A CLINICAL STUDY ON VISUAL OUTCOME FOLLOWING UVEITIS

Dissertation submitted to

THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the

regulations for the award of the degree of

M.S.OPHTHALMOLOGY

BRANCH – III

Thanjavur Medical College and Hospital

The Tamilnadu Dr. M.G.R Medical University

Chennai,India

April 2016

CERTIFICATE

This is to certify that this Dissertation entitled "**A CLINICAL STUDY ON VISUAL OUTCOME FOLLOWING UVEITIS**" is a bonafide original work done by Dr.T.MYTHILI, under my guidance and supervision in the Department of Ophthalmology, Thanjavur Medical College, Thanjavur doing her Postgraduate course from 2013 -2016.

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DECLARATION

I Dr.T.MYTHILI solemnly declare that this Dissertation "**A CLINICAL STUDY ON VISUAL OUTCOME FOLLOWING UVEITIS**" is a bonafide record of work done by me in the Department of Ophthalmology, Thanjavur Medical College, and Hospital , Thanjavur under the Guidance and Supervision of my Professor Dr.J.GNANASELVAN M.S.,D.O.,the Head of the department, Department of Ophthalmology, Thanjavur Medical College, Thanjavur between 2013 and 2016.

This Dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University , Chennai in partial fulfilment of University regulations for the award of M.S Degree (Branch – III) in Ophthalmology to be held in April 2016.

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A CLINICAL STUDY ON VISUAL OUTCOME FOLLOWING UVEITIS

submitted by Dr. T. MYTHILI **of**

Dept. of OPHTHALMOLOGY **Thanjavur Medical College, Thanjavur**

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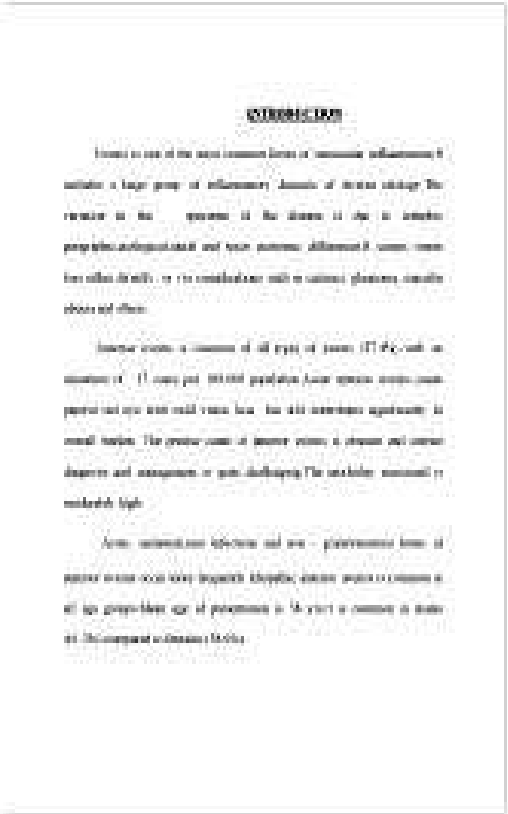

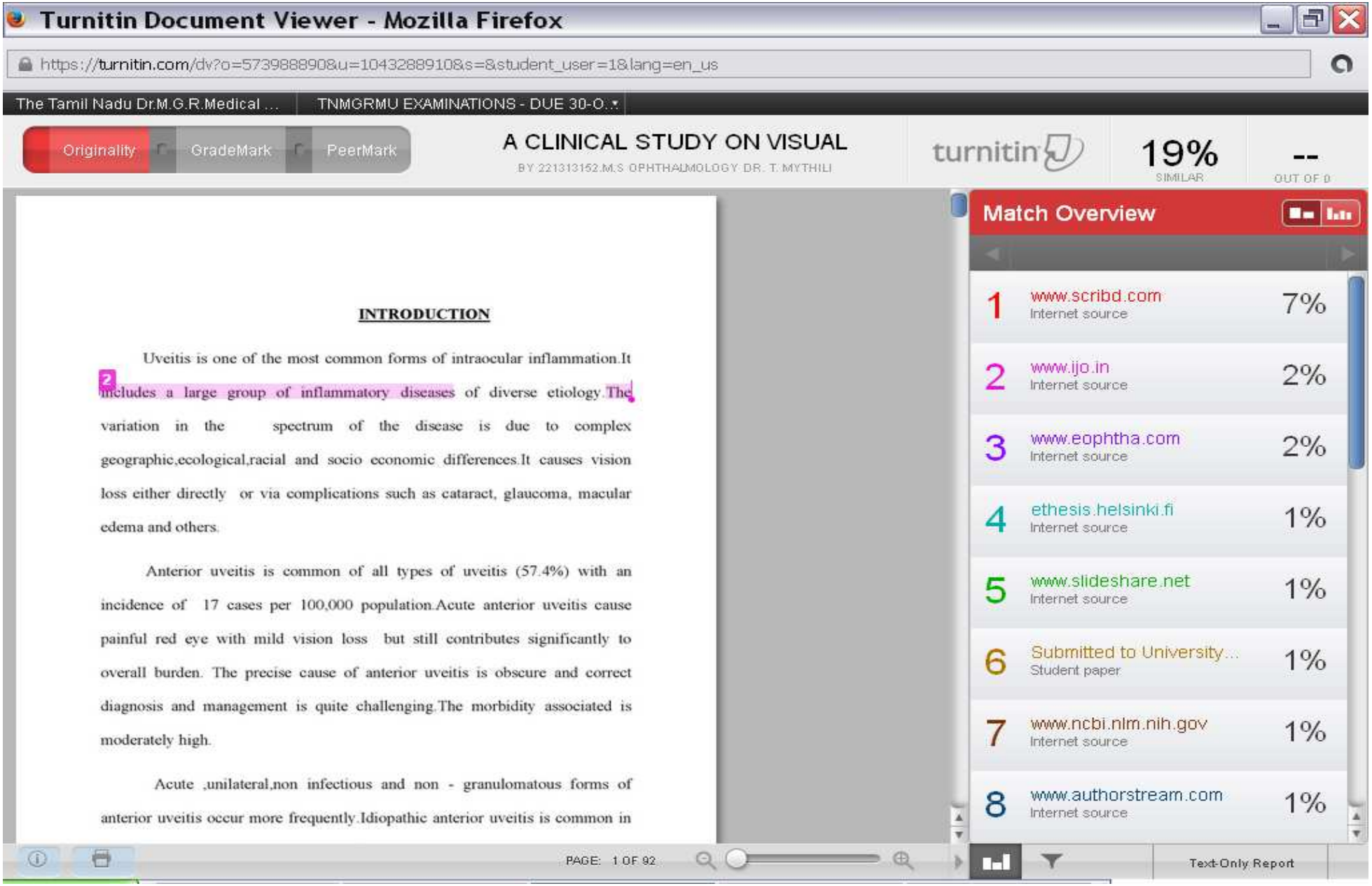
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INTRODUCTION
Uveitis is one of the most common forms of intraocular inflammation.It includes a large group of inflammatory diseases of diverse etiology.The variation in the spectrum of the disease is due to complex geographic,ecological,racial and socio economic differences.It causes vision loss either directly or via complications such as cataract, glaucoma, macular edema and others.
Anterior uveitis is common of all types of uveitis (57.4%) with an incidence of 17 cases per 100,000 population.Acute anterior uveitis cause painful red eye with mild vision loss but still contributes significantly to overall burden. The precise cause of anterior uveitis is obscure and correct diagnosis and management is quite challenging.The morbidity associated is moderately high.
Acute ,unilateral,non infectious and non - granulomatous forms of anterior uveitis occur more frequently.Idiopathic anterior uveitis is common in
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ACKNOWLEDGEMENT

I thank the All Mighty God for the abundant grace & Blessings and making me successful in every field to complete the Dissertation work.

I would like to thank the Dean, Thanjavur Medical College , Thanjavur for granting me permission to conduct this study at Thanjavur Medical College , Thanjavur.

I express my sincere gratitude to my Professor & HOD Dr.J.GNANASELVAN M.S.,D.O., for his motivation and meticulous guidance throughout the period of study.

I am extremely thankful to my teachers Dr.R.Raja, Dr.S.Amudhavadivu, Dr.V.Thaialnayaki, Dr.T.Anbuselvi and Dr.K.Rajasekaran for their encouragement and valuable support throughout the period of study.

I would like to thank the Professor & HOD , Department of Biochemistry for granting permission to use the Laboratory.

I acknowledge the support from all my postgraduate colleagues and friends in completing the study..

I thank all the children and Parents for their active participation and co-operation in conducting the study.

Finally I thank all my family members for their encouragement and moral support towards the successful completion of my Dissertation work.

CONTENTS

INTRODUCTION

Uveitis is one of the most common forms of intraocular inflammation. It includes a large group of inflammatory diseases of diverse etiology. The variation in the spectrum of the disease is due to complex geographic, ecological, racial and socio economic differences [2].It causes vision loss either directly or via complications such as cataract, glaucoma, macular edema and others.

Anterior uveitis is common of all types of uveitis (57.4%) with an incidence of 17 cases per 100,000 population. Acute anterior uveitis cause painful red eye with mild vision loss [12] but still contributes significantly to overall burden. The precise cause of anterior uveitis is obscure and correct diagnosis and management is quite challenging. The morbidity associated is moderately high.

Acute, unilateral, non infectious and non-granulomatous forms of anterior uveitis occur more frequently. Idiopathic anterior uveitis is common in all age groups. Mean age of presentation is 38 yrs. It is common in males (61.2%) compared to females (38.6%).

Herpetic anterior uveitis accounts (16.7%),lens induced uveitis (14.6%) and leprosy uveitis (4.9%) are common in elderly.Fuch’s heterochromic uveitis (9.8%),uveitis associated with spondyloarthropathy (8.2%) and traumatic uveitis(7.8%) are common in middle aged.

REVIEW OF LITERATURE

Acute anterior uveitis is the commonest of uveitis accounting for two-third of uveitis (Smith et al 1993.,Mccannel et al 1996).

Most of the uveitis literature is comprised of case series and case reports.These studies provide useful information but are highly prone for bias and lack a control population to compare the findings [14].Many forms of uveitis are rare ,therefore it is often difficult to recruit enough patients to fulfill the sample size.Patients with uveitis often have underlying systemic diseases making it difficult for patients to follow predetermined therapeutic protocol because their non ocular diseases often require systemic therapy.

Majority of patients with Acute Anterior Uveitis (AAU) have no obvious precipitating factor (Rosenbaum et al 1991).Over 50% of AAU patients have been reported to possess HLA – B27 antigen (Brewerton et al 1973b) [25].

Among patients with AAU microbes indicating infectious etiology are not often detected. Maitre jan described ocular tuberculosis in 1711,in a case presented with iris lesion eventually leading to perforation.

In 1830 Guenea de mussy first recognized choroidal tubercles.The microbes identified include gastrointestinal pathogens (Saari et al 1980),urogenital pathogen (Mattila et al 1982),respiratory pathogen(Saario and Toivanan et al 1993) .

The typical symptoms of acute presentation in anterior uveitis are pain, redness and photophobia. In severe cases patients complain of blurred vision(Nussenblatt et al 1996) [8].

Hypopyon is composed of leucocytes and it occurs occasionally in acute anterior uveitis(Nussenblatt et al 1996).

In rare cases hyphema may occur in anterior uveitis but usually resolves without permanent damage(Fong et al 1993) [22].

As the inflammation subsides,the IOP normalizes or may be increased due to synechia.But some patients are corticosteroid responders which may explain the reason for elevated intraocular pressure (Nussenblatt et al 1996) [21].

Cataract is one of the most common complications of chronic or recurrent uveitis.It occurs due to corticosteroid therapy however,may also occur due to imflammatory and cataractogenic cytokines(Hooper et al 1990).

Acute anterior uveitis may progress to chronic uveitis needing continous use of corticosteroid leading to glaucoma(BenEzra et al 1997) which is more sight threatening than the disease.It may result from blockage of trabeculum by debris,trabeculitis,persistent PAS,posterior synechia and rarely from neovascularistion of angle(Moorthy et al 1997).

In the absence of glaucoma,posterior synechia cause persistently small pupil filled with fibrin deposits (seclusio pupillae) markedly affecting the vision(Nussenblatt et al 1996) [21].

Cystoid macular edema occurs in iridocyclitis,which when left untreated results in lamellar hole and permanent decrease in central vision(Nussenblatt et at 1996).

Chronic hypotony leads to degenerative changes in the ocular tissues eventually leading to phthisis bulbi(Nusseblatt et al 1996).

Occasionally,periocular steroid injection is needed in severe AAU.In more severe case of AAU a brief course of oral steroids are the drug of choice.(Rosenbaum et al 1995). Non steroidal anti-inflammatory drugs have been used in severe forms of uveitis,considering the long term side effects with possible benefits.(Rosenbaum et al 1995).

ANATOMY OF UVEA

The eyeball comprises of three coats .The outer is fibrous coat - anterior $1/6^{th}$ is transparent cornea and posterior $5/6^{th}$ is opaque sclera , which protects the intraocular contents.The vascular coat is the uveal tissue.The nervous coat(retina) is concerned with visual functions.

The uveal tissue is divided into three parts from anterior to posterior, namely –

iris,
ciliary body and
choroid

.

IRIS –

Iris is anteriormost part of uvea. It is a thin circular disc with an average diameter of 12mm and thickness about 0.5mm.In its centre(slightly nasal) is an aperture 3 to 4mm in diameter called pupil.At the periphery,it is attached to anterior surface of ciliary body.It divides the anterior segment into anterior and posterior chambers.

Anterior surface of iris is divided into pupillary zone and ciliary zone by a circular ridge 1.5 mm away from pupillary margin called collarette.Pupillary zone extends from pupillary margin to collarette and is relatively flat.Pupillary margin is marked by a dark border known as pupillary ruff.Ciliary zone extends from collarette to root of iris.The depressions arranged in rows present in this area are known as crypts.Those crypts present near collarette are relatively larger, known as Fuchs's crypt and few crypts are seen in the periphery of iris.

Posterior surface of iris is more uniform and darker than its anterior surface and shows numerous radial contraction folds.

Microscopically, from anterior to posterior ,it has

* anterior limiting layer,

* iris stroma

*anterior and posterior (pigmented) epithelial layer.

The sphincter pupillae is a circular muscle,0.75 to 1 mm wide and 0.1 to 1.7 mm in thickness,considerably thicker than the dilator pupillae.

It is composed of spindle-shaped cells oriented parallel to pupillary margin and so its contraction causes miosis. The muscle is innervated by parasympathetic system.

The dilator pupillae lies in the stroma of ciliary zone of the iris. Because of the radial arrangement of the muscle fibres, its contraction causes mydriasis and is innervated by sympathetic system.

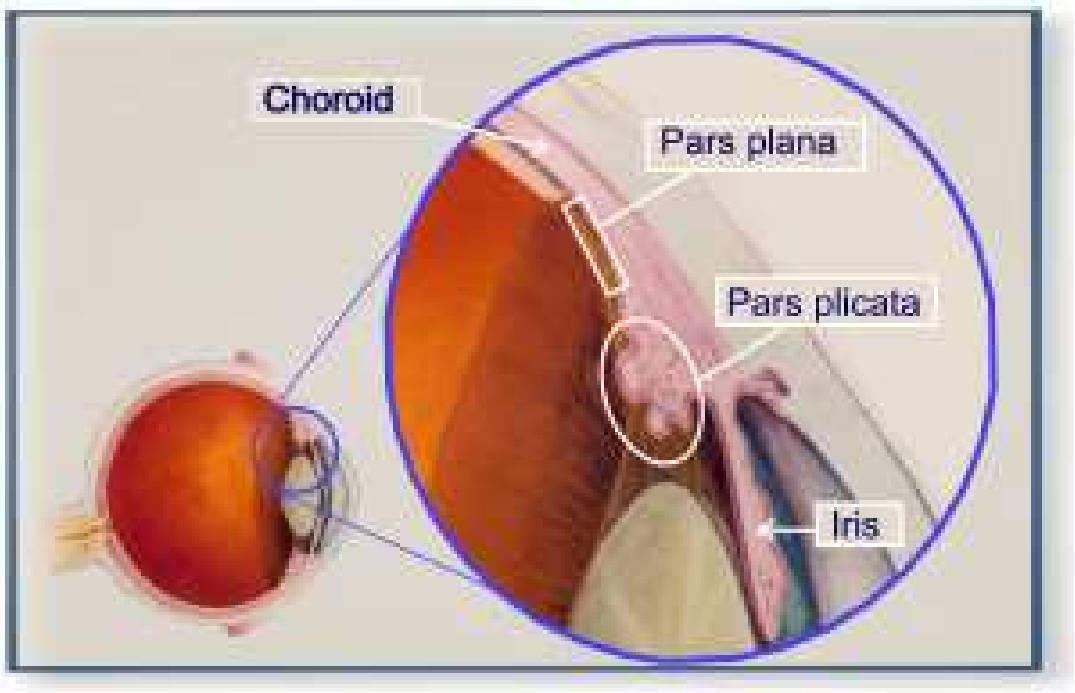


Figure i) Anatomy of uvea

CILIARY BODY – Ciliary body constitutes the middle part of the uveal tract .

It is a ring shaped structure projecting posteriorly from the scleral spur with a meridional width varying from 5.5 to 6.5 mm.

Anteriorly it is confluent with the iris root and bounds a part of the angle of anterior chamber. Posteriorly it has a crenated periphery - ora serrata, where it is continuous with the choroid and retina.It extends from about 1.5 mm posterior to the limbus to 6.5 to 7.5 mm on the temporal side and 6.5 mm on the nasal side.

Microscopically,it consists of five layers

* supraciliary lamina
* stroma,
* layer of pigmented and non-pigmented epithelium and
* internal limiting membrane.

The ciliary muscle is a non-striated muscle.Main action is to slacken the suspensory ligament of the lens and helps in accommodation.It is innervated by parasympathetic fibres.

CILIARY PROCESSES – Ciliary processes are white finger- like projections from the ciliary body (pars plicata).They are 70-80 in number and form the site of aqueous production.composed of three basic components-network of capillaries,stroma and two layers of epithelium.

CHOROID-

Choroid is the posterior most part of the uveal tract.It extends from ora serrata anteriorly to the optic disc posteriorly.Its rough outer surface is attached to sclera at the optic nerve and at the exit of vortex veins. Its smooth inner surface is attached to the pigment epithelium of the retina. It becomes continuous with the pia and arachnoid layers of the optic nerve.Choroid is 100-220 µm in thickness and is thickest at the macula (500- 1000 µm).

Microscopically ,from without inwards,the choroid consists of four layers

suprachoroidal lamina(lamina fusca),

stroma,

choriocapillaries and

basal lamina(Bruchs membrane or lamina vitrae).

BLOOD SUPPLY OF THE UVEAL TRACT-is by

SHORT POSTERIOR CILIARY ARTERIES: They arise from the ophthalmic artery as two trunks.It divides into 15 to 20 short posterior ciliary arteries , enter the sclera around the optic nerve and anastomose with other branches from the short posterior ciliary arteries to form the circle of Zinn, which run in suprachoroidal space and supply the choroid.

LONG POSTERIOR CILIARY ARTERIES: Two long posterior ciliary arteries enter the sclera- one lateral and one medial to the short ciliary arteries .It runs forward in the suprachoroidal space, enter the ciliary body and branch, anastomose with each other and with the anterior ciliary arteries to form the major arterial circle.

ANTERIOR CILIARY ARTERIES :These are 7 in number (2 each from arteries of superior, medial and inferior recti and only one from lateral rectus .

VEINS DRAINING THE UVEAL TRACT –

The veins draining uveal tract are anterior ciliary veins,smaller veins from the sclera and the venae verticosae(vortex veins or posterior ciliary veins).

PHYSIOLOGICAL CONSIDERATIONS

The uveal tissue performs the following physiological functions:

1. It is the source of blood flow to the ocular tissues .
2. It is the site of aqueous production and maintains the intraocular pressure.
3. It constitutes the blood aqueous barrier.
4. Musculature of the ciliary body plays a role in accommodation.
5. Eicosanoids are synthesized in the iris and ciliary body.
6. Uveal tissue plays a role in detoxification and antioxidation in the anterior segment.

UVEITIS

Uveitis is characterised by inflammation of the uveal tract **with** associated inflammation of the adjacent structures.

CLASSIFICATION OF UVEITIS:

i). ANATOMICAL CLASSIFICATION(SUN classification) [31]:

TYPE	SITE OF INFLAMMATION	INCLUDES
ANTERIOR UVEITIS	Anterior chamber	Iritis,iridocyclitis,anterior cyclitis
INTERMEDIATE UVEITIS	Vitreous	Pars planitis,posterior cyclitis,hyalitis
POSTERIOR UVEITIS	Choroid or Retina	Focal,multifocal or diffuse choroiditis, chorioretinitis, retinochoroiditis, retinitis,neuroretinitis
PANUVEITIS	Involves all segments of the eye without one predominating	

ii).CLINICAL CLASSIFICATION:

ACUTE UVEITIS	CHRONIC UVEITIS
1. Has a Sudden and symptomatic onset.	Has an insidious and asymptomatic onset.
2. lasts for about 6 weeks to 3 months.	lasts longer than 3 months to even years.

iii).PATHOLOGICAL CLASSIFICATION:

1.suppurative or purulent uveitis.
2.Non-suppurative uveitis. **a.Non-granulomatous uveitis** **b.Granulomatous uveitis**

iv).ETIOLOGICAL CLASSIFICATION:

1. Infective uveitis.
2. Allergic uveitis.
3. Toxic uveitis.
4. Traumatic uveitis.
5. Uveitis associated with non-infective systemic diseases .
6. Idiopathic uveitis.

SUN TERMINOLOGY FOR ACTIVITY OF UVEITIS

Although the ultimate goal of uveitis treatment is to suppress inflammation completely, it is also important to assess short term changes in inflammation, especially when evaluating the efficacy of treatment.

i)inactive disease	is defined as zero to rare anterior chamber cells(less than one cell per high power field).There was no consensus for definition of inactive vitritis on the basic of vitreous cells.

ii)**worsening activity**	-two-step increase in inflammation (anterior chamber cell or vitreous haze)or increase from grade 3+ to 4+.
iii)improved activity	-two-step decrease in inflammation (anterior chamber cell or vitreous haze) or decrease to grade 0.
iv)**remission**	In active disease for 3 months or more after discontinuing all treatments for eye disease.

PATHOLOGY OF UVEITIS

1.**Pathology of suppurative uveitis**:

Purulent inflammation of the uvea occur as a result of exogenous infection by pyogenic organisms which include *staphylococcus, streptococcus, pseudomonas, pneumococcus and gonococcus,*characterized by an outpouring of purulent exudate and infiltration by polymorphonuclear cells .So the whole uvea is thickened and necrotic and so the cavities of eye become filled with pus.

2.**Pathology of non-granulomatous uveitis**:

Non-granulomatous uveitis usually occur either due to a physical and toxic insult to the various tissue or because of hypersensitivity reactions. Iris become oedematous,muddy . Its mobility is reduced , and pupil become small due to sphincter irritation and engorgement of vessels of iris.Exudates and lymphocytes result in aqueous flare and fine KPs at the back of cornea.

Due to exudates,the posterior surface of the iris adheres to the anterior lens capsule leading to formation of posterior synechiae.The outpouring of exudate formed behind the lens is called cyclitic membrane .After healing it lead to changes like atrophy ,gliosis and fibrosis which cause scarring and eventually destruction of eye.

3.**Pathology of granulomatous uveitis:**

Granulomatous uveitis is a chronic response to anything which acts as an irritant foreign body.It can be inorganic or organic material,haemorrhage within the eye or due to certain specific organisms responsible for tuberculosis, leprosy, syphilis,etc

It is characterized by infiltration and proliferation of large mononuclear cells which eventually become epithelioid and giant cells and aggregate into nodules(koppe's and bussaca's nodules.).Leucocytes at the back of cornea are

in the form of mutton fat keratic precipitates . Aqueous flare is minimal. Necrosis leads to a reparative process resulting in fibrosis.

CLINICAL FEATURES OF UVEITIS:

SYMPTOMS OF UVEITIS:

1. **PAIN**	Dull aching or throbbing in nature and is usually referred (along the distribution of branches of the trigerminal nerve) especially to forehead and scalp.
2. **REDNESS**	Due to result of hyperaemia of anterior ciliary vessels because of the effect of toxins, histamine –like substances and axon reflex.
3. **PHOTOPHOBIA AND BLEPHAROSPASM**	Due to a reflex between sensory fibres of fifth nerve and motor fibres of the seventh nerve

4. **LACRIMATION**	occurs as a result of lacrimatory reflex mediated by fifth and the seventh nerve.
5. **DEFECTIVE VISION**	visual disturbance occur due to induced myopia , corneal haze, aqueous turbidity, pupillary block due to exudates, complicated cataract, vitreous haze, cyclitic membrane, associated macular oedema, papillitis or Secondary glaucoma. depending upon the severity and duration of the disease.

SIGNS OF UVEITIS:

I.**LID EDEMA** :

II.**CIRCUMCORNEAL CONGESTION:**

Is marked in acute iridocyclitis

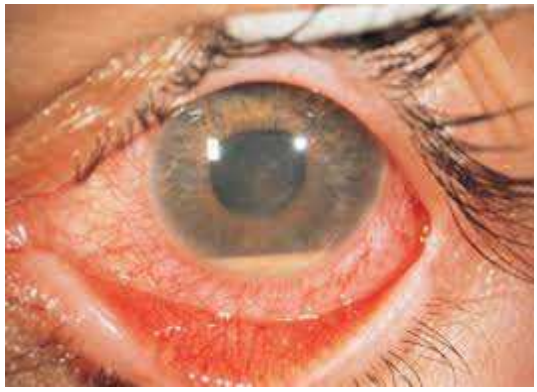


Figure ii) Circumcorneal congestion

III.**CORNEAL SIGNS** :

1. Corneal edema
2. Keratic precipitates
3. corneal opacity

Keratic precipitates (KPs) are proteinaceous-cellular deposits occurring at the back of cornea and are arranged in a triangular fashion at the centre and inferior part of the cornea known as Arlt's triangle.

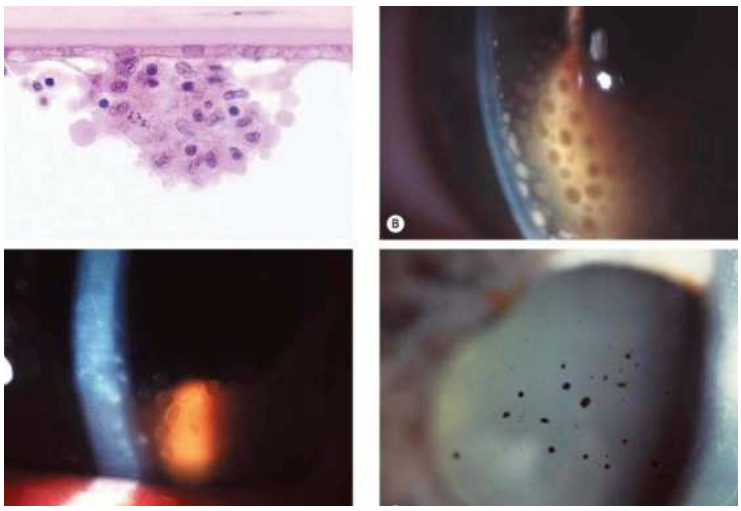


Figure iii) large mutton fat and old pigmented KPs

Types of KPs may be seen are,

i. **Mutton fat KPs**	They are large, thick, fluffy, lardaceous having a greasy or waxy appearance. usually a few in number.

ii. small and medium KPs (granular KPs)	pathognomonic of non –granulomatous uveitis These are small, discrete, dirty white arranged irregularly. may be hundreds in number and hence called endothelial dusting.
iii. Red KPs	These are formed when in addition to inflammatory cells,RBCs are present seen in haemorrhagic uveitis.
iv. Old KPs:	These are signs of healed uveitis They become pigmented and have crenated margins.

IV.**ANTERIOR CHAMBER SIGNS:**

1. **Aqueous cells**:

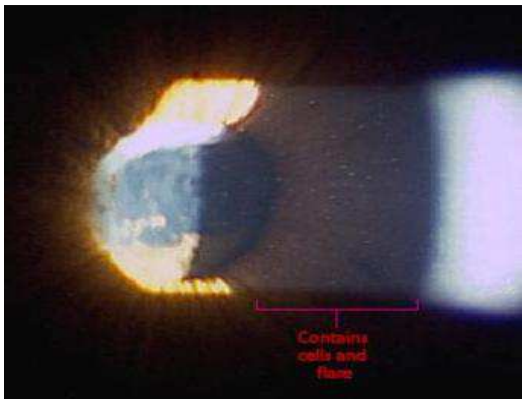


Figure iv)Anterior chamber cells

Grading of anterior chamber cells with slit lamp is as follows,

GRADE	**CELLS IN HIGH –POWER FIELD**
0	None
0.5+	1-5
1+	6-15
2+	16-25
3+	26-50
4+	>50

2.**Aqueous flare**

In the beam of light ,the protein particles are seen as suspended and moves like dust particles. This is based on the “Brownian movements” or “Tyndal phenomenon”.Flare is usually marked in non-granulomatous uveitis

Aqueous flare is graded as,

GRADE	**DESCRIPTION**
0	None
1+	Faint
2+	Moderate(iris and lens details clear)
3+	Marked(iris and lens details hazy)
4+	Intense(fibrin or plastic aqueous)

3.**Hypopyon**:The exudates settle down in dependant part of the anterior chamber as hypopyon.

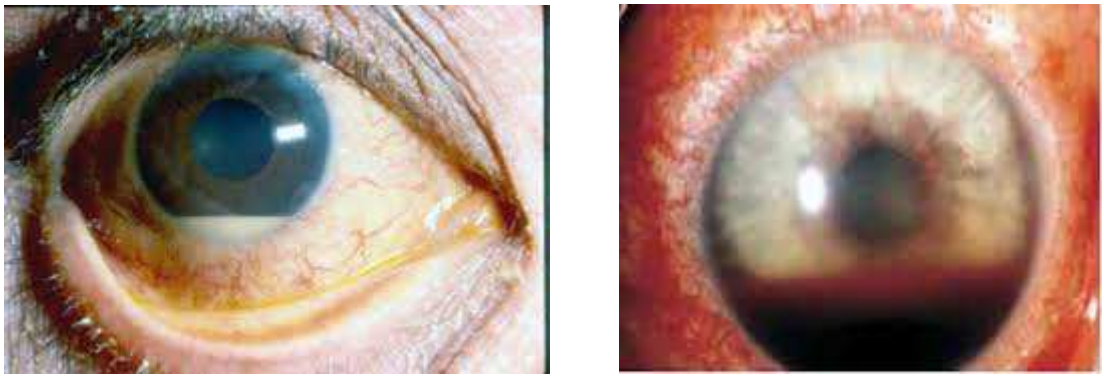


Figure v) a)Hypopyon and b)Hyphema

4.**Hyphaema :**It is the collection of blood in AC which may be seen in haemorrhagic type of uveitis.

5.Depth and shape of anterior chamber may change due to **synechiae** formation.

6. Anterior chamber angle is observed with gonioscopic for the presence of peripheral anterior synechiae .

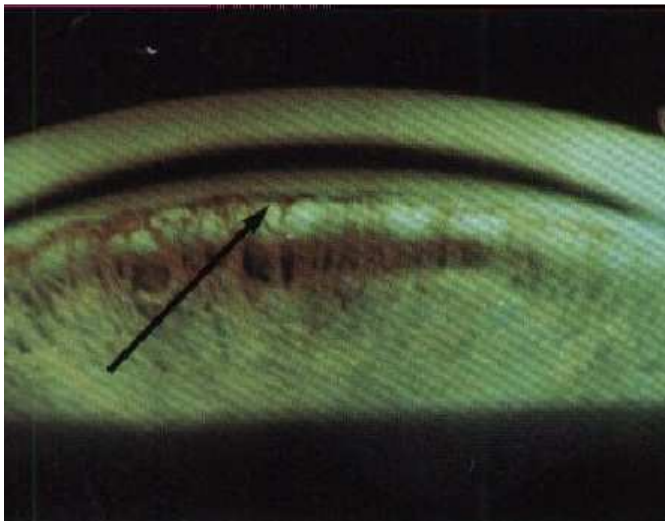


Figure vi)Gonioscope showing peripheral anterior synechiae

V.**IRIS SIGNS:**

1.**Changes in pattern**	occurs due to oedema of iris in active phase and due to atrophic changes in chronic phase.
2.**Changes in colour**:	Becomes muddy in colour during active phase and show either hyperpigmented and depigmented areas in healed stage.
3.**Iris nodules**	i) **Koeppe's nodules** are situated at the pupillary border occurs in both granulomatous and non-granulomatous uveitis. ii) **Busacca's nodules** are situated near collarette and are seen only in granulomatous uveitis

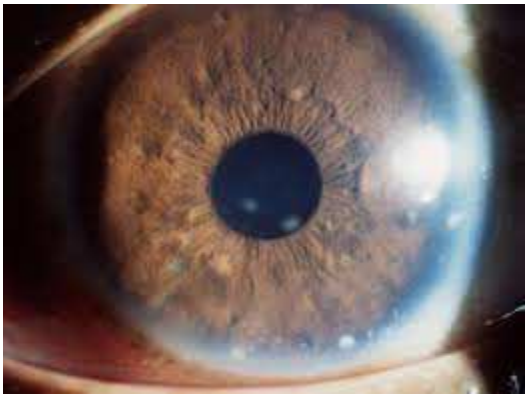


Figure vii) a)Bussaca's and b)Koppe's nodules

4.**Posterior synechiae**	These are adhesions between the posterior surface of iris and a structure posterior to it. They may be – Segmental, Annular or Total.
5. Rubeosis iridis	May be seen in chronic iridocyclitis.

VI .**PUPILLARY SIGNS:**

1.**Narrow pupil** occurs in acute attack of iridocyclitis.	2.**Irregular pupil** results from segmental posterior synechiae formation. and on dilatation with atropine results in **festooned pupil**
3.**Ectropion pupillae** develops due to contraction of fibrinous exdudate on the anterior surface of iris.	4.**Occlusio and seclusio pupillae** results when the pupil is completely occluded .

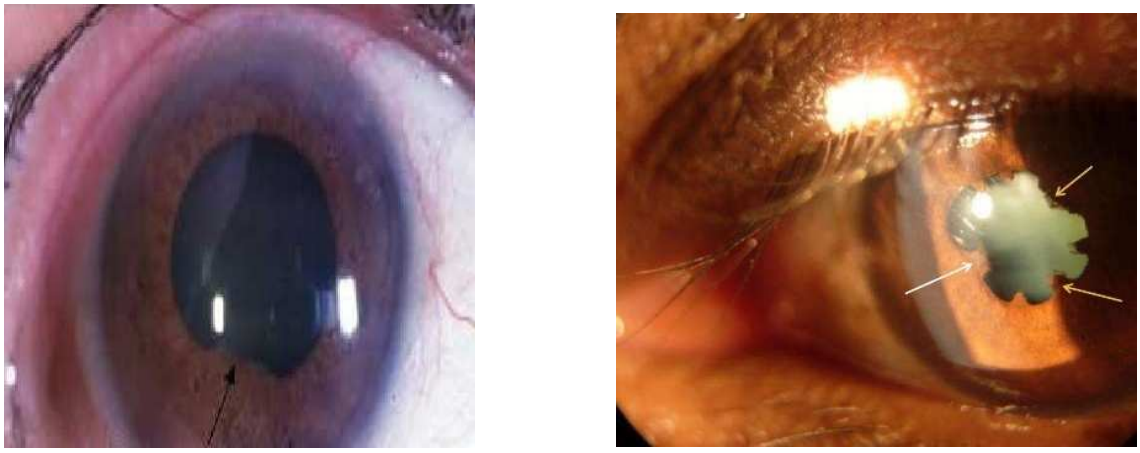


Figure viii) a)Posterior synechiae and b)Festooned pupil

VII.**CHANGES IN LENS:**

1	Pigment dispersal over the anterior lens capsule.
2	Exudates may deposit over the lens.
3	**Complicated cataract** - features in early stage are 'polychromatic luster' and 'bread-crumb'appearance of the early posterior subcapsular opacities.

VIII.**CHANGES IN VITREOUS:**

Vitreous haze is graded as follows,

GRADE	DESCRIPTION
0	Clear
0.5+	Trace
1+	Few opacities.mild blurring
2+	Significant blurring but still visible
3+	Optic nerve visible,no vessels seen
4+	Dense opacity obscures the optic nerve head

COMPLICATIONS AND SEQUELAE:

1.**Complicated cataract**
2.**Secondary glaucoma**
3.**Cyclitic membrane**..
4.**Choroiditis**.
5.**Retinal complications** include cystoid macular edema, macular degeneration, exudative retinal detachment and secondary periphlebitis retinae.
6.**Papillitis**
7.**Band –shaped keratopathy**.
8.**Phthisis bulbi**.

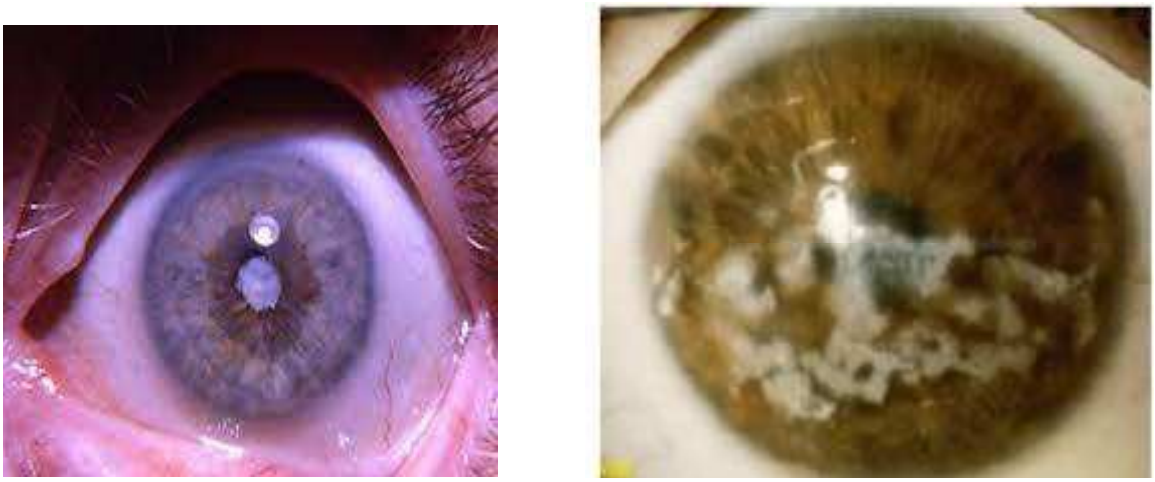


Figure ix) a)complicated cataract and b)Band keratopathy

ETIOLOGY OF UVEITIS

Anterior uveitis	**Non-granulomatous**
Granulomatous	HLA B27-associated
Sarcoidosis	(ankylosing spondylitis,Reiter's
Syphilis	syndrome, ,psoriatic arthritis, etc.,)
Tuberculosis	Fuchs heterochromic iridocyclitis
Herpes simplex	Posner –schlossman
Leptospirosis	(glaucomatocyclitic crisis)
Brucellosis	Masquerade syndromes
Phacoanaphylactic	UGH Syndrome
Idiopathic	Trauma
	Kawasaki's disease
	Drug induced

Intermediate uveitis Sarcoid,syphilis Inflammatory bowel disease Multiple sclerosis Pars planitis(idiopathic) Lymphoma, Others (tuberculosis,bechet's ,vogt-koyanagi-harada,whipple's disease,toxoplasmosis,endophthalmitis)	
Posterior uveitis **Focal retinitis** Toxoplasmosis Onchocerciasis Cysticercosis Masquerade syndromes	**Multifocal retinitis** Syphilis,sarcoidosis Herpes simplex virus (Acute Retinal Necrosis) Cytomegalovirus Masquerade syndromes Candidiasis Progressive outer retinal necrosis Eales' disease

Panuveitis Sympathetic ophthalmia Vogt-koyanagi-harada syndrome Bechet's disease Endophthalmitis Sarcoidosis Phacoanaphylaxis Lyme disease Masquerade syndromes Toxoplasmosis Syphilis,tuberculosis	
Choroiditis **Focal choroiditis** Tuberculosis Toxocariasis Nocardia Candidiasis Masquerade syndromes	**Multifocal choroiditis** Histoplasmosis,Pneumocystis choroiditis Birdshot retinochoroidopathy ,Lymphoma APMPPE Multifocal /punctate inner choroiditis Masquerade syndromes Cryptococcus,mycobacterium

SPECIFIC TYPES OF NON-SUPPURATIVE UVEITIS:

1)VIRAL UVEITIS

- **HERPES SIMPLEX VIRUS (HSV)**

It presents at all stages and occurs in both sexes with equal frequency. HSV is the leading cause of corneal blindness in developed countries.The ocular inflammation may be due to the viral infection itself or from the inflammatory response to the infection.

> The hallmark of the infection is epithelial dendritic keratitis, which is most notable with fluorescein staining

> Conjunctival injection, decreased corneal sensation ,corneal scarring and decreased visual acuity are common.

> A thickened,edematous cornea (disciform keratitis),fibrinous flare with heavy anterior chamber cell and medium sized granulomatous keratic precipitates present on the endothelium,synechiae and increased intraocular pressure arising from trabeculitis may also be seen [19].

> Most VZV and HSV infections respond to antiviral treatment ,steroids or both.

>Potential complications of HSV infection include corneal neovascularization and scarring ,cataract formation ,neurotrophic ulcers,bacterial or fungal infection ,secondary glaucoma,postherpetic neuralgia or vision loss arising from optic neuritis or chorioretinitis [35]

- **VARICELLA ZOSTER VIRUS (VZV)**

>Iridocyclitis occurs in approximately 40% to 60% cases of herpes zoster ophthalmicus [5].The life time risk of reactivation is 10-20%.

>The uveitis usually starts 1 to 2 weeks after onset of the rash .

>Decreased visual acuity ,increased intraocular pressure and miosis.

>Hutchinson's sign is a unilateral vesicular rash along the nasociliary branch of trigeminal nerve distribution.

>Anterior chamber cells and flare,granular infiltrates in the anterior corneal stroma,pseudodendrites,keratitis,ciliary injection,and corneal edema may occur.

>Sector iris stromal atrophy is a specific sign of herpetic keratouveitis.

>Mucous plaque keratopathy can occur late in the disease course.

>Patient may also develop rash and vesicles on the eyelids along with conjunctivitis,retinal necrosis ,and optic nerve involvement.

2)**LENS –INDUCED UVEITIS:**

>It can occur after cataract surgery or trauma and occasionaly occurs spontaneously if there is a hypermature cataract with a leaking capsule.

>The immune system creates inflammation against previously sequestered lens proteins.

Clinical features include,

- Circumciliary injection(ciliary flush)
- Corneal edema,'Mutton fat' keratic precipitates
- Marked flare,abundant cells,lens matter in anterior chamber
- Posterior or peripheral anterior synechiae, pupillary membrane
- Irregular lens capsule,opaque lens ,intense vitritis
- Glaucoma due to trabeculitis,lens debris in angle.
- Hypotony due to cyclitic membranes,ciliary shutdown,or choroidal effusion and Phthisis bulbi.

>Definitive treatment involves removal of the inciting agent(i.e.,the lens or residual lens matter) [23].

3)**UVEITIS IN CHRONIC SYSTEMIC BACTERIAL INFECTIONS:**

- **TUBERCULAR UVEITIS:**It still continues to be the common cause of uveitis in developing countries [4].

1.Tubercular anterior uveitis	occurs as acute non-granulomatous or granulomatous in the form of miliary tubercular iritis or conglomerate granuloma.
2.Tubercular posterior uveitis:	occur as multiple miliary tubercles in the choroid or disseminated choroiditis in chronic uveitis.
3.vasculitis	

- **ACQUIRED SYPHILITIC UVEITIS:**

It is caused by treponema pallidum.

1.syphilitic anterior uveitis	occur as acute plastic iritis or granulomatous iritis. Gummatous anterior uveitis is associated with highly vascularised multiple iris nodules.
2.syphilitic posterior uveitis	May occur as disseminated,peripheral or diffuse choroiditis

- **LEPROTIC UVEITIS:**

It is caused by mycobacterium leprae,an acid fast bacilli.

1.Acute iritis	Presents with severe exudative reaction.
2.Chronic granulomatous iritis	characterised by the presence of small glistening iris pearls.

4)UVEITIS IN NON-INFECTIOUS SYSTEMIC DISEASES:

SARCOIDOSIS	**BECHET'S DISEASE**	**VKH SYNDROME**
* affects young adults * frequently presents with bilateral hilar lymphadenopathy, pulmonary infiltration, skin and ocular lesion * presents as acute or chronic iridocyclitis.	*features are recurrent non-granulomatous uveitis with hypopyon. ,aphthous ulcers, genital ulceration and erythema multiforme. *It typically affects young males positive for HLA B51.	* presents with bilateral recurrent granulomatous uveitis with cutaneous and neurological manifestations.

5)UVEITIS IN ARTHRITIS

- **ANKYLOSING SPONDYLITIS**

  - is an idiopathic chronic inflammatory arthritis presenting with acute,recurrent, non-granulomatous iridocyclitis [20]

  - affects young males who are HLA B27 positive.

- **REITER'S SYNDROME**

  - Has a traid of urethritis,arthritis [1] and conjunctivitis with or without uveitis

  - typically involving young males positive for HLA B27.

- **JUVENILE CHRONIC ARTHRITIS**

  - is a chronic inflammatory arthritis in children below 16 years of age [7].

  - uveitis associated with this disease is bilateral , chronic ,non-granulomatous and the eye is white.

  - affects female more than male in the ratio of 4:1.

6)PARASITIC UVEITIS

• **TOXOPLASMOSIS**	• **TOXOCARIASIS**
# Systemic toxoplasmosis in humans occur in two forms - congenital and acquired. # The traid of Congenital toxoplasmosis are convulsions ,cerebral calcification and chorioretinitis. # Acquired is very rare and presents with recurrent chorio retinitis.	# It is almost unilateral which can present as, - chronic endophthalmitis, - posterior pole granuloma or - peripheral granuloma.

7)FUNGAL UVEITIS: It includes candidiasis,ocular histoplasmosis syndrome - may occur as anterior uveitis or chorioretinitis or endophthalmitis**.**

8)TRAUMATIC UVEITIS

Sympathetic ophthalmia - a rare bilateral diffuse non necrotising granulomatous panuveitis that can occur from a few days to several decades following penetrating injury.

9)UVEITIS IN INTRAOCULAR TUMORS

Acute uveitis usually associated with pseudohypopyon or hyphema may be the presenting mode in intraocular tumor (retinoblastoma masquerading as uveitis).

10) IDIOPATHIC SPECIFIC UVEITIC SYNDROMES

***Fuchs heterochromic iritis** is a rare form of unilateral non granulomatous anterior uveitis characterised by diffuse stellate keratic precipitates and iris heterochromia [24]. Rubeosis iridis with angle neovascularistion which is termed as Amsler sign can occur , eventually leading to early development of cataract and glaucoma.
***Posner schlossman syndrome** Features are mild recurrent attack of anterior uveitis with high IOP,with corneal edema and fine keratic precipitates. The eye is white with open angle [26].

TREATMENT OF UVEITIS

Anterior uveitis can be managed by medical therapy and requires surgical intervention only if structural complications like secondary glaucoma or secondary cataract supervene [6].

The goals of therapy in anterior uveitis are

* To preserve the vision
* To relieve ocular pain
* To eliminate ocular inflammation
* To prevent the synechiae formation and further sequalae.
* To manage the intraocular pressure.

Non infectious anterior uveitis is treated using the following drugs:

A)CORTICOSTEROIDS-

Corticosteroids are the drug of choice in the treatment of anterior uveitis.Steroids act by modifying and decreasing the inflammatory response in the eye.They inhibit the cycloxygenase pathway of inflammatory response.

i) **Topical steroids:** Topical corticosteroids if administered as frequent doses ,can achieve adequate therapeutic levels in the anterior chamber.

A number of topical ophthalmic corticosteroids are available.

• Prednisolone acetate 0.125% and 1%.
• Betamethasone 1%
• Dexamethasone sodium phosphate 0.1%
• Fluorometholone 0.1% and 0.25%
• Loteprednol
• Rimexolone 1%

The choice of topical steroid should be based on the severity of uveitis.In cases with severe AC reaction strong potency steroid such as prednisolone acetate should be preferred whereas in cases with mild anterior uveitis weak steroids such as betamethasone or dexamethasone can be applied.

ii) **Periocular steroids:** Periocular injection is indicated where maximum concentration of the drug is required for a longer time.The drugs used are methyl prednisolone 40mg/ml or triamcinolone acetonide 40mg/ml.Anterior subtenon injection is indicated in severe anterior uveitis.

iii) Systemic steroids: Systemic oral prednisolone 1mg/kg/day to start with and should be gradually tapered off and discontinued. Oral steroids are indicated where anterior uveitis is not responding to topical drugs alone or if the disease is recurrent and bilateral

B) MYDRIATICS/CYCLOPLEGICS-

They cause temporary paresis of sphincter pupillae and ciliary muscle and keep the pupil mobile thereby preventing the formation of synechiae. A short acting mydriatic/cycloplegic is usually preferred. Cycloplegics/mydriatics serve three purposes in the management of anterior uveitis,

- To relieve pain by immobilising the iris.
- To prevent the adhesion of iris to the anterior surface of lens.
- To stabilise the blood aqueous barrier and prevent protein leakage.

The cycloplegic/mydriatic agents used are,

➢ Atropine 0.5% ,1% and 2%
➢ Homatropine 2% and 5%
➢ Cyclopentolate 0.5% , 1% and 2%
➢ Tropicamide 0.5% and 1%
➢ Phenyl ephrine 2.5% ,5% and 10%.

C)<u>NON-STEROIDAL ANTI INFLAMMATORY DRUGS</u>:

They act by inhibiting the arachidonic acid metabolism.The drugs used are flurbiprofen,indomethacin,diclofenac sodium.

D)<u>IMMUNOSUPPRESSIVES</u>-

They are used as steroid sparing agents (intolerable to the side effects of steroids or steroid non-responders) and in vision threatening uveitis. They are not routinely used in acute anterior uveitis except in JRA associated iridocyclitis [27] and Bechets disease.

The commonly used immunosuppressive agents are,

▪ Methotrexate 7.5 -25 mg/week with folic acid.
▪ Azathioprine 2-2.5mg/kg/day.
▪ Mycofenolate mofetil 1g/day.
▪ Cyclosporine 3 -5mg/kg/day.
▪ Tacrolimus 0.05 mg/kg/day.
▪ Cyclophosphomide 1-3 mg /kg.
▪ Chlorambucil 0.1 -0.2 mg/kg.

E)BIOLOGICS-

Biologics represent newer class of drugs used for the treatment of autoimmune diseases.

The currently used biologics are,

- **Tumor necrosis factor alpha antagonists –** Infliximab and adalimumab.
- **Interleukin receptor antagonists –**etarnacept, Anakinra and daclizumab.

FUTURE DIRECTIONS

Currently there are many uveitic therapeutic drug trials in clinical investigation. There are several revolutions in medicine today that are bound to make marked improvement in uveitis diagnosis and management. Advances in structural and functional imaging have an impact in early detection of disease activity. Novel drug delivery system holds the promise to provide sustained local therapy with minimal systemic toxicity.

Understanding of genetics, microbes or environmental factors involved in the pathogenesis of various diseases will afford newer diagnostic tools and chip away large proportion of disease which are termed as IDIOPATHIC.

Finally a better molecular understanding of pathogenesis of disease will lead to development of novel therapies with desirable efficacy minimising the adverse effects.

AIMS AND OBJECTIVES

1)To evaluate the etiology,clinical features,systemic associations and Severity of uveitis.

2)To analyse the visual outcome following uveitis.

INCLUSION CRITERIA:

- Anterior uveitis (acute and chronic) of all etiologies
- Unilateral and bilateral
- Male and Females, all ages.

EXCLUSION CRITERIA:

- Intermediate uveitis
- Posterior uveitis
- Recurrent uveitis.

MATERIALS AND METHODS

This is a prospective study which includes 80 patients with anterior uveitis of various etiologies presented to our department at Thanjavur medical college from july 2014 – September 2015.They were subjected to detailed clinical examination and relevant investigations.

A proforma was drawn up and following details were recorded for each patient :age and sex, history related to trauma,surgery and specific systemic associations of uveitis,clinical examination of signs and symptoms of uveitis incuding general systemic examination.

Ocular examination in this study included:

- A thorough examination with diffuse illumination.
- Visual acuity by snellens chart .
- Slit lamp examination.
- Intraocular pressure with applanation tonometry.
- Gonioscopy.
- Direct ophthalmoscopy.
- Slit lamp biomicroscopy with 90D lens
- Indirect ophthalmoscopy.

- B-scan ultrasonography in patients suspected to have posterior segment abnormality.
- Radiography of chest,sacro iliac joint and lumbar spine.
- Routine investigations of blood and urine.
- Skin tests.

Patients were examined by other specialities like general medicine, ENT,Dental,Dermatology,Obstretics and gynecology for specific systemic examination.

Patients with mild uveitis at presentation were treated as out patients and those with severe uveitis were hospitalized for evaluation and appropriate management.They were reviewed according to the severity of uveitis and followed up for a period of 3 months.According to improvement/worsening of the disease activity they were managed.

CLINICAL EVALUATION AND MANAGEMENT AT FOLLOWUP:

SEVERITY OF UVEITIS	FOLLOW UP	VISUAL ACUITY	SLIT LAMP EXAM	IOP	IDO	MANAGEMENT
MILD	Every 7 days	Yes	yes	Yes	yes	Topical steroids and mydriatics /cycloplegics
MODERATE	Every 2-4 days	Yes	Yes	Yes	yes	Topical and systemic steroids, mydriatics/cycloplegics, beta blockers(if IOP raised)
SEVERE	Every 1-2 days	Yes	yes	yes	yes	Topical,systemic and periocular steroids, mydriatics/cycloplegics, beta blockers(if IOP raised)

The cases were followed up periodically according to the severity of uveitis for the period of 15 months and managed.

CLINICAL INVESTIGATIONS AND PROCEDURES:

1)SLIT LAMP EXAMINATION:

Slit- lamp biomicrosopy is a dynamic examination in which the eye is scanned anteroposteriorly and horizontally..

- An optical section of peripheral cornea and anterior chamber is made with illumination and viewing arms at 60degree and viewing arm perpendicular to cornea using magnification 10x.A comparison of depth of peripheral anterior chamberto the peripheral cornea is used to assess the shallowness of anterior shamber in the Van Herricks method.
- Keratic Precipitates were examined by slit lamp examination.
- Aqueous cells and flare is visible on slit lamp when its beam is narrowed to 1 x 1 mm and graded according to SUN working group grading and the severity of inflammation was assessed.
- The colour of the iris and clarity of its pattern was examined.Special attention was paid for the presence of anterior or posterior synechiae.
- The pupils were examined at the earliest for size,shape reaction to light and sphincter tear before mydriatic was applied.
- The intraocular pressure estimation was done with the schiotz / goldmann applanation tonometry for all the patients.

2)**GONIOSCOPY:**

Gonioscopy is a clinical technique used to examine structures in angle of the anterior chamber. The Goldmann three mirror gonioscope lens was used.

Procedure:

The cornea was anaesthetized with 4% xylocaine eye drops. The patient was seated comfortably in adjustable stool with the chin resting on chin rest of the slit lamp.Gonioscope was placed against the cornea with methylcellulose.After focusing the slit lamp ,the angle was studied by rotating the lens,to visualize 360degree of the angle . Both the eyes were examined.

3) **INDIRECT OPHTHALMOSCOPY**:

The Keeler binocular indirect ophthalmoscope with the lens strength of +20D was used in this study. The beam penetrates most of the media opacities.

With the pupils dilated, the patients were examined in the supine position in a dark room. The periphery of the retina was viewed with scleral indenter.

4) **B-SCAN ULTRASONOGRAPHY**:

In B-scan ultrasound, contact technique is performed with the probe held directly against the closed lids

It is useful to plan surgery in hazy media due to complicated cataract and to diagnose masquerade syndromes like lymphoma,etc.,

It is also used to identify vitreous disorders like vitreous haemorrhage,inflammation,pars planitis,retained lens fragments.Useful in visualization of optic disc edema,exudative detachment,choroidal thickening, uveal effusion and hypotony and also to rule out intra ocular tumors.

5) **LABORATORY INVESTIGATIONS:**

The following laboratory tests were performed for the patients involved in this study,

i) HAEMATOLOGICAL INVESTIGATIONS:

- Complete blood count(TC,DC,ESR)- to rule out infection and to ascertain the existence of chronic inflammation.
- Random blood sugar – to rule out diabetes mellitus.
- Renal/liver function tests
- Serological tests for syphilis – VDRL.
- Serum calcium
- RA factor,C-reactive protein.

ii) **URINE ROUTINE -** to rule out urinary tract infections.

iii) SKIN TESTS:

Mantoux tuberculin skin test (TST) is the standard method of determining whether the patient is infected with mycobacterium tuberculosis.The TST is performed by injecting 0.1 ml of PPD into the inner surface of forearm with the tuberculin syringe intradermally.The test reaction should be read between 48-72 hours after administration in millimeters of induration.

6)**RADIOLOGICAL INVESTIGATIONS**:

- X-RAY CHEST:useful in identifying diseases like tuberculosis,sarcoidosis and histoplasmosis.
    - In tuberculosis it shows fibro cavity lesions or military lesions.
    - In sarcoidosis it shows hilar lymphadenopathy with reticulonodular parenchymal infiltrates and eventually progressive pulmonary fibrosis.
- X-RAY SACROILIAC JOINT:It is done in all young patients with acute unilateral anterior uveitis irrespective of the presence or absence of low backache.This is because X ray may be positive before patient is symptomatic in case of ankylosing spondylitis.
- X-RAY HAND AND FEET –for sarcoidosis.
- CT/MRI BRAIN is indicated in lymphoma.
- X-RAY SKULL: To find out cerebral calcifications in case of toxoplasmosis,CMV.

PERIOCULAR INJECTIONS

Periocular steroid injections are indicated in case of severe anterior uveitis with severe hypopyon/fibrin membrane, adjuvant to topical and systemic treatment and in case of poor patients compliance to achieve therapeutic concentration.

Types of periocular injections used in this study include,

i)**ANTERIOR SUBTENON TECHNIQUE**:For cases with severe persistent anterior uveitis.

Procedure – Under strict aseptic precaution, with the patient in supine position,topical 4% xylocaine eye drops is instilled before the procedure. The inferotemporal quadrant is preferred usually. With a 25 gauge needle,5/8th of the needle facing the sclera, bulbar conjunctiva is penetrated 2 to 3 mm from fornix,ensure that the needle has not penetrated sclera attempt to place the needle near posterior sclera. The piston of the syringe is withdrawn to ensure the needle is not intravascular and the contents are injected and the needle is removed.

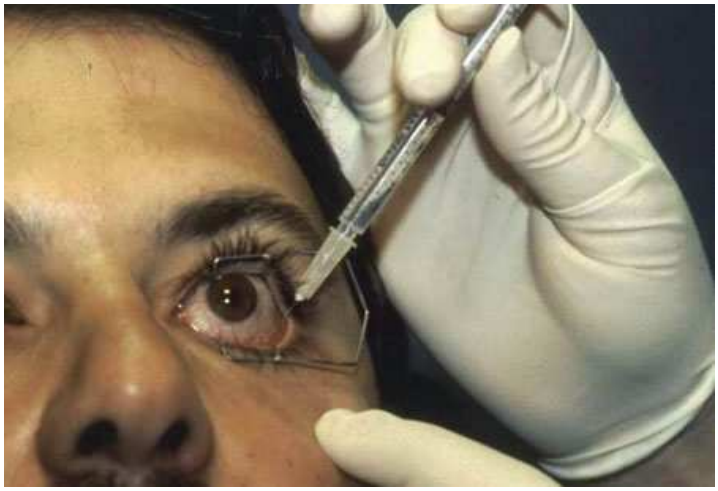


Figure x)Anterior subtenon injection

Triamcinolone acetonide 40mg or methylprednisolone acetate 40mg were the periocular steroid used in this study.

ii)**SUB CONJUNCTIVAL TECHNIQUE**:

By subconjunctival route,mydricaine(proparacaine,adrenaline and atropine) and steroids(dexamethasone phosphate) were injected for some patients involved in this study.

Procedure-Under strict aseptic precaution,with the patient in supine position topical 4% xylocaine drops is applied.Subconjunctival space is penetrated with 25 gauge needle several millimeters below the limbus at 4 – 8 ‘0 clock position.The piston is withdrawn to ensure the needle is not intravascular , the contents are injected and the needle is removed.

Figure xi)Sub conjunctival injection

The advantages of the periocular injections are they can achieve therapeutic concentration and have a long lasting effect.

OBSERVATION AND RESULTS

This is a prospective study which includes 80 patients with anterior uveitis of various etiologies presented to our department at Thanjavur medical college from july 2014 – September 2015.They were subjected to detailed clinical examination and relevant investigations.

The following parameters were analyzed.

TABLE NO: 1 AGE DISTRIBUTION

Age in years	**No of cases**	**Percentage (%)**
1 to 10	2	2.50
11 to 20	28	35.00
21 to 30	8	10.00
31 to 40	13	16.25
41 to 50	8	10.00
>50	21	26.25

In this study of 80 patients the maximum i.e 28 cases were seen in 11 – 20 age group accounting for 35% followed by 21 cases (26.25%) in the age group of 50 years and above, 13 cases (16.25%) in 31 -40 age group, 8 cases (10%) in 21 -30 and also 41 – 50 age group.

FIGURE 1: AGE DISTRIBUTION

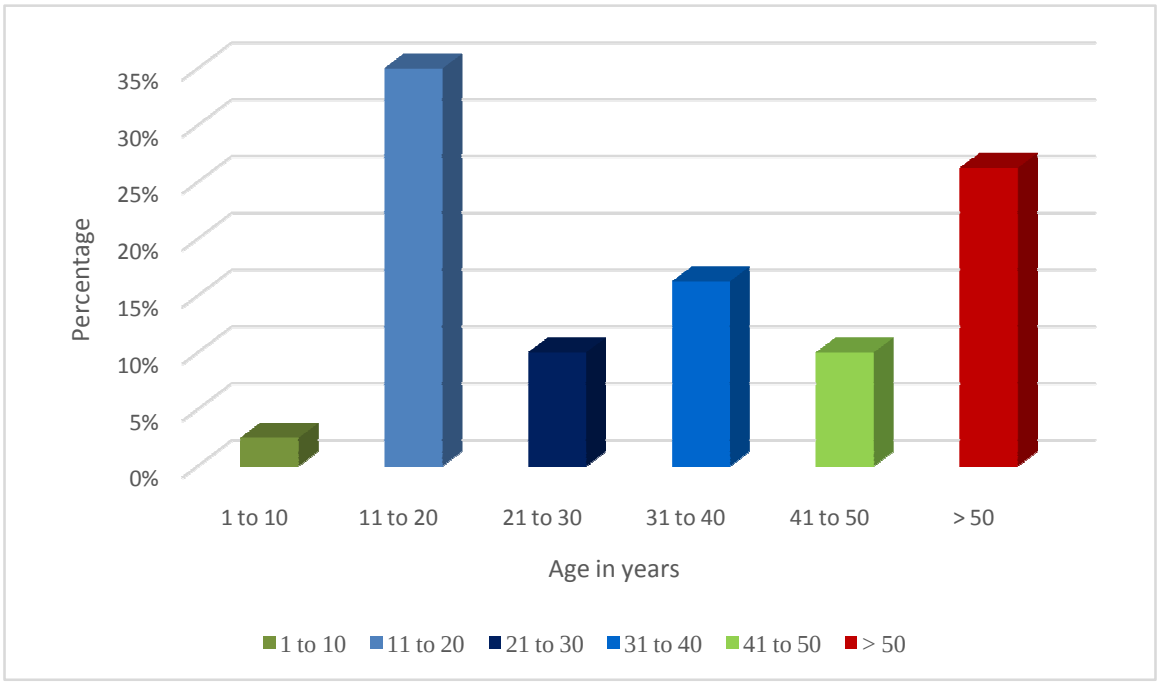


TABLE 2 : SEX DISTRIBUTION

Sex	No of cases	Percentage (%)
Male	50	62.50
Female	30	37.50

Out of 80 patients, 50 patients (62.5%) were male, 30 patients were female (37.5%) with male female ratio being 1.6:1.

FIGURE 2 : SEX DISTRIBUTION

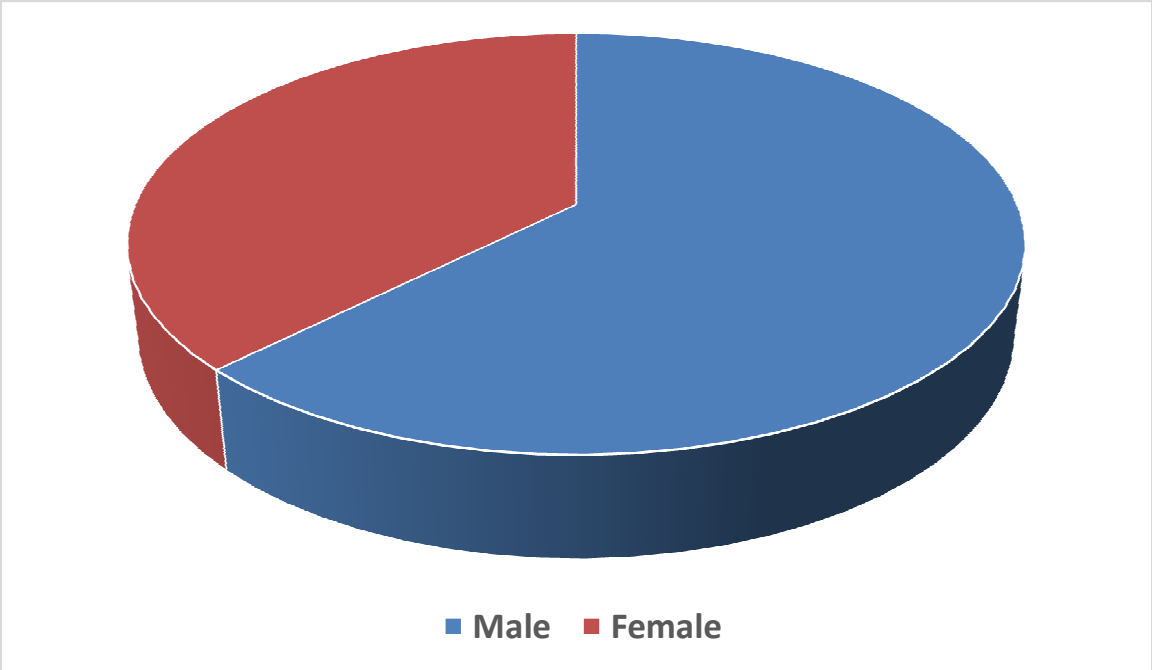


TABLE NO 3 : EYE INVOLVED – LATERALITY

Eye involved	No of cases	Percentage %
Unilateral	77	96.25
Bilateral	3	3.75

Out of 80 patients, 77 patients (96.25%) had unilateral involvement and 3 patients (3.75%) had bilateral involvement.

FIGURE 3: EYE INVOLVED – LATERALITY

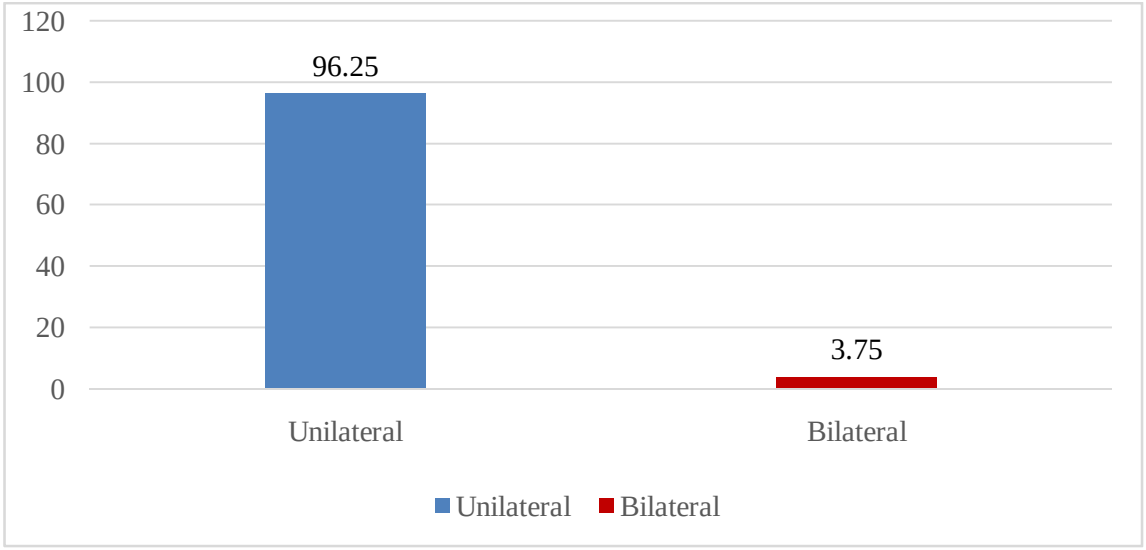


TABLE NO 4 : CLASSIFICATION BASED ON ETIOLOGY

Etiology	No of cases	Percentage %
Trauma	38	47.50
Lens induced	4	5.00
Infections	7	8.75
Idiopathic	14	17.50
Postoperative uveitis	17	21.25

Out of 80 patients, 38 patients (47.50%) were due to post traumatic, 4cases (5.00%) due to Lens induced , 17 cases(21.25%) due to postoperative uveitis,7 cases (8.75%) due to infective etiologies and 14 cases (17.50%) were idiopathic.

FIGURE 4 : CLASSIFICATION BASED ON ETIOLOGY

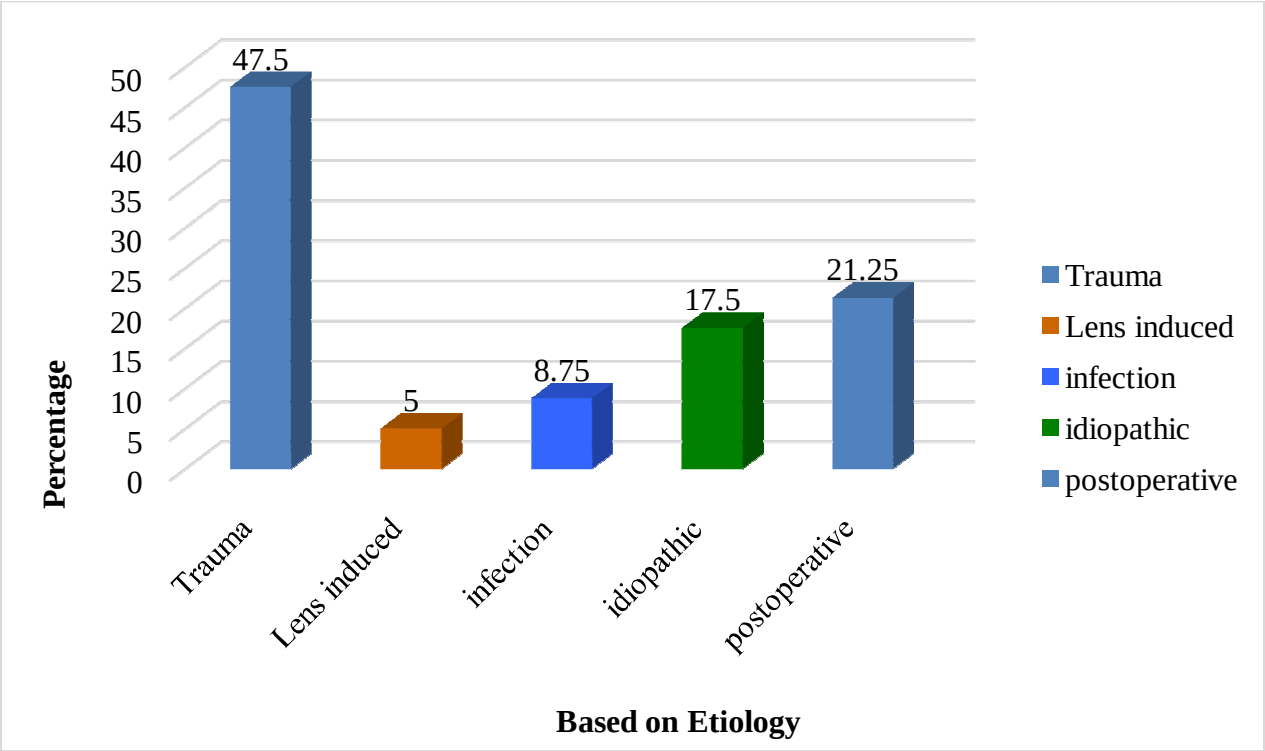


TABLE 5: VISION AT THE TIME OF PRESENTATION

Vision	No of cases	Percentage %
No PL	1	1.25
PL +	1	1.25
CFCF	17	21.25
1/60 to 6/60	24	30.00
6/36 to 6/18	22	27.50
6/12 to 6/9	15	18.75
6/6	-	-

At the time of presentation , Out of 80 patients, 1 patient (1.25%) had no perception of light , 1 patient (1.25%) had perception of light, 17 patients (21.25%) had counting figures close to face, 24 patients (30%) had visual acuity between 1/60 – 6/60 , 22 patients (27.50%) had between 6/36 – 6/18, 15 patients (18.75%) had between 6/12 – 6/9 and none had visual acuity of 6/6.

FIGURE 5a : VISION AT THE TIME OF PRESENTATION

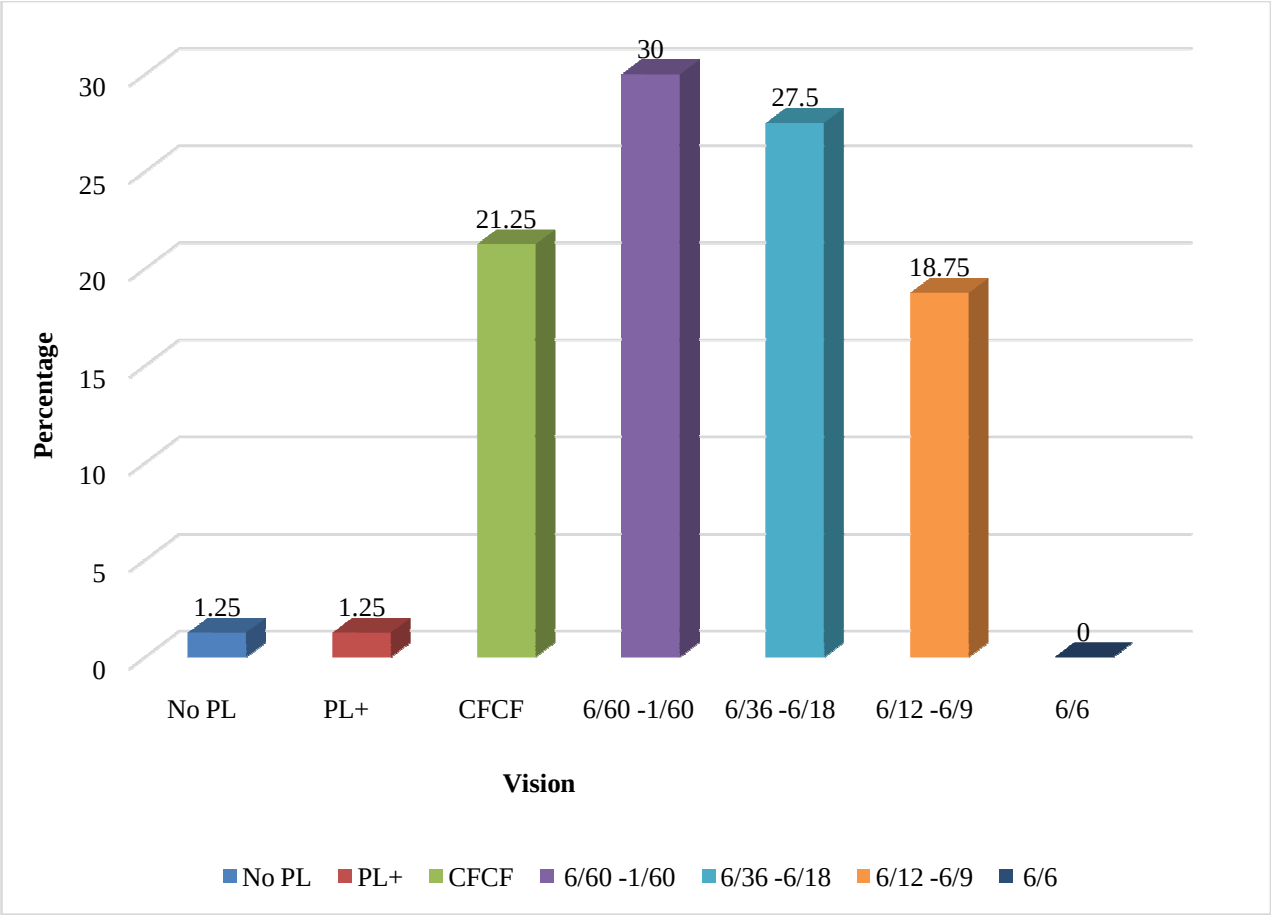


TABLE 6: FINAL VISUAL OUTCOME

Vision	No of cases	Percentage
No PL	2	2.50
PL +	-	-
CFCF	2	2.50
1/60 to 6/60	13	16.25
6/36 to 6/18	19	23.75
6/12 to 6/9	24	30.00
6/6	20	25.00

Out of 80 patients, 2 patients (2.5%) had final visual outcome of no perception of light, 2 patients (2.5%) had counting fingers close to face, 13 patients (16.25%) had visual acuity between 1/60 – 6/60, 19 patients (23.75%) had between 6/36 – 6/18, 24 patients (30%) had between 6/12 – 6/9 and 20 (25%) patients had visual acuity of 6/6.

FIGURE 5b: FINAL VISUAL OUTCOME

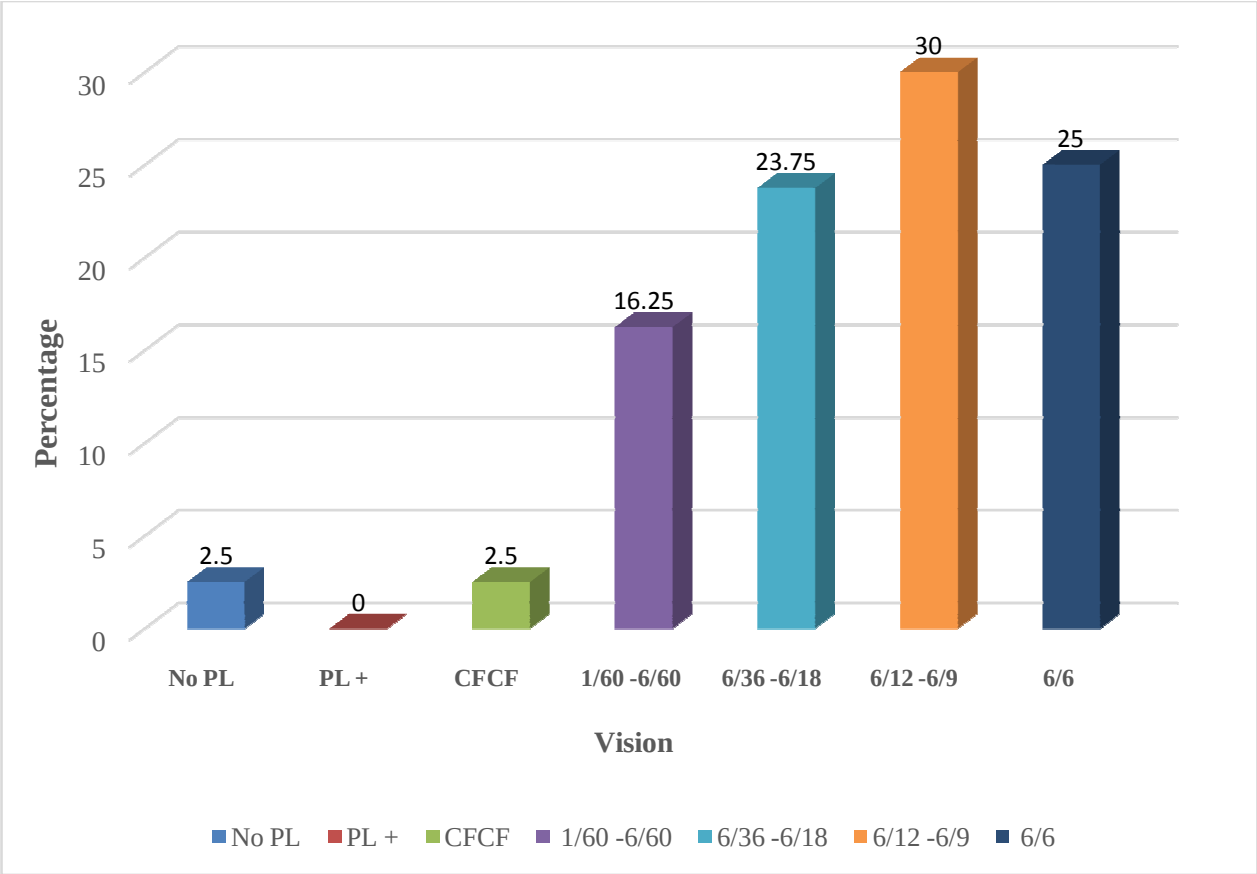


FIGURE 6: COMPARISON OF VISUAL ACUITY

AT PRESENTATION AND FINAL VISUAL OUTCOME

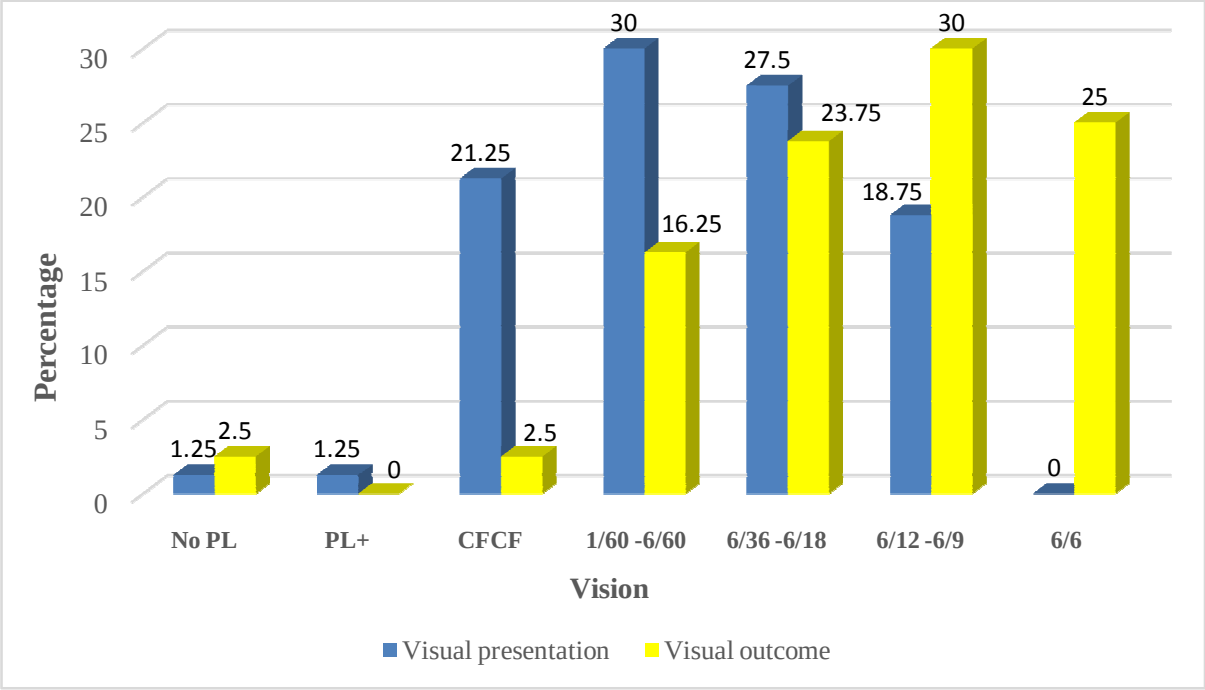


TABLE 7: ANTERIOR CHAMBER EXAMINATION

Anterior chamber	No.of cases	Percentage %
Hyphema	9	11.25
Hypopyon	16	20.00
Cortex	2	2.50
AC cells/flare	53	66.25

Out of 80 patients,9 patients(11.25%) had hyphema,16 patients (20%) had hypopyon and 2 patients(2.5%) had lens material(cortex) in anterior chamber and 53 patients (66.25%) presented with only AC reaction.

FIGURE 7:ANTERIOR CHAMBER EXAMINATION

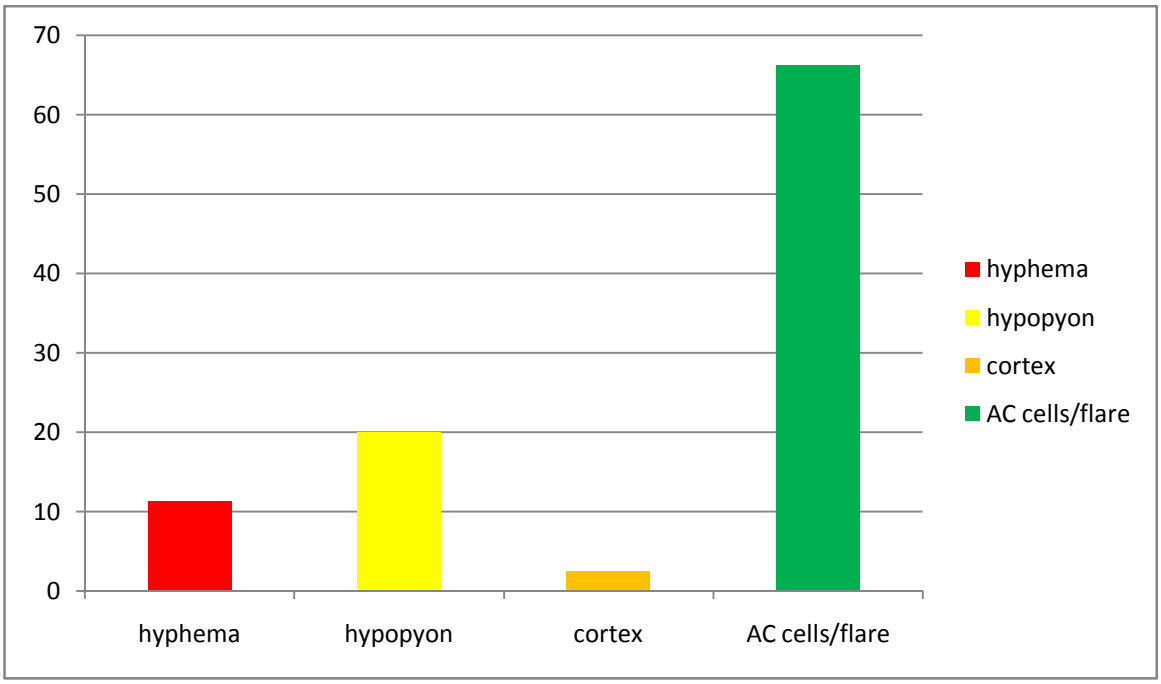


TABLE 8: PUPILLARY EXAMINATION

PUPIL	NO. OF CASES	PERCENTAGE %
Sphincter tears	8	10.00
Posterior synechiae	10	12.50
miosis	11	13.75
Circular (3 to 4 mm)	51	63.75

Out of 80 patients,8 patients (10%) presented with sphincter tears,11 had miotic pupil (13.75%)and 10 (12.50%) had posterior synechiae and 51 patients (63.75%) had circular pupil of 3 t0 4 mm diameter.

FIGURE 8: PUPILLARY EXAMINATION

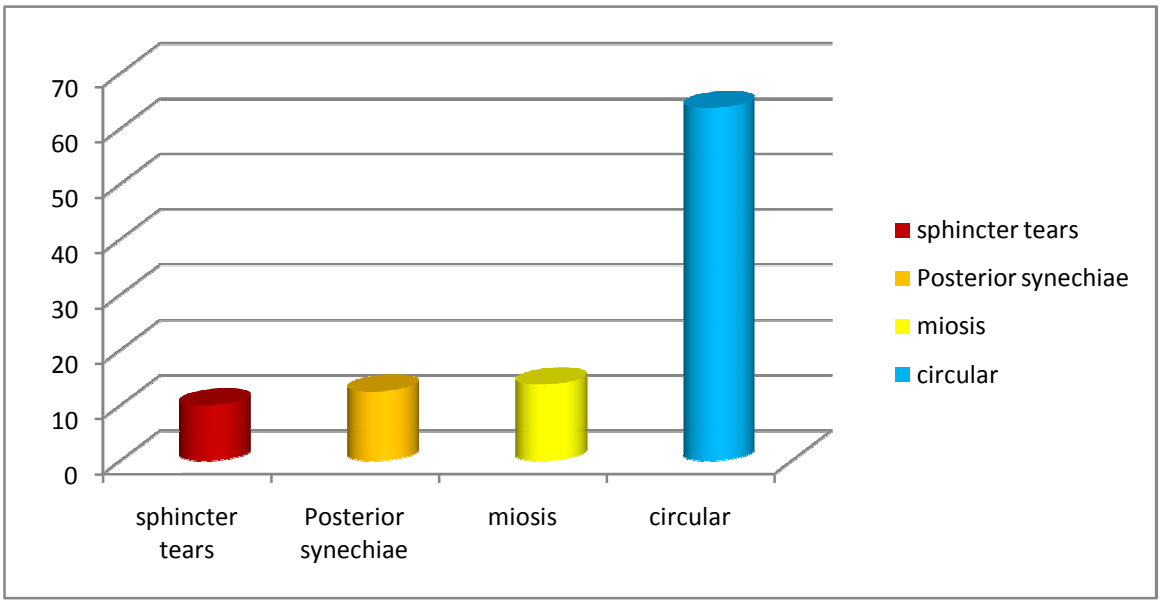
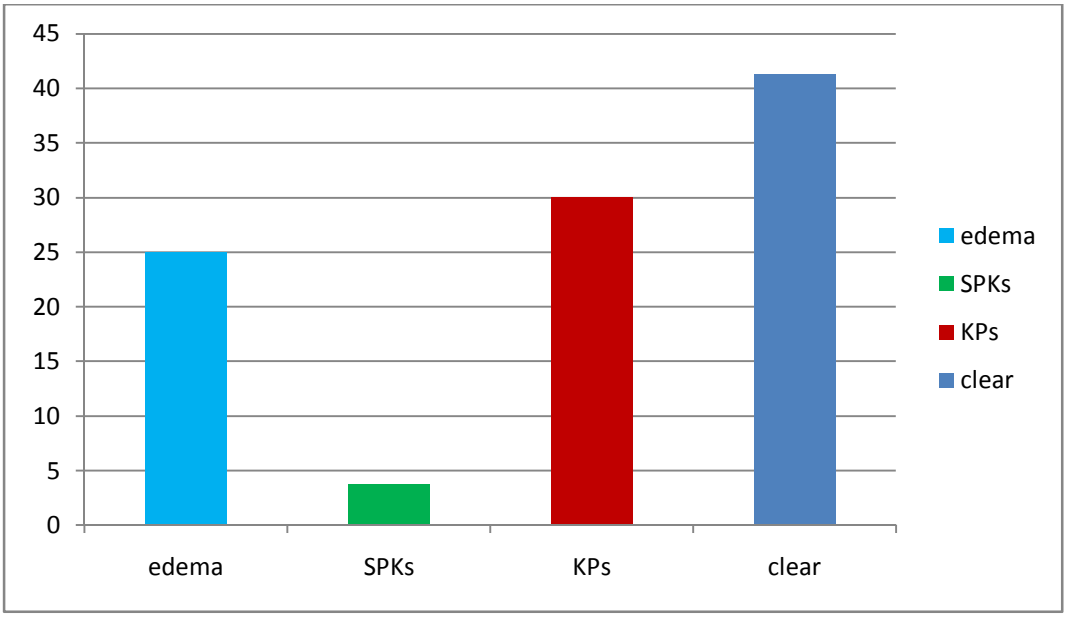


TABLE 9: CORNEAL EXAMINATION

CORNEA	NO.OF CASES	PERCENTAGE %
Edema	20	25.00
SPKs	3	3.75
KP s	24	30.00
Clear	33	41.25

Out of 80, 20 patients (25.00%) had corneal edema , 3 patients (3.75%) had superficial punctuate keratitis (SPKs) and 24 patients(30.00%) had keratic precipitates(KPs) and 33 patients(41.25%) had clear cornea.

FIGURE 9: CORNEAL EXAMINATION



CLINICAL GRADING OF ANTERIOR UVEITIS

PARAMETERS	MILD	MODERATE	SEVERE
SYMPTOMS	Mild	moderate	Severe
CONJUNCTIVA	Superficial CCC	Deep CCC	Deep CCC
CORNEA	No KPs	Scattered KPs	Dense KPs
AC REACTION/ PUPIL/IRIS	Trace to 1+ cells	1+to 3+ cells/flare Miotic pupil,mild PS.	3+ to 4+ cells/flare Sluggish/fixed pupil, Fibrous PS
VISION	6/6 – 6/9	6/9 – 6/36	<6/36
IOP	Reduced <4mmHg	Reduced 3-4mmHg	Raised IOP

TABLE 10: DEGREE OF SEVERITY OF ANTERIOR UVEITIS

SEVERITY	NO. OF CASES %	PERCENTAGE %
MILD	13	16.25
MODERATE	24	30.00
SEVERE	43	53.75

Out of 80 cases of, 13 cases (16.25%) presented with mild ,24 cases(30.00%) with moderate and 43 cases(53.75%) with severe degree of anterior uveitis.

FIGURE 10: DEGREE OF SEVERITY OF ANTERIOR UVEITIS

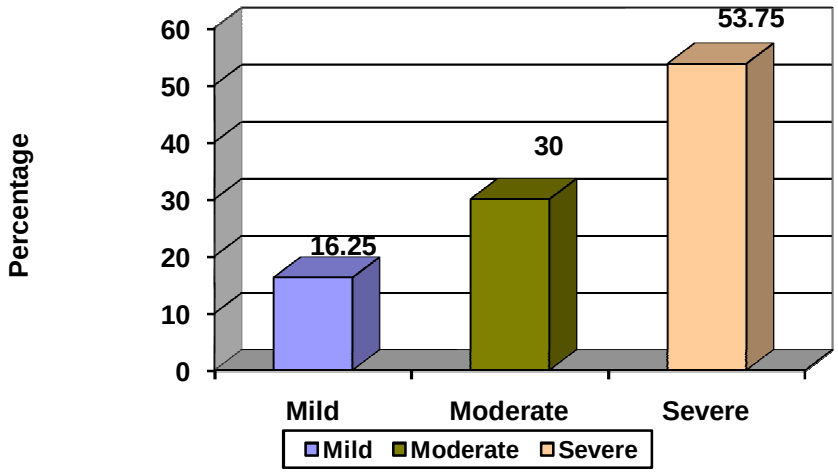


TABLE 11: COMPLICATIONS OF UVEITIS

COMPLICATIONS	NO. OF CASES	PERCENTAGE %
Cataract	6	7.50
Glaucoma	8	10.00
Macular edema	5	6.25
Postoperative flare up	9	11.25

Out of 80patients,6patients(7.50%) developed cataract,8patients(10.00%) developed secondary glaucoma,5 patients(6.25%) had macular edema and 9 patients (11.25%) had postoperative flare up of uveitis.

FIGURE 11: COMPLICATIONS OF UVEITIS

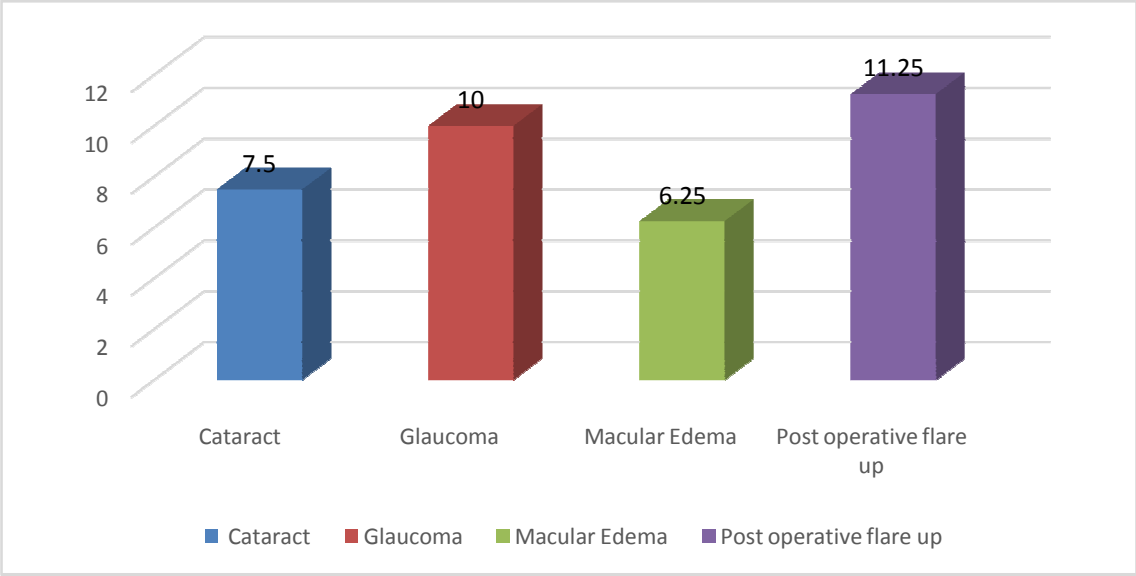


TABLE 12:VISUAL OUTCOME IN TRAUMATIC ANTERIOR UVEITIS

Vision	No of Cases at presentation	Percentage %	No of cases at Follow up	Percentage %
NO PL	-	-	1	2.63
PL +	-	-	-	-
CFCF	5	13.15	2	5.26
1/60 -6/60	5	13.15	1	2.63
6/36 -6/18	14	36.84	4	10.52
6/12 -6/9	14	36.84	12	31.57
6/6	-	-	18	47.36

Out of 38 patients with traumatic anterior uveitis,1 patient (2.63%) had no improvement and 18 patients (47.36%) had final visual outcome of 6/6 **.**

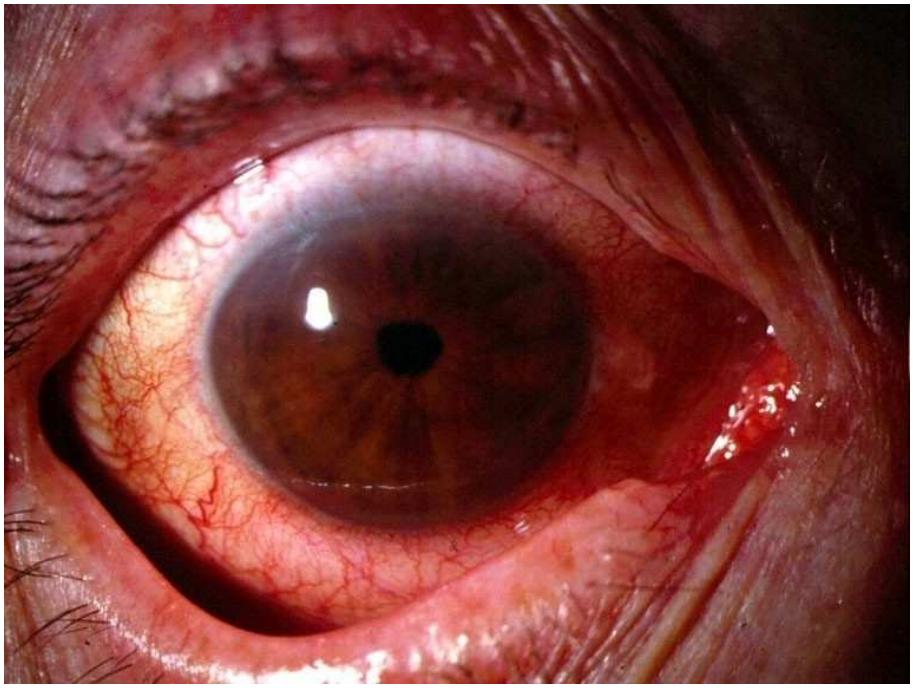


Figure xii) Traumatic anterior uveitis

FIGURE12:VISUALOUTCOME IN TRAUMATIC ANTERIOR UVEITIS

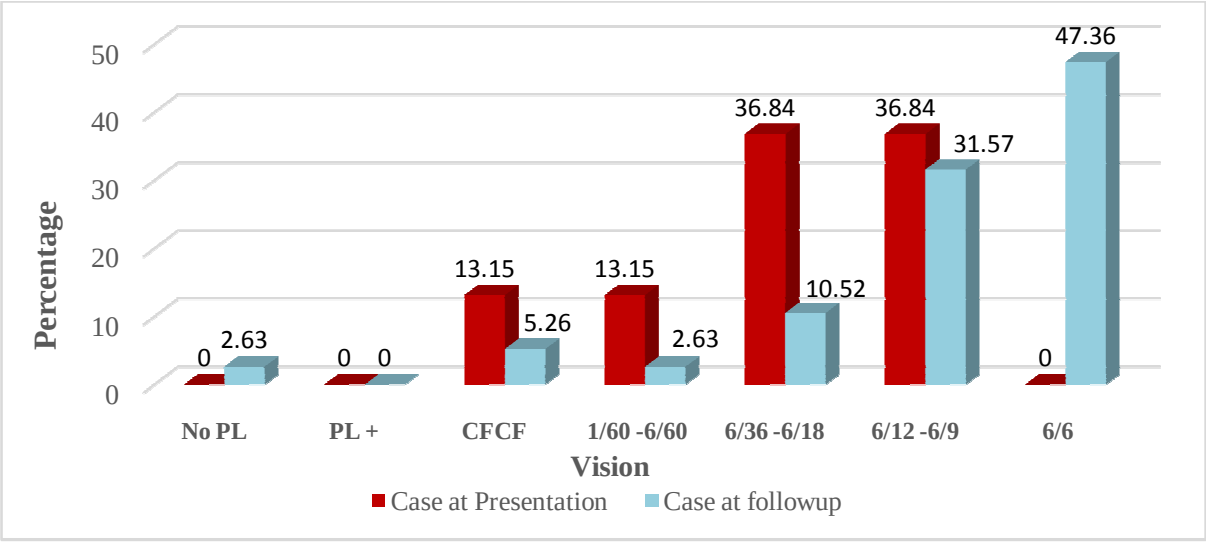


TABLE 13:VISUAL OUTCOME IN LENS INDUCED UVEITIS

Vision	No of Cases at presentation	Percentage %	No of cases at Follow up	Percentage %
NO PL	-	-	-	-
PL +	1	25	-	-
CFCF	3	75	-	-
1/60 -6/60	-	-	3	75
6/36 -6/18	-	-	1	25
6/12 -6/9	-	-	-	-
6/6	-	-	-	-

Out of 4 patients , 1patient (25%)had PL and 3 patients(75%) had CFCF visual acuity at presentation and final visual outcome was in 3 patients (75%) between 1/60 – 6/60 and in 1 patient(25%) between 6/36 – 6/18 .

FIGURE 13:VISUAL OUTCOME IN LENS INDUCED UVEITIS

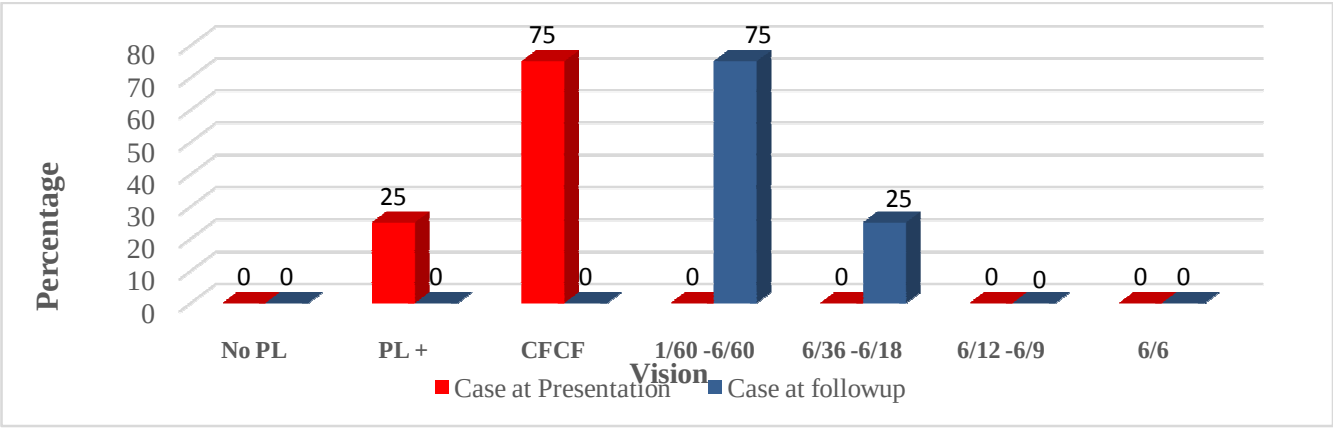


TABLE 14:VISUAL OUTCOME IN INFECTIOUS UVEITIS

Vision	No of Cases at presentation	Percentage %	No of cases at Follow up	Percentage %
NO PL	1	14.29	1	14.29
PL +	-	-	-	-
CFCF	1	14.29	-	-
1/60 -6/60	2	28.57	1	14.29
6/36 -6/18	3	42.85	1	14.29
6/12 -6/9	-	-	2	28.57
6/6	-	-	2	28.57

Out of 7 patients with infectious uveitis , 1patient (14.28%) had no improvement in vision and 2 patients(28.57%) had finsl visual outcome between 6/12 and 6/9 and 2 patients (28.57%) had 6/6 visual outcome after treatment.

FIGURE 14:VISUAL OUTCOME IN INFECTIOUS UVEITIS

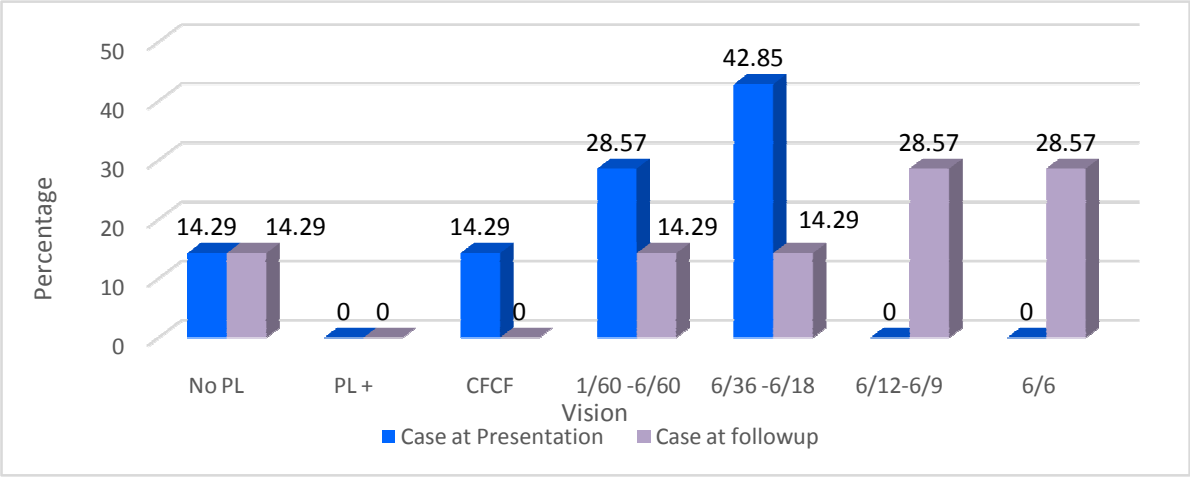


TABLE 15:VISUAL OUTCOME IN POSTOPERATIVE UVEITIS

Vision	No of Cases at presentation	Percentage %	No of cases at Follow up	Percentage %
NO PL	-	-	-	-
PL +	-	-	-	-
CFCF	5	29.41	-	-
1/60 -6/60	9	52.94	5	29.41
6/36 -6/18	3	17.64	7	41.17
6/12 -6/9	-	-	5	29.41
6/6	-	-	-	-

Out of 17 patients with postoperative anterior uveitis, 5patients(29.41%) had CFCF at presentation and 7 patients(41.17%) had final visual outcome between 6/36 and 6/18 and in 5patients(29.41%) vision improved between 6/12 and 6/9.

FIGURE 15:VISUAL OUTCOME IN POSTOPERATIVE UVEITIS

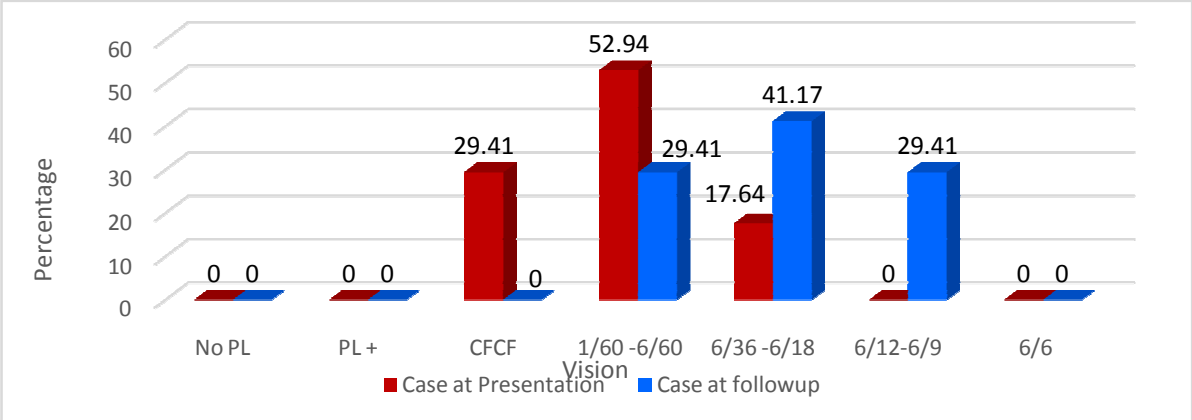
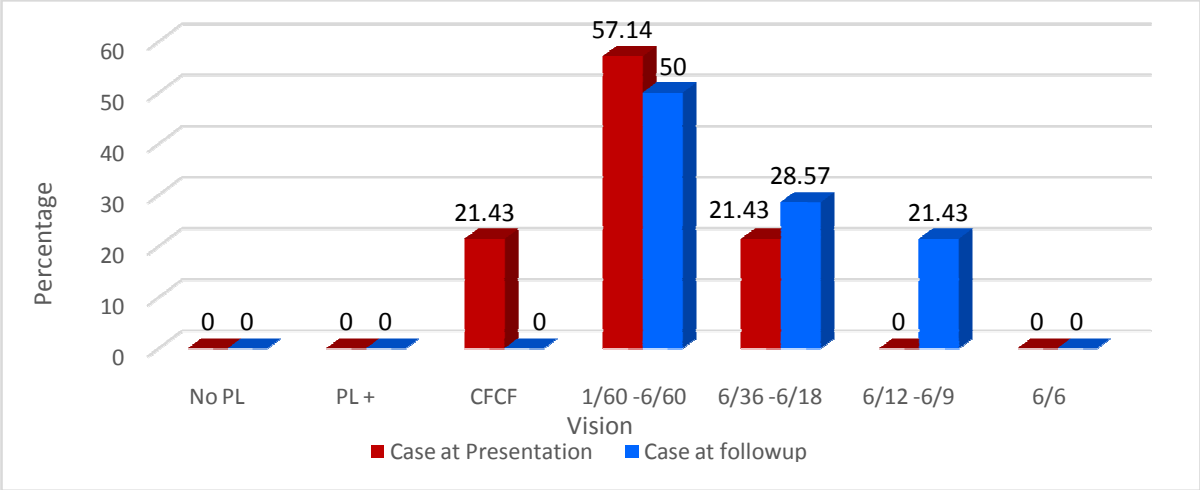


TABLE 16:VISUAL OUTCOME IN IDIOPATHIC UVEITIS

Vision	No of Cases at presentation	Percentage %	No of cases at Follow up	Percentage %
NO PL	-	-	-	-
PL +	-	-	-	-
CFCF	3	21.43	-	-
1/60 -6/60	8	57.14	7	50.00
6/36 -6/18	3	21.43	4	28.57
6/12 -6/9	-	-	3	21.43
6/6	-	-	-	-

Out of 14patients ,vision improved between 6/36 and 6/18 in 4 patients (28.57%) and 3 patients (21.43%) had vision between 6/12 and 6/9.

FIGURE 16:VISUAL OUTCOME IN IDIOPATHIC UVEITIS



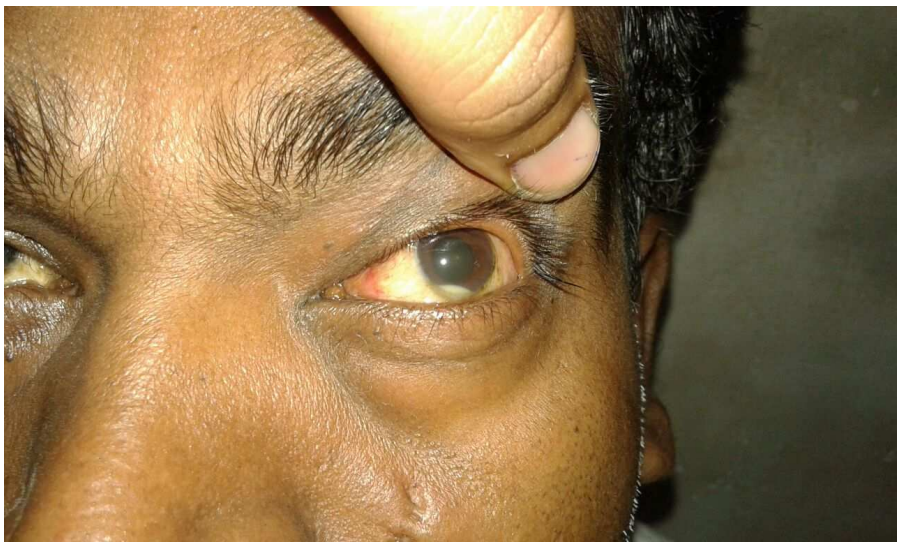


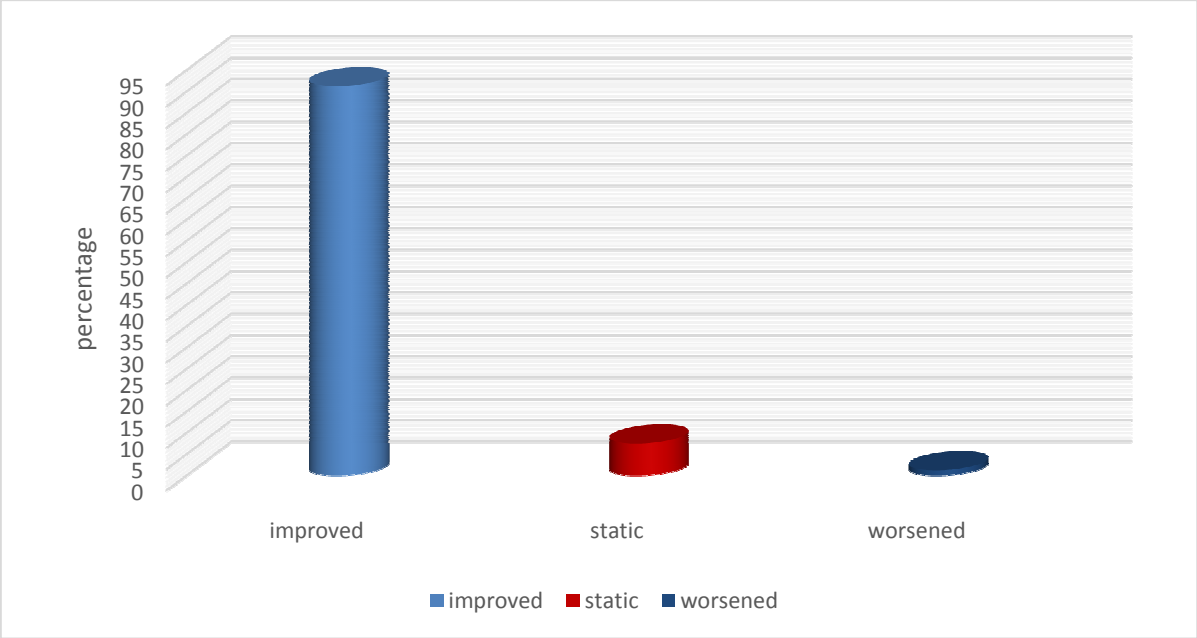
Figure xiii) Idiopathic anterior uveitis

TABLE 17:VISUAL OUTCOME FOLLOWING MANAGEMENT

VISUAL OUTCOME	NO. OF CASES	PERCENTAGE %
IMPROVED	73	91.25
STATIC	6	7.50
WORSENED	1	1.25

Out of 80 patients with anterior uveitis,73 patients (91.25%) showed improvement in visual outcome following treatment,6 (7.50%) patients showed no improvement and in 1 patient (1.25%)visual outcome got deteriorated.

FIGURE 17:VISUAL OUTCOME FOLLOWING MANAGEMENT



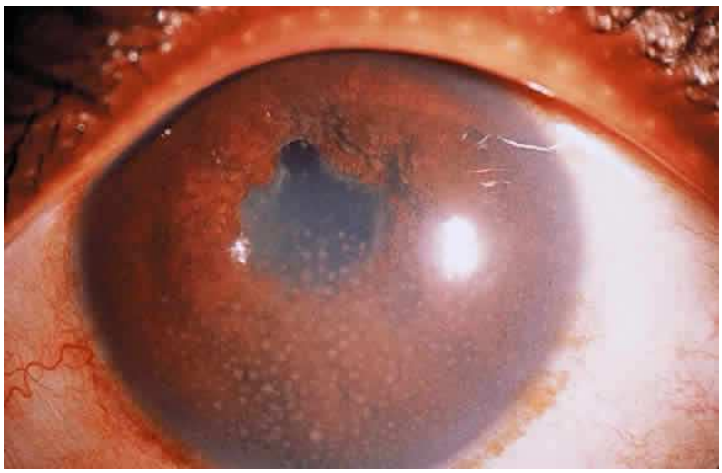


Figure xiv)uveitis not responding to treatment

DISCUSSION

This prospective study of 80 patients with anterior uveitis was done to analyse the final visual outcome following proper treatment.

In the earlier studies conducted in South India the ratio of uveitis in male to female was 1.5 :1. In our study the ratio is found to be 1.6:1.

In a study conducted by Rathinam et al about global variation in pattern and changes in epidemiology of uveitis (2007)[9], uveitis is 85.3% unilateral and 14.7% bilateral.

In our study 96.25% had unilateral and 3.75% had bilateral presentation of uveitis.

Regarding the study conducted by Rathinam et al(2007) ,60 – 80% of the patients were in the age group between 30 and 60 years .Among 23,980 patients included in the study, 5 – 16% belong to age group less than 16years and 6 – 21.8% were more than 60 years [15].

In our study 52.50% were in the age group between 30 and 60 years,22.50% belong to the age group less than 16 years and 26.50% were more than 60 years.

In a study of 155 patients with anterior uveitis by Leonardo (1991) 7.1% presented with hypopyon In our study 20% of patients presented with hypopyon.

In a study conducted by Rathinam et al about global variation in pattern and changes in epidemiology of uveitis (2007) [2] ,with regard to etiology in 44.6% uveitis is idiopathic,27.3% had infectious uveitis,3.9% had traumatic and 10.6% had lens induced uveitis.

In our study, 17.50% is idiopathic,8.75% had infective uveitis,47.50% had traumatic ,5% lens induced uveitis and 21.25% had post operative uveitis.

Holland and Helm (1993) made several conclusions from their study on uveitis in tuberculosis .The conclusions were – age ranged from 1-75,no gender predeliction, chronic granulomatous uveitis and majority had unilateral presentation.Iris nodules were rare.

In our study 2 cases of uveitis had tuberculosis etiology in the age group of 5 and 45 years , in female.Conglomerate granuloma was noted in one case.

In a data collected from 12 countries,Hormoz et all (1960 – 2009).The prevelance of idiopathic uveitis is 17.4% in 1960 and more recent investigation

to be reported to be 52%.In almost all these reports non -infectious causes predominated (45 – 94.8%) than infectious causes which are common in developing countries.

In our study non- infectious causes(73.75%) predominated infectious causes of uveitis.

In a study by OM Durrani regarding severity,duration and vision loss in uveitis ,vision loss in uveitis accounted 17.7% due to cataract and 26.8% due to macular edema [30]. In our study 7.50% had cataract, 6.25% had macular edema and 10% glaucoma.

In studies conducted earlier,Anterior uveitis had good visual prognosis showing improvement in 80% [17] and in our study visual outcome improved in 91.50% of the patients .

Blindness due to anterior uveitis is 4% and in our study blindness due to anterior uveitis is 1.25%

This study definitely indicates that uveitis threatens to become the major cause of ocular morbidity but patients get good final visual outcome with detailed clinical examination and initiation of proper management.

SUMMARY

A total of 80 patients with anterior uveitis of various etiologies who presented at Thanjavur Medical College,Thanjavur were studied from july 2014 – September 2015.

- The preponderance of male patient was noted with male : female ratio of 1.6:1.
- Unilateral eye involvement was more commoner than bilateral involvement.
- The commonest age group was found to be 11-20 yrs.
- Non-infectious etiology predominates the infectious etiology.
- In non- infectious etiology trauma was more commoner.
- Traumatic uveitis was commoner in children.
- Keratic precipitates and AC reaction was the common clinical sign.
- Complications like cataract and glaucoma were commoner.
- Postoperative uveitis were common in patients presented with preoperative uveitis.
- Prognosis of Visual outcome was good when treated earlier.
- Blindness due to anterior uveitis was 1%.

The etiology of anterior uveitis are diverse and varies with age and geographic location.

Proper evaluation of anterior uveitis involves careful consideration of its clinical features along with well planned tailored diagnostic approach.

The diagnosis and treatment of uveitis which is a major cause of visual morbidity is quite challenging inspite newer diagnostic techniques and treatment modalities.

Blindness due to its complications are inevitable but final visual outcome after initiation of meticulous treatment in anterior uveitis is generally good.

CONCLUSION

To conclude the study, Anterior uveitis can have benign clinical presentation but can cause serious complications if not diagnosed and treated on time.Recognition of ocular diagnostic clues is essential in making clinical diagnosis of specific entities as well as in guiding investigations

Corticosteroids have been the cornerstone in the treatment of uveitis.Steroids are used aggressively in a step ladder algorithm for resolution of inflammation through various routes and a gradual taper is essential to achieve a level of remission.Other treatment modalities include cycloplegics, NSAIDS, immunomodulators,systemic therapy with close monitoring of side effects.

Cases presented earlier even with severe uveitis had favourable response to treatment and good prognosis in visual outcome than those presented late with uveitic sequalae and with co-morbid risk factors irrespective of etiology.

Uveitis of unknown etiology still accounts as a cause of anterior uveitis with significant number of cases contributing to it.

The recommendations to improve the visual outcome in patients presenting with uveitis include :

- ❖ Detailed history taking including occupation,contact with pet animals,history for specific systemic disorders.
- ❖ Complete evaluation including general examination and ocular examination (slit lamp examination, tonometry, gonioscopy, indirect ophthalmoscopy,ultrasound B scan).
- ❖ Identification of high risk individuals at presentation.
- ❖ Investigations include serological and imaging studies.
- ❖ Clinical suspicion and Early diagnosis of uveitis.
- ❖ Therapeutic guidelines should be followed targeting complete elimination of active inflammation.
- ❖ Proper patient education to achieve therapeutic compliance
- ❖ Co-ordination among ophthalmologist, general physician, paediatrician and rheumatologist.

Though the proportion of blindness due to anterior uveitis is decreasing because of emerging treatment options, some patients experience vision loss at some point during their clinical course.

Visual morbidity following anterior uveitis can be prevented by prompt diagnosis and judicious management, achieving gratifying results in visual outcome.

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ANNEXURE I

A CLINICAL STUDY ON VISUAL OUTCOME FOLLOWING UVEITIS

PROFORMA

Name of the patient: **age/sex:**

Date:

Hospital no: **Address:**

Occupation:

History of present illness:

Onset and progression:

C/O Pain:

C/O Photophobia / redness / watering:

C/O Defective vision:

C/O Floaters:

H/O Trauma:

H/O Fever /cough/evening rise of temperature:

H/O Exanthematous rash:

H/O Joint pain/backache:

H/O Oro genital ulcers:

H/O contact with pet animals:

Past history:

H/O previous similar episodes:

H/O ocular surgery.

DM/HT/TB/SYPHILIS/SKIN DISEASES/ARTHRITIS

Treatment history:.

H/O Systemic medications.

Treatment for present illness.

Personal history:

Diet

Bladder and bowel habits.

General examination:

Anemia - pedal edema- Lymphadenopathy –

Examination of bone & joints –

Examination of skin-

Cardiovascular system – BP-

Respiratory system

GI/GU system –

Central nervous system –

Ocular examination:

OD **OS**

Vision-

Forehead / adnexa -

Lids-

Conjunctiva –

Cornea-

Edema:

KPs:

Others:

Anterior chamber –

Depth:

Cells:

Flare:

Hypopyon:

Others:

Pupil –

Size:

Shape:

Reaction to light:

Synechiae:

Iris –

Colour /pattern:

Nodules:

Lens -

Anterior vitreous-

EOM-

IOP-

Fundus –

B scan –

Clinical impression:

Investigations :

1.**Complete blood investigations** :

complete blood count –

RBS - ESR - LFT-

VDRL - RFT - lipid profile –

2.Urine routine :

3.Radiological investigations:

4.**skin tests:**

5.others:

Treatment

1.Mydriatics / Cycloplegics:

2.Steroids :

Topical:

Periocular:

Systemic:

3. Immunosuppressives:

4.Antibiotics:

Topical:

Systemic:

5.Antiglaucoma drugs:

Visual outcome :

ANNEXURE - II

KEY TO MASTER CHART

S.No.	:	Serial Number
M	:	Male
F	:	Female
BE	:	Both Eye
RE	:	Right Eye
LE	:	Left Eye
Conj.	:	Conjunctiva
AC	:	Anterior Chamber
IOP(I)	:	Intra Ocular Pressure (Initial)
IOP(F)	:	Intra Ocular Pressure (Follow Up)
F	:	Fundus
HZO	:	Herpes Zoster Ophthalmicus
SICS	:	Small Incision Cataract Surgery
PHACO	:	Phaco Emulsification
CCC	:	Circum Corneal Congestion
CT	:	Conjunctival tear
KPs	:	Keratic Precipitates
IP	:	Iris Pigment

RTL	:	Reacting To Light
SRTL	:	Sluggishly Reacting To Light
ST	:	Sphincter Tear
PS	:	Posterior Synechiae
SPS	:	Segmental Posterior Synechiae
APS	:	Annular Posterior Synechiae
C	:	Clear
LC	:	Lens Changes
IMC	:	Immature Cataract
MC	:	Mature Cataract
PCIOL	:	Posterior Chamber Intraocular Lens
ME	:	Macular Edema
RE	:	Retinal Edema
RD	:	Retinal Detachment
PL	:	Perception of Light
CFCF	:	Counting Fingers Close To Face

ANNEXURE III

LIST OF ABBREVATIONS USED

SUN	-	Standardization of Uveitis Nomenclature.
AAU	-	Acute Anterior Uveitis
KP	-	Keratic Precipitate.
IOP	-	Intra Ocular Pressure.
SPK	-	Superficial Punctate Keratitis
CCC	-	Circum Corneal Congestion.
AC	-	Anterior Chamber.
PL	-	Perception of Light.
CFCF	-	Counting Fingers Close to Face.
HLA	-	Human Leucocyte Antigen
NSAID	-	Non Steroidal Anti-Inflammatory Drugs.
APMPPE	-	Acute Posterior Multifocal Placoid Pigment Epitheliopathy
JRA	-	Juvenile Rheumatoid Arthritis
PPD	-	Purified Protein Derivative
TC	-	Total Count
DC	-	Differential Count
ESR	-	Erythrocyte Sedimentation Rate
VDRL	-	Veneral Disease Research Laboratory

CONSENT FORM

Name : Date :

Age : Sex :

I have been explained clearly about the research and its objectives. I understand the facts and I give full consent to be included as a participant in the " A CLINICAL STUDY ON VISUAL OUTCOME FOLLOWING UVEITIS."

- I have been explained about the nature of the study,clinical and laboratory investigations required for this study.
- I have been explained about my rights and responsibilities by the investigator.
- I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the regulatory authorities, Govt. agencies, and IEC.

- ❖ I have understood that my identity will be kept confidential .

- ❖ I have my questions being answered to my satisfaction.

- ❖ I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

Date : Signature of the patient

S.No.	Name	Age	Sex	Specific History	Eye	forehead/adexa	Lid	Conj.	Cornea	AC	Accells	Acflare	Pupil	Iris	Lens	IOP(I)	F	VISION at presentation	Diagnosis	IOP(F)	Visual outcome
1	Arun	17	M	Trauma	RE	N	N	CCC,CT	C	HYPHEMA	3+	3+	SRTL, ST	A	C	N	ME	6/60	Traumatic iritis/ME	N	6/12
2	Sethu	15	M	Trauma	LE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
3	Siva	14	M	Trauma	LE	N	N	CCC	EDEMA	N	2+	1+	RTL	N	C	N	N	6/18	Traumatic iritis	N	6/6
4	Arjun	16	M	Trauma	LE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
5	Sugumar	14	M	Trauma	RE	N	N	CCC	C	N	3+	2+	miosis	N	C	N	N	6/18	Traumatic iritis	N	6/6
6	Sankar	15	M	Trauma	RE	N	N	CCC	C	N	3+	2+	miosis	N	C	N	N	6/18	Traumatic iritis	N	6/6
7	Annamalai	62	M	Trauma	RE	N	N	CCC, CT	EDEMA	HYPHEMA	3+	3+	SRTL, ST	A	IMC	N	ME	6/60	Traumatic iritis/ME	N	6/18
8	Karthick	12	M	Trauma	RE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
9	Kumar	18	M	Trauma	RE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
10	Shankar	13	M	Trauma	LE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
11	Sethu	15	M	Trauma	RE	N	N	CCC, CT	C	HYPHEMA	2+	1+	SRTL, ST	N	C	N	N	6/18	Traumatic iritis	N	6/9
12	Kannan	14	M	Trauma	LE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
13	Elangovan	46	M	Trauma	LE	N	N	CCC	sealed CT,KP	HYPOPYON	4+	4+	PS	A	MC	26	-	HM	Traumatic iritis/MC	N	6/18

14	Surender	27	M	Trauma	LE	N	O	CCC	CT IP	HYP OPY ON	3+	3+	SRTL, ST	A	LC	28	-	4/60	Traumatic iritis/C	N	6/12
15	Sankaran	15	M	Trauma	LE	N	N	CCC, CT	C	HYP HEM A	3+	2+	SRTL, ST	A	C	N	ME	6/18	Traumatic iritis/ME	N	6/12
16	Manoharan	32	M	Trauma	RE	N	N	CCC	IP	N	2+	2+	PS	A	LC	1O	RD	CFCF	Traumatic iritis/RD	8	NO PL
17	Manikandan	15	M	Trauma	RE	N	O	CCC	IP	HYP HEM A	4+	4+	SRTL, ST	A	C	24	RE+ ME	CFCF	Traumatic iritis/ME	18	2/60
18	Indiragandhi	41	F	HZO	RE	VES ICL ES	O	CCC	KPs	HYP OPY ON	2+	1+	RTL	N	C	N	N	6/18	infectious uveitis	N	6/6
19	Shanthi	36	F	-	RE	VES ICL ES	O	CCC	SPKs	N	2+	1+	RTL	N	C	N	N	6/36	infectious uveitis	N	6/6
20	Arayee	63	F	HZO	LE	VES ICL ES	O	CCC	KPs	HYP OPY ON	3+	2+	RTL	A	MC	N	-	CFCF	infectious uveitis	N	3/60
21	Kumar	65	M	-	LE	N	N	CCC	EDE MA	N	4+	4+	SRTL	A	PCI OL	N	-	CFCF	Lens ind.uveitis	N	6/36
22	Thavamani	40	F	PHACO	RE	N	N	CCC	EDE MA	HYP OPY ON	4+	4+	SRTL	A	PCI OL	20	-	CFCF	postop uveitis	26	6/60
23	Muthulakshmi	46	F	PHACO	RE	N	N	CCC	KPs	HYP OPY ON	4+	4+	SRTL	A	PCI OL	18	-	CFCF	postop uveitis	22	6/18
24	Periyasamy	61	M	SICS	LE	N	N	CCC	KPs	N	3+	3+	RTL	A	PCI OL	N	-	1/60	postop uveitis	N	6/36
25	Chinnathal	67	F	SICS	LE	N	N	CCC	EDE MA	HYP OPY ON	4+	4+	RTL	A	PCI OL	N	-	1/60	postop uveitis	N	2/60
26	Jothivel	61	M	SICS	LE	N	N	CCC	KPs	HYP OPY ON	3+	2+	RTL	A	PCI OL	N	-	6/36	postop uveitis	N	6/12
27	Idumbaiyan	65	M	-	RE	N	N	CCC	EDE MA	N	4+	3+	SRTL	A	PCI OL	N	-	PL	Lens ind.uveitis	N	3/60

28	Savitri	55	F	-	RE	N	N	CCC	EDEMA	N	4+	4+	RTL	A	PCIOL	N	-	CFCF	Lens ind.uveitis	N	6/60
29	Ammakannu	70	F	SICS	RE	N	N	CCC	EDEMA	HYPOPYON	3+	3+	RTL	A	PCIOL	N	-	CFCF	postop uveitis	N	6/24
30	Kamachiyammal	60	F	SICS	LE	N	N	CCC	EDEMA	N	3+	3+	RTL	A	PCIOL	N	-	1/60	postop uveitis	N	6/60
31	Sivakkannu	62	F	SICS	LE	N	N	CCC	EDEMA	N	3+	3+	RTL	A	PCIOL	N	-	3/60	postop uveitis	N	6/24
32	Dhanakodi	65	M	SICS	RE	N	N	CCC	EDEMA	HYPOPYON	4+	3+	SRTL	A	PCIOL	18	-	CFCF	postop uveitis	24	5/60
33	Muthaiyan	62	M	SICS	RE	N	N	CCC	KPs	HYPOPYON	4+	4+	SRTL	A	PCIOL	N	-	1/60	postop uveitis	N	6/12
34	Shruti	5	F	TB	LE	N	N	CCC	KPs	HYPOPYON	4+	4+	RTL	CG	C	N	-	6/60	infectious uveitis	N	6/18
35	Dhanalakshmi	45	F	TB	BE	N	N	C	KPs	shallow	3+	3+	APS	A	MC	12	-	NO PL	infectious uveitis	10	NO PL
36	Suguna	34	F	-	BE	N	N	C	KPs	irregular	2+	2+	SPS	A	IMC	18	-	1/60	idiopathic uveitis	24	3/60
37	Muthukumar	36	M	-	BE	N	N	C	KPs	irregular	2+	2+	PS	A	IMC	N	-	6/24 5/60	idiopathic uveitis	N	6/60
38	karthickeyan	28	M	Trauma	LE	N	O	CCC	C	HYPHEMA	4+	3+	SRTL, ST	A	C	N	ME	4/60	Traumatic iritis/ME	N	6/36
39	Akash	18	M	-	LE	N	N	CCC	KPs	N	2+	2+	RTL	A	C	N	-	6/24	idiopathic uveitis	N	6/9
40	Shanthiya	17	F	-	LE	N	N	CCC	KPs	N	2+	2+	RTL	A	C	N	-	6/18	idiopathic uveitis	N	6/9
41	Saraswathi	55	F	-	LE	N	N	CCC	KPs	N	4+	4+	PS	A	IMC	20	-	6/60	idiopathic uveitis	26	6/60
42	Ganesan	52	M	-	LE	N	N	CCC	KPs	HYPOPYON	4+	3+	RTL	A	LC	N	-	HM	idiopathic uveitis	N	6/18

43	Leo	25	M	-	LE	N	N	CCC	EDEMA	HYPOPYON	4+	4+	PS	A	C	N	-	HM	idiopathic uveitis	N	6/12
44	pavunambal	55	F	-	RE	N	N	CCC	KPs	N	3+	3+	RTL	A	IMC	N	-	2/60	idiopathic uveitis	N	6/60
45	Yellambal	50	F	-	RE	N	N	CCC	KPs	N	2+	1+	RTL	A	IMC	N	-	3/60	idiopathic uveitis	N	6/60
46	Bala	23	M	-	RE	N	N	CCC	KPs	N	3+	2+	RTL	A	C	N	-	6/60	idiopathic uveitis	N	6/18
47	Shanthi	40	F	-	LE	N	N	CCC	KPs	N	3+	3+	RTL	A	LC	20	-	CFCF	idiopathic uveitis	28	6/18
48	Dinesh	16	M	Trauma	RE	N	N	CCC	EDEMA	HYPHEMA	3+	2+	miosis	N	C	N	N	6/36	Traumatic iritis	N	6/9
49	Mukil	14	M	Trauma	RE	N	O	CCC	C	N	3+	2+	miosis	N	C	N	N	6/60	Traumatic iritis	N	6/12
50	Anbu	16	F	Trauma	LE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
51	Akash	17	M	Trauma	LE	N	N	CCC	C	N	2+	2+	SRTL	N	C	N	N	6/36	Traumatic iritis	N	6/9
52	Barani	16	F	Trauma	LE	N	N	CCC	C	N	3+	1+	miosis	N	C	N	N	6/36	Traumatic iritis	N	6/9
53	Ezhil	32	M	Trauma	LE	N	O	CCC	C	N	3+	1+	miosis	N	C	N	N	6/18	Traumatic iritis	N	6/9
54	Harish	12	M	Trauma	RE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/9	Traumatic iritis	N	6/6
55	Saravanan	14	M	Trauma	LE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6
56	Ravi	30	M	Trauma	RE	N	N	CCC, CT	C	HYPHEMA	3+	1+	miosis	N	C	N	N	6/36	Traumatic iritis	N	6/9
57	Madhan	12	M	Trauma	RE	N	N	CCC	C	N	1+	2+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/9
58	Ilayaraja	26	M	Trauma	LE	N	O	CCC	C	N	2+	1+	miosis	N	C	N	N	6/18	Traumatic iritis	N	6/9

59	Sundar	18	M	Trauma	LE	N	O	CCC	C	N	2+	1+	miosis	N	C	N	N	6/36	Traumatic iritis	N	6/9
60	Prabu	8	M	Trauma	RE	N	O	CCC	C	N	1+	1+	RTL	N	C	N	N	6/9	Traumatic iritis	N	6/6
61	Archana	18	F	Trauma	RE	N	N	CCC	C	N	1+	1+	RTL	N	N	N	N	6/12	Traumatic iritis	N	6/6
62	Divya	13	F	Trauma	LE	N	N	CCC	C	N	1+	1+	RTL	N	N	N	N	6/12	Traumatic iritis	N	6/6
63	Vignesh	14	M	Trauma	RE	N	O	CCC	C	N	3+	2+	miosis	N	N	N	N	6/18	Traumatic iritis	N	6/6
64	Shenbagavalli	39	F	HZO	LE	VESICLES	O	CCC	SPKs	N	3+	2+	SRTL	N	N	N	N	6/36	infectious uveitis	N	6/9
65	Sathish	19	M	Trauma	RE	N	N	CCC, CT	C	N	3+	3+	SRTL	N	N	12	RD	CFCF	Traumatic iritis/RD	10	CFCF
66	Sundari	37	F	-	LE	VESICLES	O	CCC	SPKs	N	2+	1+	SRTL	N	N	N	N	6/60	infectious uveitis	N	6/9
67	Nagalakshmi	52	F	-	RE	N	N	N	KPs	N	2+	2+	PS	A	IMC	22	N	6/60	idiopathic uveitis	24	6/60
68	Parvathy	42	F	SICS	RE	N	N	CCC	EDEMA	N	2+	1+	SRTL	N	PCIOL	N	N	6/60	postop uveitis	N	6/9
69	Saroja	57	F	SICS	LE	N	N	CCC	EDEMA	N	3+	2+	RTL	N	PCIOL	N	ME	3/60	postop uveitis	N	6/36
70	Valli	62	F	SICS	LE	N	N	CCC	EDEMA	HYPOPYON	4+	4+	RTL	N	PCIOL	N	ME	CFCF	postop uveitis	N	3/60
71	Chandrasekar	61	M	SICS	LE	N	N	CCC	EDEMA	HYPOPYON	3+	2+	RTL	N	PCIOL	N	N	6/60	postop uveitis	N	6/24
72	Govindasamy	52	M	SICS	RE	N	N	CCC	EDEMA	N	2+	1+	RTL	N	PCIOL	N	N	6/36	postop uveitis	N	6/12
73	Kulandaisamy	60	M	-	LE	N	N	CCC	EDEMA	N	4+	4+	RTL	N	PCIOL	20	N	CFCF	Lens ind.uveitis	28	2/60
74	kamalam	46	F	SICS	RE	N	N	CCC	EDEMA	N	2+	1+	RTL	N	PCIOL	N	N	6/36	postop uveitis	N	6/9

75	Saithambal	68	F	-	RE	N	N	C	KPs	N	_	-	PS	N	IMC	16	N	6/60	idiopathic uveitis	24	6/60
76	Prabakaran	36	M	Trauma	RE	N	O	CCC	KPs	N	3+	1+	SRTL, ST	N	C	14	RD	CFCF	Traumatic iritis/RD	12	CFCF
77	Geetha	44	F	-	RE	N	N	C	KPs	N	-	-	PS	N	IMC	N	N	6/36	idiopathic uveitis	N	6/18
78	Kumarasamy	60	M	SICS	RE	N	O	CCC	KPs	N	3+	1+	RTL	N	PCIOL	N	N	6/60	postop uveitis	N	6/24
79	Praveen	27	M	Trauma	LE	N	N	CCC	C	HYPHEMA	2+	1+	miosis	N	C	N	N	6/18	Traumatic iritis	N	6/6
80	Venkatesh	24	M	Trauma	RE	N	N	CCC	C	N	1+	1+	RTL	N	C	N	N	6/12	Traumatic iritis	N	6/6