A CLINICAL STUDY OF UVEITIS IN CHILDREN AND ADOLESCENTS

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Certificate

This is to certify that the dissertation entitled "**A CLINICAL STUDY OF UVEITIS IN CHILDREN AND ADOLESCENTS"** is the bonafide original work of **Dr. MAHARAJA DAVID GIDEON** in partial fulfillment of the requirements for **M.S. Branch – III (Ophthalmology)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in September 2006.

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Declaration

I, **Dr. MAHARAJA DAVID GIDEON,** solemnly declare that dissertation titled, "**A CLINICAL STUDY OF UVEITIS IN CHILDREN AND ADOLESCENTS"** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2003-2006 under the expert guidance and supervision of **Prof. A. PRIYA, M.S., D.O.** Head of the Department, Department of Ophthalmology.

The dissertation is submitted to The Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.S. Degree (Branch – III) in Ophthalmology.**

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KEY TO MASTER CHART

Anb	:	Antibiotics (Systemic)
AS	:	Ankylosing Spondylitis
AS	:	Anterior Synechiae
ATT	:	Antituberculous Treatment
AU	:	Anterior uveitis
AV	:	Anterior vitreous
BE	:	Both Eyes
BK	:	Band keratopathy
C	:	Cells
CC	:	Complicated cataract
CF	:	Counting fingers
Ch	:	Choroiditis Patch
Ch-C	:	Chest Clinic
CxR	:	Chest X-ray
d	:	Days
DE	:	Divergent Eye
De-C	:	Dental Consultation
DV	:	Defective vision
ECCE	:	Extra Capsular Cataract Extraction
ELISA	:	Enzyme Linked Immuno Sorbant Assay
End	:	Endophthalmitis
Ex	:	Exudates
F	:	Flare
Fl	:	Floaters
FL	:	Follows light
HM	:	Hand Movements
IU	:	Intermediate uveitis
IV Ab	:	Intravitreal antibiotics
Jra	:	Juvenile Rheumatoid Arthritis
KP	:	Keratic Precipitates
LE	:	Left Eye
m	:	Months

Myd	:	Mydriatic and cycloplegics
Nys	:	Nystagmus
P	:	Pain
PA	:	Psoriatic Arthritis
PaU	:	Pan Uveitis
PeS	:	Periocular Steroids
PL	:	Perception of light
PoU	:	Posterior Uveitis
PPL	:	Pars Plana Lensectomy
PPV	:	Pars Plana Vitrectomy
PR	:	Projection of light
PRP	:	Pan Retinal Photocoagulation
PS	:	Posterior Synechiae
R	:	Redness
RE	:	Right Eye
Rh-C	:	Rheumatology Clinic
SB	:	Snow banking
SICS	:	Small incision cataract surgery
Sk-C	:	Skin Consultation
SS	:	Systemic Steroids
T.S.	:	Topical Steroids
Tox	:	Toxocara
Toxo	:	Toxoplasmosis
Tr	:	Traumatic
Tub	:	Tuberculosis
VC	:	Vitreous cells
VF	:	Vitreous flare
W	:	Watering
wk	:	Weeks

ABBREVIATIONS

AC	:	Anterior Chamber
AFB	:	Acid Fast Bacilli
ANCA	:	Antinuclear Cytoplasmic Antibody
D	:	Dioptre
DC	:	Differential Count
ELISA	:	Enzyme Linked Immuno Sorbant Assay
ENT	:	Ear, Nose and Throat
Hb	:	Haemoglobin
HLA	:	Human Leucocyte Antigen
HM	:	Hand Movements
Ig	:	Immunoglobulin
IUSG	:	International Uveitis Study Group
P	:	Partial
PCIOL	:	Posterior Chamber Intra Ocular Lens
PL	:	Perception of Light
PPV	:	Pars Plana Vitrectomy
PR	:	Projection of Light
RA	:	Rheumatoid Arthritis
RD	:	Retinal Detachment
SLE	:	Systemic Lupus Erythematosis
TB	:	Tuberculosis
TC	:	Total Count
TORCH	:	Toxoplasma, Rubella, Cytomegalo Virus, Herpes
VDRL	:	Veneral Disease Research Laboratory

CONTENTS

INTRODUCTION

Uveitis refers to the inflammation of the middle vascular tunic of the eye of called uvea.

The terminology uvea is derived from the latin word uva or grape and consists of the iris, ciliary body and the choroid.

The frequency of uveitis in children under the age of 18, is relatively low, the approximate incidence of 8-10% of the over all uveitis cases from different world literature.

Unlike adults, the identification, history and symptoms, of uveitis in a child is a diagnostic challenge, the complications are severe leading to vision threatening problems, and the peculiar problem of development of amblyopia in children.

SOME HISTORICAL FEATURES

1500 BC Hippocrates mentioned the typical finding of uveitis.

1801 AD – name Iritis introduced by Johann Adam Schmidt.

1853 – Vontolt classified uveitis aetiologically as scrofula 30%, rheumatism 21.5% syphilis 25% and idiopathic the rest.

1931 – Kolmer (USA) put forward the view, that inflammation of uveal tissue was due to bacterial toxins. The place of allergy as a potent aetiological factor thus was established.

1950 – Toxoplasmic uveitis became a proven infection, thus parasitic infection played a considerable role in etiology of posterior uveitis.

REVIEW OF LITERATURE

ANATOMY OF UVEAL TRACT

The middle coat of the eye ball is between the sclera on the outside and retina in the inside and is called the uveal tract.

It is divided into the choroid, ciliary body and the iris.

CHOROID

This extends from the ora serrata to the optic disc. Posteriorly it merges with border tissue of elsching which surrounds the optic disc. Anteriorly it continues as the ciliary body. The scleral surface of choroids is shabby and irregular and the retinal surface is smooth and shiny.

The choroid measures 0.22 mm in thickness, posteriorly and 0.1mm in thickness anteriorly. The choroid has a high blood flow with pressure in the choroidal veins exceeding 20mm of Hg.

The following are the different layers of the choroid.

a) Suprachoroid lamina of FUSCA contains a delicate mesh work of elastic fibres. It is lined by endothelium and contains potential lymph spaces.

b) Layers of Haller and Sattler : These are layers of blood vessels. Larger layer of blood vessels are called layer of Haller and the layer of Sattler consists of medium sized vessels.

Stroma of these layers consist of collagen and elastic fibres – Pigment granules are evenly distributed in the stroma. They are responsible for absorbing excess light to prevent it from scattering.

LAYER OF CHORIOCAPILLARIES

The capillaries in this layer are of wide bore. The chorio capillaries end in the ora serrata whereas other layers continue to the ciliary body.

MEMBRANE OF BRUCH

It is a glossy homogenous membrane about 1.5μ thickness and is also called lamina vitrea. It lies next to retina and lies closely adherent to it.

It helps in passage of nutrients to retina and also helps in removal of debris and metabolites.

CILIARY BODY

This is the part of the uvea which extends beyond the ora serrata to the base of iris. This is black in colour. It extends upto 6-7mm

nasally. Extends more (8mm) temporally. The posterior part appears smooth and is called pars plana. The anterior part is called pars plicata. It presents as 70 radiating ridges with valleys in between.

CILIARIS

This is the muscle of the ciliary body. It is triangular in cross section. It consists of bundle of muscle fibers running antero-posteriorly in the outer layer and circular in the inner layer. Anterioposterior fibres are called longitudinal fibres. They are attached to the scleral spur and posteriorly they extend upto the equator.

Circular fibres occupy anterior and posterior parts of the ciliary muscle. They are called Muller's muscle. Muscle is supplied by short ciliary nerve from the ciliary ganglion containing parasympathetic fibres. Longitudinal fibres draw ciliary ring forward thus reducing circumference. Circular fibres act as a sphincter and reduce circumference of ciliary ring. They both slacken suspensory ligament of lens and decrease the capsular tension in the lens.

CILIARY PROCESSES

Each process is a ridge about 2mm long and 0.5mm high. Responsible for secreting aqueous into the posterior chamber.

IRIS

This is the anterior most part of the choroid. This forms a circular disc in front of the lens with a central circular aperture called the pupil.

Peripherally this is attached to the anterior surface of ciliary body. The anterior surface is divided into inner pupillary zone and outer ciliary zone divided by the collarette. There are crypts adjoining the collarette the epithelium is defective here and hence fluid can easily leave the iris.

It consists of anterior endothelium, anterior limiting layer, stroma, posterior membrane, epithelium. Anterior surface of iris is smooth and velvety. Anterior surface presents with depression adjoining the collarette called crypts of Fuchs. Minor arterial circle is found in the region of collarette.

Posterior membrane consists of smooth muscle fibres called dilator pupillae supplied by sympathetic fibres.

Sphincter pupillae is present in the stroma along with the blood vessels.

PATHOGENESIS OF UVEITIS

The etiopathogenesis of uveitis are several, and many theories have been put forward.

INFECTIVE EXOGENOUS INFECTIONS

Can be due to introduction into the eye of organisms through a perforating wound or ulcer. This results in suppurative iridocyclitis and may progress to endophthalmitis and panophthalmitis.

Secondary Infections

This inflammation of the uveal tract is due to its spread from one of the other ocular tissues (Cornea, sclera, or retina).

ENDOGENOUS INFECTIONS

This is a common cause of uveitis in which an infection lodged any where else in the body can be transmitted to the uvea by the blood stream.

Common infections are tuberculosis, syphilis, brucellosis, urinary infection, osteomyelitis, toxoplasmosis, etc.

IMMUNE RELATED INFLAMMATION

They are very common. Primary source of infection, exists some where in the body and the uveal tissue gets sensitized to the

infection. If it occurs again an immune process is set on the uveal tissues, with deposition of immune complexes. Commonly they are due to sinusitis, dental infections, pharyngotonsillitis, etc.

Acute iridocyclitis may be comparable to the antibody formation in a lymph node wherein they are sensitized with thymus dependent memory cells which may be implanted in the uvea. These cells proliferate on contact with the antigen, should it reappear in the blood stream and differentiate to form cytotoxic lymphocytes. This may explain recurrence of uveitis in chronic inflammatory disease after the infective form is treated.

HLA (Human Leucocyte Antigen) Associated : They occur more frequently in persons with specific HLA antigens. Thus patients with ankylosing spondylitis, juvenile chronic arthritis belonging to HLA B27 group present with iridocyclitis.

SYSTEMIC DISEASES

Uveitis are also associated with systemic etiologies like Stills' disease, Wegener's granulomatosis, sarcoidosis, Reiter's disease. They all have an autoimmune component in their etiology.

TRAUMATIC

Blunt or penetrating ocular trauma can produce features of iridocyclitis, surgical procedures involving iris and rest of uvea can also produce iritis.

IDIOPATHIC

A large percentage of uveitis can present without any identifiable aetiological factor and are hence termed idiopathic.

SPECIFIC UVEITIS

Fuch’s uveitis syndrome, sympathetic ophthalmitis. VKH Syndrome are examples of specific uveitis.

CLASSIFICATION

Anatomical Classification (IUSG)

Anterior uveitis :	Iritis
	Anterior cyclitis
	Iridocyclitis
Intermediate Uveitis :	Posterior cyclitis
	Hyalitis
	Basal retinochoroiditis
Posterior Uveitis :	Choroiditis
	Retinochoroiditis
	Neuroretinitis
	Pan Uveitis

ETIOLOGICAL CLASSIFICATION

Idiopathic, infective, immune related, neoplastic, traumatic.

PATHOLOGY

Granulomatous

Non-Granulomatous

Chronicity	Acute
	Recurrent-acute
	Chronic
Pattern	Focal
	Multifocal
	Diffuse

DIAGNOSIS OF UVEITIS IN CHILDREN AND ADOLESCENTS

HISTORY

PRESENT HISTORY

In the case of uveitis, the chief complaint is usually unilateral or bilateral, photophobia, redness, floaters, blurred or decreased vision, either alone or in combination.

Although blurred or diminished vision is the most common and least localizing symptom, other symptoms can suggest a primary site of intraocular inflammation. Juvenile idiopathic arthritis is a notable exception in which pain, photophobia and redness and frequently absent.

These conditions can be very taxing for the ophthalmologist, since the patient is asymptomatic and the child may not be able to come forward with complaints of floaters or blurring.

Hence only periodic examination of these patients can help to identify the disease.

PAST HISTORY

Previous attacks of uveitis, trauma, previous treatment history, history of defective vision, amblyopia or strabismus since childhood that might indicate vision loss from childhood are important. A history of

herpes simplex or previous attacks of varicella zoster give an important clue to the diagnosis.

Previous attacks of sinusitis, tonsillitis, systemic infections, parasitic infections, attacks of arthritis and skin lesions are important in further management.

PERSONAL HISTORY

A thorough dietary history, drug use, exposure history should be elicited. History of contact with pets in lieu of toxoplasmosis and toxocariasis and also pica habit in children which might lead to parasitic infections are very pertinent.

FAMILY HISTORY

Genetics does appear to play a role in some forms of uveitis as evidenced by their increased association with HLA. HLA B27 with anterior uveitis often occur together with ankylosing spondylitis. Reiter's syndrome or inflammatory bowel disease. Other HLA associations are HLA B51 with Bechet's disease and HLA B53 and HLA DR4 with VKH syndrome.

CLINICAL FEATURES AND EXAMINATION OF A CASE OF UVEITIS

The examination should start with the general condition of the child, the behaviour, the nutritional status and the development.

The child should be examined for anemia, lymphadenopathy.

The skin of the child should be thoroughly examined. The presence of rashes, nodules or vitiligo can be diagnostic in children.

Hair loss, poliosis, granulomas and nodules of the lid has to be noted.

VISUAL ACUITY

In pre verbal children,

If the child objects to occlusion of one eye it indicates poor visual acuity in the other eye. Fixation test with prisms, covering one eye and keeping a prism over the other eye is done. Now the cover is removed from the eye and ability to maintain fixation is noted.

Preferential looking, rotation test, optokinetic nystagmus are other methods of assessing visual acuity. Verbal children can undertake picture naming test such as Kay pictures. Older children can be assessed with a Sheridan-Gardner test and a E Snellen chart.

OCULAR EXAMINATION

After examining the lids and eye brows for hair loss, nodules, granulomas and vitiligo, the conjunctiva is examined for congestion,

dilated vessels, ulceration, nodules, phylcten. The congestion is divided into ciliary and conjunctival congestion.

Ciliary congestion is pathognomonic of corneal lesion or inflammation of the iris and ciliary body and also acute rise in intra ocular pressure.

Cornea is examined for edema, band keratopathy, degeneration and ulcers. Keratic precipitates are accumulations of inflammatory cells over the desquamated sticky endothelium. Identification is important as they some times may be the only signs of inflammation. They are mostly found in the form of a triangle between the 4 – 8 o' clock position. This triangle is formed with the base towards the limbus. It is called the Arlt's triangle.

Keratic precipitates are classified as

Pigmented and crenated	-	old and inactive
White or yellow and round	-	Recent or currently active non granulomatous inflammations.
Larger and frequently greasy appearance.	-	Granulomatous
Red KPs	-	following hemorrhage into the anterior chamber.

ANTERIOR CHAMBER

It is examined for depth and the clarity. Presence of aqueous flare in graded by its intensity. Flare is due to disruption of the blood aqueous barrier causing leakage of the proteins from the iris and ciliary vessels. The aqueous cells are due to the extravasations of the inflammatory cells from the vessels. They can be graded as follows (by HOGAN'S Method)[1].

AC cells –

0	-	0
1+	-	5-10 Cells
2+	-	11-20 cells
3+	-	21-50 cells
4+	-	> 50 cells

AC Flare

0	-	Normal
1+	-	Faint (Just detectable)
2+	-	Moderate (Iris details clear)
3+	-	Marked (iris details hazy)
4+	-	Severe (Fibrin, coagulated aqueous)

In addition to the above finding there may be collection of macro phages and polymorphs which may be so severe that they accumulate at

the base of the anterior chamber. This is called hypopyon. Usually this hypopyon is sterile and hence once the inflammation is brought under control they get spontaneously absorbed. Some times in viral iritis there may be accumulation of blood in the anterior chamber. This is called hyphaema.

IRIS AND PUPIL

The iris colour and pattern may be lost due to inflammation and leakage of the blood vessels. The whole iris becomes boggy and edematous and thus they loose their architecture. The colour might be lost in atrophic patches and also in heterochromia iridis as in Fuch's iritis.

Nodular deposits in the iris may be due to granulomatous inflammation (ex. Bussaca nodules, Berlin's nodules) and due to both granulomatous and non-granulomatous uveitis (Koeppe's nodules).

There may be synechiae (anterior and posterior) secondary to the inflammation. Anterior synechiae is due to attachment between the iris and the cornea where as posterior synechiae is due to attachment between the iris and the lens. This posterior synechiae is very dangerous because this blocks the flow of aqueous humour from the posterior chamber to the anterior chamber. It results in anterior bowing of the iris

resulting in shallow anterior chamber and formation of peripheral anterior synechiae. It produces severe secondary angle closure glaucoma. The pupil is irregular in appearance in these cases producing a festooned pupil when they are being dilated.

LENS

It may contain pigment deposits in its anterior capsule due to deposition of iris pigments and also due to broken posterior synechiae. Previous attacks of raised intra ocular pressure produces a grayish white discolouration in the lens surface. This is called glaucomflecken. Cataractous changes especially anterior sub capsular cataract and posterior sub capsular cataract are common following chronic iritis and due to prolonged steroid therapy.

VITREOUS

Vitreous inflammation may be diffuse or may be anterior or posterior. The vitreous opacities are either floaters or cells. These vitreous opacities can be graded by Hogan's method.

The patient is examined using indirect ophthalmoscopy or slit lamp bio microscopy using a 78, 90 D lens or a H ruby lens.

0	–	Normal
1+	-	Few Scattered fine and coarse opacities. Clear fundus details seen.
2+	-	Scattered fine and coarse opacities fundus details can still be seen.
3+	-	Many opacities and marked blurring of fundus details.
4+	-	Dense opacities no view of the fundus.

Apart from this vitreous bands fibrous adhesions are also noted.

Fungal uveitis produces cotton ball like exudates floating in the vitreous.

Vitreous hemorrhage has to be identified if present.

RETINA AND CHOROID

Retinal periphlebitis, vascular sheathing, neovascularizations, occluded vessels are common things that has to be searched for in the retinal vasculature. Both the posterior and the peripheral vessels have to be scrutinized for new vessels.

Retinal lesions are manifested as yellow lesions with irregular margins obscuring the vessels. They have associated superficial hemorrhages.

Retinal hemorrhages, retinal necrosis and cystoid macular edema are other associated features. In addition to the above clinical findings

there may be retinal folds and epiretinal membrane formation due to chronic uveitis.

Fibrous bands originating from one part of the retinal surface to the other and also from the retina into the vitreous can also be elicited using an indirect ophthalmoscope. Choroidal inflammation are yellow or grayish white lesions lying deeper to the retina with normal over lying retinal vessels. Active inflammation is characterized by hazy margins and surrounding hemorrhages. Old lesions are punched out, well demarcated and are surrounded by pigments. The background sclera is usually visible as the retina and choroid are usually destroyed by the inflammation.

Choroidal inflammation can be focal, multifocal or diffuse. They are caused by a wide variety of parasitic, bacterial and fungal infections. The choroid is the vascular coat of the eye ball and hence blood from the rest of the body pass through the choroid. Hence they are easily liable to get infected by organisms from various parts of the body. Thus choroidal inflammation is usually infective in origin whereas an anterior uveitis is usually immunologically mediated and not usually due to a direct infection.

OPTIC NERVE

It can be secondarily inflamed after severe retinits. Most of the chorioretinitis are associated with subsequent papillitis. This results in severe loss of vision and associated scotomas. Colour vision is also severely affected as is contrast sensitivity. Papillitis has to be identified early and treated vigorously with steroids. Failure to do so will lead to secondary optic atrophy. This is the ultimate course of events in severe fulminent chorioretinits due to cytomegolovirus, sarcoidosis, herpetic retinal necrosis etc.

GONIOSCOPY

This is done to look for peripheral anterior synechiae, Berlin's nodules, new vessels in the angle and also membranes. Both the eyes are compared with each other and the angle width is documented. Associated pigmentation of the trabeculum is also documented in four grades.

INTRA OCULAR PRESSURE

Intra ocular pressure is measured using Shiotz, indentation tonometry. It can also be measured using an applanation tonometer. Usually the intra ocular pressure is elevated in episodes of acute iritis. This is because of inflammatory cells occluding the anterior chamber

angle. It may also be due to formation of posterior synechiae and also peripheral anterior synechiae. This has to be identified and vigorously treated with tension reducing measures like beta blockers. The intra ocular pressure is also elevated in patients who are undergoing long term treatment with steroid drops and they have to be regularly screened for raised pressure.

These examination are usually a great challenge in young children and pre verbal children and they have to be sedated or even anesthetized to perform these intricate examinations.

DIAGNOSTIC TESTS

Complete blood cell count with differential count

It is necessary to find out the presence of infection in the blood and also to find out the presence of acute or chronic inflammatory cells. Also it is needed to find out the response to treatment as the cell count comes back to normal if the treatment is successful. In patients with long term steroid therapy or immunosuppressive therapy regular monitoring of the blood cells has to be performed as they are likely to suffer from reduced cell count as a result of toxicity to the drugs given.

Erythrocyte sedimentation rate

It is a non specific indicator of plasma fibrinogen in the blood and also the globulin levels in the blood. This is elevated in chronic infections and inflammations especially granulomatous inflammations and systemic malignancies. Systemic malignancies can cause masquerade syndromes in the eye where by they mimic regular inflammatory conditions and can be confused with simple iritis. Using the Westergren method normal ESR for children should be less than 10 mm per hour.

SPECIFIC TESTS

Syphilis serology. As syphilis forms a major part of both anterior and posterior uveitis serological examination of this disease place a major part in management of uveitis. VDRL test forms the corner stone of serological examination of syphilis. The VDRL titre can be tested by taking the patient's, blood and agglutinating with a known antigen like cardiolipin. This test however gives false positive and false negative results. Antitreponemal antibody test such as FTA-ABS, MHATP has 100% sensitivity and specificity for syphilis regardless of the stage of the disease. Unfortunately these tests remain positive for life and hence do not signify on going disease activity. The RPR and VDRL titres in

contrast do reflect disease activity and therefore are used primarily to gauge disease activity and response to treatment.

RHEUMATOID FACTOR (RF) TEST

Rheumatoid factor is an auto antibody directed against the Fc fragment of the human IgG. About 80% of patients with rheumatoid arthritis are RF positive defined as a titre of more than 1:80. Rheumatoid factor sero positivity is non specific and best used to support a clinical diagnosis of rheumatoid arthritis.

ANTINUCLEAR ANTIBODIES (ANA) :

The ANA test is typically performed by applying serial dilutions of the patients serum to cultured tumour cells and then titrating for the presence and pattern of nuclear auto antibody staining. The ANA test is generally used to confirm the collagen vascular diseases particularly systemic lupus erythematosus or Juvenile rheumatoid arthritis. Along with ANA other tests like ANCA, Anti-double stranded DNA are more specific tests for the diseases like Poly arteritis, Wegener's Granulomatosis.

HERPES VIRUS ANTIBODIES :

The prevalence of herpes virus antibodies is so high in the general populations that a positive antibody titre is virtually

meaningless. A negative test, however eliminates herpes virus infections and therefore can be useful in selected instances. Herpes virus serology remains positive for life. Hence in cases where the clinical diagnosis is not very obvious herpes virus inclusion bodies can be seen by using Giemsa Stains for the affected cells. The inclusion bodies are usually seen in the nucleus of the affected cells.

HIV ANTIBODIES :

Most commonly detected by using an enzyme linked immuno sorbant assay (ELISA), positive results are confirmed by a Western blot test. HIV testing in uveitis is usually ordered in patients with known HIV risk factors, severe or bilateral retinitis or choroiditis and suspected Herpes Zoster Uveitis in children. HIV testing requires patient consent.

TOXOPLASMA ANTIBODIES :

Tests available to detect and quantify antitoxoplasma gondii antibodies are Sabin Feldman (SF) dye test, immuno fluorescence antibody (IFA) test and ELISA. Of these SF dye test remains the most sensitive and specific test but it is technically difficult and of limited availability, whereas IFA and ELISA are relatively easy and economical and can be used to distinguish IgG and IgM anti toxoplasma gondii antibodies. When interpreting positive titres, it is important to

remember that IgM anti toxoplasma antibodies may be elevated for upto 1 year after infection, limiting the accuracy with which they can date acute infection and that antibody titres are generally less reliable in patients with AIDS.

TOXOCARA ANTIBODIES :

Detected by haemoagglutination, complement fixation and immunofluorescent antibody test. But the ELISA has the sensitivity and specificity of 90%. Although a titre of more than 1:8 is considered diagnostic for ocular toxocariasis, it is important to remember that ocular toxocariasis is primarily a clinical diagnosis and negative titre does not rule out the disease.

ANGIOTENSIN CONVERTING ENZYME (ACE LEVEL) :

ACE is produced primarily by capillary endothelial cells, abundant in both lungs and liver and by macrophages. Clinically ACE levels are elevated in more than two thirds of patients with active disease of sarcoidosis.

Uveitis and a negative purified protein derivative with increased ACE is fairly specific for sarcoidosis. Normal serum ACE level is 12-55 mol/min/ml in men and 11-19mol/min/ml in women.

HUMAN LEUCOCYTE ANTIGEN (HLA) STUDY :

The surface membrane of human leucocyte contain HLA. These are regulated by gene loci on chromosome 6. The reaction of these antigens with specific antisera causes lysis of the cell membrane and this is the basis of cell typing. HLAB-27 has been associated with Ankylosing spondylitis, Reiter's syndrome, Psoriatic arthritis and arthritis associated with inflammatory bowel disease. HLAB-5 is associated with Bechet's disease.

DIAGNOSTIC PARACENTESIS :

Aqueous and vitreous samples are useful for polymerase chain reaction (PCR) testing for toxoplasmosis and herpes virus particularly in AIDS patients in whom diagnosis can be difficult.

To demonstrate local production of antibody the specific antibody in the eye is measured relative to the total amount of globulin in the eye. The formula for determining the value is

$$C = \text{Antibody titre } \frac{\text{Aqueous humor}}{\text{Serum}} \times \text{Ig } \frac{\text{Serum}}{\text{Aqueous humor}}$$

It is significant if the coefficient is more than 8^{2}.

CONJUNCTIVAL AND LACRIMAL GLAND BIOPSY :

Reserved for those patients with visible conjunctival masses or lacrimal gland enlargement as can occur with sarcoidosis, tuberculosis or coccidio domycosis.

MUCOSAL BIOPSY :

The greatest use of oral mucosal biopsy is for the diagnosis of Bechet's syndrome, evidence of an occlusive vasculitis can greatly support the diagnosis.

SKIN TEST :

Kveim's Test :

Suspension of antigenic preparation from human sarcoid tissue is injected intradermally and read at the end of 6 weeks when a papule develops. The papules is biopsied for evidence of granuloma and giant cells and epitheloid cells, with no caseation. This test is not easily available.

Mantoux Test :

Purified protein derivative of tuberculin is injected intradermally. It is a non-specific test. Because of prior exposure to tuberculosis a large number (8-30%) of healthy adults have positive PPD skin test

representing inactive infection. Therefore it is disadvantageous as it yields more false positive than true positive results.

Pathergy Test :

This is a scratch test done for Bechet's syndrome. In this scratch or a line drawn on the skin produces a wheal, the phenomenon is called dermographism.

X-RAY CHEST, X-RAY SACROILIAC JOINTS AND SKULL X-RAYS :

Chest X-ray taken in patients suspected of having sarcoidosis or tuberculosis.

Skull x-ray to evaluate patients with suspected congenital toxoplasmosis and rubella for calcification.

X-ray sarcoiliac joints and x-rays of the peripheral joints are taken in patients with suspected ankylosing spondylitis and juvenile rheumatoid arthritis.

B – SCAN :

Echoes are plotted as dots instead of spikes and the brightness of the dot indicates the sides of the received echo. The transducer is moved in an arc above the eye and whole series of intensity registration are plotted. B-scans may be taken in the horizontal, sagittal, oblique

planes. B-scan produces a real time two dimensional gray scale display of the eye and orbit. It is useful in delineating intraocular structures with opaque media such as severe cataract and vitreous haemorrhage and in evaluating vitreoretinal and orbital mass lesions. Dynamic ultrasound allows differentiation of detachments of the retina from those of the vitreous as well as identification of vascular abnormalities.

ULTRASOUND BIO-MICROSCOPY[3] :

This is another method of ultrasound examination of eye in which high frequency ultrasound waves of 50-80Mhz are used that allows histological evaluation of the anterior segment structures. It is used specifically for defining abnormalities of anterior chamber angle, limbus, ciliary body and anterior part of the retina. Snow banking and organized exudates in the peripheral retina and also peripheral retinal detachment can be clearly made out with ultrasound bio-microscope.

FLUORESCEIN ANGIOGRAPHY :

It is an invaluable aid in evaluating numerous pathological changes in the eyes.

Fluorescein used are usually in 10% or 25% solutions. Major uses of fluorescein angiography are :

a) Cystoid macular oedema

b) Subretinal neovascular membranes

c) Disc leakage

d) Staining of retinal vessels

e) Neovascularisation of retinal vessels

f) Retinal pigment epithelium (window defects)

Fluorescein angiogram can cause anaphylactic shock due to allergy to the dye. Hence all precautions to revive the patients in case of shock should be kept ready before the test is performed.

INDOCYANINE GREEN (ICG):

It is used to mainly identify choroidal neovascular membranes. It is well bound to plasma proteins and hence does not leak from the normal choroidal vessels. The retinal pigment epithelium does not block the choroidal fluorescence when ICG dye is illuminated with infrared light. ICG has a longer wave length when compared to fluorescein. Hence it is not blocked by the pigment layer. The disc is usually seen as a dark shadow in the ICG angiogram. ICG is also used to study patients with serpiginous choroiditis, bird shot choroiditis.

Laser Flare Photometry, optical coherence tomography are now used to assess uveitis and retinal edema and disc edema.

COMMON CAUSES OF SPECIFIC UVEITIS IN CHILDREN AND ADOLESCENTS

ANTERIOR UVEITIS

Juvenile Rheumatoid Arthritis (JRA)

JRA is the most frequently identifiable etiology of pediatric anterior uveitis. It is usually divided into systemic, polyarticular and pauciarticular. Pauciarticular arthritis is defined as the involvement of four or fewer joints in the first 6 months after the onset of the disease. Uveitis is more common in pauciarticular type of JRA. JRA and juvenile chronic arthritis together form the clinical spectrum of juvenile idiopathic arthritis. It produces chronic inflammation of the synovium. B-lymphocytes infiltration and expansion occurs in the joint space. This produces a thick pannus in the joints leading to destruction of the articular cartilage and leading to fusion of the adjoining bones. Pauciarticular and polyarticular JRA affects girls more than the boys. Systemic JRA is equal in both boys and girls. Patients with chronic uveitis develop joints symptoms early in life, some by about 6 years[4].

Laboratory diagnosis :

There is an elevated erythrocyte sedimentation rate and lymphopenia. ANA titre is positive in 25% of the children with JRA.

This is more common in children with pauci artricular JRA. Titres are usually in the range of 1:40 or 1:80. RA factor is usually rare and its presence indicates persistent disease into adulthood. There is also increased fibrinogen and D-dimer levels in the blood. Clinical diagnosed is based on presence of arthritis in a child with negative RA factor[5].

JRA uveitis is most frequently a chronic non-granulomatous iridocyclitis, bilateral in 70% of patients, common in female children. Ocular inflammation is usually asymptomatic and eyes are non-congested. Complications include cataract, glaucoma, band keratopathy, posterior synechiae and phthisis bulbi.

The arthritis usually precedes the uveitis. Iridocyclitis occur in approximately half of the affected patients within 2 years of the onset of the arthritis. There is no correlation between the degree of arthritis activity and ocular inflammation.

According to American Association of Paediatrics pauciarticular individuals should be checked every 3 months[6]. Polyarticular patients may be evaluated at 6 months intervals for the development of uveitis, while children with systemic onset JRA are examined annually.

Management is by use of topical steroids and also periocular steroids[7]. If the patients do not respond to the above methods systemic steroids has to be started. In severe cases where intraocular damage is rampant immunosuppressants and antimetabolites like methotrexate and etanercept has to be used.

Ankylosing Spondylitis :

The onset of juvenile ankylosing spondylitis is usually at 8-10 years of age and males are more affected, 95% associated with HLA B27. Frequently presents as a peripheral arthropathy before physical or radiographic findings of sacroiliac joint involvement.

An acute, recurrent non-granulomatous iridocyclitis affects 10-20% of patients with juvenile ankylosing spondylitis. Typically unilateral and may precede or follow the arthritis.

The patient might have severe iridocyclitis resulting in formation of a hypopyon.

Management is by topical or systemic steroids or in resistant cases by immunosuppressants.

Reiter's Syndrome :

Triad of Reiter's syndrome is non-infectious urethritis, arthritis and conjunctivitis. It is infrequent in children, males are more affected.

Although patients with Reiter's syndrome may have sacroilitis and HLA B 27 positivity, the presence of uveitis, keratoderma blenorrhagica of palms and soles and ulceration of the mouth and genitals differentiates this disorder from ankylosing spondylitis. Unlike Bechet's disease the mucosal lesions are usually not painful. A non-granulomatous anterior uveitis affects 3%-12% of patients.

Bechet's Disease :

In this condition the patient usually manifests with recurrent oral ulceration, genital ulceration and severe uveitis. Patients might have hypersensitivity and dermographism of the skin which is demonstrated by the Pathergy test. The uveitis might manifest as a simple anterior uveitis or may produce a severe vasculitis of the retina. This may lead to vascular occlusion of the retina resulting in retinal infarcts and finally leading to consecutive optic atrophy. In cases with vasculitis, management has to be with immunosuppressants and antimetabolites as only these can be vision saving.

Fuch's Heterochromic Iridocyclitis :

Frequently asymptomatic, with chronic low grade iridocyclitis which is almost always unilateral. Hyperchromia is a result of iris

atrophy is typical but not invariable. The keratic precipitates have a characteristic small, stellate appearance.

Treatment with topical corticosteroids is usually ineffective. Mydriatics are often not required because posterior synechiae are uncommon. This commonly causes anterior subcapsular cataract which may be the only symptom the patient might have as the eye is usually quiet. These patients might have new vessels in the iris and anterior chamber angle and when a paracentesis is done a typical type of haemorrhage (Filiform) called Amslers sign is present. This is pathognomonic of this disease.

Lens Induced Uveitis :

The phacolytic reaction usually occurs in the presence of a hypermature cataract. The lens material acts as a chemical irritant, probably acting directly on the iris and ciliary body. Macrophages enter to engulf the liberated material. No polymorphonuclear cells are seen. No keratic precipitates are usually seen in this condition. Management is usually by means of steroids to control the inflammation and removal of the cataractous lens with thorough washing of the anterior chamber.

In phacoanaphylatic uveitis, typically a break in the lens capsule occurs in one eye, the second eye develops a severe anterior

granulomatous uveitis after surgery or trauma. Poly Morpho Nuclear cells and macrophages are found in the aqueous, iris and lens. There may be also be white fluffy lens material seen in the anterior chamber and also over the surface of the lens and in the angle of anterior chamber. Patients have to be intensively treated with steroids and the offending lens material has to be removed.

Acute Interstitial Nephritis :

It is an uncommon renal disorder, as a result of an immune reaction to antibiotics, non-steroidal anti-inflammatory drugs or infection. It is characterized by non-specific systemic complaints of low-grade fever, pallor, fatigue and weight loss and may be associated with increased Erythrocyte sedimentation rate, increased serum creatinine level, proteinuria, glycosuria, microhematuria, leucocyturia, and excretion of casts. A bilateral anterior uveitis may precede, follow or occur concomitantly with this renal disorder. This diagnosis is established by renal biopsy. The prognosis in childhood is good.

Tuberculosis :

This may cause severe granulomatous and also non-granulomatous iridocyclitis usually a result of systemic tuberculosis. These patients manifest with tubercles in the iris in the solitary form or

the conglomerate form. These patients along with treatment by topical steroids also need to be treated with antituberculous drugs for control of the disease process.

Sarcoidosis :

This causes both non-granulomatous and granulomatous iridocyclitis. Lesion may be present all throughout the body. Biopsy of the sarcoid nodules may reveal non-caseating granulomas. These cases respond well to systemic and topical steroids.

Leprosy :

It causes severe anterior uveitis both in the multi bacillary and pauci bacillary forms. In multi bacillary form there is direct invation of the anterior chamber by the bacilli. It forms an iris pearl in the anterior surface of the iris. Treatment is both with steroids and anti-leprosy drugs.

Herpes :

Herpes iridocyclitis can be caused by both herpes zoster and herpes simplex viruses. Sometimes they follow severe corneal dendritic ulcerations. They also may accompany interstitial and disciform keratitis. The anterior chamber reaction may be due to immunologically mediated sensitivity to the viruses and also by direct invasion of the

viruses in to the anterior chamber. Haemorrhagic uveitis is a common manifestation of herpetic iritis.

INTERMEDIATE UVEITIS

Early in the course of the disease, the presence of anterior vitreous cells is the only sign. Initially there are fine and separate, but as the disorder progresses they aggregate and may acquire a fibrillar state in the anterior 1/3rd of the vitreous. There is usually posterior subcapsular cataract formation at the time of presentation. The patient usually has no acute symptoms of redness or pain and the only symptoms may be floaters in front of the eye while the patient is viewing a light background. Progressively the patient may slowly develop a decrease in visual acuity as the vitreous cells and floaters become more organized.

Scleral depression early in the course of illness will reveal a graywhite exudates in the pars plana. This is done by using either an indirect ophthalmoscope or a three mirror lens using a slit lamp. Other signs are peripheral retinal vasculitis with sheathing of both the venules and the arterioles. When vision is reduced below a level of 6/18, ophthalmoscopically visible cystoid macular edema is usually seen.

Local corticosteroid are used only if the patients vision is 6/12 or better and significant anterior chamber reaction is present. Patients with minimal anterior chamber reaction and vision of 6/12 or better receive no treatment. If vision is less than 6/12 and subretinal exudation, optic nerve papillitis or disturbing floaters are found, periocular steroids are given weekly (upto 6 doses). The commonly used periocular steroids is triamcinolone acetonide in doses of 20-40mg/dose. This can be given until visual improvement occurs. If these fails to achieve the desired result, retinal cryopexy should be applied, if cryopexy also fails and the disease is bilateral, cyclosporine should be employed. The common causes of intermediate uveitis are usually idiopathic in nature where the etiology is not usually found. Other specific etiologies are sarcoidosis, sympathetic ophthalmitis, multiple sclerosis and also secondary to septic foci else where in the body.

POSTERIOR UVEITIS

Toxoplasmosis :

It is the most common identifiable cause of pediatric uveitis. Infection during the first trimester is associated with neonatal convulsions, intracranial calcifications and chorioretinitis. Infants infected in the later trimesters often develop the chorioretinitis only.

Majority of the cases in children are recurrences of a congenital infection[8].

This protozoa is usually found in cats and may be acquired by close contact with the animals or with contamination of the cat feces. It is present in three forms, the sporozoites, bradyzoites and tachyzoites. Tachyzoites are the most virulent form of the protozoa. Tachyzoites are responsible for causing severe tissue damage. Bradyzoites are usually the dormant stage of the protozoa.

Inactive atrophic scars usually affecting the posterior pole, occur in approximately 80% of infected new borns. Ocular toxoplasmosis in older children and adults represents a reactivation of the subclinical congenital infection of the retina. The posterior uveitis may be associated with a granulomatous or non-granulomatous iridocyclitis. Congenital toxoplasmosis usually occurs in and around the macula. The patients usually develops a divergent squint as they usually have the disease from their intrauterine life and are not able to use the eye to fix light. This divergence may be the only sign of the macular choroiditis in these children. Also children might get affected by close contact with infected cats and may have the same manifestation as the adult form of the disease. The disease might present with focal, multifocal and diffuse forms.

Treatment consists of the combined use of pyrimethamine, folinic acid supplement and sulfadiazine. Clindamycin may be used in addition to these medications or in substitutions for pyrimethamine. The ELISA is the most sensitive and specific test. Any positive titer is significant in evaluating the diagnosis of toxoplasmosis. Systemic corticosteroids used under antimicrobial coverage. Not all cases are treated in immunocompetent children. The cases that are treated either have a lesion in the papulo macular bundle or near a major vessel or close to the optic disc. In immunosupressed children all the lesions irrespective of locations has to be treated vigorously.

Toxocariasis :

It is primarily a disease of children, acquired by the ingestion of soil containing the eggs of the canine intestinal round worm, toxocara canis. It is unusual for visceral larva migrans and ocular toxocariasis to affect the same person.

Ocular form is typically found in patients approximately 7 – 10 years old. Usually unilateral and presents as strabismus, leucocoria, or decreased vision. May be present as a chronic endophthalmitis, peripheral chorioretinal granuloma or posterior pole chorioretinal granuloma. Peripheral and posterior granuloma ultimately results in

formation of fibres bands which leads to tractional retinal detachment and dragging of the macula and the disc.

Lab diagnosis is by the light sensitivity and specificity (90%) of an ELISA titre for toxocara. Oral or periocular corticosteroid are used to treat toxocara endophthalmitis. Antihelminthics are not effective for ocular toxocariasis[9]. In cases with tractional bands and macular traction viterectomy with release of the traction bands has to be performed to prevent a tractional retinal detachment.

Congenital Syphilis :

Infants with congenital syphilis have been born with active disease, in some active choroiditis may be seen, but in most the only evidence of choroiditis, is the presence of segmental pigmentation in the posterior pole. Cataract may begin earlier, than would be expected. Glaucoma is a late sequelae. Decades later, recurrence of iritis and interstitial keratitis are puzzling but they respond to topical corticosteroid and cycloplegics.

Fungal Disease

Candidiasis and other fungal entities are rare causes of posterior uveitis in children. Vitrectomy and systemic and intravitreal antifungal agents are used for treatment.

Viral Disease :

Rubella retinitis is the most common ocular features of the maternal rubella syndrome. Unilateral or bilateral pigment deposits, usually limited to the posterior pole, vary from powdery or granular to more discrete shapes. Cytomegalo virus retinitis is a common cause of blindness in individuals with immunosuppression. They may occur in the indolent form as well as the fulminating form. Early antiviral treatment is vision saving. Herpes virus can produce progressive outer retinopathy in immunosuppressed children and acute retinal necrosis in immuno competent healthy children. This leads to severe irreversible damage to the retina with subsequent consecutive optic atrophy. Early treatment with systemic acyclovir is useful to reduce the damage caused by the virus.

PAN UVEITIS

Endophthalmitis :

Endophthalmitis is intraocular inflammation predominantly involving the vitreous cavity and anterior chamber of the eye. Contiguous ocular structures such as the retina or choroid may also be involved.

Endogenous endophthalmitis results from blood borne spread of bacteria or fungi during generalized septicemia, characterized by an acute onset with pain, decreased vision, hypopyon and vitritis. Most common organisms are streptococcus pyogenes, staphylococcus aureus, neisseria meningitides, H. influenza and E. coli, Candida is the most common fungal aetiology.

Infectious endophthalmitis is an exogenous infection in which organisms enters through a surgical incision, a traumatic laceration or a conjunctival filtering bleb. The incidence of post traumatic endophthalmitis ranges between 4% - 8%. Most common organisms found are S. epidermidis, bacillus species, streptococcus species, S. Aureus and various fungi.

Most common signs of endophthalmitis are decreased vision, anterior chamber reaction (hypopyon) and vitritis. Conjunctival chemosis, hyperemia, eyelid edema and corneal edema may also be observed. There will be no fundal red glow. Patients will have symptoms of severe pain.

Diagnosis is by culture and sensitivity of aqueous tap and vitreous tap. Drops of the sample should be placed into blood agar, Sabaroud's agar, chocolate agar and thioglycollate broth. One drop each

of the aqueous and vitreous specimens should be placed in clean slides for Gram and Giemsa stains for bacteria and fungi. Management should be instantaneous and one should not wait for the culture reports. If the patient has visual acuity of hand movements or better they should be subjected to intravitreal antibiotics usually a cephalosporin (third generation) and an aminoglycoside (amikacin). If the visual acuity is less than hand movements then the patient should be subjected to immediate core vitrectomy.

Sarcoidosis :

Sarcoidosis is a chronic multisystemic disorders of unknown etiology. Paediatric sarcoidosis has two peaks of incidence, occurring between ages 10 and 15 and in children younger than 5 years old.

It is a common cause of panuveitis consisting of granulomatous and non-granulomatous anterior uveitis, intermediate uveitis and also posterior uveitis. Complications are band keratopathy, cataract and retinal periphlebitis and optic neuritis. Management is with systemic, topical and periocular steroids.

Sympathetic Ophthalmia :

It is a bilateral granulomatous panuveitis that develops after a penetrating injury to one eye. A gradual onset of inflammation will

develop within 3 months after trauma in 70% of the cases and within one year in 90% of the cases.

Initially, photophobia and blurred vision secondary to loss of accommodation and later a bilateral granulomatous uveitis develops. Multiple signs of posterior segment inflammation including cells and haze in the vitreous, choroidal thickening and optic nerve edema also develops.

The first sign of sympathetic ophthalmia is retrolental flare. The pathogenesis is usually the exposure of the uveal tissue to the superficial conjunctival and episcleral vessels which sensitizes the blood to the uveal antigen and triggers an inflammatory reaction in the other eye.

Dalen – Fuchs nodules which are small yellow-white infiltrates in the retinal periphery at the level of retinal pigment epithelium are characteristic, but not pathognomonic for sympathetic ophthalmia. The Dalen – Fuchs nodules contain chronic inflammatory cells like macrophages plasma cells and epitheloid giant cells.

Vogt – Koyanagi – Harada Syndrome

Vogt-Koyanagi – Harada syndrome is a multisystemic disorder of unknown etiology. Although it usually affects young and middle aged adults there have been occasional reports in paediatric patients. Asian

and heavily pigmented persons are more often affected than whites. The Vogt-Koyanagi syndrome consists of skin lesions like hypopigmented patches and poliosis of the lashes along with anterior uveitis. Whereas the Harada syndrome usually consists of posterior uveitis along with retinal detachment (exudative).

TREATMENT

Mydriatics and Cycloplegics:

Topical cycloplegics are beneficial for breaking or preventing the formation of posterior synechiae and for providing relief of photophobia secondary to ciliary spasms. It also relieves the pain associated with spasm of the ciliary body. It also reduces the leakage of the vessels thus reducing the inflammation in the aqueous. The stronger the inflammatory reaction the more frequent is the dosage of cycloplegics. Short acting drop such as cyclopentolate or long acting drops such as atropine may be used.

Corticosteroid :

Corticosteroid are the mainstay of uveitis therapy. The amount and duration of corticosteroid therapy must be determined on individual basis. The minimum amount needed to control inflammation should be prescribed to reduce complications of the treatment. If steroid therapy is used longer than 2-3 weeks, the dosage should be tapered before discontinuation. The dosage should be increased when surgical intervention is required to prevent exacerbation of uveitis postoperatively.

Topical steroids are used only for anterior uveitis because they do not reach a therapeutic level in tissues behind the lens. Strong steroids such as dexamethasone, betamethasone and prednisolone must be used.

Periocular steroids are able to reach therapeutic concentrations behind the lens. A long-lasting effect can be achieved if a depot preparation such as triamcinolone acetonide or methylprednisolone acetate is used.

Periocular sub-tenon injections can be given either anteriorly or posteriorly. Anterior subtenon injections are usually given for severe anterior uveitis, whereas the main indication for posterior subtenon injection is intermediate and posterior uveitis.

Indications for systemic corticosteroid are intractable anterior uveitis which has failed to respond to both topical therapy and anterior subtenon injection, intermediate uveitis which has failed to respond to posterior subtenon injections, and in certain types of posterior or pan uveitis with severe bilateral involvement.

They are usually started with a large dose and then reduced. The initial dose of prednisolone is 1-1.5mg/kg body weight. The total dose should be taken before eating breakfast. Once the inflammation is brought under control, reduce the dose gradually over several weeks. If

steroids are given for less than 2 weeks there is no need for gradual reduction of the dose.

Ocular complications of prolonged corticosteroid therapy are cataract formation glaucoma, corneal / scleral thinning or perforation, ptosis, scarring of conjunctiva / Tenon's capsule.

Systemic complications of prolonged corticosteroid therapy are weight gain, sodium and fluid retention, peptic ulcers, diabetes mellitus, hypertension, osteoporosis, mental status changes, acne and exacerbation of systemic infections.

Methotrexate :

Commonly used drug. It is an anti-metabolite. It inhibits dihydrofolate reductase enzyme. Toxicity is specifically on the bone marrow and the epithelial cells on the body. There may be suppression of the normal function of the bone marrow leading to aplastic anaemia and also toxicity involving the gastric and intestinal mucosa which results in ulcerations and gastrointestinal bleeding. It is used in severe cases of juvenile rheumatoid arthritis, other types of spondylo arthropathies, Bechet's disease. Methotrexate is given along with folinic acid which neutralizes most of the cytopathic effects of the drug.

Dosage is 10-25mg/m^2 per week either in a single weekly dose or divided daily dosage with a break of 2 days for every 5 days.

Etanercept :

This drug inhibits the tumour necrosis factor- α receptor. Thus it prevents the binding of the factor to its receptor and thus prevents the severe inflammatory effects of the TNF α. Dosage is 0.8 to 1mg/ kg body weight / week. It is used in steroid resistant cases and also in severe vision threatening uveitis. It is very useful to prevent joint damage in children with juvenile rheumatoid arthritis as this factor is responsible for the irreversible damage of the joint articular surface.

Cyclosporin, Tacrolimus, Azathioprine and Cyclophosphamide are useful in modulating and controlling the devastating complications of immunologically mediated uveitis and also in children who unable to tolerate steroids.

AIM

The aim is to analyse uveitis in children and adolescents with reference to age, sex, laterality, chronicity, severity, etiology, clinical presentation, complications and the various treatment modalities employed and the final visual outcome.

MATERIALS AND METHODS

The study design was a prospective case study of 100 children and adolescents (<18 years) with uveitis presenting themselves in Stanley Medical College during the period of October 2003 to December 2005 for a period of 26 months.

These patients were questioned for presenting complaints (if they were quite small and not reliable) history was taken from their parents.

The duration of illness, the symptoms, (causative factor if any) associated symptoms esp. joint pain, loss of weight, appetite evidence of focal sepsis were enquired in detail.

Contact history among the children were looked into. Any exposure history in parents were elicited.

A thorough, systemic examination with special attention to musculo cutaneous, musculo skeletal, gastrointestinal, cardio pulmonary and neurological systems was made.

Complete ocular examination was done with special attention to visual acuity, slit lamp examination, intraocular tension, direct and indirect ophthalmoscopy was performed.

All these patients were subjected to a battery of investigations including total blood count, differential blood count, erythrocyte sedimentation rate, blood sugar, motion for ova and cysts.

Suspected cases with history of contact and systemic signs were further subjected to, mantoux test, chest x-ray, x-ray sacro-iliac joints and x-ray of peripheral joints. Investigations in the order of rheumatoid factor, antinuclear antibody, C-reactive protein, anti-cytoplasmic nuclear antibody were done.

ELISA tests (IgM) for toxoplasma and toxocara were done for necessary cases to confirm the clinical diagnosis.

Opinions of consultants in fields like, rheumatology, skin, neurology were obtained, for the patients.

The patients were sent to ENT, skin, dental and chest clinics to rule out any septic foci that might be the cause for the uveitis.

In patients with endophthalmitis, vitreous tapping was done and sent for culture and sensitivity.

Some patients were referred for ultrasound B-Scan of the eye especially cases with opaque media to rule out posterior segment problems.

Regular screening of patients from the rheumatology clinic was done to identify cases of uveitis even though their eyes were quiet.

The patients were treated symptomatically, while they were investigated for any underlying systemic disorder.

The patients with anterior uveitis were treated with cycloplegics and topical steroids. Patients with severe grade of iritis were treated with systemic and periocular steroids along with the topical medications.

Cases with endophthalmitis were managed with cycloplegics, topical antibiotics, systemic antibiotic and intravitreal antibiotics. Unresponsive cases were subjected to core vitrectomy.

Cases with band keratopathy underwent chelation with sodium versenate for their removal.

Surgery in the form of cataract removal with intraocular lens implantation was done in cases with complicated cataract.

Post inflammatory glaucoma was managed with antiglaucoma measures along with steroids and followed up, regularly. Chorioditis that were active were followed up after management of active phase. Cases needing vitrectomy for traction bands, were referred for surgery and later followed-up

ANALYSIS AND OBSERVATION

These were the observations of our study.

Table 1
Age Incidence

Age in Years	No. of Cases	Percentage
0-4	5	5%
5-9	35	35%
10-14	44	44%
15-18	16	16%

In our study the maximum number of cases of uveitis in children were found to be within the age groups of 10-14 years and 5-9 years, comprising 44% and 35% of the cases respectively. Least number of cases were found to be in the age group of 0-4 years.

Table 2
Sex Incidence

Sex	No. of cases	Percentage
Male	54	54%
Female	46	46%

Out of the 100 cases in this study. 54 cases were males and 46 cases were females.

Table 3
Laterality

Laterality	No. of Cases	Percentage
Unilateral	52	52%
Bilateral	48	48%

Regarding the laterality out of 100 cases in our study, 52 cases were unilateral and 48 cases were bilateral.

Table 4
Duration and Onset

Duration	No. of Cases	Percentage
Acute	54	54%
Chronic	46	46%

Out of our 100 cases 54% presented with acute onset of less than 6 weeks and 46% with chronic duration. Acute on chronic cases were included with chronic uveitis.

Table 5
Aetiological Analysis (Anterior Uveitis)

Type	No. of cases	Percentage
Idiopathic	31	48%
Allergic	10	15%
Arthropathy associated	13	20%
Traumatic	8	12%
Infective	3	5%

In our study out of the 65 cases of anterior uveitis (31 cases). 48% were formed by idiopathic causes. Iritis secondary to septic foci in the body formed 15% of cases. Anterior uveitis associated with juvenile rheumatoid arthritis and sero negative arthropathies amounted to 20% of cases (13 cases). Trauma was the cause for 12% of cases. Infective causes amounted to 5% of cases (3 cases). They belonged to children with herpes simplex uveitis (two) and one with fungal corneal ulcer with fungal iritis and hypopyon.

Table 6
Aetiological analysis of posterior uveitis

Aetiological Type	No. of cases	Percentage
Tuberculosis	4	19%
Toxoplasmosis	12	57%
Toxocara	1	5%
Others	5	19%

In our study out of the 21 cases of posterior uveitis. Tubercular etiology was found in 4cases, 12cases of toxoplasmosis involving both old and fresh cases and one case of Toxocara endophthalmitis, four cases of chorio retinal vasculitis namely one each of Eale's disease, SLE, cytomegalovirus retinitis in immunosuppressed child and one due to systemic leptospirosis.

Table 7
Anatomical Classifications

Type	No. of Cases	Percentage
Anterior	65	65%
Intermediate	6	6%
Posterior	21	21%
Pan-uveitis	8	8%

Based on anatomical classification (IUSG) in our study 65 cases (65%)were anterior uveitis, 6 cases (6%) were intermediate uveitis, 21 cases (21%) were posterior uveitis and 8 cases (8%) were pan uveitis.

In intermediate uveitis 5 cases were idiopathic and one case was due to focal sepsis.

In pan uveitis four cases were due to infection with bacterial seeding, ultimately leading on to endophthalmitis. 3 cases were due to tuberculosis, 1 case was due to active toxoplasmosis.

Table 8
Age incidence (Anatomical Classification)

Age in years	Anterior Uveitis	Intermediate Uveitis	Posterior Uveitis	Pan Uveitis
	No. of Cases	No. of cases	No. of Cases	No. of Cases
0 – 4	1	-	3	1
5 – 9	19	2	11	3
10 – 14	36	2	5	1
15 – 19	9	2	2	3

In our study anterior uveitis was more prevalent in the age group of (5-14 years). Among the very young (0-4 years) posterior uveitis form the bulk of the cases. Intermediate uveitis was not seen in the age group of 0-4 years.

Table 9
Laterality based on Anatomical Classifications

Laterality	Anterior Uveitis	Intermediate Uveitis	Posterior Uveitis	Pan Uveitis
	No. of Cases	No. of cases	No. of Cases	No. of Cases
Unilateral	39	3	5	5
Bilateral	26	3	16	3

In our study majority of the anterior and pan uveitis cases were unilateral. Whereas the converse was true in cases of posterior and intermediate uveitis.

Table 10
Complications

	No. of Cases
Band keratopathy	3
Complicated cataract	5
Divergent Eye	4
Exudative retinal detachment	2
Phthisical eye	1

Among the most serious complications complicated cataract accounted for five cases, followed by Divergent eye due to early choroiditis 4 cases. Band keratopathy accounted for 3 cases, exudative RD for 2 cases and 1 eye was phthisical.

Table 11
Visual Outcome After Surgery

	Cataract Surgery					**PPV**	
Pre-Op Vision	4/60	4/60	2/60	6/36	3/60	HM +	PL/PR+
Post-Op Vision	6/18P	6/9	6/12P (with glasses)	6/9	6/9P (with glasses)	6/18 P	6/24

Most patients after cataract surgery had a good post-op visual acuity. Two cases had recurrent post-op iritis but have useful vision.

Two cases after pars plana vitrectomy had useful post-op vision with contact lenses.

Table – 12.
Etiology in Pan Uveitis and Intermediate Uveitis

Etiology	Pan Uveitis No. of Cases	Intermediate Uveitis No. of cases
Idiopathic	-	5
Infective :		
Endogenous	2	-
Exogenous	2	
Specific Infections :		
Tuberculosis	3	-
Toxoplasmosis	1	-
Septic Foci	-	1

In our analysis, the cause of pan uveitis was infection. They comprised of exogenous and endogenous infections. Exogenous infections were due to corneal ulcer and trauma. Endogenous infection producing pan uveitis were due to septicemia.

Specific etiological cause for pan uveitis, was due to tuberculosis (3 cases) and 1 case of Toxoplasmosis.

As for intermediate uveitis they were mainly parsplanitis secondary to idiopathic causes as no etiological factor could be found out after extensive investigations. One case was due to allergens from septic foci in the dental cavity.

DISCUSSION

In our study of 100 paediatric and adolescent uveitis it was observed that uveitis is more common in the age group of 5-14 years, which forms nearly 80% of the cases.

More than half the cases were of acute onset.

Least number of cases were found in the age group of 0-4 years.

On analyzing the aetiology of uveitis in our study the majority of cases were of unknown aetiology (36 cases), followed by juvenile spondyloarthropathies (13 cases), next comes toxoplasmosis (12 cases). Focal sepsis were also common manifesting about 15% of anterior uveitis.

ANATOMICAL CLASSIFICATION

Our anatomical classification is based on the international uveitis study group (IUSG).

Classification :

Anterior Uveitis	:	65%
Intermediate	:	6%
Posterior Uveitis	:	21%

Pan Uveitis : 8%

Comparative Studies :

In a study by Paivonsalo-Heitonen; Tuominen J, (Uveitis in children Scandinavia 78(1) 84-8, 2000) shows out of 55 paediatric uveitis, 50(90%) had anterior uveitis, 3(6%) had posterior uveitis and one case was found to have intermediate uveitis[10].

In a study conducted by Soylu et al (Paediatric Uveitis – Ocular immunology 5(3) : 197 – 202 Sep. 1997) out of 90 patients 30 (33.3%) had anterior uveitis, 21(23.3%) had posterior uveitis, 8(8.9%) had intermediate uveitis and 31(34.4%) had pan uveitis[11].

Thus there are wide variations among regional and ethnical groups.

AETIOLOGICAL ANALYSIS

In the study by Soylu et al (1997 Sep) Idiopathic causes, constituted 48.4%, of pan uveitis and 46.7% of anterior uveitis cases. The most common cause of posterior uveitis in his study was toxoplasmosis 39%.

In our study, idiopathic causes played a major part of anterior uveitis 48%. Arthropathy associated causes, constitutes 20% of cases,

septic foci producing allergic uveitis was 15%, traumatic constituted 12%.

A study was done in North India at PGI, Chandigarh, between Jan. 98 to Jun. 01 using 1233 patients out of which 52% were males, and 48% were females. Anterior uveitis comprised of 49.3%, posterior 20.3%, intermediate 16% and pan uveitis 14.5%. Out of the total idiopathic causes amounted to 51.2% and specific etiology was found out in 48.8% of infective cases. TB was the common cause, followed by, toxoplasmosis. In non-infective etiology JRA formed the most common etiology.

In out study arthropathy associated cases formed a major part of non-infective uveitis. They were secondary to juvenile rheumatoid arthritis, which formed a major bulk, followed by one each of early onset ankylosing spondylosis and psoriatic arthritis.

These cases were referred from the rheumatology and Paediatric Departments of Govt. Stanley Hospital. JRA cases, were of both ANA positive, ANA negative and both pauci and poly articular type. As per literature it was the pauci articular type that had the most severe presentations.

The children who had band keratopathy underwent chelation with Sodium versanate and some children who had complicated cataract, underwent cataract extraction with PCIOL implantation. Some children with PCIOL implantation suffered from sever post-op iritis leading to formation of exudative membrane . Two children underwent pars plana lensectomy with anterior vitrectomy and had few post-op complications.

J. Paed. Ophthal. Strabismus 2001, May-Jun (38)3 129, 176, by Paulos P, Totopoulos M et al, Greece, states that Pars Plana Lensectomy with anterior vitrectomy as a safer option for children[12].

Graefe's Arch, Clinical Exp. Ophthalmol 1996, Oct. 234 (10) 618-22, Analyses complications in cataract surgery in children and identifies pars plana lensectomy with anterior vitrectomy as the best choice for complicated cataract in children.

In our study, trauma cases were usually due to blunt trauma producing a mild to moderate iritis which cleared with topical steroids.

In our study, cases following septic foci else were in the body formed about 15% of cases of anterior uveitis and are secondary to mostly dental cases, sinusitis and pharyngitis. These cases had no recurrence, after the primary cause was treated.

Tuberculosis was a common cause of uveitis. These cases were either having primary tuberculosis or had reactivation of tuberculosis. They were treated by the chest physician at the chest clinic here at Stanley Hospital and they had no recurrences. Tuberculosis was responsible for both anterior, posterior and pan uveitis.

Two cases of infective iritis secondary to herpetic keratitis leading to kerato uveitis were assessed. They were put on systemic acyclovir along with topical acyclovir. Once their corneal lesion healed they were put on steroids.

Intermediate uveitis had idiopathic aetiology as leading cause. One case was secondary to septic foci.

Most common cause of posterior uveitis were due toxoplasmosis. Acute cases had positive IgM by ELISA test (for toxoplasma antibody). Other cases were clinically diagnosed to have toxoplasmosis.

COMPLICATIONS

Complicated cataract was found in five patients. Most of the cataract cases were due to JRA which still causes the most debilitating complications in the uveitis patients. One of the reasons could be that these patients had minimal symptoms and they did not present themselves for regular followup.

Cases with band keratopathy – 3 cases had band keratopathy and were secondary to JRA. They were taken up for chelation.

Inflammatory glaucoma were seen in a few cases and they got controlled as the inflammation was controlled.

Exudative retinal detachment as complication of posterior uveitis occurred in 2 cases which resolved spontaneously.

Four patients had divergent eyes mostly secondary to early onset of macular choroiditis due to toxoplasmosis.

One eye became phthisical, unresponsive to treatment.

Cases with old choroiditis and scars were meticulously followed up for recurrence and they were educated about the symptoms of reactivation.

Another study by Ben Ezra, E. Cohen, (Uveitis in Children, Hebrew University 2004) examining 825 patients showed that 33.3% of cases were infections and 66.7% cases were non infectious. JRA was present in 14.7cases. JRA cases had the most anterior segment complications[13].

TREATMENT

In our study cases were graded according to severity into mild, moderate and severe eyes.

Mild and moderate cases responded well to topical steroids and cycloplegics. Few cases esp. those secondary to septic foci, and arthropathy required periocular steroids.

Severe cases required, periocular steroids and systemic steroids.

Case of vasculitis due to SLE required anti metabolites.

Similarly few cases of JRA required T. Methotrexate with folinic acid for suppression of systemic disease and to prevent ocular recurrence. Cases on antimetabolites were regularly followed up with hemogram to rule out complications. Some cases were lost as they did not follow-up. So far no systemic toxicity following methotrexate administration was found.

Other cases (Cases secondary to other etiological causes) were meticulously followed up for recurrences and referred to appropriate depts. for management of primary causes. Cases with cataract and opaque media were referred for B. Scan to rule out posterior segment pathology.

Complicated cataract were subjected to cataract surgery three months after control of inflammation. Two cases had chronic postoperative iridocyclitis. An Indian study showed that patients performed well with intraocular lenses[14].

In a study of (Ben Ezra D and Cohen E. Cataract surgery in chronic uveitis) : Ophthalma 2000, showed out of 17 cases with pre-op visual acuity of < 3/60, visual acuity improved in many cases. Some cases with posterior chamber intraocular lenses had recurrent iritis with exudative membrane and secondary glaucoma[15].

Some cases underwent pars plana lensectomy fared better with less post-op complications and had good vision with contact lenses[16].

In our study some cases of endophthalmitis were taken up for pars plana vitrectomy and they had useful post operative vision.

Vitreous samples were taken up for culture and sensitivity and appropriate intravitreal antibiotics used.

Cases of posterior uveitis received appropriate antimicrobials as per the etiology once it was found out after extensive investigations. Depot steroids were given under antibiotic cover.

CONCLUSION

- Uveitis beginning in childhood is a serious disease associated with sight threatening complications.
- Paediatric uveitis comprising of anterior uveitis is common in the age group between 5-15 years and posterior uveitis is common in children younger than ten years.
- Anterior uveitis is the most common anatomical type amounting to more than 50% of the cases.
- Idiopathic non specific etiology forms a major part of anterior uveitis.
- Toxoplasmosis active and old cases formed a major part of the posterior uveitis.
- Toxoplasmosis and tuberculosis caused both posterior as well as pan uveitis.
- Traumatic anterior uveitis due to blunt trauma were mild and resolved without any sequelae.

- Uveitis anterior associated with juvenile rheumatoid arthritis, juvenile ankylosing spondylitis, had the most severe prognosis leading to vision threatening complications.

- Intermediate uveitis were commonly due to idiopathic causes, which were asymptomatic until the disease was well advanced, leading to visual morbidity. This stresses the importance of routine school screening of visual acuity and slit lamp exam of children to identify the problems early.

- In some cases of JRA, uveitis preceded the arthritis thus stressing the importance of paediatric screening.

- Early identification and management and a thorough search of treatable causes is paramount to prevent most of the complications of uveitis.

- Routine follow-ups, stressed on the parents and guardians of the patients is mandatory to recognize and manage recurrences to prevent vision threatening complications.

- Children with old choroiditis due to toxoplasma, were, meticulously followed up for reactivation, as it manifests with minimal symptom or none at all.

- In children with complicated cataract, those managed with IOL after surgery, some, manifested with persistent iritis post-operatively.

- In few other cases they had no morbidity post operatively and had good visual outcome.

- In cases of endophthalmitis and tractional vitreal bands, vitrectomy was performed and they had useful vision.

- There has to be an increased awareness among paediatricians, rheumatologists and also in ophthalmologists about early detection, and screening of young susceptible children and careful follow up treated cases for recurrences would go about a long way in preventing the devastating visual complications that arise out of the disease.

- Further the development of a safer immuno suppressant in cases of arthropathy induced uveitis and retinal vasculitis will take away the gloomier picture associated with long term supportive management of these cases.

- Advocation of antenatal screening of mothers to detect the presence of Torch infections and manage them before ocular complications arise.

- Children with low vision have to be trained for special vocation and employment and have to be provided low vision aids at subsidized cost.

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3) Indian Journal of Ophthalmology
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5) Community Eye Health Journal

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PROFORMA

UVEITIS IN CHILDREN AND ADOLESCENTS

Name : Age / Sex : Hospital No. :

Address :

Present Complaints : Pain / Redness / Photophobia / Defective Vision/ Watering / Injury / Floaters

Duration :

Other Complaints :

Past History : Previous Episodes and Treatment

: Viral Illness / Joint Pain / Low backache / Skin lesions / Drug allergy / Injury

Personal History: - H/o Contact with tuberculosis or leprosy
- H/o. Pica
- H/o. Contact with pet animals
- Antenatal H/o. exanthematous fever / Torch Inf. / drug intake / Recurrent abortions.

General examinations : Anaemia / Lymphadenopathy

Cardiovascular System :

Pulse : /min Respiratory System :

BP : mmHg Central nervous system :

Resp. rate : / min

Oral :

ENT :

Skin :

Genitals :

Ocular Examination : Right Eye Left Eye

Vision:

Extraocular Movements :

Eyebrows, Lids, Lashes

Conjunctiva :

Cornea :

AC :

Pupils :

Iris :

Lens :

Anterior Vitreous :

Fundus :

Intraocular Pressure :

Visual Fields :

Investigations

Blood : TC Mantoux Test

DC Sputum for AFB

Hb% X-ray joints

Blood – Sugar, RA Factor, VDRL, ANA factor, Elisa for toxoplasmosis.

AC/Vitreous Tap.

B-Scan (if necessary)

Urine : Albumin

Sugar

Motion : Ova

Cyst

ENT Opinion :

Dental Opinion :

Rheumtology opinion :

Dermatology Opinion :

Chest Clinic Opinion :

Diagnosis

- Acute / Intermittent / Chronic
- Unilateral / Bilateral
- Granulomatous / Non Granulomatous
- Mild / Moderate / severe / Quiet eye
- Anterior / Intermediate / Posterior / Pan uveitis
- Aetiology

Treatment

Medical - Cycloplegics & Mydriatics
Topical Steroids
Systemic steroids
Periocular steroids
Others.

Surgical -

Follow-up

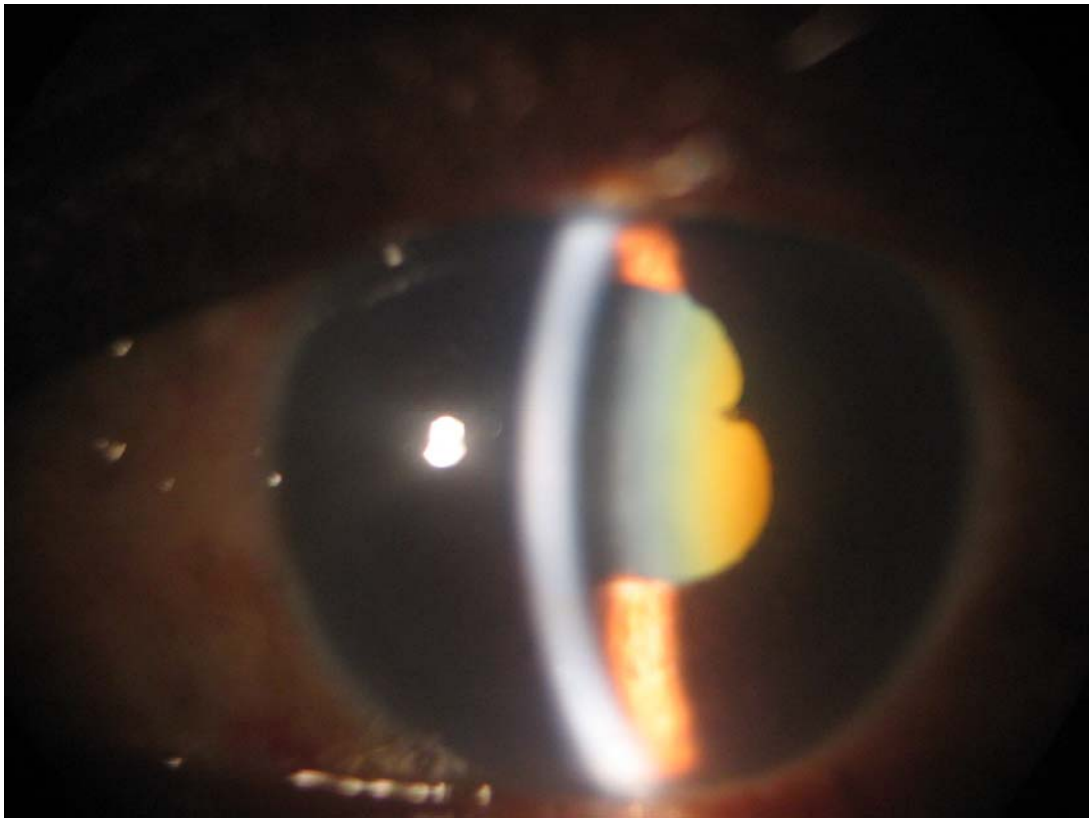
Ist

IInd

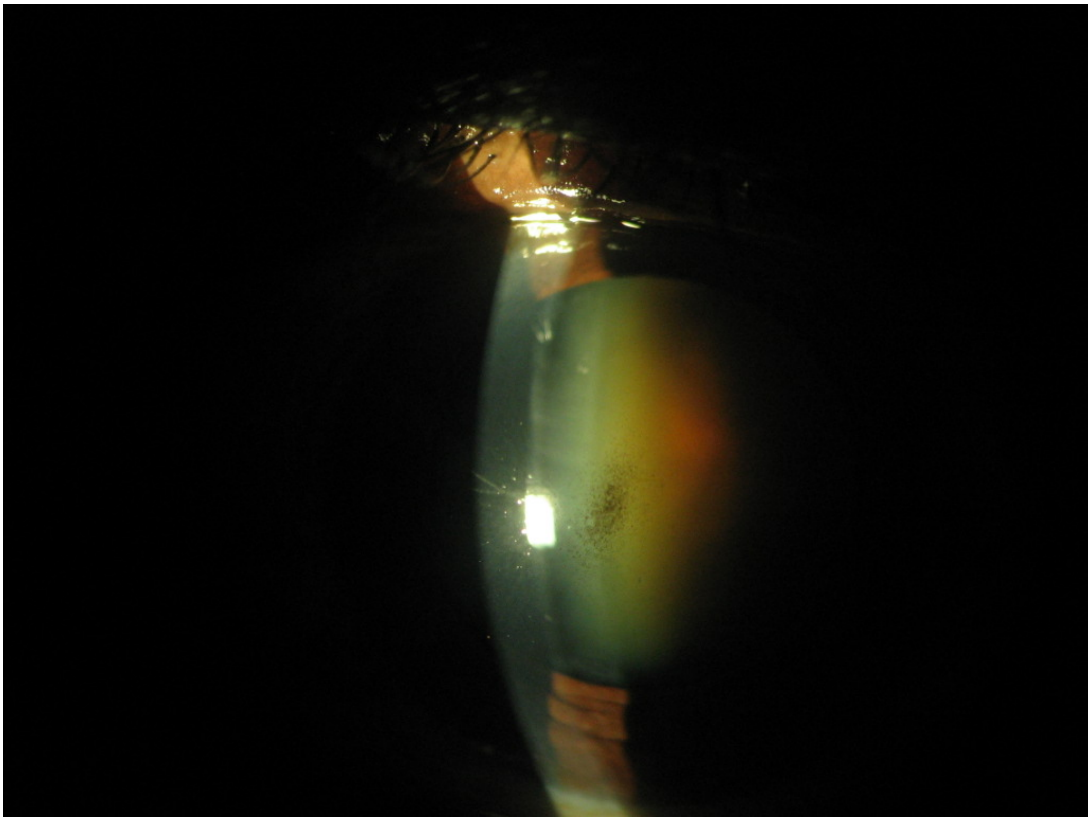
IIIrd

BAND KERATOPATHY IN A 13 YEAR OLD GIRL

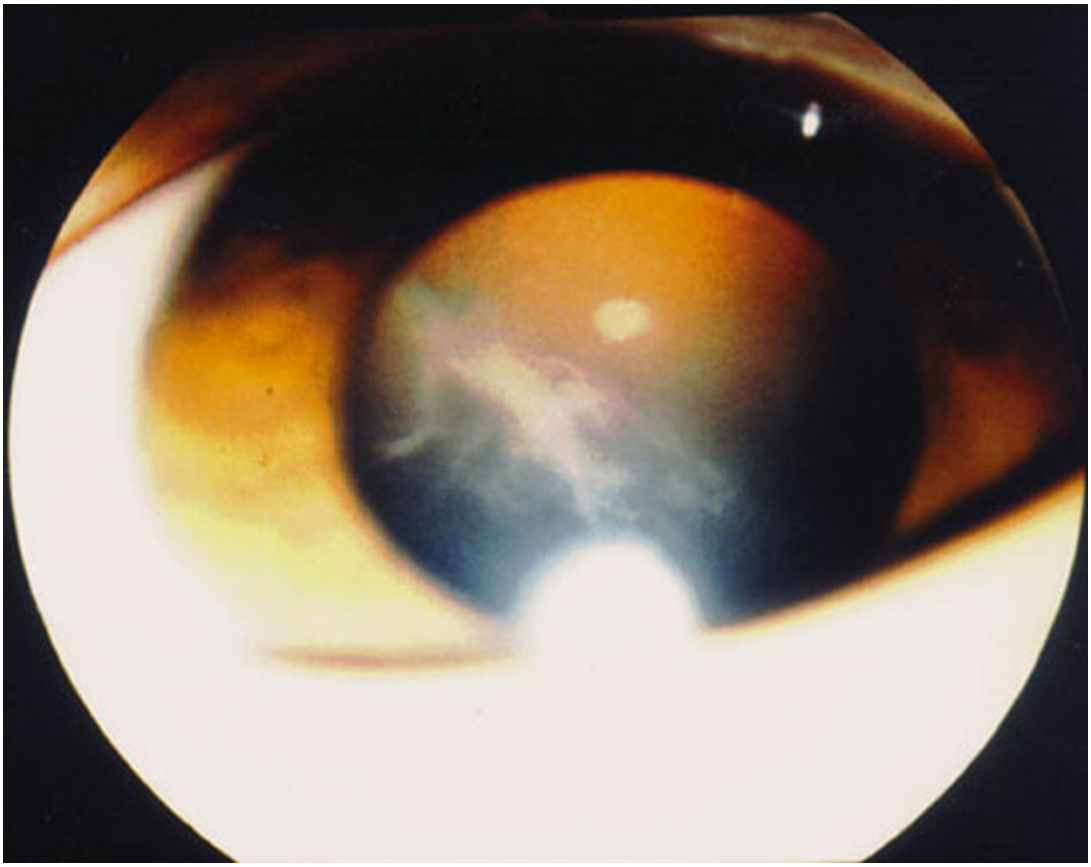
POSTERIOR SYNECHIAE IN A 16 YEAR OLD MALE



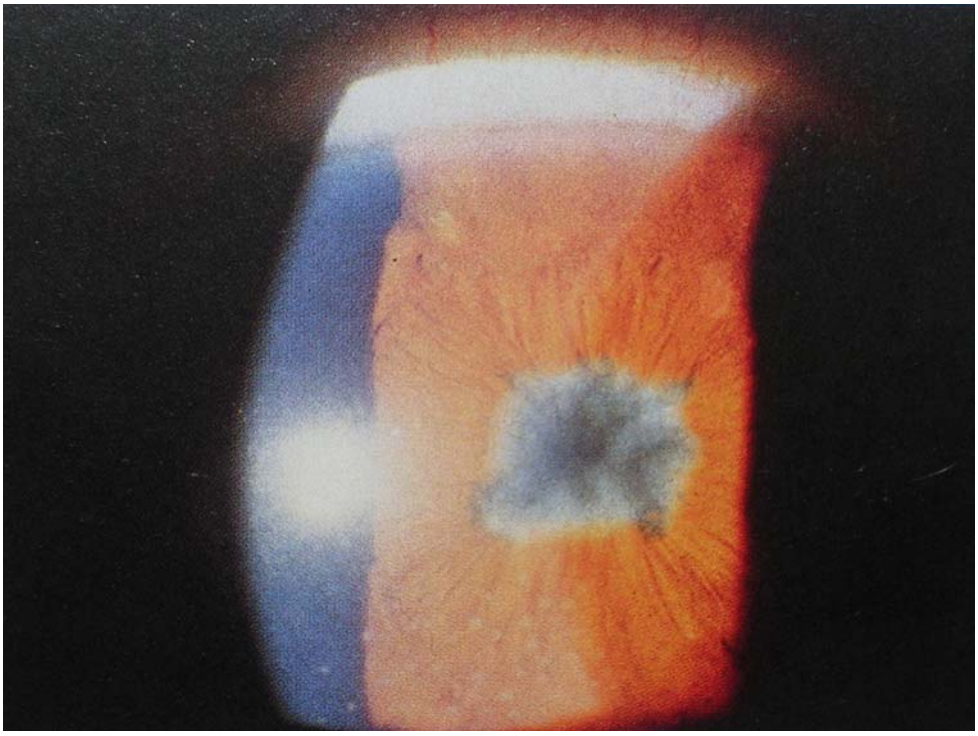
IRIS PIGMENT DEPOSIT IN ENDOTHELIUM



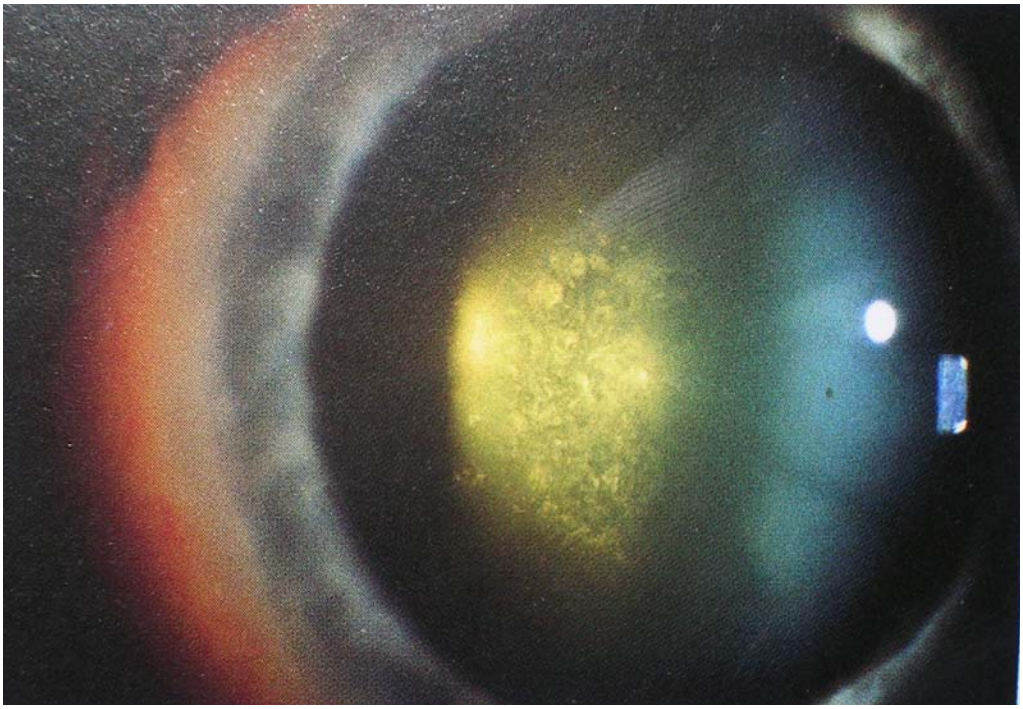
VITREOUS FLOATERS IN A 9 YEAR OLD MALE



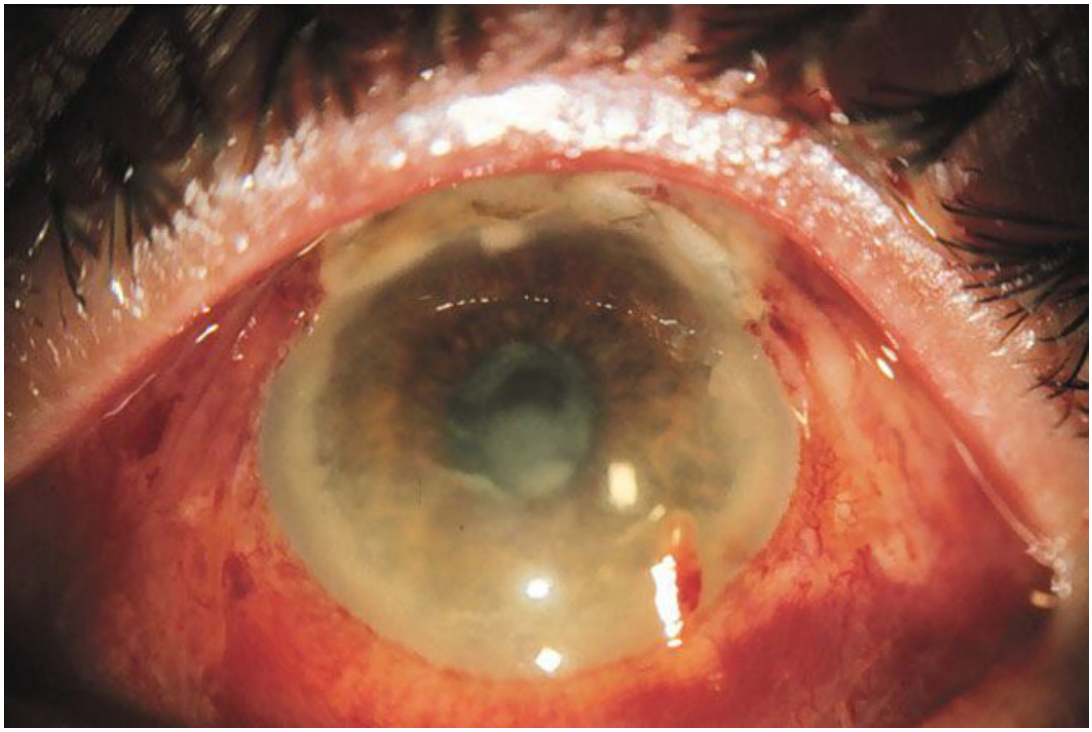
POSTERIOR SYNECHIAE WITH CATARACT
IN A 13 YEAR OLD GIRL



POLYCHROMATIC LUSTRE IN A COMPLICATED CATARACT

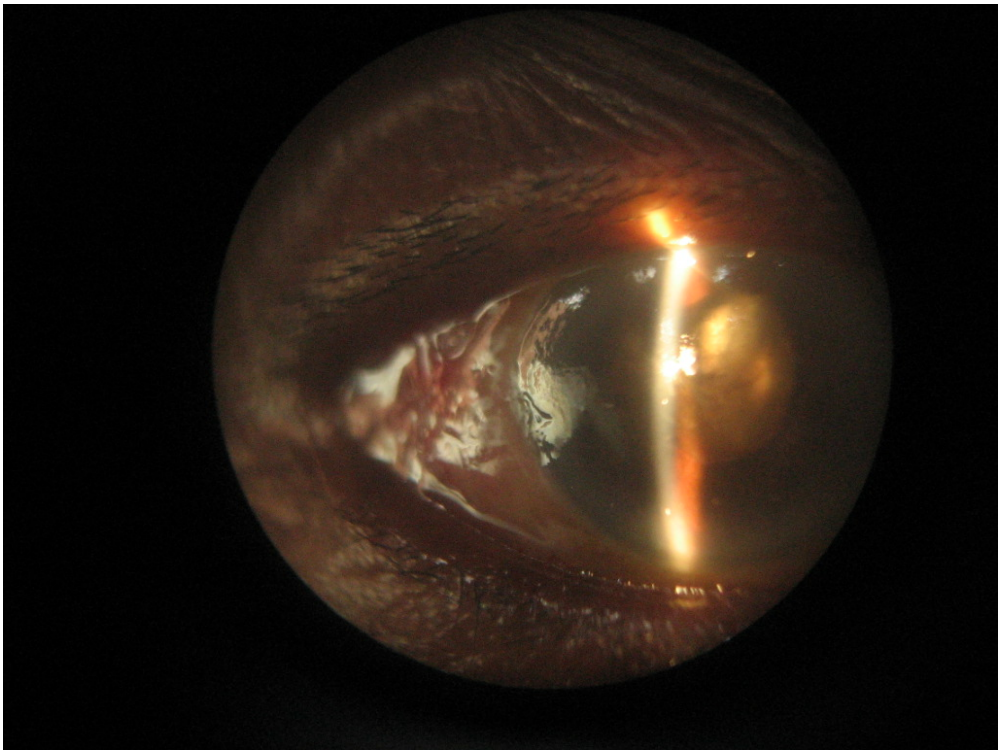


ENDOPHTHALMITIS IN A 16 YEAR OLD MALE

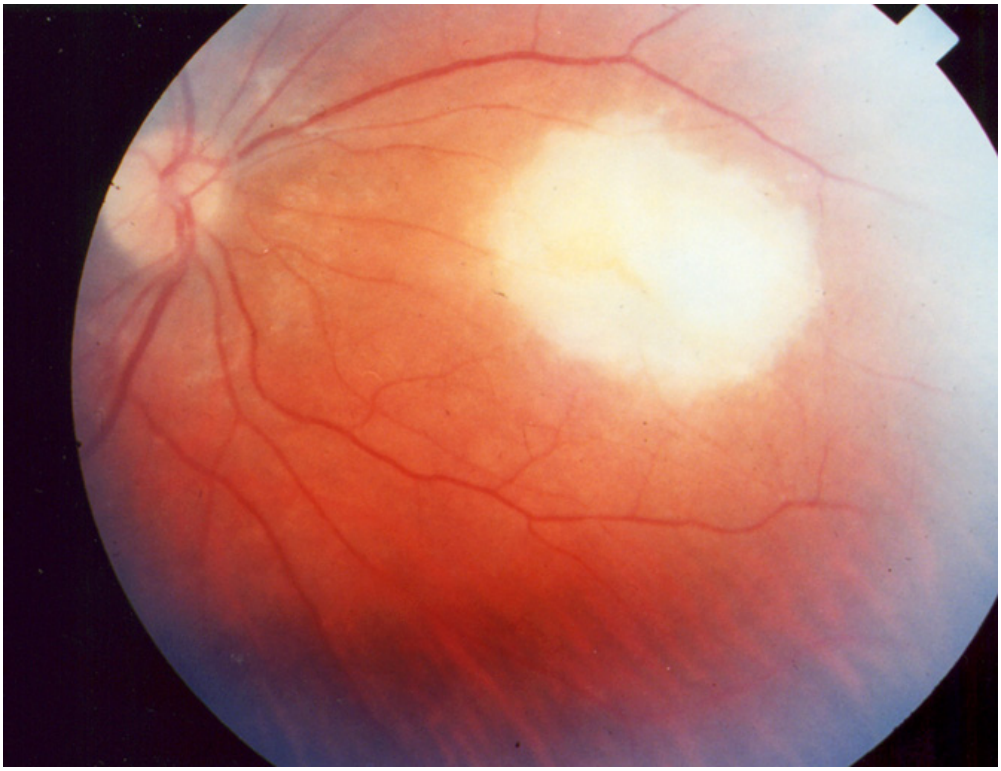


SEVERE POST-OP IRITIS WITH HYPOPYON IN A 13 YEAR OLD GIRL

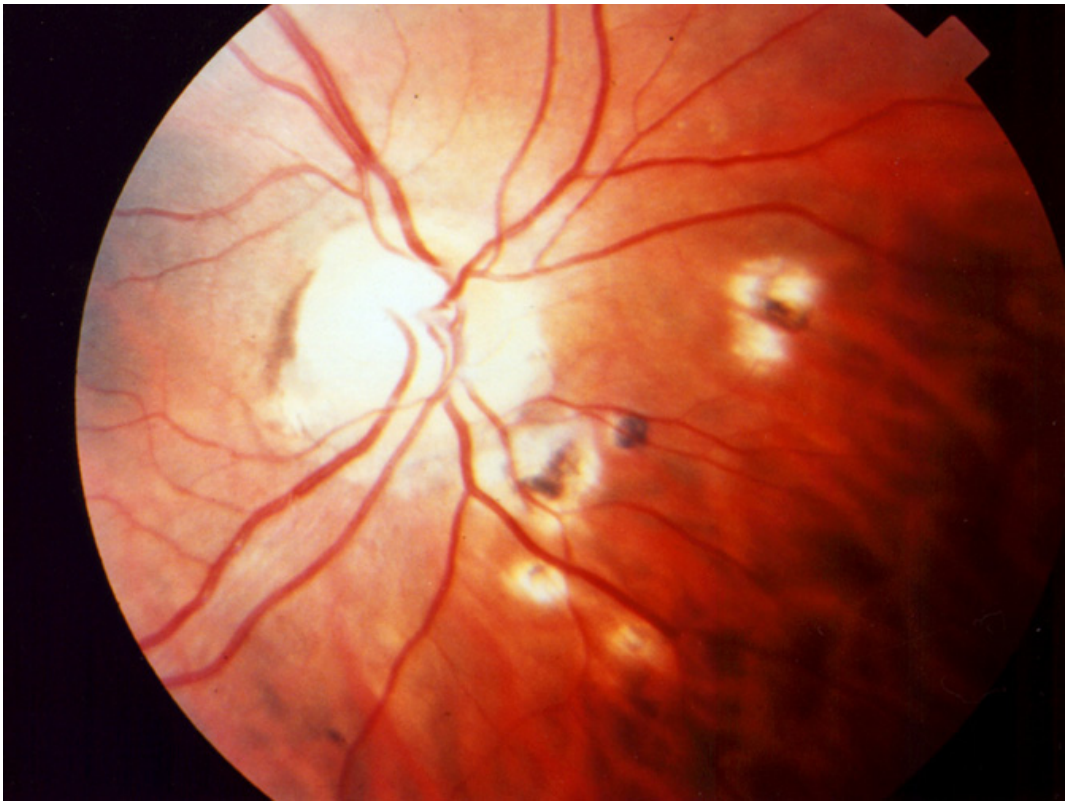
POST-OP IRITIS WITH EXUDATIVE MEMBRANE



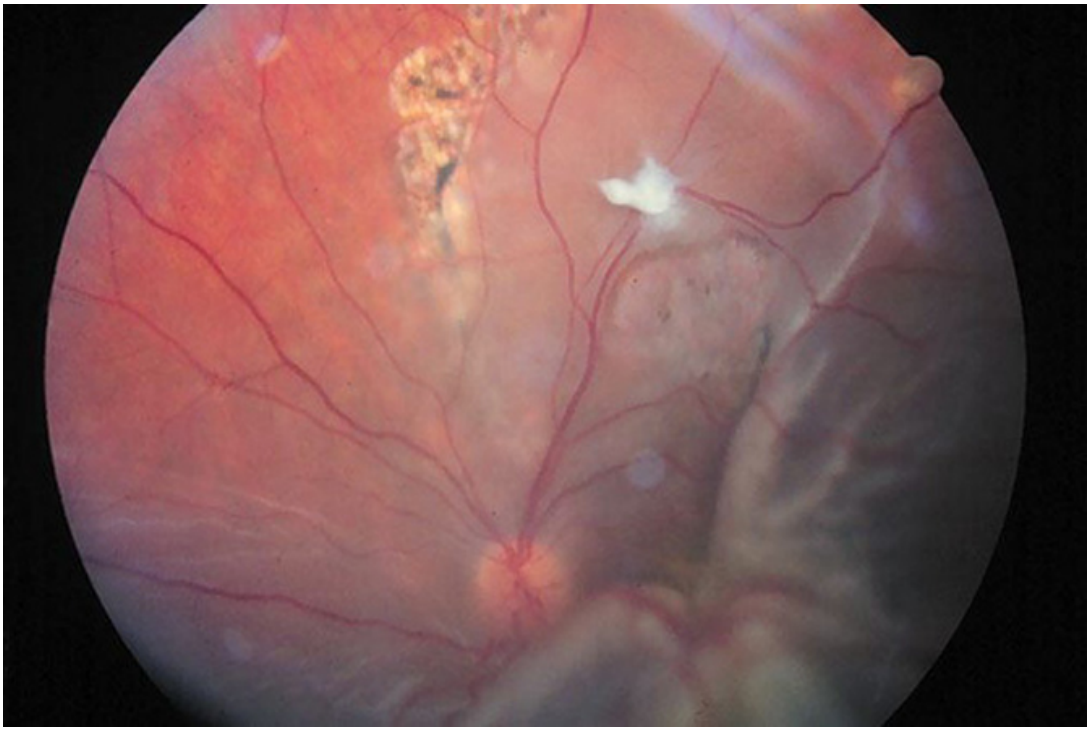
ACTIVE TOXOPLASMA CHORIO RETINITIS IN A 8 YEAR OLD BOY



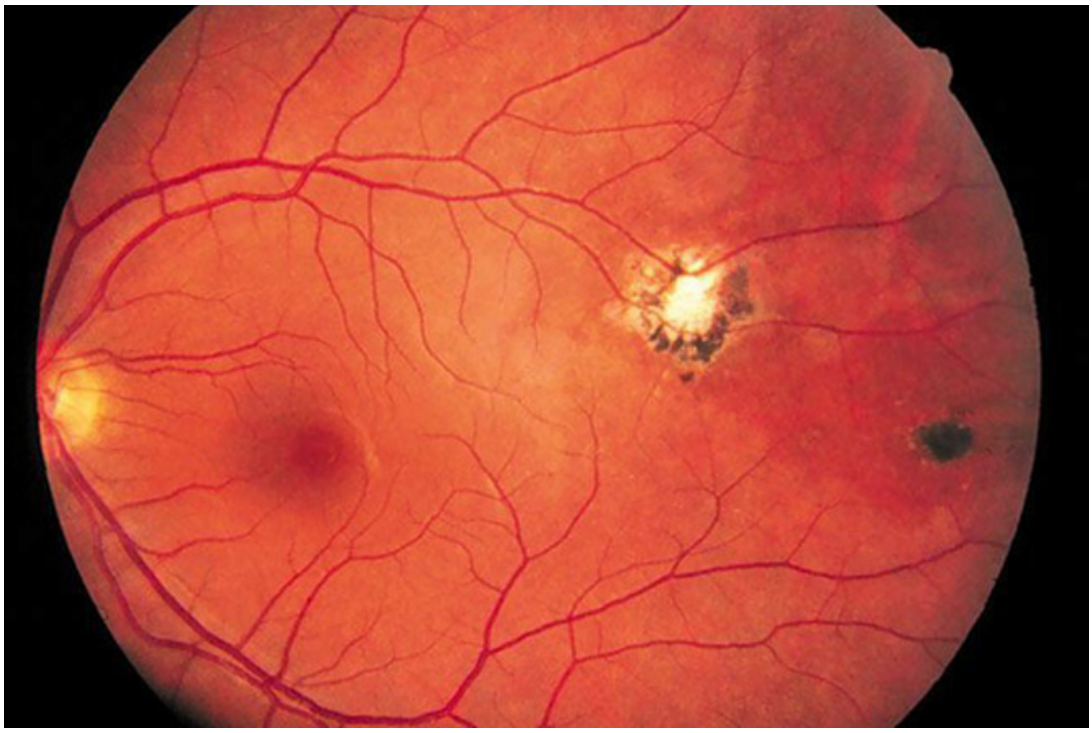
OLD TOXOPLASMOSIS SCARRING WITH REACTIVATION



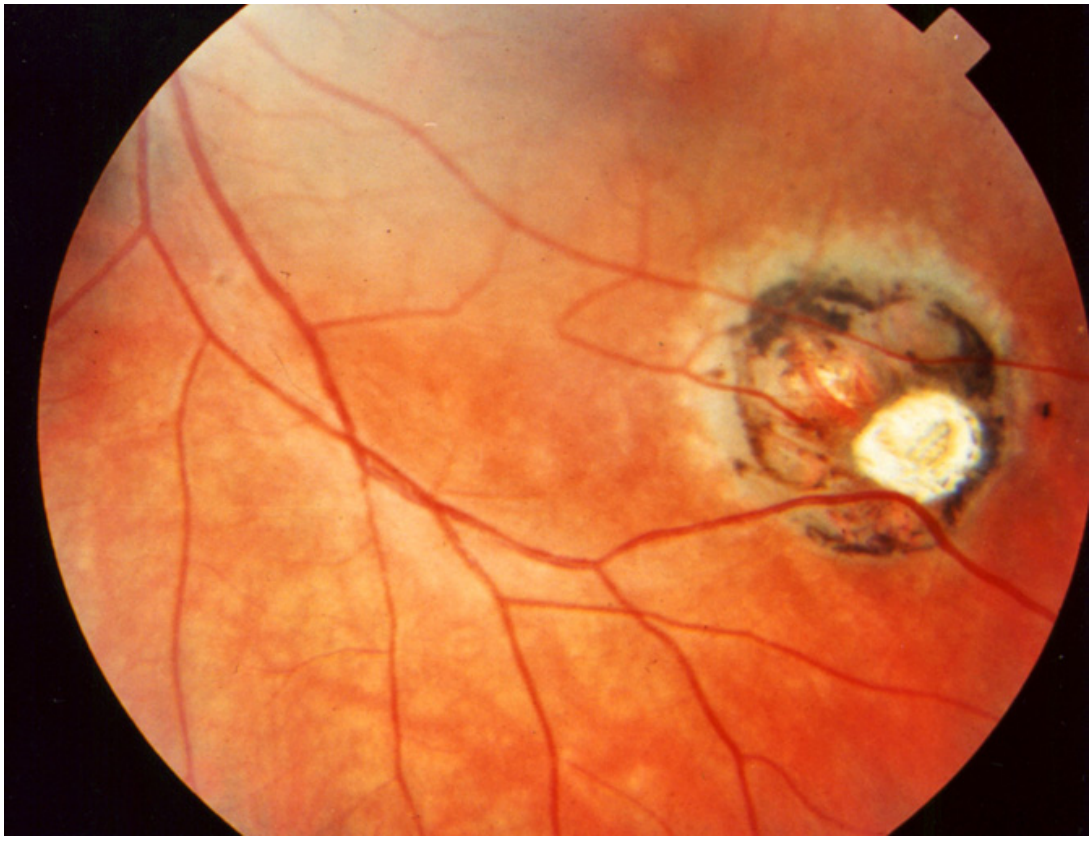
EXUDATIVE RETINAL DETACHMENT FOLLOWING CHOROIDITIS



HEALED CHORIORETINAL SCAR FOLLOWING TOXOPLASMOSIS



HEALED CHORIORETINAL SCAR SHOWING PIGMENTARY BORDER



MASTER CHART

S. No	Name / OP NO.	Age/ Sex	Complaints / Duration	Vision		Anterior Segment		Fundus/ Vitreous		Investigation	Diag-nosis	Treat-ment	Final Vision	
				RE	LE	RE	LE	RE	LE				RE	LE
1	Parvathy 920016	9 /F	R,W,P,LE 1 wk	6/6	6/9	-	F+, C+	-	-	-	LE AU	Myd +, TS +	6/6	6/6
2	Stephen 920144	13/M	R,W,P,Ph,LE 5 d	6/6	6/9 P	-	KP+,F+,C+	-	-	-	LE AU	Myd +, TS +	6/6	6/6
3	Manikandan 920163	14/M	R,W,P,Ph,DV, BE 4 wk	6/12	6/12	C+, KP+,F+,PS	C+, KP+, F+	-	-	TC, DC, ESR, Mx, Rh-C, De-C	BE AU	Myd +, TS, Den. Ext	6/6	6/6
4	Chellappan 922341	9/M	R,P,RE 2-wk	6./9 P	6/6	C+, KP+,F+	-	-	-	-	RE AU	Myd +, TS +	6/6	6/6
5	Saranya 923788	11/F	R,W,DV, Ph, Tr, LE 3 d	6/6	6/12	-	C+, KP+, F+	-	-	-	LE AU (Tr)	Myd +, TS+	6/6	6/6
6	Suresh 927373	15/M	W,Ph,DV,BE (on / off) 6 m	6/18	6/12 P	C+, KP+,F+	C+, KP+, F+, PS	-	-	TC, DC, ESR, Mx, Rh-C, ANA, RF	BE AU (Jra)	Myd +, TS+, Pe.S+	6/6 P	6/6
7	Suhashini 931462	11/F	DV,R,Fl,RE - 4 m	2/60	6/9	-	-	Fl, Vc, Ch	-	TC, DC, ESR, ELISA, CxR	RE PoU (Toxo)	PeS +, Anb	6/12	6/9
8	Raghavi 932848	7/F	R,DV,W,Ph,BE - 2wk	6/9	6/12	C+, F+,KP+	C+, F+	-	-	TC, DC, ESR, Mx, Ch - C, Rh-C	BE AU	Myd +, TS +	6/6	6/6
9	Ravi 933023	16/M	R,DV,W,Ph,LE - 5 d, Tr	6/6	6/12	-	C+,F+,KP+	-	-	-	LE (Tr) AU	Myd + TS +	6/6	6/6
10	Selvi 936681	11/F	R,DV,W,Ph,BE - 2wk	6/9 P	6/12 P	C+, F+,KP+	C+, F+, KP+, PS	-	-	TC, DC, ESR, Mx, Ch - C, Rh-C, De-C	BE AU	Myd + TS +, Den. Ext	6/6	6/6
11	Kumaran 937001	12/M	DV, W, Ph (on/off) - 12 wk	6/18	6/18	C+, F+,KP+, PS	C+, F+, KP+, PS	-	-	TC, DC, ESR, Mx, Ch - C, Rh-C	BE AU (Jra)	Myd +, TS+, Pe.S+	6/6 P	6/9
12	Gajalakshmi 939343	8/F	DV, W, Ph, R-1wk, LE	6/6	6/9 P	-	C+, F+, KP+	-	-	-	LE AU	Myd +, TS+	6/6	6/6
13	Mari 939600	10/F	P,W,R,RE-1 wk	6/9	6/6	C+. F+	-	-	-	-	RE AU	Myd +, TS+	6/6	6/6

14	Naresh 940067	6/M	Fl, DV - 3 yrs	6/9	1/60	-	-	-	Ch, VFl	TC, DC, ESR, ELISA, CxR	LE PoU (Toxo)	Observation	6/9	1/60
15	Chamundeswari 940866	8/F	DV, Fl, BE-3 wk	2/60	4/60	-	-	Ch, VH, VC	CH, VH, VC	TC,DC, ESR, ELISA	BE PoU (Toxo)	Myd +, TS+, Anb	6/12	6/18
16	Sivakumar 943579	17/m	P,R,Ph,W,BE-2 wk	6/18	6/18	C+, F+, KP+	C+, F+, KP+	-	-	TC,DC, ESR	BE AU (Inf)	Myd +, AnV	6/6	6/6 P
17	Varadharajan 945432	16/M	Fl,W, DV, BE - 8 wk	6/18	6/12	F+	F+	VC, VF, SB	VC, VF, SB	TC, DC, ESR, Mx, CxR, Ch - De - C	BE IU	Myd + TS+, Pe.S	6/9	6/9
18	Varsha 946621	2/F	R,Ph,W,RE - 3 d	-	-	-	Hypopyon, F+, C+	-	VH, VF, Yellow reflex	-	RE PaU	Anb, PPV	PHTHISICAL EYE	-
19	Rajan 947225	12/M	P,R,Ph,W,RE-1 wk	6/12	6/6	C+, F+	-	-	-	-	RE AU	Myd +, TS+	6/6	6/6
20	Sivagami 948352	9/F	P.R.Fl,W,DV, BE - 10 wk	6/18	6/9 P	C+, F+, KP+, PS, BK	C+, F+, KP+, PS	-	-	TC, DC, ESR, Mx, CxR, RA, ANA	BE AU (Jra)	Myd +, TS+, Pe.S+, BK - Chelation	6/6 P	6/6 P
21	Swapna 948556	12/F	P,R,W,DV - 5 d	6/6	6/9 P	-	C+, F+	-	-	-	LE AU	Myd +, TS+	6/6	6/6
22	Vijay 951324	6/M	P,R,W,Tr, DV, RE - 2d	6/12	6/6	C+, F+, KP+	-	-	-	-	RE AU (Tr)	Myd +, TS+	6/6	6/6
23	Ramesh 952999	8/M	P, Ph, R, W, DV, BE - 10wk	6/12	6/12	C+, F+, KP+	C+, F+, KP+, PS	-	-	TC, DC, ESR, Ch-C, ENT - C	BE AU	Myd +, TS+ Anb	6/6	6/6 P
24	Ajit 954001	12/M	P, R,W,RE - 3d	6/9 P	6/6	C+, F+	-	-	-	-	RE AU	Myd +, TS+	6/6	6/6
25	Kamlesh 954098	9/M	DV, Fl, P, BE-3m	6/60	4/60	-	-	Ch, VH, VC	CH, VH, VC	TC, DC, ESR, Mx, CxR, ELISA	BE PoU (Toxo)	Myd +, TS+, Anb	6/12	6/12
26	Mark 955896	15/M	FL, W, R, BE-8wk	6/12	6/24	F+	F+	VC, VF, SB	VC, VF, SB	TC, DC, ESR, Mx, CxR, VDRL, ELISA	BE IU	Myd +, TS+, Pe.S	6/6 P	6/9
27	Ranjit 957563	13/M	P, Ph, R, W, LE - 3 d	6/6	6/12	-	F+, C+, KP+	-	-	-	LE AU	Myd +, TS+	6/6	6/6
28	Priyakumari 958925	6/F	P,Ph,W,R,RE - 5d	6/9 P	6/6	F+, KP+, C+	-	-	-	-	RE AU	Myd +, TS+	6/6	6/6

29	Elizabeth 959320	8/F	W,Ph,DV,BE - 8 wk	6/12	6/12	F+, KP+, C+	F+, C+, KP+	-	-	TC, DC, ESR, Mx, CxR, ENT - C, Ch-C	BE AU	Myd +, TS, Anb	6/6 P	6/6 P
30	Priyanka 959699	14/F	P,W,R,LE - 3 wk	6/6	6/9	-	F+, C+	-	-	-	LE AU	Myd +, TS	6/6	6/6
31	Iyshwarya 961062	12/F	W,R,DV, BE - 6m (On / Off)	6/36	4/60	F+, KP+, C+	F+, KP+,C+, CC+	-	-	TC, DC, ESR, ANA, CRP, RAF, Rh-C	BE AU (Jra)	Myd+, TS+, ECCE+PCIOL	6/12	6/18
32	Sivamuthu 962488	8/M	P, Ph, W, R, LE - 2 d	6/6	6/6P	-	F+, C+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
33	Nandhakrishnan 963967	8/M	DV, Fl, BE - 5 yr (Divergent LE)	3/60	2/60	-	-	Ch, VFl,	Ch	TC, DC, ESR, Mx, ELISA	BE PoU (Toxo)	Observation	3/60	2/60
34	Marimuthu 965223	13/M	P, Ph, W, R, BE - 12 wk (on / off)	6/12	6/12	F+, KP+, C+, PS+	F+, KP+, C+	-	-	TC, DC, ESR, Mx, Ch-C	BE AU	Myd+, TS+, ATT+	6/6	6/6
35	Chinnappa 967474	11/M	P, Ph, W, R, LE - 2 wk	6/6	6/6P	-	F+, KP+, C+	-	-	TC, DC, ESR, Mx, Ch-C, Rh - C	LE AU	Myd+, TS+	6/6	6/6
36	Jannet 968206	8/F	P, Ph, W, R, RE - 1 wk	6/9	6/6	F+, C+,	-	-	-	-	RE AU	Myd+, TS+	6/6	6/6
37	Shaik 968351	9/M	B, BE - 6 yr (Divergent RE)	1/60	6/36	-	-	Ch	Ch	-	BE PoU	Followup	1/60	6/36
38	Sarvanan 968690	12/M	P, Ph, W, R, Re - 5 d	6/9	6/6	F+, C+,	-	-	-	-	RE AU	Myd+, TS+	6/6	6/6
39	Vyashali 971206	7/F	P, Ph, W, R, LE - 3 d (Tr)	6/6	6/12	-	F+, C+, KP+	-	-	-	RE AU	Myd+, TS+	6/6	6/6
40	Esther 973002	13/F	P, Ph, W, R, RE - 1 wk	6/9	6/6	F+, C+	-	-	-	-	RE AU	Myd+, TS+	6/6	6/6
41	Dinaker 976341	14/M	P, Ph, W, R, LE - 1 wk	6/6	6/9P	-	F+, C+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
42	Rajalakshmi 977742	10/F	W, Ph, DV, RE - 5 m	6/24	6/9	F+	-	VC, VF, SB	-	TC, DC, ESR, Mx, CxR, ELISA, ANA	RE IU	Myd+, TS+, PeS	6/9	6/9
43	Simon 978165	12/M	P, W, DV, Fl, BE - 3 m	3/60	5/60	-	F+	Ch, VC, VF	Ch, VC, VF	TC, DC, ESR, MX, CxR, ELISA	BE PoU (Toxo)	Myd+, TS+, Anb	6/18	6/18

44	Rajendran 978444	7/M	P, Ph, W, R, RE - 1 wk (Tr)	6/18	6/6	F+, C+, KP (Foreign body)	-	-	-	B-Scan, TC, DC, ESR, Hb%	RE AU (Tr) (FB)	Surgical removal, TS, Anb	6/6	6/6
45	Seethalakshmi 979090	14/F	P, W, R, RE - 3 d	6/6P	6/6	F+, C+	-	-	-	-	RE AU	Myd+, TS+	6/6	6/6
46	Viramuthu 980844	12/M	P, Ph, W, R, DV, LE - 1 wk	6/6	6/9P	-	F+, C+, KP+	-	-	-	LE AU	Myd+, TS+	6/6	6/9
47	Shakeela 981243	17/F	P, Ph, W, R, DV, RE - 3d	½/60	6/9P	F+, C+, KP+, Hypopyon	-	Yellow reflex VC, VF	-	TC, DC, ESR, Bld. Culture, Hb%, LP	RE PaU	Anb, IV - Ab	6/24	6/9
48	Saravanan 983384	16/M	P, Ph, W, R, DV, LE - 2 d (Tr)	6/6	PL+, PR+	-	F+, C+, KP+ Hypopyon	-	Yellow reflex VC, VF	Hb%, Bld. Culture, B-scan	RE PaU	PPV, Anb, FB removal	6/6	6/24
49	Murugesh 983640	11/M	W, PH, DV, BE - 3 m	6/18	6/12	F+, C+, KP+, PS+	F+, C+, KP+	-	-	TC, DC, ESR, Mx, ANA, RF, Rh-C	BE AU	Myd+, TS+, PeS	6/9	6/6P
50	Alexander 985000	14/M	P, R, Ph, DV, BE - 10 wk	6/36	6/36	F+, C+, KP+, Ulcer	F+, C+, KP+, Ulcer	-	-	TC, DC, ESR	BE AU	Myd+, antifungal	6/18	6/12
51	Ranjeetha 987212	13/F	W, DV, R, BE - 12 wk	4/60	6/60	F+, C+, PS+, CC+	F+, C+, PS+, CC+	-	-	TC, DC, ESR, RF, ANA, CRP, Rh-C	BE AU	Myd+, TS+, ECCE+PCIOL	6/9	6/12
52	Nalini 988013	16/F	Ph, R, W, DV, P, BE - 12 wk (on / off)	6/12	6/18	C+, KP+	C+, KP+, F+, PS+	-	-	TC, DC, ESR, Mx, Ch-C, CxR	BE AU	Myd+, TS+, PeS, ATT	6/6	6/6
53	Mary 989300	13/F	P, R, W, RE - 5 d	6/6P	6/6	C+, F+,	-	-	-	-	RE AU	Myd+, TS+	6/6	6/6
54	Varadharajan 990013	9/M	DV, BE - 5 yr	3/60	4/60	-	-	Ch	Ch	TC, DC, ESR, Mx, CxR, VDRL, ELISA	BE PoU (Toxo)	Observation	3/60	4/60
55	Karthick 990740	6/M	DV, Fl, P, BE - 4 m	6/60	1/60	F+	F+	Ch, VC, VF	Ch, VC, VF	TC, DC, ESR, Mx, CxR, VDRL, ELISA	BE PoU (Toxo)	Myd+, TS+, Anb	6/12	6/24
56	Charan 993655	11/M	P, R, W, Ph, BE - 8 wk	6/12	6/24	C+, F+, KP+	C+, F+, KP+, PS+	-	-	TC, DC, ESR, De-C, Ch-C	BE AU	Myd+, TS+, Den. Ext.	6/9	6/6
57	Jawahar 995409	8/M	P, R, W, Ph, LE (Corneal Ulcer) - 1 wk	6/6	HM +	-	C+, F+, K:+, Hypopyon	-	VC, VF, Yellow Reflex	TC, DC, ESR, Hb%, Vitreous Culture	LE PaU	Myd+, Anb, PPV	6/6	6/18P

58	Arun 997251	9/M	P, R, W, Ph, DV, BE - 12wk	6/60	4/60	C+, F+, KP+	C+, F+, KP+	Ch, VC	Ch, VC	TC, DC, ESR, Bld. Culture, CxR	BE PaU	Myd+, TS+, ATT+	6/18	6/24
59	Ramesh 997894	16/M	P, R, W, Ph, RE - 5 d (Tr)	6/9P	6/9	C+, F+, KP+	-	-	-	-	RE AU	Myd+, TS+	6/6	6/9
60	Vanaja 998163	12/F	P, R, W, LE - 1 wk	6/6	6/9	-	C+, F+, KP+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
61	Jeniffer 998579	14/F	P, R, W, Re - 2 d (Tr)	6/6	6/12	-	C+, F+, KP+	-	-	-	RE AU	Myd+, TS+	6/6	6/6
62	Kokila 999002	14/F	P, R, W, DV, LE - 1 wk	6/6	6/9P	-	C+, F+, KP+	-	-	-	LE AU	Myd+, TS+	6/6	6/9
63	Manoj 999541	8/M	P, R, W, Ph, BE - 2 wk	6/9	6/9P	C+, F+, KP+	C+, F+, KP+	-	-	TC, DC, ESR, Mx, Ch-C, De-C	BE AU	Myd+, TS+, ATT+	6/6	6/6P
64	Nalini 999832	4/F	P, DV, Fl, BE -	6/60 (PC)	6/60 (PC)	-	-	Ch, VF, VC	Ch, VF, VC	TC, DC, ESR, CxR, VDRL, ELISA	BE PoU (Toxo)	Myd+, TS+, Anb	6/36	6/24
65	Aparna 100048	13/F	Fl, DV, R, BE - 12 wk	6/36	6/36	F+	F+	VF, SB	VF, VC	TC, DC, ESR, De-C	BE IU	Myd+, TS+, PeS, Den. Ext.	6/18	6/12
66	Micheal 100181	17/M	P, R, Ph, W, DV, BE - 2 wk	6/18	6/12	C+, F+, KP+, Ulcer	F+, C+, KP+, Ulcer	-	-	TC, DC, ESR, Hb%	BE AU	Myd+, antiviral	6/9	6/6P
67	Santhosh 101088	3/M	BE (Leukocoria), Ph, W - 2wk	Not elicited		-	-	VC, VF, Ch	Ch (old)	TC, DC, ESR, Torch, CxR	BE PoU (Tox)	Myd+, TS+, Anb, PPV	-	-
68	Vinetha 103856	12/F	W, P, R, Ph, DV, BE - 10wk	6/60	2/60	C+, F+, KP+	C+, F+, KP+	VC, Ch	VC, Ch	TC, DC, ESR, Mx, CxR	BE PaU	ATT+, Myd+, TS+, PeS	6/36	6/36
69	Shakeel 104654	10/M	P, R, Ph, LE - 1 wk	6/6	6/9	-	C+, F+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
70	Sunaja 105890	12/F	P, R, LE - 3 d	6/6	6/6P	-	C+, F+	-	-	-	RE AU	Myd+, TS+	6/6	6/6
71	Tamilarasi 106932	6/F	P, R, W, RE - 3d	6/6P	6/6	-	C+, F+	-	-	-	RE AU	Myd+, TS+	6/6	6/6
72	Masillamani 107101	8/M	Fl, DV, W, BE - 6m	6/60	6/60	-	F+	VC, VF, Ch	VC, VF, Ch	TC, DC, ESR, Mx, VDRL, CxR, ELISA	BE PoU (Toxo)	Myd+, TS+, Anb	6/9	6/18

73	Narayanan 108807	7/M	Fl, W, RE - 6 m	6/36P	6/9	-	-	VC, VF, SB	-	TC, DC, ESR, Mx, CRP, ANA, CxR	RE IU	Myd+, TS+, PeS	6/12	6/9
74	Mani 109460	16/M	W, DV, Ph, BE - 6m (on / off)	6/18	2/60	C+, F+, KP+, PS+	C+, F+, KP+, PS+, CC+	-	-	TC, DC, ESR, CRP, ANA, RF, Rh-C	BE AU	Myd+, TS+, PP lensectomy	6/6P	6/12
75	Harini 109681	16/F	DV, Fl, LE (Divergent RE) - 3 m	1/60	6/36	-	-	Ch	Ch, VC, VF	TC, DC, ESR, CxR, VDRL, ELISA	BE PoU	Myd+, TS+, Anb	1/60	6/9
76	Sankaran 110068	12/M	R, W, DV, BE - 3 m	6/12	6/9P	C+, F+, KP+	C+, F+, KP+	-	-	TC, DC, ESR, RF,ANA, Rh-C	BE AU	Myd+, TS+, SS+, PeS	6/6P	6/6
77	Ashok 111640	13/M	P, R, Ph, LE - 1 wk	6/6	6/9	-	C+, F+, KP+	-	-	TC, DC, ESR, Mx, CxR	LE AU	Myd+, TS+	6/6	6/6
78	Ravindran 113547	9/M	Fl, DV, P, LE - 3 m	6/6	6/24	-	F+	-	VC, VF, SB	TC, DC, ESR, Mx, CxR, CRP, ANA	LE IU	Myd+, TS+ PeS+	6/6	6/9P
79	Manikandan 116554	16/M	P, R, W, Ph, RE - 3 d	6/9	6/6	C+, F+, KP+	-	-	-	-	RE AU	Myd+, TS+	6/6	6/6
80	Josephine 116668	13/F	W, Ph, DV, BE - 6m	6/24	3/60	C+, F+, KP+, BK	C+, F+, KP+, CC+	-	-	TC, DC, ESR, RF, ANA, CRP, Rh-C	BE AU	Myd+, TS+, PeS+, PP lensectomy BK	6/9	6/9P
81	Chitra 117321	12/F	W, DV, BE - 1 yr	6/18	6/18P	C+, F+, PS+	C+, F+, PS+	-	-	RF, ANA, CRP, Rh-C, x-ray (joints)	BE AU	Myd+, TS+, PeS+	6/9	6/9
82	Vandhana 118567	12/F	W, DV, Fl, BE - 6 m	6/60	4/60	F+	-	VC, VF, Ch	VC, VF, Ch	TC, DC, ESR, Mx, CxR, Ch-C	BE PoU (Tub)	Myd+, TS+, PeS+, ATT	6/12	6/18
83	Suresh 118568	15/M	W, DV, Fl, P, R, BE - 6 m	3/60	2/60	F+, C+, KP+	F+, C+, KP+	VC, VF, Ch	VC, VF, Ch	ESR, ELISA, CxR	BE PaU	Myd+, TS+, Anb	6/24	6/24
84	Sanjana 119463	13/F	W, DV, Ph, BE - 4 m	6/24	6/9P	C+, F+, KP+, PS+	C+, F+, KP+, PS+	-	-	RF, ANA, CRP, Rh-C, x-ray (joints)	BE AU	Myd+, TS+, PeS+, BK Chelation	6/6	6/6
85	Radhika 119674	8/F	P, Ph, R, LE - 1wk	6/6	6/6P	-	C+, F+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
86	Ravi 119899	3/M	Fever, SCH, Hepatomegaly - 1 wk	-	-	-	-	VC, VF, Hge (vasculitis)	VC, VF, Hge (vasculitis)	MSAT, Leptospira +ve	BE PoU	Penicillin, Supportive Mgt.	-	-
87	Kalavathy 120846	14/F	DV, Fl, LE - 3 m	6/9	6/60	-	-	-	VC, VF, Ch	TC, DC, ESR, Mx, CxR, Ch-C	LE PoU (Tub)	Myd+, TS+, ATT+, PeS+	6/9	6/12

88	Naresh 123481	12/M	P, R, W, Ph, RE - 1 wk	6/9	6/6	C+, F+, KP+	-	-	-	TC, DC, ESR	RE AU	Myd+, TS+	6/6	6/6
89	Vidhya 125674	9/F	P, R, Ph, LE- 1 wk (Tr)	6/6	6/12	-	C+, F+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
90	Narashimman 126677	8/M	Fl, DV, BE - 4m	6/60	6/60	-	-	VC, VF, New vessels, Sheathing		TC, DC, ESR, Mx, CxR	BE PoU (Eales)	PeS+, TS+, Myd+, PRP	6/18	6/18
91	Sakthi 128003	14/F	W, Ph, DV, BE - 8 m	6/36	6/24	C+, F+, KP+, CC+	C+, F+, KP+, BK	-	-	RF, ANA, CRP, Rh-C, x-ray (joints)	BE AU	Myd+, TS+, PeS+, SICS + PCIOL, BK chelation	6/9	6/9
92	Saradha 129463	13/F	W, DV, BE - 3 m	6/18	6/24	C+, F+, KP+	C+, F+, KP+, PS+	-	-	RF, ANA, CRP, Rh-C, x-ray (joints)	BE AU	Myd+, TS+, PeS +	6/6P	6/9
93	Rajesh 130341	7/M	W, Ph, R, P, LE - 2 d (Tr)	6/6	6/9P	-	C+, F+, KP+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
94	Babu 132885	7/M	Ph, Fl, DV, BE - 3m	6/60	4/60	-	-	VC, VF, Ch	VC, VF, Ch	TC, DC, ESR, Mx, CxR	BE PoU (Tub)	Myd+, TS+, ATT+	6/18	6/24
95	Krishnaveni 1334636	17/F	Fl, DV, BE - 12 wk	5/60	4/60	-	-	VC, VF (Vasculitis)	VC, VF (Vasculitis)	ANA, ESR, CRP, ANCA	BE PoU (SLE)	SS+, TS+, Antimetabolites	6/60	6/36
96	Harikrishnan 138981	9/M	P, R, W, DV, LE - 8 wk	6/9	6/60	-	C+, F+, KP+	-	VC, VF, Ch	TC, DC, ESR, Mx, CxR	LE PaU	ATT+, TS+, Myd+	6/9	6/24
97	Ahamed 141643	8/M	W, Ph, R, P, BE - 2 wk	6/18	6/12	C+, F+, KP+	C+, F+, KP+	-	-	TC, DC, ESR, Mx, CxR, Ch-C	BE AU	ATT+, TS+, Myd+	6/9	6/6
98	Sureka 142432	6/F	P, R, Ph, LE - 1 wk	6/6	6/9	-	C+, F+	-	-	-	LE AU	Myd+, TS+	6/6	6/6
99	Ajay 143556	13/M	Fl, DV, LE - 6 wk	6/12	6/18	-	-	-	Ch, VC, VF	TC, DC, ESR, Mx, CxR	LE PoU (Tub)	ATT+, TS+, Myd+	6/9	6/12
100	Sheeba 143841	9/F	DV, Fl, P, LE (Immunosuppressed)	6/9P	2/60	-	-	-	Vasculitis VC, VF, Hge	Diagnosed as CMV Retinitis	LE PoU	Sys. Antiviral	6/9	6/60

0-4 years	5-9 years	10-14 years	15-18 years
5%	35%	44%	16%

Idiopathic	Allergic	Arthropathy	Traumatic	Infective
31	10	13	8	3

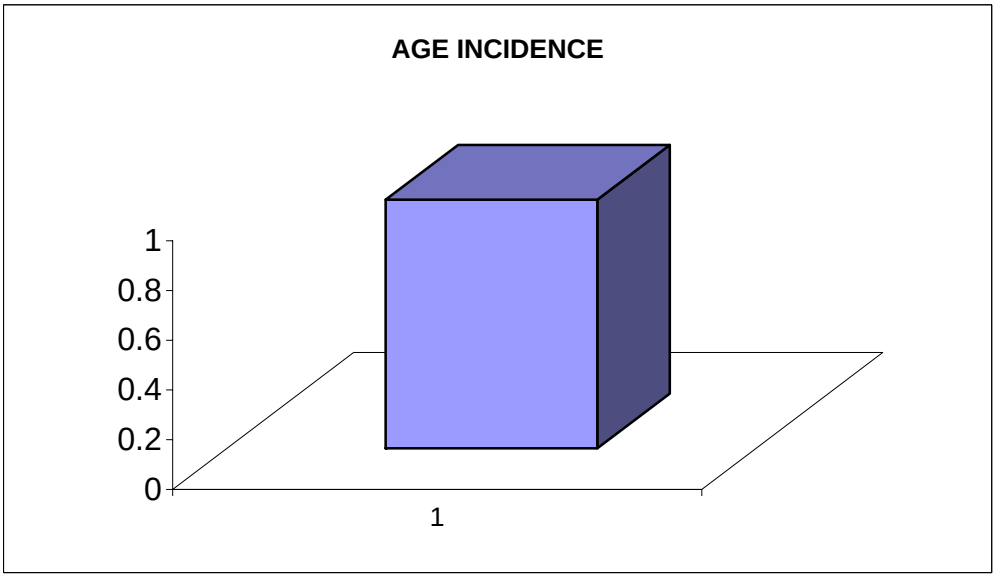
Tuberculos	Toxoplasm	Toxocara	Others
4	12	1	5

Anterior	Intermediat	Posterior	Pan Uveitis
65	6	21	8

	0-4 years	5-9 years	10-14 years	15-18 years
Anterior Uv	1	19	36	9
Intermediat	0	2	2	2
Posterior U	3	11	5	2
Pan Uveitis	1	3	1	3

Band Kerat	Complicate	Divergent E	Exudative I	Phthisical eye
3	5	4	2	1

	Idiopathic	Infective	Specific Inf	Septic Foci
Pan Uveitis	0	4	4	0
Intermediat	5	0	0	1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0.4
0.2
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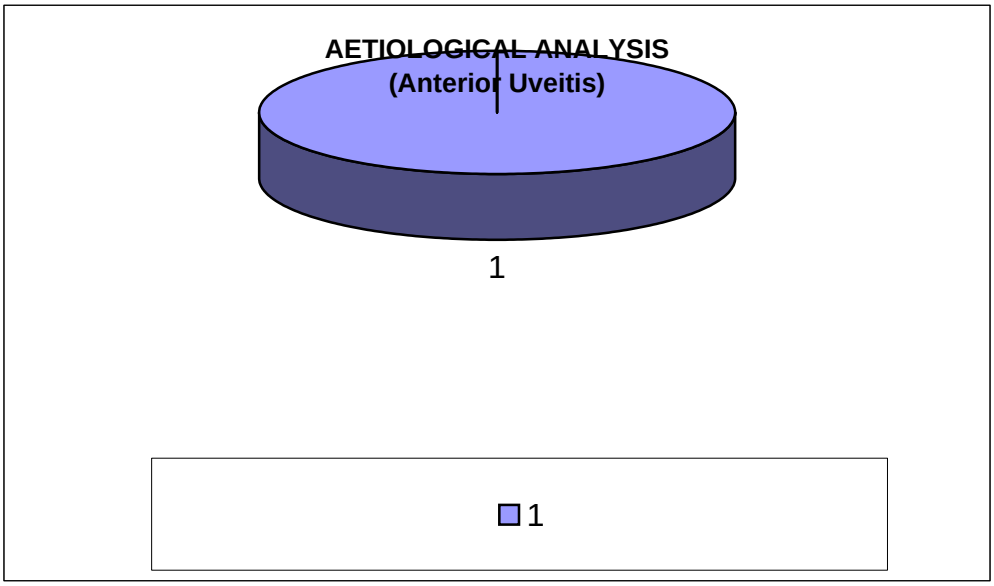
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(Anterior Uveitis)
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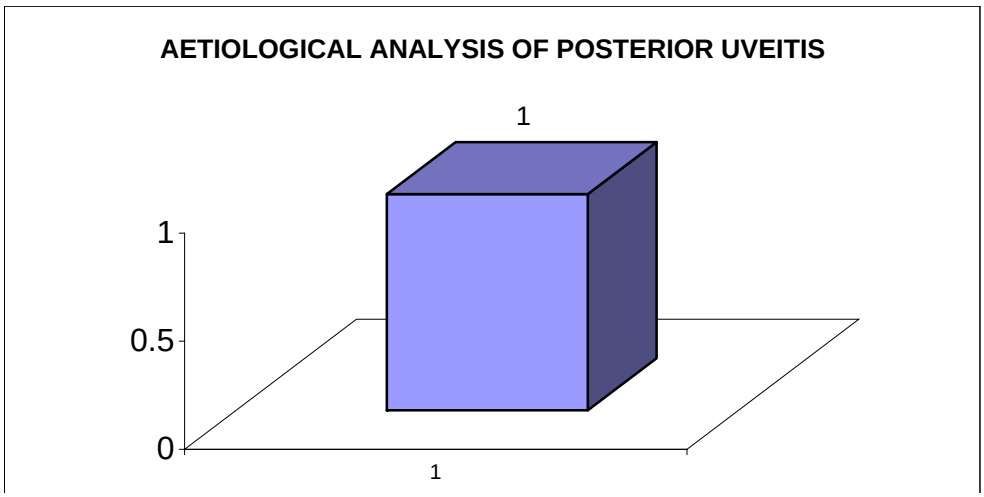
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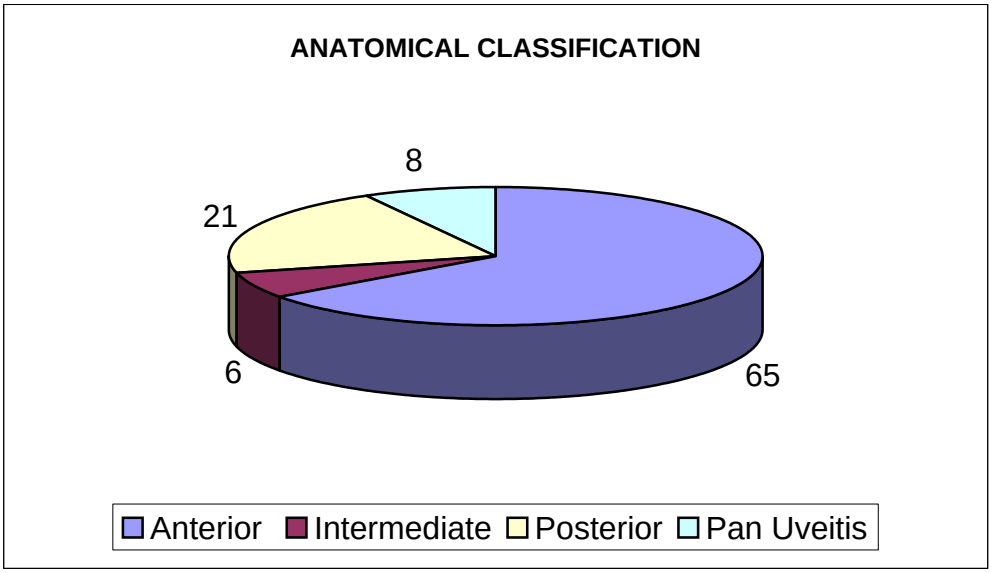
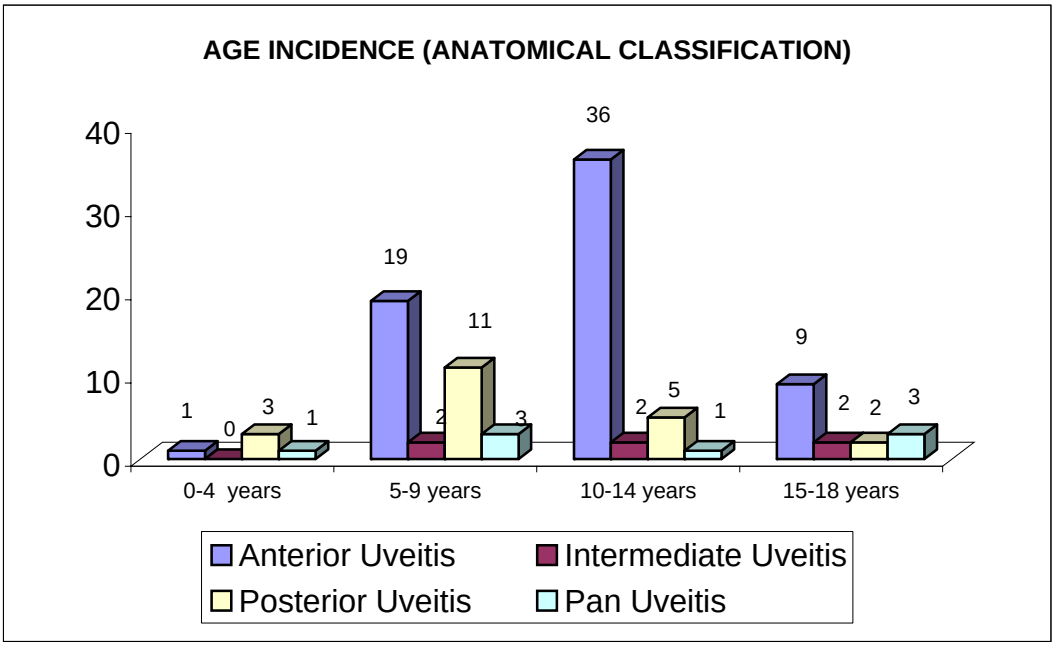
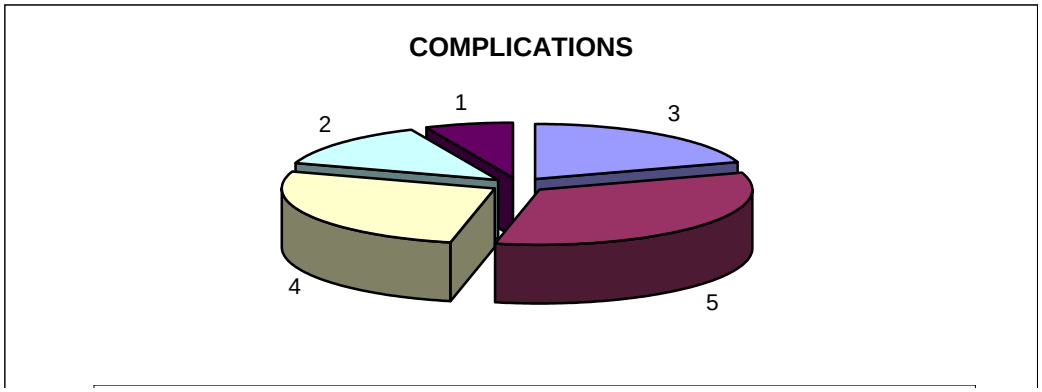

ANATOMICAL CLASSIFICATION
8
21
6
65
Anterior
Intermediate
Posterior
Pan Uveitis


AGE INCIDENCE (ANATOMICAL CLASSIFICATION)
40
30
20
10
0
1
0
3
1
19
2
11
3
36
2
5
1
9
2
2
3
0-4 years
5-9 years
10-14 years
15-18 years
Anterior Uveitis
Intermediate Uveitis
Posterior Uveitis
Pan Uveitis


COMPLICATIONS
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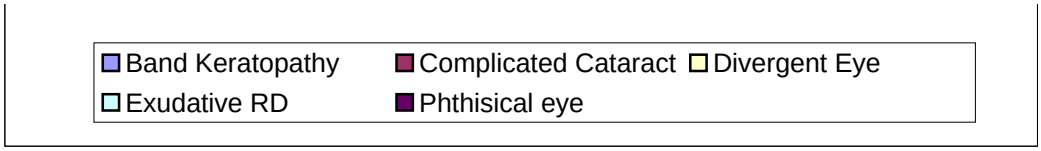
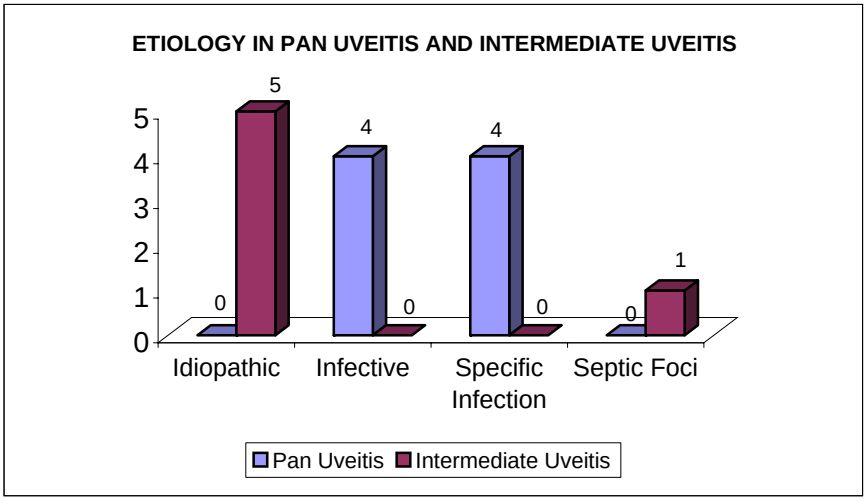
2
4
5


Band Keratopathy
Complicated Cataract
Divergent Eye
Exudative RD
Phthisical eye


ETIOLOGY IN PAN UVEITIS AND INTERMEDIATE UVEITIS
5
4
3
2
1
0
0
5
4
0
4
0
0
1
Idiopathic
Infective
Specific Infection
Septic Foci
Pan Uveitis
Intermediate Uveitis