gram staining, one for 10% KOH or KOH calcoflour mount wet mount and the third for culture. The reported rate of sensitivity of simple KOH wet mount for the presumptive diagnosis of fungal keratitis varies between 33 and 92%. Gram stain, though has been reported to yield an accuracy of 60–75% in detecting the causative organism, is undoubtedly a simple and rapid method.

Culture and identification: The yeasts, particularly *Candida* species appear as oval cells measuring about three to four microns in size and their mode of reproduction is by budding. The yeasts appear Gram positive in Gram stained smear (Figure 1) and also can be seen as distinct oval structures in Geimsa stained smear and appear brownish black in colour in GMS stained smears. In culture the yeast colonies appear cream in colour (Figure 2), pasty in consistency and can grow in all types of culture media, though the growth is rapid on Sabouraud's Dextrose Agar.

Yeasts can be speciated by looking for chlamydospore formation on cornmeal agar and germ tube production (Figure 3) as well as various sugar fermentation and assimilation tests, urease test and other biochemical tests.

Limitations of fungal culture: The limitations to culture in establishing an etiological diagnosis of fungal endophthalmitis are due to the small size of the sample as reported culture positivity of

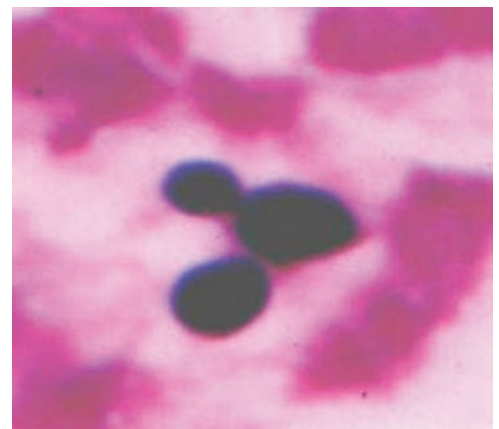


Figure 1: Gram stain smear of Vitreous Aspirate in which intra-cellular budding yeast cells are seen.

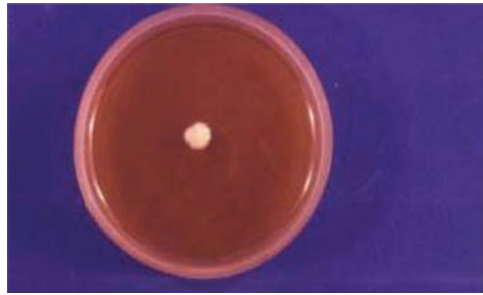


Figure 2: *Candida albicans* grown right on the inoculum of the same Vitreo aspirate.



Figure 3: *Candida albicans* showing germ tube test positive.

only 64% success rate of isolation in intra-ocular specimens from suspected fungal endophthalmitis, prior use of antifungal agents may also yield result in negative culture results and fungi take a longer time to grow to generate a report on time to decide on therapy.

Molecular methods for the diagnosis of ocular candida infections: Polymerase chain reaction (PCR) is universally accepted as most popular technique as it can yield quick results, confirming the diagnosis of mycotic infection within a few hours, whereas culture takes at least 5–6 days for a positive detection PCR, PCR-single stranded conformational polymorphism and PCR-restriction fragment length polymorphism techniques have also been standardized for fungal identification [35]. In a recent study, a PCR-based assay, developed to amplify a part of the fungal 18S r-RNA gene, was used for detection of fungal DNA in corneal scrapings. PCR and fungal culture results matched in 74% of cases. Thus, PCR assay, presently, seems quite promising for the diagnosis of ocular fungal infections offering definite

advantage over culture methods. However, its main drawback is its occasional false positivity that can be overcome by application of stringency in laboratory procedures and proper standardization of the techniques. Anand *et al.* [8] recently studied 27 intra-ocular specimens from 22 cases of suspected fungal endophthalmitis, by conventional microscopy and culture as well as by PCR assay, for the detection of fungi. Average time required for culture was 10 days, whereas PCR needed only 24 h.

Treatment for ocular candida infections: Ocular candidiasis can be treated with systemic antifungal therapy, or with intra-vitreous injection of an antifungal agent, sometimes combined with vitrectomy [9]. Systemic Amphotericin B and echinocandins do not penetrate well in the vitreous humour, whereas fluconazole and voriconazole reach vitreous concentrations between 25 and 100% of their serum concentrations. However, few prospective studies have addressed the outcome of antifungal therapy on the incidence and course of ocular Candida infection. First choice: Amphotericin B 0.15% eye drops; Fluconazole (0.5% drops, 200 mg orally) [1].

Alternatives

Flucytosine (1% drops, 150 mg/kg orally); Miconazole 1% drops, subconjunctival/injection; Ketoconazole 1% drops; Ketoconazole 200 mg BD orally. Species of *Candida* other than *C. albicans* are reportedly showing *in vitro* resistance to fluconazole. In addition, *C. tropicalis* is intrinsically resistant to many azole compounds.

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The Sankara Nethralaya Academy (TSNA) is education arm of Sankara Nethralaya(a unit of Medical Research foundation)

TSNA is avowed to employ a system of educating people in a community or society, arts, and science colleges, imparting knowledge through instruction courses, training classes, and usher in newer training/teaching methodologies including the use of IT. The objective is to teach courses that deal with A to Z in healthcare services.

TSNA offers career oriented programs in the field of allied healthcare and Hospital administration. Our allied health science courses are run in collaboration with The Tamil Nadu Dr MGR medical University. The programs have been designed to meet the growing demand for the well trained health care professionals.

Key Features of our Academy are

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| <ul style="list-style-type: none"> • Modern air conditioned classroom built for aesthetic comfort • Library facility • Interactive classroom session with eminent faculties • Hostel facility available on request • Audio Video classroom facility • Hi speed internet and Computer lab with latest systems. | <ul style="list-style-type: none"> • No Capitation / Donation Fees • Transportation facilities, • Nominal Fee structure • Flexible fee payment option (Instalments) • Faculty and student collaborative projects • Education loan through City Union Bank (for applicable courses) • Tie up with Multi Speciality Hospital |
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The following courses are offered by our SN Academy are:

S No	Degree and Diploma Courses	Course details like :
1	Diploma in Operation Theatre & Anaesthesia Technology.	Affiliation / Association / Eligibility Criteria / Fees Structures to be referred our website : www.thesnacademy.ac.in or Write to: A Mahalingam Academic Officer / Asst Registrar The Sankara Nethralaya Academy No 9, Vanagaram – Ambattur Road Ayanambakkam, Chennai – 600 095 Ph No : 044 4908 6000 Fax No : 044 2825 4180 E – Mail : mahali@snmail.org
2	Diploma in Ophthalmic Nursing Asst(ONA)	
3	Diploma in Refraction and Dispensing	
4	BSc Medical Laboratory Technology	
5	MS Medical Laboratory Technology	
6	MBA in Hospital and Health System Management.	
Short tem / Certificate Courses		
1	Certificate Course in Hospital Management(weekend)	
2	Certificate Course in Hospital Infection Control(Distance Learning)	
3	Certificate Course in Optical Sales	
4	Certificate course in Ophthalmic Dispensing	
5	Certificate Course in Optical Retail Management	
6	Certificate Course in Basic Life Support (American Heart Assn)	
7	PG Program in Hospital management(Distance Learning)	
8	PG Program in Optical Retail Management(Distance Learning)	
Optometry - Short tem / Certificate Courses		
1	Certificate Course in Retinal Diagnosis – 2 weeks	
2	Certificate Course in Glaucoma Diagnosis – 2 weeks	
3	Certificate Course in Corneal Diagnosis – 2 weeks	
4	Certificate Course in Low Vision Aids – 3 months	
5	Certificate Course in Binocular Vision & Vision Therapy	
6	Certificate Course in Clinical Optometry – 3 months	
7	Fellowship in Low Vision Aids – 6 months	
8	Fellowship in Contact Lens – 6 months	
9	Fellowship in Binocular Vision & Vision Therapy – 6 months	
10	Fellowship in British Dispensing Opticians (FBDO) – 8 months	

FELLOWSHIP EXAMINATION IN OPHTHALMOLOGY

Applications are now being accepted for Part B and Part C of this examination which will be conducted from Monday 23 to Friday 27 June 2014 in Chennai.

Applications, together with the examination fee and appropriate documentation, must be received in the College no later than Friday 28 March 2014.

The fees for the examination are as follows:

Part B – Full Examination: £ 1,050

Part B – Resit Examination: 5 or more sections: £ 1,050
 Up to 4 section: £ 685

Candidates who are successful in Part B are eligible to apply for election as a Member of the College (MRCSEd (Ophth)).

Part C – Full Examination: £ 1,450

Part C – Resit Examination: 5 or more sections: £ 1,450
 Up to 6 sections: £ 945

Candidates who are successful in Part C are eligible to apply for election as a Fellow of the College (FRCSEd (Ophth)).

IMPORTANT: Candidates may not apply for both Part B and Part C at any diet of this examination.

The numbers for this examination are limited. Please apply early to avoid disappointment. Candidates who apply after the number of available places are filled will be included on a waiting list.

Further information is available at: www.rcsed.ac.uk/examinations/ophthalmology.aspx

Or by emailing: ophthalmology.exams@rcsed.ac.uk



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