"PATTERN OF UVEITIS IN A RURAL TERTIARY EYE CARE CENTRE"

By

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MASTER OF SURGERY

IN

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Under the Guidance of
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Tamaka, Kolar

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Date: **Signature of the Candidate**

LIST OF ABBREVIATIONS USED

MHC	»	Major Histocompatibility Complex
APC	»	Antigen Presenting Cells
IOP	»	Intraocular Pressure
HLA	»	Human Leucocyte Antigen
KP	»	Keratic Precipitates
MS	»	Multiple Sclerosis
HIV	»	Human Immunodeficiency Virus
VDRL	»	Veneral Disease Research Laboratory
TPHA	»	Treponema Pallidum Haemagglutination
ELISA	»	Enzyme linked Immunosorbent Assay
FFA	»	Fundus Flurosceine Angiography
VKH	»	Vogt Koyanagi Harada
CME	»	Cystoid Macular Edema

ABSTRACT

Aims:

The pattern of uveitis in different populations across the globe is influenced by the environmental, geographical and genetic factors with consequent unique epidemiology for the given geographical region. The aim of our study was two-fold (a) To study the pattern of uveitis in Kolar region and compare it with the other regions. (b) To identify common causes of uveitis in our region.

Materials and Methods:

In this study 50 new cases of uveitis, hailing from Kolar region were examined and evaluated for detecting type and etiology of uveitis and their complications. SPSS software and the Chi-square test were used for statistical analysis.

Results:

The mean age of onset of uveitis was around 40 years. Anterior uveitis was the most frequently occurring form accounting for (48%) of all patients, followed by posterior uveitis (36%), panuveitis (10%) and intermediate uveitis (6%). The etiology remained undetermined in majority of the case (52%). The most common infective cause was

Tuberculosis; Rheumatoid arthritis being the leading non- infective etiology.

Conclusion:

The most common types of uveitis in our patient population were idiopathic, anterior, chronic, non-granulomatous, and noninfectious with no significant gender predisposition. Improving the laboratory facilities in the rural setup for early diagnosis and treatment of uveitis is the need of the hour so as to prevent visual complications.

KEYWORDS

Uveitis, Epidemiology, Etiology, Referral pattern, Complications.

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INTRODUCTION

INTRODUCTION

The term *uveitis*, derived from *uva*, the Latin word for grape, encompasses a broad range of disease processes involving the uveal tract (i.e. the iris, ciliary body, and choroid) and its associated ocular structures including the retina, optic nerve and vitreous. It may also be a part of systemic inflammatory disease.

The etiology is difficult to establish since the exact cause of uveitis frequently remains unknown. Therefore the current International Uveitis Study Group classification system is based on the anatomical location of the inflammation: anterior uveitis (iris and ciliary body), posterior uveitis (choroid and retina), intermediate uveitis (peripheral retina and pars plana of the ciliary body), and panuveitis (generalized inflammation of the whole uvea).[1]

Causes of uveitis are known to vary in different populations depending upon the ecological, racial and socioeconomic variations of the populations studied. The cause of inflammation might be infections agent or trauma, but in most cases it the underlying mechanism appears to be autoimmune in nature.

Tropical countries are unique in their climate, prevailing pathogens and existing diseases, which further influence the epidemiological and geographical distribution of specific entities. Awareness of such regional differences in the disease pattern is essential in deriving a region-specific list of differential diagnosis which in turn facilitates the final diagnosis.[2]

The importance of diagnosing uveitis lies in the fact that Uveitis is estimated to account for ~10% of visual handicap in the Western world and is responsible for 30000 new cases of legal blindness each year. A number of retrospective studies have examined the incidence and prevalence of uveitis in various parts of the world. The annual incidence of uveitis is estimated between 17 and 52 per 100 000 person-years

with a prevalence of 38-714 cases per 100000 persons.[3-5] The incidence of uveitis in India as reported by the various hospital based studies ranges from 0.8-1.8% of all new ophthalmic patients.[2,6] In order to enhance understanding and management of ocular inflammation International ocular Inflammation Society (IOIS) has been founded .[7]

Deleterious effects on vision, either by acute ocular inflammation or by its sequelae, such as cataracts, glaucoma and retina vascular ischemia, make uveitis one of the major causes of visual loss.[8]

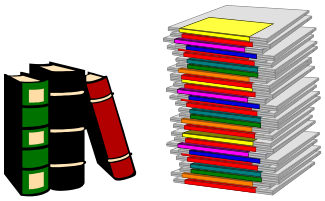
Uveitis may occur in any age group, but most commonly seen in the 30 to 39 year old age group. It is less common in children below 10 years and in patients over 60 years.[6]

We performed this study in a tertiary eye center in rural Kolar region, South India with the objective of determining the pattern and causes of uveitis, the associated systemic conditions and to compare this pattern seen elsewhere in the world. This study will facilitate in deriving a region specific list of differential diagnosis which in turn will help in taking preventive steps towards visual complications.

AIMS AND OBJECTIVES

The aim of our study is two-fold.

- To study the pattern of uveitis in Kolar region and compare it with the other regions.
- To identify common causes of uveitis in our region.



REVIEW OF LITERATURE

REVIEW OF LITERATURE:

ANATOMY

The uveal tract is the middle vascular coat of the eye, lying between the sclera and the neuroepithelium. It consists of the iris, ciliary body, and choroid. The former represents the anterior part, the latter the posterior part, and the ciliary body forms the middle part. However, the entire uveal tract is developmentally, structurally and functionally one indivisible structure. The uvea contains nerves, supporting connective tissue, and a variable number of melanocytes that are responsible for its distinctive color.

THE IRIS

The iris forms a diaphragm in front of the crystalline lens. The iris controls the amount of light transmitted into the eye by changes in the pupillary size. The vascular supply to the iris originates in the anterior and long posterior ciliary arteries. **Histologically** the iris is made up of three layers: (1) an anterior layer composed of fibroblasts, melanocytes, and collagen, with its anterior surface folded into many ridges and crypts; (2) a middle stromal layer containing fibroblasts, melanocytes, and collagen; and (3) a posterior layer composed of the dilator muscle and pigment epithelium. The stroma, or middle layer, makes up the bulk of the iris and contains blood vessels, nerves, melanocytes, and clump cells in a loose extracellular matrix of collagen and mucopolysaccharides.[9] The iris color is determined by the number and degree of melanin granules in the superficial stromal melanocytes.[10]

The posterior epithelium is velvety smooth and uniform. It consists of two layers of densely pigmented cells, which are arranged apex to apex.[11]

The dilator muscle of the pupil extends from the region of the sphincter muscle to the base of the iris and is located in the posterior portion of the iris. The sphincter muscle is located in the posterior iris stroma in the pupillary zone and consists of a circular band of smooth muscle fibers. The dilator muscle is innervated by parasympathetic nerves and the sphincter muscle by the sympathetic nervous system.

CILIARY BODY

The ciliary body extends from the base of the iris and becomes continuous with the choroid at the ora serrata. It is approximately 6-6.5 mm in antero-posterior dimension. It consists of an anterior portion called the pars plicata and a posterior portion called the pars plana. The pars plicata contains approximately 70 finger like projections called ciliary processes, which are covered by the ciliary body epithelium. The pars plicata is the flat part of the ciliary body and ends at the ora serrata. The ciliary processes are thin and fingerlike in children. With age they become thickened and reveal hyalinization of the stroma. The stroma of these areas is filled with fibrous connective tissue, rich vascular networks, melanocytes, and bundles of smooth muscle.

The smooth muscle of the ciliary can be divided into three group of fibers; the outer longitudinal portion that attaches anteriorly to the scleral spur and trabecular meshwork fibers, a middle oblique portion, and the inner circular portion.[12] Accommodation is the result of a parasympathetic stimulus that is followed by contraction of the ciliary muscle, which decreases the zonular tension on the crystalline lens. This allows the lens to move forward and assume a more spherical shape increasing the dioptric power of the eye.

The innermost part of both pars plana and pars plicata is covered by a bilayer epithelium: the outer pigmented epithelium cells and the inner nonpigmented cells. The cells of these two layers are arranged apex to apex with tight junctions between them. The zonula occludens near the apices of the nonpigmented epithelial cells from the blood-aqueous barrier. The principal source of aqueous humor is the nonpigmented ciliary epithelium of the pars plicata. Production of the vitreous mucopolysaccharide has been attributed to the nonpigmented ciliary epithelium of the pars plana.

CHOROID

The choroid is the principal vascular and pigmented tissue that forms the middle coat of the posterior part of the eye. It extends from the ora serrata to the optic nerve.[13] It is attached to the sclera by connective tissue strands and especially posteriorly, by numerous blood vessels and nerves that enter the choroid from the sclera. Small amounts of choroidal tissue may extend into the scleral canals through which ciliary vessels and nerves enter the eye.

Histologically the choroid reveals four layers: the lamina fusca, the stroma, the choriocapillaris, and Bruch's membrane. Bruch's membrane can be divided into five components: the basement membrane of the retinal pigment epithelium, an inner collagenous zone, an elastic layer, the outer collagenous zone, and the basement membrane of the endothelium of the choriocapillaris. The choriocapillaris is the capillary layer of the choroid and provides nutrition to the retinal pigment epithelium and outer retinal layers (photoreceptor cell and outer plexiform layers and the outer aspect of the inner nuclear layer). It appears to be arranged in lobules with a central arteriolar feeder.[14] The endothelial cells lining the choriocapillaris are fenestrated and

are joined by gap junctions. The stroma of the choroid contains larger arteries and veins. These vessels are not fenestrated. The lamina fusca is the transition zone between sclera and choroid. It consists of a delicate meshwork of elastic fibers, fibrocytes, and melanocytes, traversed by long posterior ciliary nerves and vessels.

BLOOD SUPPLY OF UVEAL TRACT[15]

Arterial supply: The uveal tract is supplied by three sets of arteries-

1. Short posterior ciliary arteries. These arise as two trunks from the ophthalmic artery; each trunk divides into 10-20 branches which pierce the sclera around the optic nerve and supply the choroid in a segmental manner.

2. Long posterior ciliary arteries. These are two in number, nasal and temporal. These pierce the sclera obliquely on medial and lateral side of the optic nerve and run forward in the suprachoroidal space to reach the ciliary muscle, without giving any branch. At the anterior end of ciliary muscle these anastomose with each other and with the anterior ciliary arteries; and gives branches which supply the ciliary body.

3. Anterior ciliary arteries. These are derived from the muscular branches of ophthalmic artery. These are 7 in number; 2 each from arteries of superior rectus, inferior rectus, and medial rectus muscle and one from that of lateral rectus muscle. These arteries pass anteriorly in the episclera, give branches to sclera, limbus and conjunctiva; and ultimately pierce the sclera near the limbus to enter the ciliary muscle; where they anastomose with the two long posterior ciliary arteries to form the circulus arteriosus major, near the root of iris. Several branches arise from the circulus arteriosus major and supply the ciliary processes (one branch for each process). Similarly, many branches from this major arterial circle run radially through the iris towards pupillary margin, where they anastomose with each other to form circulus arteriosus minor.

Venous drainage: A series of small veins which drain blood from the iris, ciliary body and choroid join to form the vortex veins. The vortex veins are four in number– superior temporal, inferior temporal, superior nasal and inferior nasal. They pierce the sclera behind the equator and drain into superior and inferior ophthalmic veins which in turn drain into the cavernous sinus.

EPIDEMIOLOGY

Uveitis is of global distribution. The reported prevalence of uveitis has varied widely. A number of retrospective studies have examined the incidence and prevalence of uveitis in various parts of the world. Various factors may influence these statistics, including age, gender, ethnicity, endemic infectious disease, and genetic background within a specific area or as yet unknown factors; the clinical criteria used to detect early stages and/or more subtle changes, the method and thoroughness of examination and awareness of the examiner

Uveitis is estimated to account for ~10% of visual handicap in the Western world and is responsible for 30000 new cases of legal blindness each year.[3] Uveitis may occur in any age group, but most commonly affects those aged 20 and 44 years. The annual incidence of uveitis is estimated between 17 and 52 per 100 000 person-years with a prevalence of 38-714 cases per 100000 persons.[5,16,17]

In the Indian scenario various studies have reported that new cases of uveitis amount to 0.8%-1.8% of all new ophthalmic patients.[2,6]

Anterior uveitis has been reported to be the most common form of uveitis, accounting for 50-60% of all uveitis cases in tertiary referral centers. Posterior uveitis remains the

second most common form of uveitis, accounting for 15-30% of cases. Intermediate uveitis remains the least common form of uveitis and most cases are idiopathic.[1,18]

However, the relative frequencies of various types of uveitis may not reflect the experience of community-based ophthalmologists because many of the published case series come from tertiary referral centers. According to the UCLA Community-Based Uveitis Study Group, the anatomic distribution of uveitis differed significantly between community-based practices (anterior 90.6%, intermediate 1.4%, posterior 4.7%, panuveitis 1.4%)[19] and university referral practices (anterior 60.6%, intermediate 12.2%, posterior 14.6%, and panuveitis 9.4%).

Childhood onset uveitis accounts for fewer than 10% of all cases.[20]

Uveitis in children accounts for 5-10% of patients with uveitis seen in a tertiary referral center and occurs with greater frequency in girls than in boys. Anterior uveitis accounts for 30-40% of cases, posterior uveitis accounts for 40-50%, and intermediate uveitis accounts for 10-20%.[21] According to a study from Finland, the annual incidence of uveitis in children is four per 100,000 persons and the prevalence is 28 per100,000 persons.[22]

According to a survey of patients in the United States adults 65 years and older, the incidence of uveitis averaged 340.9/100000 persons/year. Anterior uveitis was the most common form of uveitis diagnosed, with an incidence of 243.6 cases per 100,000persons/year. In the 9-year study period from 1991-99, the prevalence of uveitis in elderly patients doubled from 511/100000 in 1991 to 1231/100000 in 1999.[23]

CLASSIFICATION

Uveitis is largely classified according to the classification scheme recommended by the International Uveitis Study Group[1] and the Standardization of Uveitis Nomenclature (SUN) Working Group[24] based on anatomic location.

This classification is not only useful in aiding with the preparation of a differential diagnosis in clinical setting but also provides for a more standard international classification that will enable clinicians and investigators to discuss and compare their cases with greater uniformity.[5]

Table 1: The classification of Uveitis

TYPE	PRIMARY SITES of INFLAMMATION	INCLUDES
Anterior uveitis	Antrerior chmber	Iritis, Iridicyclitis, anterior cyclitis
Intermediate uveitis	Vitreous	Pars planitis, posterior cyclitis, Hyalitis
Posterior uveitis	Retina or choroid	Choroiditis, chorioretinitis, retinochoroidits, retinitis, neuroretinitis
Panuveitis	Anterior chamber, vitreous, and retina or choroid	

The SUN Working Group also reached consensus on clinical descriptors of the timing of uveitis regarding onset, duration, and course of uveitis. Onset may be divided into

acute and *insidious* onset. Duration of the uveitis attack is classified as *limited*, if it lasts 3 months or less, and *persistent*, if it lasts greater than 3 months. The course of disease may be characterized as *acute* if the episode is of sudden onset and limited duration, *recurrent*, if repeated episodes are experienced with periods of inactivity without treatment lasting 3 months or greater in duration, or *chronic*, if persistent uveitis is present with relapse in less than 3 months after discontinuing therapy.

Table 2: The SUN Working Group Descriptors of Uveitis

CATEGORY	DESCRIPTOR	COMMENT
Onset	Sudden	
	Insidious	
Duration	Limited	≤3 months duration
	Persistent	> 3 months duration
Course	Acute	Episode characterized by sudden onset and limited duration.
	Recurrent	Repeated episodes separated by periods of inactivity without treatment <3 months in duration.
	Chronic	Persistent uveitis with relapse in <3 months after discontinuing treatment.

Table 3: **International Uveitis Study Group (IUSG) Proposed Clinical Classification of Uveitis**

Infectious	**Non-infectious**
Bacterial	Known systemic association
Viral	No known systemic association
Fungal	**Masquerade**
Parasitic	Neoplastic
Others	Nonneoplastic

Etiological or Duke Elder classification:

1) Uveitis wherein infective element is dominant:

   a) Exogenous

      i) Wound infection

      ii) Parasitic entry

   b) From neighboring structure by direct continuity

      i) Extra ocular

      ii) Ocular

   c) Endogenous: metastatic or occurring in the course of a general infection – bacterial, rickettsial, viral, mycotic or parasitic.

2) Uveitis wherein element of hypersensitivity is dominant

   a) Anaphylactic and atopic uveitis

   b) Uveitis due to bacterial (delayed) allergy

   c) Autoimmune uveitis

Focal infections

3) Toxic uveitis:

a) Endogenous toxins

i) Auto intoxication

ii) Organismal toxins

b) Endocular toxins – atrophic, hemorrhagic, neoplastic

c) Exogenous chemical irritants

4) Traumatic uveitis

5) Uveitis associated with non-infective systemic disease

a) Sarcoidosis

b) Collagen and related diseases

c) Disease of central nervous system

d) Diseases of skin

6) Uveitis of unknown etiology

a) Sympathetic ophthalmitis

b) Heterochromic iridocyclitis

D) Pathological classification:

1) Suppurative or purulent uveitis.

2) Non suppurative uveitis (Wood's classification)

i) Non granulomatous

ii) Granulomatous

ETIOLOGY AND PATHOGENESIS OF UVEITIS[25]

In the various types of uveitis encountered in humans in whom the mechanism is known, there main underlying causes occur.

- Reaction to trauma
- Autoimmune reaction
- Response to an infectious agent

Reaction to trauma:

Uveitis related to trauma could follow a blunt trauma or a penetrating injury.

Related to blunt trauma[26]:

The pathological basis of traumatic iridocyclitis following blunt trauma is a spasmodic and temporary ischaemia followed by a prolonged paretic vasodilatation associated with much endothelial damage. The reactions may be transient resulting in an inflammatory reaction in anterior uveal tract.

After more severe injury a plastic iridocyclitis may develop with the formation of exudative pupillary and cyclitic membranes which may excite a secondary glaucoma.

Related to penetrating injury:

Sympathetic ophthalmitis is a serious bilateral granulomatous panuveitis which follows a penetrating injury to one eye. The injured eye is called exciting eye and fellow eye which also develops uveitis is called sympathizing eye. Exact etiology is not known. Sympathetic ophthalmitis is believed to represent an autoimmune reaction to component or components of retina, retinal pigment epithelium or choroids (retinal 'S' antigen tyrosinase related protein).[27]

Cell mediated immune responses to retinal antigens also have been found in patients who have sympathetic ophthalmitis.[28] The specific antigen or antigens involved are not identified. A penetrating wound appears to be essential for development of uveitis. Additional insults, such as vitrectomy and laser or radiation therapy, may increase the incidence.[29]

Autoimmune reactions:

In the case of autoimmune component, this could be either a direct response to autoantigens as a consequence of tolerance breakdown, or a secondary reaction to autoantigens that results from damage to ocular structures from other causes.

Possible mechanisms of tolerance breakdown and autoimmunity[25]:

- Presence of certain MHC genotype that could lead to:
  - Presence of autoreactive T cells
  - Absence of immunoregulatory T cells.
- Release of normally sequestered auto antigens due to:
  - Trauma
  - Infection
- Alteration of autoantigen resulting from:
  - Tissue injury
  - Inflammation
- Expression of co stimulators on APCs can overcome peripheral tolerance.
- Polyclonal stimulation of self reactive lymphocytes by
  - Lipopolysaccharides for B cells
  - Bacterial superantigens for T cells.
- Molecular mimicry due to homology between :

- Pathogens and host tissue antigens
- Microbial proteins and certain HLA antigens.

Infectious uveitis:

In matter of infectious uveitis, toxoplasma and other infectious agents have been implicated in many cases.

The inflammatory response observed in infectious uveitis could result from following scenarios:

1) A reaction to noxious agents such as toxins produced by pathogens.

2) An immune response to a particular pathogen.

3) Cause of similarity or cross reaction in antigenic structure between the two. This cross reaction or homology is termed molecular mimicry and is postulated to be of the mechanisms by which an autoimmune response could be initiated.

There has been speculation about molecular mimicry playing a role in certain diseases with genetic predisposition correlation between expression of various HLAs and certain immune diseases have been noted for antigens of HLA class I and II.

Because MHC class II molecules are involved in selection and activation of CD4+ T cells and these T cells play an integral part in the regulation of immune response an association of MHC class II antigens and disease prevalence is believed. This is demonstrated in studies of sympathetic ophthalmia, Vogt-Koyanagi Harada syndrome. In these instances disease process is initiated by cross reactivity between microbial proteins and certain HLA antigens resulting in autoimmune response.

Studies have analyzed disease induction by HLA peptides that have a homology in their sequence to certain retinal antigens.[30]

Although the expression of particulars HLA gene product may not by itself be the cause of any autoimmune disease, it may be one of several factors that contribute to breakdown of tolerance.

Idiopathic uveitis:

1) Idiopathic specific uveitis group include conditions which have certain special characteristics of their own. Eg. Fuch's heterochromic iridocyclitis.
2) Non specific idiopathic uveitis group includes conditions which do not belong to any known etiologic groups.

CLINICAL FEATURES

ANTERIOR UVEITIS

SYMPTOMS[15]

1. **Pain.** It is dominating symptom of acute anterior uveitis. Patients usually complain of a dull aching throbbing sensation which is typically worse at night. The ocular pain is usually referred along the distribution of branches of fifth nerve, especially towards forehead and scalp.

2. **Redness**. It is due to circumcorneal congestion, which occurs as a result of active hyperaemia of anterior ciliary vessels due to the effect of toxins, histamine and histamine-like substances and axon reflex.

3. **Photophobia and blepharospasm** observed in patients with acute anterior uveitis are due to a reflex between sensory fibers of fifth nerve (which are irritated) and motor fibers of the seventh nerve, supplying the orbicularis oculi muscle.

4. **Lacrimation** occurs as a result of lacrimatory reflex mediated by fifth nerve (afferent) and secretomotor fibres of the seventh nerve (efferent).

5. **Defective vision** in a patient with iridocyclitis may vary from a slight blur in early phase to marked deterioration in late phase. Factors responsible for visual disturbance include induced myopia due to ciliary spasm, corneal haze (due to oedema and KPs), aqueous turbidity, pupillary block due to exudates, complicated cataract, vitreous haze, cyclitic membrane, associated macular oedema, papillitis or secondary glaucoma.

SIGNS

Slit lamp biomicroscopic examination is essential to elicit most of the signs of uveitis.

I. Lid oedema usually mild, may accompany a severe attack of acute anterior uveitis.

II. Circumcorneal congestion is marked in acute iridocyclitis and minimal in chronic iridocyclitis. It must be differentiated from superficial congestion occurring in acute conjunctivitis.

III. Corneal signs include; corneal oedema, KPs and posterior corneal opacities

1. Corneal oedema is due to toxic endothelitis and raised intraocular pressure when present.

2. Keratic precipitates (KPs) are proteinaceous cellular deposits occurring at the back of cornea. Keratic precipitates are the most common corneal findings in iridocyclitis. They are small aggregates of inflammatory cells that accumulate on the endothelial surface of the cornea.[31] Histologically the KPs are made up of cells enmeshed in a network of fibrin, usually lymphocytes, plasma cells and large mononuclear phagocytes, sometimes associated with granules of uveal pigment.[32] In most inflammatory reactions, the neutrophils are the first cells to appear, and the transformed macrophages (epithelioid cells) and lymphocytes accumulate as the

inflammation becomes chronic. After resolution of active inflammation, the KPs may disappear completely or become smaller, translucent or pigmented.[31]

The following observations should be made and recorded.

Distribution of KPs:

Very often they are found in the central and lower half of the cornea, often in a base-down triangle configuration (Arlt's triangle) or in a linear vertical formation (Turk's line).[33] This distribution is caused by the aqueous humour convection current in the anterior chamber. They may also disseminate diffusely all over the cornea as in Fuch's heterochromic iridocyclitis. The composition and morphology of KPs varies with the severity, duration and type of uveitis. The KPs vary in size from flecks, the size of cornea guttata to 1 mm in diameter.[34]

Following types of KPs may be seen:

i. Mutton fat KPs. These typically occur in granulomatous iridocyclitis and are composed of epithelioid cells and macrophages. They are large, thick, fluffy, lardaceous KPs, having a greasy or waxy appearance. Mutton fat KPs are usually a few (10 to 15) in number.

ii. Small and medium KPs (granular KPs). These are pathognomic of non-granulomatous uveitis and are composed of lymphocytes. These small, discrete, dirty white KPs are arranged irregularly at the back of cornea. Small KPs may be hundreds in number and form the so called endothelial dusting.

iii. Red KPs. These are formed when in addition to inflammatory cells; RBCs also take part in composition. They may be seen in haemorrhagic uveitis.

iv. Old KPs. These are sign of healed uveitis. Either of the above described KPs with healing process shrink, fade, become pigmented and irregular in shape (crenated

margins). Old mutton fats KPs usually have a ground glass appearance due to hyalinization.

3. Posterior corneal opacity may be formed in longstanding cases of iridocyclitis.

IV. Anterior chamber signs

1. Aqueous cells. Presence of cells in the aqueous indicates an active inflammation in the iris and ciliary body. The pattern of circulation of cells in anterior chamber should always be noted. The cells in aqueous move in same pattern as aqueous.[32] Polymorphonuclear cells are the predominant cells in the acute cases; in chronic cases they tend to be lymphocytes, plasma cells, monocytes and macrophages. Inflammatory cells are small, spherical, glistening and non-pigmented. Macrophages are larger, especially when they have ingested blood or melanin.

The cells should be graded according to the number observed in the oblique slit beam. The light intensity and magnification of the slit lamp should be maximal and the wide beam and narrow slit of 3 x 1 mm

Table 4: **Grading anterior chamber cells**

Cells in field	Grade
<1	0
1–5	±
6–15	+1
16–25	+2
26–50	+3
>50	+4

When exudation within the anterior chamber is quite severe the cellular elements tend to layer down, along the inferior zone giving rise to a hypopyon. This is normally sterile and may be seen to shift into the dependent zone, on changes in head position.

2. Aqueous flare. Increased protein content in the aqueous humour is a manifestation of a breakdown of the blood-ocular barrier and produces a Tyndall effect similar to the effect produced by moving picture projector beam when it crosses a darkened smoky room.[32] When the slit-lamp beam is obliquely aimed across the anterior-chamber, the ability to visualize the path of the beam is termed flare. The amount of light scattering is proportional to the, concentration of protein in a solution and hence more flare indicates increased protein in the anterior-chamber fluid. The flare is graded according to the method of Hogan and co-workers from 0 to 4+ as shown in the table, using the same slit-lamp setting as used for grading cells.

Table 5: Grading of anterior chamber flare (Hogan et al)[35]:

Description	Grade
Complete absence	0
Faint – just detectable	1 +
Moderate – Iris and lens details clear	2 +
Marked – iris and lens details hazy	3 +
intense – fixed coagulated aqueous with considerable fibrin	4 +

Occasionally the aqueous has an exceptionally high fibrin content and gives rise to iritis called “plastic” iritis.[36]

3. Hypopyon. When exudates are heavy and thick, they settle down in lower part of the anterior chamber as hypopyon (sterile pus in the anterior chamber).

4. Hyphema (blood in the anterior chamber): It may be seen in haemorrhagic type of uveitis.

5. Changes in depth and shape of anterior chamber may occur due to synechiae formation.

6. Changes in the angle of anterior chamber are observed with gonioscopic examination. In active stage, cellular deposits and in chronic stage peripheral anterior synechiae may be seen.

V. Iris signs

1. Loss of normal pattern. It occurs due to oedema and waterlogging of iris in active phase and due to atrophic changes in chronic phase.

2. Changes in iris colour. Iris usually becomes muddy in colour during active phase and may show hyperpigmented and depigmented areas in healed stage.

3. Iris nodules Cellular aggregates on the iris are termed as nodules. Pathologically these nodules differ from granulomata in that they consist of accumulations of epitheloid cells and lymphocytes which have been deposited without involving the loss of tissue. Nodules may be divided into two types – (i) those appearing on the ectodermal layers of the iris at the pupillary border are called "ectodermal nodules of Koeppe" and (ii) those appearing on the anterior mesodermal layers of the iris are called "mesodermal floccules of Busacca". Busacca nodules are less common than Koeppe nodules. Occurrence of nodules, especially the Busacca type usually denotes a granulomatous type of inflammation. Their presence indicates severity of inflammation.

4. Iris granuloma[36]: Rarer than either Koeppe or Busaca nodules. They are much larger than other two types. Appear single anywhere on surface of iris or pupillary margin. It is pink, vascularized and opaque, is pathognomic of granulomotous disease.

5. Iris atrophy[31]: It is an important feature of Fuch's uveitis syndrome and it also occurs in uveitis due to herpes simplex and herpes zoster.

6. Posterior synechiae. These are adhesions between the posterior surface of iris and anterior capsule of crystalline lens or anterior hyaloid face. Posterior synechiae may be formed during an acute attack, as the pupil is small and exudates poured from iris and ciliary body, tend to stick the iris to the anterior lens capsule so that it becomes fixed. If the pupil is not dilated pharmacologically, the fibroblasts organize these adhesions, converted into fibrous band and make them unable to rupture with mydriatics. When such synechiae are localized, and a mydriatics causes the intervening portions of the circle of pupil to dilate and pupil assumes festooned appearance.

Due to contraction of the organised exudates upon the iris, the pigment epithelium in its posterior surface may be pulled around the papillary margin so that patches of pigment may be seen on the anterior surface of iris – ectropion of uveal pigment.

Morphologically, posterior synechiae may be segmental, annular or total.

i. Segmental posterior synechiae refers to adhesions of iris to the lens at some points.

ii. Annular posterior synechiae (ring synechiae are 360^{o} adhesions of pupillary margin to anterior capsule of lens. These prevent the circulation of aqueous humour from posterior chamber to anterior chamber (seclusio pupillae). Thus, the aqueous collects behind the iris and pushes it anteriorly leading to 'iris-bombe' formation. This is usually followed by a rise in intraocular pressure.

iii. Total posterior synechiae due to plastering of total posterior surface of iris with the anterior capsule of lens are rarely formed in acute plastic type of uveitis. These result in deepening of anterior chamber.

7. Neovascularsation of iris (rubeosis iridis) develops in some eyes with chronic anterior uveitis and in Fuchs' heterochromic iridocylitis. In a few cases neovascularisation also develops in the angle. Iris neovascularisation is frequently associated with a persistent flare due to the continuous leakage of proteins into the aqueous humour.

VI. Pupillary signs

1. Narrow pupil. It occurs in acute attack of iridocyclitis due to irritation of sphincter pupillae by toxins. Iris oedema and engorged radial vessels of iris also contribute in making the pupil narrow.

2. Irregular pupil shape. It results from segmental posterior synechiae formation. Dilatation of pupil with atropine at this stage results in festooned pupil.

3. Ectropion pupillae (evertion of pupillary margin). It may develop due to contraction of fibrinous exudates on the anterior surface of the iris.

4. Pupillary reaction The pupillary reactions become sluggish or abolished and pupil is constricted. Due to inflammation of iris, there is dilatation of blood vessels with impairment of capillary walls and exudation of protein rich fluid into the tissue spaces of iris occurs. The hyperemia tends to cause the pupil to contract mechanically on account of radial arrangement of blood vessels. The extreme vascularity and looseness of tissue of iris causes swelling and accumulation of exudation. The iris becomes a water lodged sponge, so that freedom of movement is impaired and reactions become sluggish or abolished. The fluid also contains toxic substances, which act as irritant causing muscle fibers to contract, since the sphincter muscle overcome dilator muscle, constriction of pupil results.

5. Occlusio pupillae results when the pupil is completely occluded due to organisation of the exudates across the entire pupillary area.

VII. Changes in the lens

1. Pigment dispersal on the anterior capsule of lens is almost of universal occurrence in a case of anterior uveitis.

2. Exudates may be deposited on the lens in cases with acute plastic iridocyclitis.

3. Complicated cataract may develop as a complication of persistent iridocyclitis. Typical features of a complicated cataract in early stage are 'polychromatic luster' and 'bread-crumb' appearance of the early posterior subcapsular opacities. In the presence of posterior synechiae, the complicated cataract progresses rapidly to maturity.

VIII. Change in the vitreous

Inflammation in the vitreous, as in the anterior chamber, is characterized by increased cells and protein. The vitreous is rarely the source of inflammatory cells, which instead arise from the choroids, retina and ciliary body. Both vitreous cells and vitreous haze are more difficult to quantify than aqueous cells and flare.

IX. Fundus examination:

Every patient with uveitis should have a complete fundus examination. It is best done with a combination of indirect, the Hruby lens, the +90D lens and a mirrored contact lens. It includes –

1) The clarity of the media, the type of any vitreous opacity noted.
2) Optic nerve head is examined for evidence of engorgement, oedema or glaucomatous changes.
3) The foveal reflex should be examined and compared with that of the other eye. Look for any macular oedema or cystic changes at the macula.
4) Anterior uveitis is often associated with perivasculitis, sometimes with sclerosis or sheathing.

5) Note the presence or absence of any atrophic choriorctinal scars and their relation to any active lesion.
6) Any peripheral lesions should be searched with indirect binocular ophthalmoscope and examination of pars plana with scleral depression.

INTERMEDIATE UVEITIS

1. Presentation is with the insidious onset of blurred vision often accompanied by vitreous floaters. The initial symptoms are usually unilateral, but the condition is typically bilateral and often asymmetrical. Careful examination of the apparently normal eye may reveal minor abnormalities of the peripheral retina, such as vascular sheathing or localized vitreous condensations.

2. Anterior Uveitis

In Pars planitis there may be a few cells and small scattered KP which occasionally have a linear distribution in the inferior cornea.

3. Vitreous

Vitreous cells with anterior predominance are universal. Vitreous condensation and haze in more severe cases. Vitreous snowballs are usually most numerous in the inferior peripheral vitreous.

Table 6: Grading of vitreous haze

Haze severity	Grading
Good view of nerve fibre layer (NFL)	0
Clear disc and vessels but hazy NFL	+1
Disc and vessels hazy	+2
Only disc visible	+3
Disc not visible	+4

4. **Posterior segment**

Peripheral periphlebitis is common, particularly in patients with MS. Snowbanking is characterized by a grey-white fibrovascular plaque which may occur in all quadrants, but is most frequently inferior.

Neovascularization may occur on the 'snowbank' or the optic nerve head; the latter usually resolves when inflammatory activity is controlled. Subtle disc oedema may be seen, especially in young patients

POSTERIOR UVEITIS

Symptoms: Choroiditis is a painless condition, usually characterised by visual symptoms due to associated vitreous haze and involvement of the retina. Therefore, small patches situated in periphery may be symptomless and are usually discovered as healed patches on routine fundus examination. On the contrary, a central patch produces marked symptoms which draw immediate attention. Various visual symptoms experienced by a patient of choroiditis are summarised below:

1. **Defective vision.** It is usually mild due to vitreous haze, but may be severe as in central choroiditis.

2. **Photopsia**. It is a subjective sensation of flashes of light resulting due to irritation of rods and cones.

3. **Black spots** floating in front of the eyes. It is a very common complaint of such patients. They occur due to large exudative clumps in the vitreous.

4. **Metamorphopsia**. Herein, patients perceive distorted images of the object. This results due to alteration in the retinal contour caused by a raised patch of choroiditis.

5. **Micropsia** which results due to separation of visual cells is a common complaint. In this the objects appear smaller than they are.

6. **Macropsia**, i.e., perception of the objects larger than they are, may occur due to crowding together of rods and cones.

7. **Positive scotoma,** i.e., perception of a fixed large spot in the field of vision, corresponding to the lesion may be noted by many patients.

Signs: Usually there are no external signs and the eye looks quiet. However, fine KPs may be seen on biomicroscopy due to associated cyclitis. Fundus examination may reveal following signs:

1. Vitreous opacities due to choroiditis are usually present in its middle or posterior part. These may be fine, coarse, stringy or snowball opacities.

2. Features of a patch of choroiditis.

i. In active stage it looks as a pale-yellow or dirty white raised area with ill-defined edges. This results due to exudation and cellular infiltration of the choroid which hide the choroidal vessels. The lesion is typically deeper to the retinal vessels. The overlying retina is often cloudy and oedematous.

ii. In atrophic stage or healed stage, when active inflammation subsides, the affected area becomes more sharply defined and delineated from the rest of the normal area. The involved area shows white sclera below the atrophic choroid and black pigmented clumps at the periphery of the lesion.

INVESTIGATIONS[36,37]

Laboratory evaluation of the patient with intraocular inflammation demands a thoughtful, organized approach-one that is designed to achieve results that are likely to represent the true diagnosis. Ordering the standard battery of tests for the uveitis patient leads to a delay in diagnosis and excessive expense. A more fruitful method is to first consider which diagnoses are more likely and then perform well planned

laboratory evaluation. This approach takes into account such factors as the patient's age, sex, ethnicity, history, physical examination findings, review of systems, geographic area of patient's home, and history of travel. The major challenge is to :1) recognize which tests can narrow the diagnosis in a patient with uveitis; 2) properly choose tests based on individual patient factors and on the results of Bayes' theorem; 3) identify new testing modalities that are rapidly enhancing the clinician's ability to accurately diagnose the patient with uveitis.

Bayes' theorem calculates the likelihood that a positive test result represents true positive result (i.e., the patient actually has the disease). This post-test probability depends on the sensitivity and specificity of the test and, most importantly, on the prevalence of the disease.[31]

It is important to understand the reasons for ordering any investigation. It should help in (1) identifying any underlying systemic disease process or association, (2) providing a 'definitive' etiology, (3) confirming or rejecting a diagnosis, and (4) managing the patient.

Investigations which are performed in a case of uveitis are:

1) Haematological Tests:

Estimation of Hb, TLC, DLC, and ESR are non-specific and of no help in the diagnosis. However, they are important when the patient is to be started on antimetabolites. In these patients baseline and repeated values are necessary in detecting bone marrow suppression and dose adjustment.

2) Immunological investigation:

a) Rheumatoid factor: In case of rhematoid arthritis.

b) Antinuclear atnibodies (ANA): The presence of ANA shows that there is possibility of an existing autoimmune disease. Antinuclear antibody detection is the most sensitive laboratory test for SLE and is present in 99% of these cases.

c) Anti-DNA antibodies: These are found in 40-80% cases of SLE and only rarely in other connective tissue disorders.

d) Anti-neutrophil cytoplasmic antibodies (ANCA): ANCA seen in patients with systemic small vessel vasculitis. The common conditions are Wegener's granulomatosis, and microscopic polyarteritis nodosa.

e) Angiotensin converting enzyme (ACE): ACE levels are elevated in 85% of patients with active pulmonary disease due to sarcoidosis.

f) Serum globulin: 75% of patients with sarcoidosis have elevated globulin levels. Due to this serum protein increase and albumin-globulin ratio decreases.

g) Serum Lysozyme: In sarcoidosis serum levels of lysozyme have been found to be elevated in 70% of cases irrespective of whether the disease is active or inactive. Increased levels may also be present in tuberculosis.

h) Serum C-reactive protein (SCRP): The values of SCRP generally parallel that of ESR but the former is not influenced by anaemia. It is a non-specific indicator of inflammatory activity in the body.

i) Important serological tests include those for syphilis (VDRL, TPHA, FTA-ABS), toxoplasmosis (IgG/IgM antibodies) and HIV (western blot, ELISA). In non-endemic areas they could be useful in conditions like toxocariasis. Serological tests may also be performed on aqueous fluid obtained by paracentesis. On such samples, modern tests like polymerase chain reaction (PCR) could be more revealing.

3) Radiological Investigations:

Chest x-ray: For, Tuberculosis, Sarcoidosis, Histoplasmosis, malignancy

Sacroiliac joint and spinal x-ray: For ankylosing spondylitis

Skull x-ray: For congenital toxoplasmosis

Gallium scan: For sarcoidosis.

4) HLA typing[38]:

HLA antigens are now considered to be the genetic markers for disease susceptibility. The classical example is association of acute anterior uveitis and ankylosing spondylitis in HLA-B27 positive individuals.

5) Skin Tests:

The basis for all skin tests is delayed hyper sensitivity reaction of type-IV. Its main use has been in the diagnosis of TB and histoplasmosis. It has also been used to indicate anergy in cases of sarcoidosis. The tests are

- Mantoux test for : Tuberculosis
- Histoplasmin test for : Histoplasmosis
- Kveim test for : Sarcoidosis

6) Ultrasonography:

It is helpful to diagnose a masquerading anterior uveitis with hazy media. It can rule out long standing retinal detachment, intraocular tumour or coat's disease. It can help in planning surgery in patients with hazy media or complicated cataract due to uveitis.

7) Fluorescein Angiography:

Fundus Fluorescein angiography helps to regulate treatment and detection of sequelae or complications. Any case of anterior uveitis or intermediate uveitis with

unexplained loss of vision must be investigated by FFA to rule out cystoid rnacular oedema.

8) Neurologic tests:

In uveitis evaluation of the brain and cerebrospinal fluid can give important clues to the underlying disease. For example, lumbar puncture is useful in evaluating patients with possible Vogt-Koyanagi-Harada syndrome or intraocular lymphoma.

9) Diagnostic Surgeries[39]:

Invasive procedures are usually indicated in uncontrolled vision threatening uveitis, doubtful malignancy, endophthalmitis and viral keratitis. The procedures are-

a) Paracentesis of anterior chamber: It provides a small amount of fluid (200-250 μl). The fluid can be used for immunohistology, cytology, antibody estimation and for culture depending upon the case.

b) Vitreous aspiration: Identification of the inflammatory cells in vitreous may be of great value in establishing the diagnosis of a uveal tract disease eg: eosinophils in ocular toxocariasis. A vitreous biopsy may also differentiate intraocular masquarade syndrome and primary uveitis. It is done by aspirating directly by a syringe with 18-20 gauge needle.

c) Vitrectomy: It is the preferred method as compared to aspiration due to following factors: collection of more material, better follow-up due to clear media, less risk of retinal tear due to controlled traction on vitreous base.

COMPLICATIONS OF UVEITIS[40]

1) CORNEA:

- Corneal Oedema: It is proportionate to the degree of damaged corneal endothelium and the height of intraocular pressure.
- Band shaped keratopathy: It is seen in advanced chronic long standing uveitis and in Stills disease. It appears as a white band in the palpebral aperture running from nasal to temporal side. The calcified opacity is at the level of Bowman's membrane. Cornea above and below this band is clear.
- Sclerosing keratouveitis: This is a rare complication and may appear during acute stage of anterior uveitis. It appears usually in the temporal side of the limbus. It assumes the shape of triangular wedge with the apex towards the cornea, involving the cornea, sclera, and uveal tract.
- Deep keratitis: Especially in the central area due to toxic action, corneal involvement occur at the periphery by direct spread through the angle, a complication most readily seen in granulomatous lesion e.g Tuberculosis.

2) SECONDARY GLAUCOMA:

- It may be an early or late complication.

3) COMPLICATED CATARACT

4) CYCLITIC MEMBRANE

5) VITREOUS OPACITIES, DEGENERATION AND DETACHMENT:

Vitreous opacities, consists of protein rich plasmoid fluid and inflammatory cells. Opacities may be fine or coarse. Inflammatory cells include lymphocytes, plasma cells or macrophages. Vitreous may be liquefied and shrinkage occurs due to break

down of collagenous framework from severe cases of anterior uveitis resulting in vitreous detachment especially at the posterior pole.

6) OEDEMA OF THE DISC AND MACULA: It is seen in more severe anterior uveitis and panuveitis. Disc oedema does not cause much visual impairment when compared to macular oedema.

7) OPTIC NEURITIS AND SECONDARY OPTIC ATROPHY: Seen in severe forms of uveitis like panuveitis.

8) SECONDARY PERIPHLEBITIS RETINAE

9) RETINAL DETACHMENT: It may be exudative as in VKH syndrome. Uveitis produce retinal detachment by shrinkage of vitreous which result in traction and tears in the retina. Detachment can also result from contraction of exudative fibrous bands extending between the vitreous and retina.

9) ATROPHIC BULBI: This is the final stage of any form of chronic uveitis. As a result of repeated inflammatory attacks ciliary processes become disorganized, fibrosed and hyalinized. This results in diminution or abolition of aqueous secretion resulting in hypotony and atrophic bulbi.

10) PHTHISIS BULBI: As a result of hypotony the eye becomes soft, shrunken and quadrilateral in shape due to pressure by recti muscles.

TREATMENT OF UVEITIS[41]

Before starting treatment it is extremely important to identify the parameters (like dose, duration) to decide treatment response, resistance and the end point of

treatment. This decision would help in preventing several ocular and systemic side effects of the treatment itself.

OBJECTIVES OF THE TREATMENT:

- To curtail the inflammatory process so as to prevent or decrease risk of vision threatening complications.
- To relieve the patients discomfort.
- To manage associated systemic disease.
- To restore vision when possible, (Eg. Cataract surgery).

I) NON-SPECIFIC TREATMENT:

Non-specific measures include corticosteroids, mydriatic and cycloplegics, NSAIDS and immunosuppressive drugs.

A) Corticosteroids[42-44]**:** Corticosteroids are very effective, they are easy to administer and inexpensive. Therefore they are most commonly used. Corticosteroids should not be used in the following conditions; they are -

- In inactive disease with chronic flare but no cells.
- In mild anterior uveitis with not more than a 1+ cells.
- In intermediate uveitis with normal vision.
- In Fuchs' heterochromic iridocyclitis.
- When antimicrobial therapy is more appropriate, Eg. Candidiasis.

Corticosteroids can be administered topically in the form of drops or ointment, by periocular injection, or by systemic route.

TOPICAL ADMINISTRATION:

This is the most common route used in the treatment of anterior uveitis. They can be administered in the form drops or ointments.Severe forms of anterior uveitis can be treated with the powerful steroids, such as dexamethasone (0.1%), betamethasone (0.1%), and prednisolone (0.5%), while the weaker preparations such as fluorometholone (0.1%), clobetasone (0.1%) and loteprednol (0.5%) arc reserved for relatively mild uveitis in patients who are steroid reactors.

Indications:

Topical steroids can only be used in the treatment of anterior uveitis, because they cannot reach a therapeutic level in tissues that are behind the lens.

PERIOCULAR INJECTIONS:

Preparations:

The main steroids available for periocular injection are shown in table.

Table 7: Main steroids used for periocular injections

Short-acting (1 day)	Long-acting (several weeks)
Betamethasone 4mg/ml	Methylprednisolone acetate 40mg/ml
Dexamethasone 4mg/ml	Triamcinolone acetonide 40mg/ml

Advantages over drops:

- It ensures high concentration of steroids in posterior segment at the same time avoids systemic side effects.[45]

- Drugs that are only water soluble and incapable of penetrating the cornea, when given topically can enter the eye by penetrating the sclera, when given by periocular injection.
- A long lasting effect can be achieved if a depot preparation such as methylprednisolone (Depomedrone) is used.

Disadvantages[46]:

- Potential for high intraocular pressure.
- Conjunctival scarring
- Globe perforation
- Orbital cellulitis
- Ptosis and orbital fat atrophy

Indications:

- Severe acute anterior uveitis, especially in patients with ankylosing spondylitis with a marked fibrinous exudate in the anterior chamber or hypopyon.
- As an adjunct to topical or systemic therapy in resistant cases of chronic anterior uveitis.
- Intermediate uveitis
- Poor patient compliance.
- At the time of surgery in eyes with uveitis.

Techniques of Administration:

Periocular injections can be subconjunctival, anterior sub-Tenon, posterior sub-Tenon, and retrobulbar.

SYSTEMIC THERAPY:

Preparations:

The main oral preparation for systemic use is prednisolone 5mg.

Indications:

The main indications for systemic therapy are:

- Intractable anterior uveitis which has failed to respond to both topical therapy and anterior Sub-Tenon injections.
- Intractable intermediate uveitis which has failed to respond to posterior sub-Tenon injections.
- Posterior uveitis which has failed to respond to posterior sub-Tenon injections.

Side effects of Corticosteroid therapy:

a) **Topical Therapy:** Potential side effects of topical therapy include, temporary partial lid ptosis, pupillary mydriasis of 0.6-2.0mm, increased IOP in approximately 30% of patients treated for 3 weeks or more, posterior subcapsular cataract formation, bacterial or fungal super infection secondary to suppression of cellular defense systems, and decreased wound healing.

b) **Complications of systemic therapy :** They are all the complications of topical therapy above plus Cushing's syndrome, peptic ulcers, systemic hypertension, sodium retention, hyperglycemia in diabetics, psychosis, failure of growth, amenorrhea, aseptic joint necrosis, osteoporosis, myopathy, fluid retention and Addison's disease.

B) MYDRIATIC AND CYCLOPLEGICS:

They are used to give comfort by relieving iris sphincter and ciliary muscle spasm and to prevent posterior synechiae formation and to break down the synechiae if they are formed. Atropine (1%) the strongest mydriatic cycloplegic is used for 1-4 times daily, usually not more than 1-2 weeks. As uveitis lessens, the atropine dosage may be reduced or the patient placed on homotropine (5%) 2-4 times daily, will suffice to move the pupil. Bedtime use is beneficial only in milder cases, since the pupil is mobilized primarily during sleep and iatrogenic presbyopia is not present during the day. Pupil that fails to dilate on atropine should have pad and bandage applied, to increase the effect of atropine. Scopolamine (0.5%) has almost the same cycloplegic strength as atropine and may be substituted for it, in patients with urinary retention problems. The shorter acting agents, such as tropicamide (0.5-1%), helps to keep the iris moving and therefore decreases the possibility of posterior synechiae. Cyclopentolate may aggravate iritis because of its neutrophilic chemotactic effect.[47] Therefore cyclopentolate should be avoided in uveitis patients.

C) NON STEROIDAL ANTIINFLAMMATORY DRUGS (NSAIDs) :

They appear to act by blocking the local mediators of the inflammation.

1) **Oral NSAIDs:** They are often steroid sparing agents. These are useful in, long term therapy of recurrent anterior uveitis, or macular oedema, initially controlled by steroid therapy.

2) **Topical NSAIDs:** They are equivocally effective in iritis but may have a steroid-sparing effect. They also used in treatment of CME. Commonly used agents are flurbiprofen (0.03%), diclofenac (0.1%), Ketorolac (0.5%) and suprofen (1%).

D) IMMUNOSUPPRESSIVE AGENTS[48]:

These agents should usually not to be prescribed by an ophthalmologist alone but in concert with an oncologist or hematologists, who are familiar with them. The main indications are;

- Those who have progressive, usually bilateral vision threatening diseases.
- Failed to respond to conventional steroid therapy, or have an unacceptable side effect from them.
- Those who have Wegener's granulomatosis, polyarteritisnodosa or Behcet's disease.

Three classes of immunosuppressive agents commonly used in ocular inflammatory diseases are -

1) **Alkylating agents,** they work by suppression of T-cell lymphocytes and to lesser extent, B-cell function. The drugs used are Cyclophosphamide and Chlorambucil.
2) **The antimetabolite** Azathioprine interferes with purine metabolism and Methotrexate interferes with folate action, both functions are essential to nucleic acid synthesis.
3) **The antibiotic** Cyclosporine A probably interferes with T-cell lymphocyte activation and interleukin activity.

II) SPECIFIC TREATMENT OF THE CAUSE:

The non-specific treatment described above is very effective and usually eats away the uveal inflammation, in most of the cases, but it does not cure the disease, resulting in relapses. Therefore all possible efforts should be made to find out and treat the underlying cause. So, a full course of antitubercular drugs, for underlying Koch's

disease, adequate treatment for syphilis, toxoplasmosis etc, when detected should be carried out. When no cause is ascertained, a full course of broad spectrum antibiotics may be helpful by eradicating some masked focus of infection in patients, with uveitis.

III. Treatment of complications

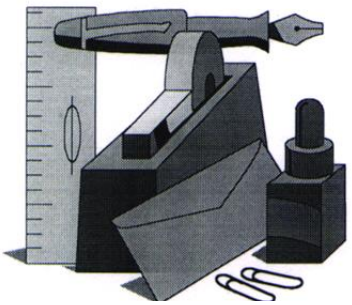
1. Inflammatory glaucoma (hypertensive uveitis) In such cases, drugs to lower intraocular pressure such as 0.5 percent timolol maleate eyedrops twice a day and tablet acetazolamide (250 mg thrice a day) should be added, over and above the usual treatment of iridocyclitis. Pilocarpine and latanoprost eye drops are contraindicated in inflammatory glaucoma.

2. Post-inflammatory glaucoma due to ring synechiae is treated by laser iridotomy. Surgical iridectomy may be done when laser is not available. However, surgery should be performed in a quiet eye under high doses of corticosteroids.

3. Complicated cataract requires lens extraction with guarded prognosis in spite of all precautions. The presence of fresh KPs is considered a contraindication for intraocular surgery.

4. Retinal detachment of exudative type usually settles itself if uveitis is treated aggressively. A tractional detachment requires vitrectomy and management of complicated retinal detachment, with poor visual prognosis.

5. Phthisis bulbi especially when painful requires removal by enucleation operation.



METHODOLOGY

MATERIALS AND METHODS

TITLE OF THE STUDY:

"Pattern of uveitis in a rural tertiary eye care centre, South India"

SOURCE OF DATA: A total of 50 patients of uveitis were studied, source being routine OPD patient presenting to the R.L.JALAPPA HOSPITAL AND RESEARCH CENTRE,TAMAKA, KOLAR attached to SRI DEVRAJ URS MEDICAL COLLEGE between the period from December 2010 upto September 2012 after their informed consent.

INCLUSION CRITERA:

All patients with uveitis.

EXCLUSION CRITERIA:

1. Cases of Endophthalmitis and Panophthalmitis.
2. Cases of Eale's disease.
3. Cases having glaucoma with uveitis.
4. Cases of traumatic and post-operative uveitis.

EVALUATION

A standard clinical proforma was filled in all cases for analytical study which included salient points in symptoms, history, clinical findings and anticillary testing.

Ophthalmic examination included:-

1. Visual acuity testing for distance and near using Snellen's distant chart and near vision chart respectively.
2. External ocular examination.

3. Slit lamp biomicroscopic examination for evidence of the following findings.
    - Morphological alterations of the cornea, corneal transparency and oedema, evidence of Keratic precipitates.
    - Anterior chamber depth and pigment dispersion in the anterior chamber.
    - Aqueous flare and cells
    - Presence of posterior synechiae.
    - Iris colour and pattern, nodules, atrophic patches.
    - Pupillary size and shape; reactions.
    - Lens transparency, complicated cataract.
4. Tonometry using Applanation tonometer.
5. Gonioscopy with Goldmann three mirror lens, to evaluate the angle of anterior chamber, which was graded as follows:

Table 8: Shaffer's grading of angle width

Grade	**Angle width**	**Configuration**	**Chance of closure**	**Structure visible on Gonioscopy**
4	35-45	Wide open	Nil	From Schwalbe's line to cilliary body
3	20-35	Open	Nil	From Schwalbe's line to sclera spur
2	20	Moderately Narrow	Possible	From Schwalbe's line to Trabecular meshwork
1	10	Very narrow	High	Schwalbe's line only
0	0	Closed	Closed	None of the structures visible.

5. The pupils were then dilated with a combination of 5% phenylephrine and tropicamide 0.8% drop was instilled every 5 minutes for 15 minutes interval.
6. This was followed by slit lamp examination for
    - Measuring pupil size, evidence of festooned pupil.
    - Posterior segment evaluation with +90D lens
7. Ophthalmoscopy: direct and indirect.

An anatomical diagnosis was made in all the cases according to International Uveitis Study Group System as having anterior uveitis, posterior uveitis, intermediate uveitis, or panuveitis.

Details on disease severity, laterality, chronicity, ocular signs and associated systemic conditions were noted.

Acute uveitis was defined as sudden onset of intraocular inflammation lasting less than 3 months. Chronic uveitis was defined as intraocular inflammation lasting longer than 3 months. Recurrent uveitis if two or more episodes of inflammation separated by a disease free period.

Patients were also classified with (a) non-granulomatous uveitis if they exhibited small endothelial keratic precipitates and the absence of iris nodules and/or choroidal granulomas, or (b) granulomatous uveitis if large ‘mutton-fat’ keratic precipitates, iris nodules and/or optic disc, and choroidal granulomas were seen.

The unilateral or bilateral character of uveitis was also recorded. Presentation was considered as unilateral if active inflammation was present in only one eye and bilateral if both eyes presented with active inflammation.

Cases of endopthalmitis, traumatic uveitis and post-op uveitis were excluded from the study.

A short differential diagnosis was made in each case. Subsequently, a tailored laboratory investigation was carried out. Investigations included total and differential counts, erythrocyte sedimentation rate, urine and stool examination, mantoux test. Serological tests for, syphilis, HIV, rheumatoid factor was done in all cases. Radiological investigations included x-ray of chest, lumbosacral and knee joints.

Patients with diagnostic leads provided by the history, review of systems, or examination underwent specific directed tests. Fluorescein angiography was undertaken in all patients with definite or questionable posterior segment involvement. Visual field testing, electrophysiology tests, and ocular ultrasonography were performed when indicated.

The final diagnosis was based on chronological history, clinical manifestations and the result of the laboratory investigations systemic evaluation by other medical specialities. The term idiopathic uveitis was used whenever the intraocular inflammation could not be attributed to an underlying systemic disease or specific ocular entity.

After the diagnostic procedures were completed, the patients were classified as having infectious uveitis, uveitis associated with noninfectious systemic diseases, specific ocular entities, or idiopathic uveitis.

As this was a descriptive study design, for statistical analysis descriptive statistical method of proportions was used. Chi-square test was used to compare the results amongst different populations.

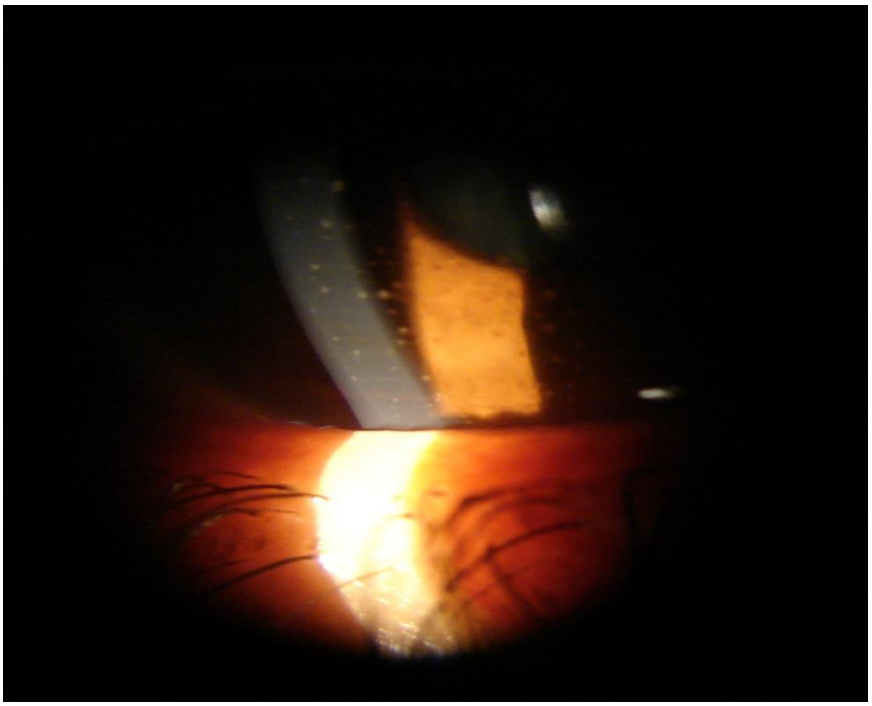


Fig. 1: Small and medium size KPs on direct illumination

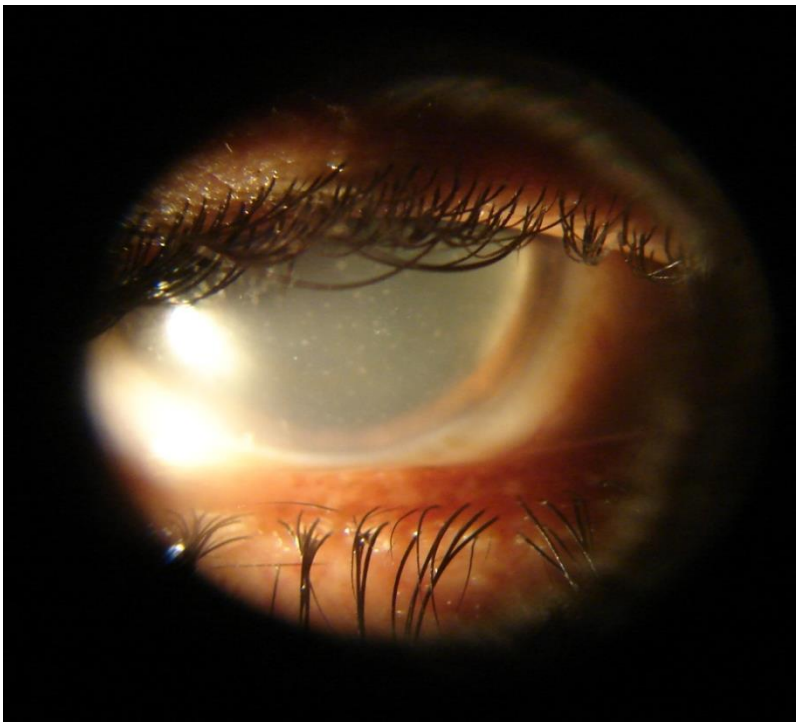


Fig. 2: KPs all over cornea

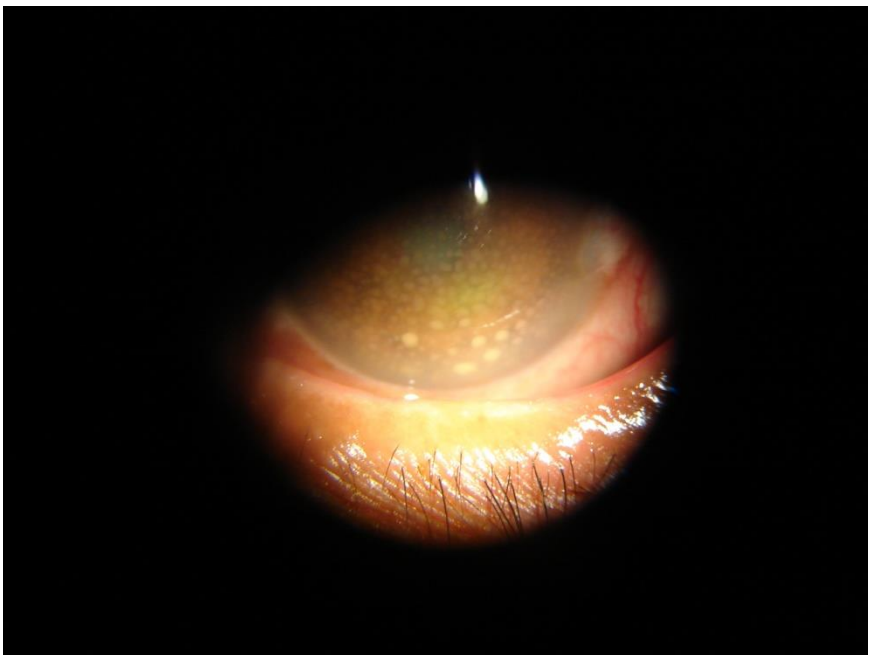


Fig. 3: Mutton fat KPs

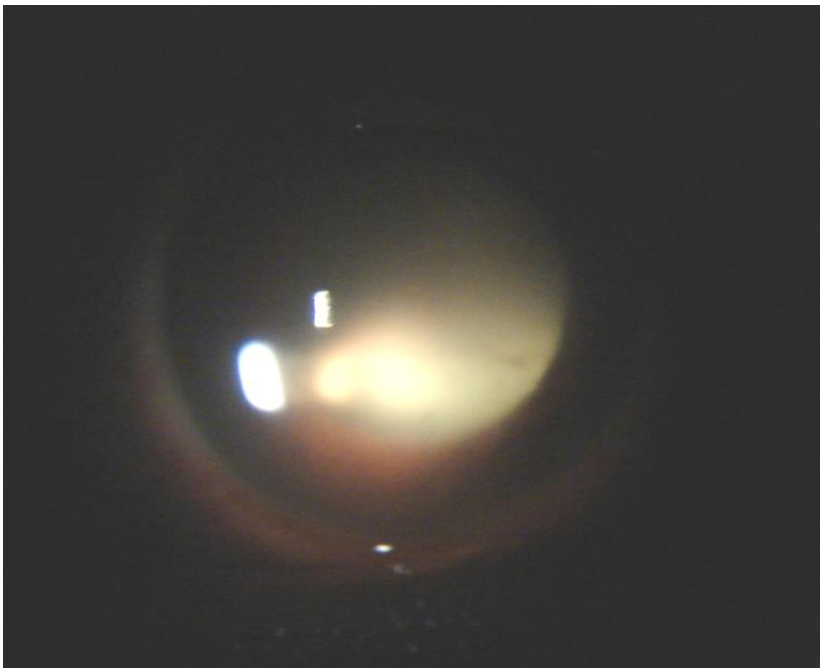


Fig. 4: Flare in AC

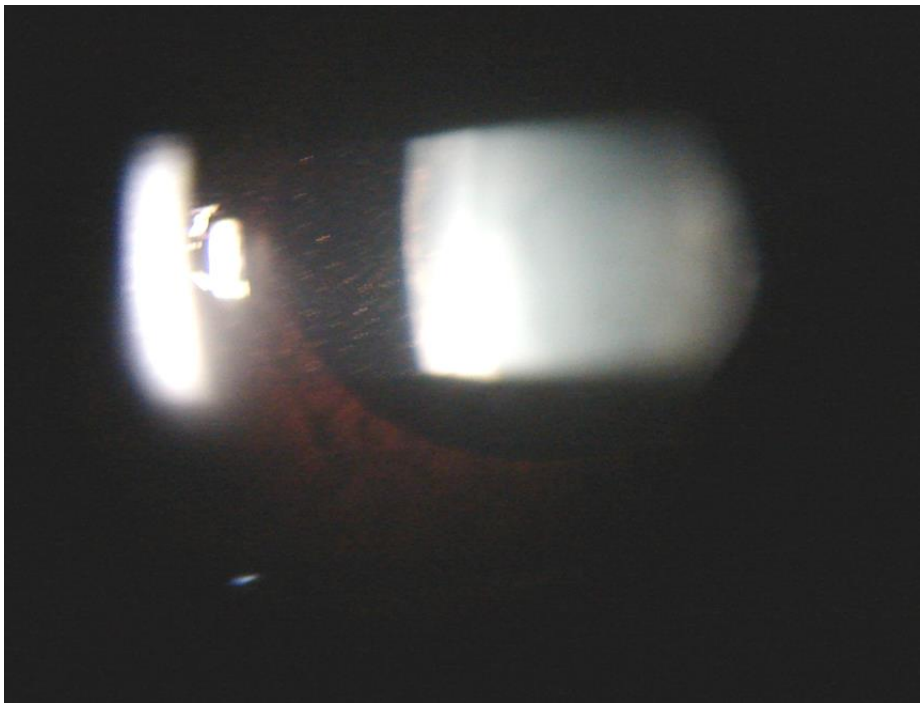


Fig. 5: Cells in AC 3mm x 1mm beam

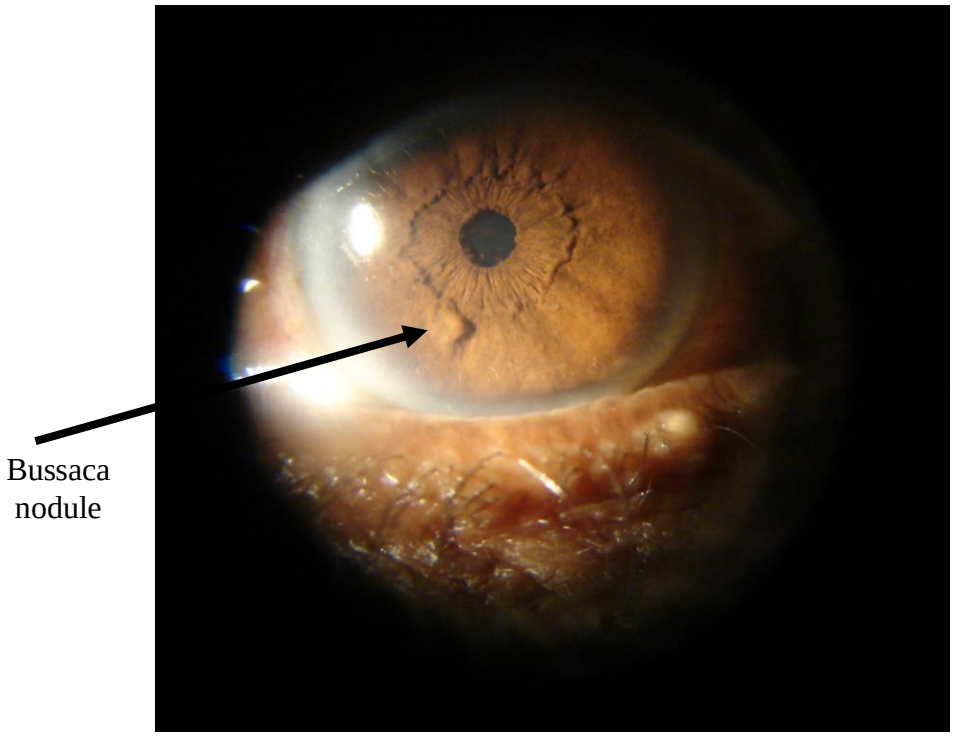


Fig. 6: Iris nodules

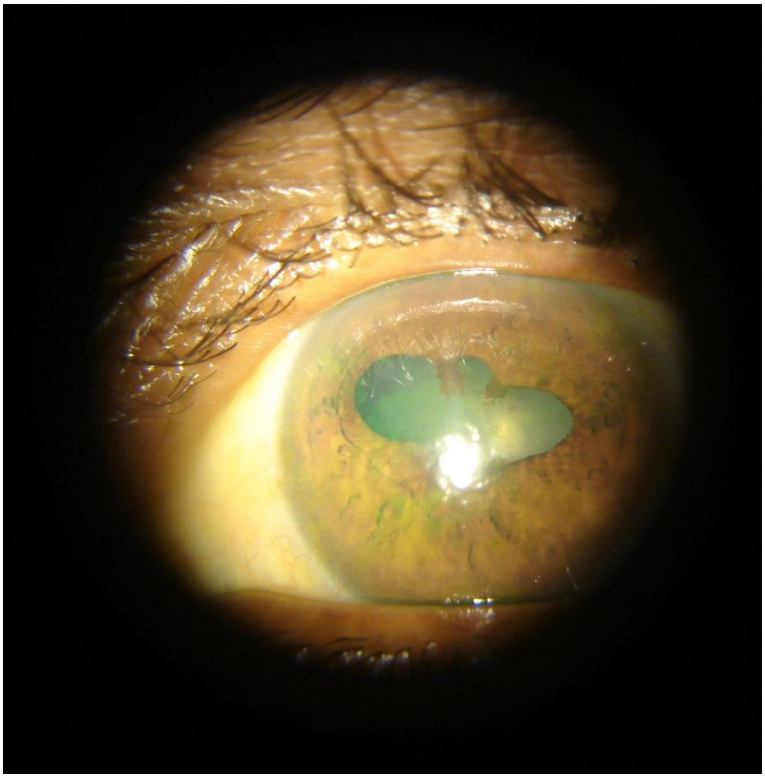


Fig.7: Posterior synechiae “Festoon pupil”

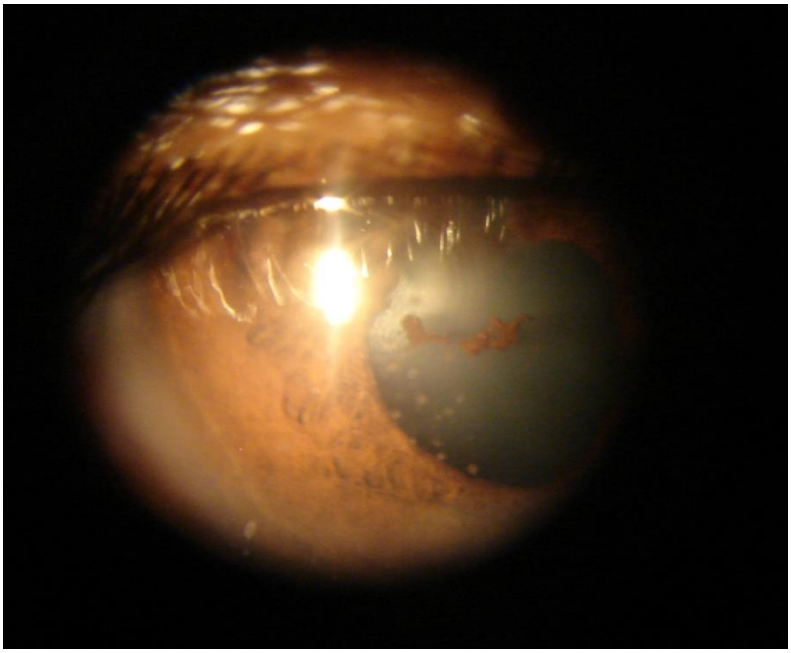


Fig. 8: Broken posterior synechiae

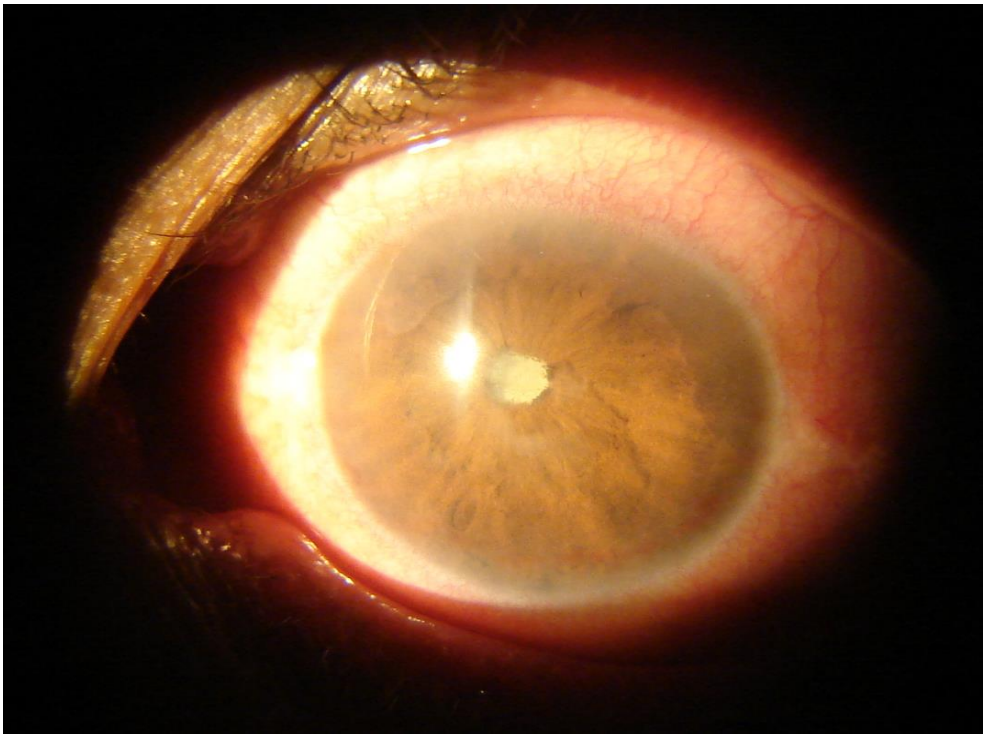


Fig. 9: Occlusio pupillae

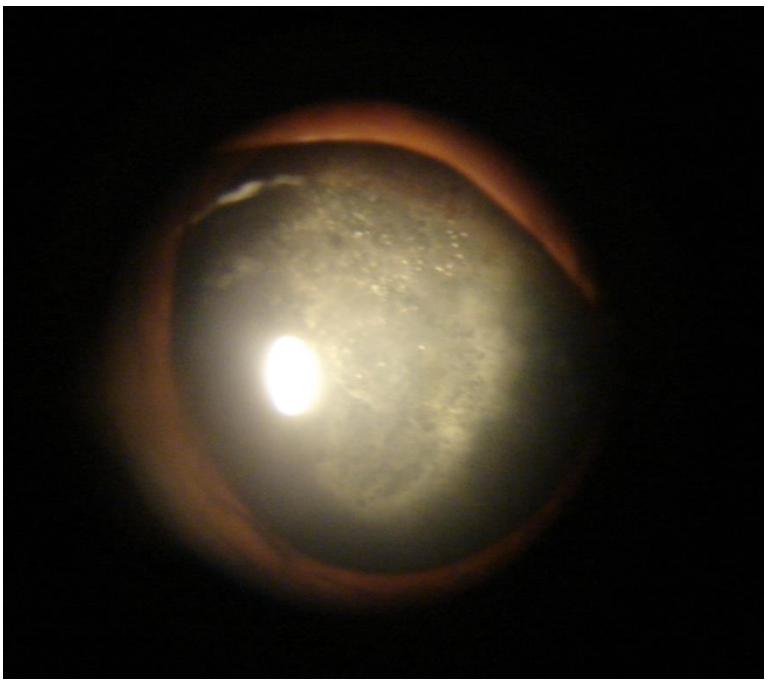


Fig. 10: Complicated cataract "Bread crumb appearance"

OBSERVATION AND RESULTS

RESULTS

In the present study 50 patients aged 10-80 yrs of both sexes were studied and following observations were made. The mean age at onset of uveitis was 40.3 years in males and 38.7 years in females. In all, 20 patients (40%) were between the ages of 20 and 50 years. In total, 2 (4%) of our patients were younger than 18 years and 16 (32%) were aged more than 60 years. No significant gender predominance was found (male–female ratio, 1: 1.7). This is shown in following tables.

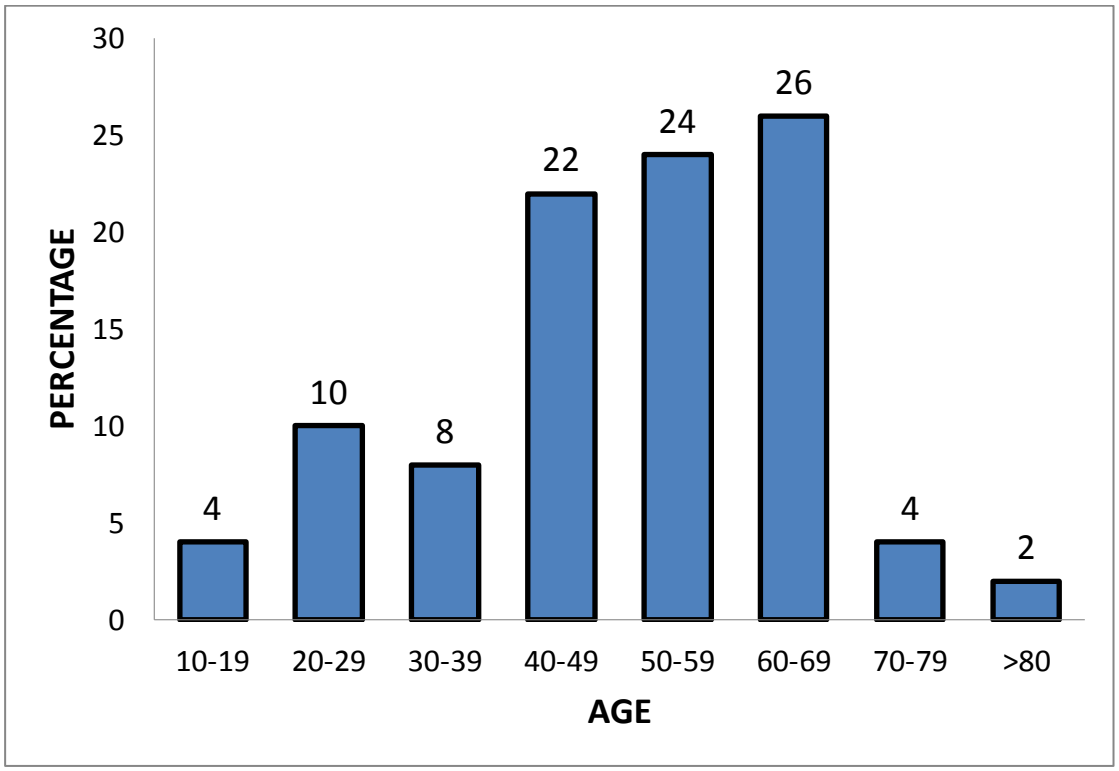
TABLE 9: AGE DISTRIBUTION

Sl. No.	Age (yrs)	Number	Percentage (%)
1	10-19	2	4
2	20-29	5	10
3	30-39	4	8
4	40-49	11	22
5	50-59	12	24
6	60-69	13	26
7	70-79	2	4
8	$\geq$80	1	2

TABLE 10: SEX DISTRIBUTION

Sl. No.	Sex	Number	Percentage (%)
1	Male	23	46
2	Female	27	54

GRAPH 1: AGE DISTRIBUTION



GRAPH 2: SEX DISTRIBUTION

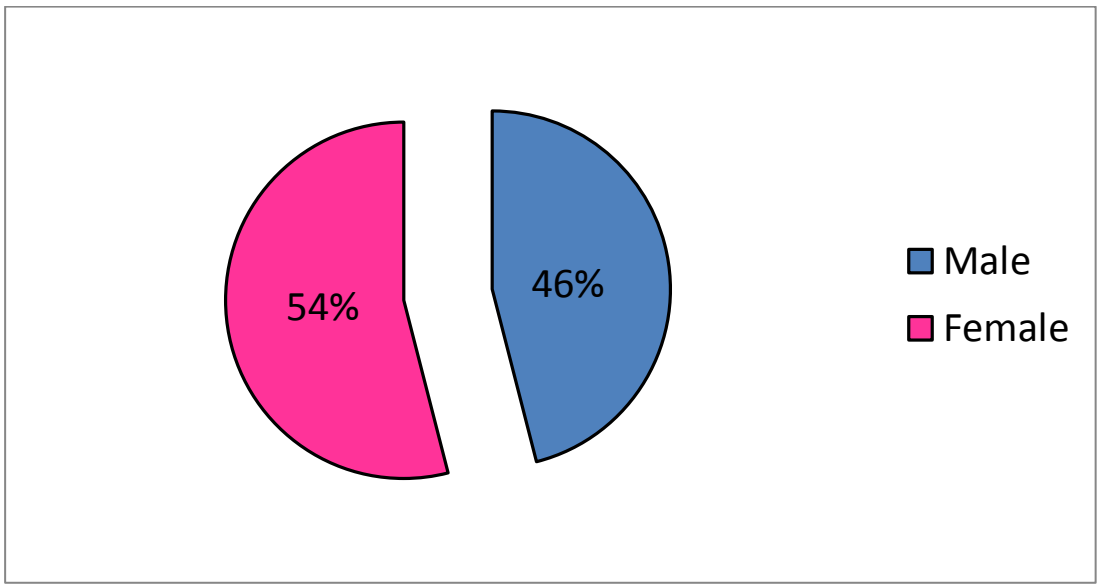


TABLE 11: LATERALITY

Sl. No.	Age (yrs)	Number	Percentage (%)
1	Unilateral	39	78
2	Bilateral	11	22
Total		50	100

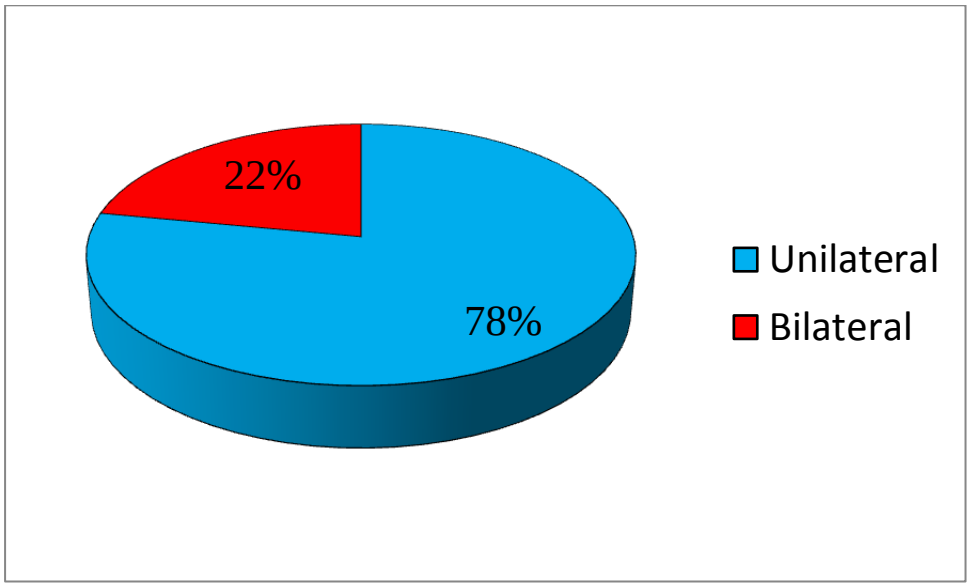
In this study unilateral involvement was more common compared to bilateral. Uveitis was unilateral in 39 patients (78%) and bilateral in 11 patients (22%).

TABLE 12: CLINICAL PRESENTATION

Sl. No.	Presentation	Number	Percentage (%)
1	Acute	16	32
2	Chronic	34	68
Total		50	100

In our study it was observed that most common presentation was chronic in 34 patients (68%).

GRAPH 3: LATERALITY



GRAPH 4: CLINICAL PRESENTATION

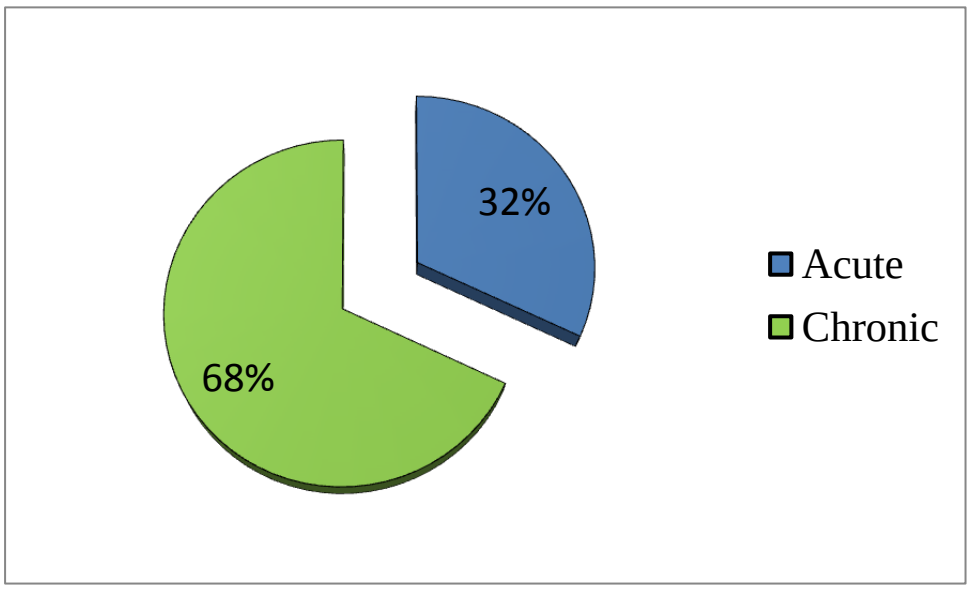


TABLE 13: TYPE OF INFLAMMATION

Sl. No.	Type	Number	Percentage (%)
1	Nongranulomatous	33	66
2	Granulomatous	17	34
Total		50	100

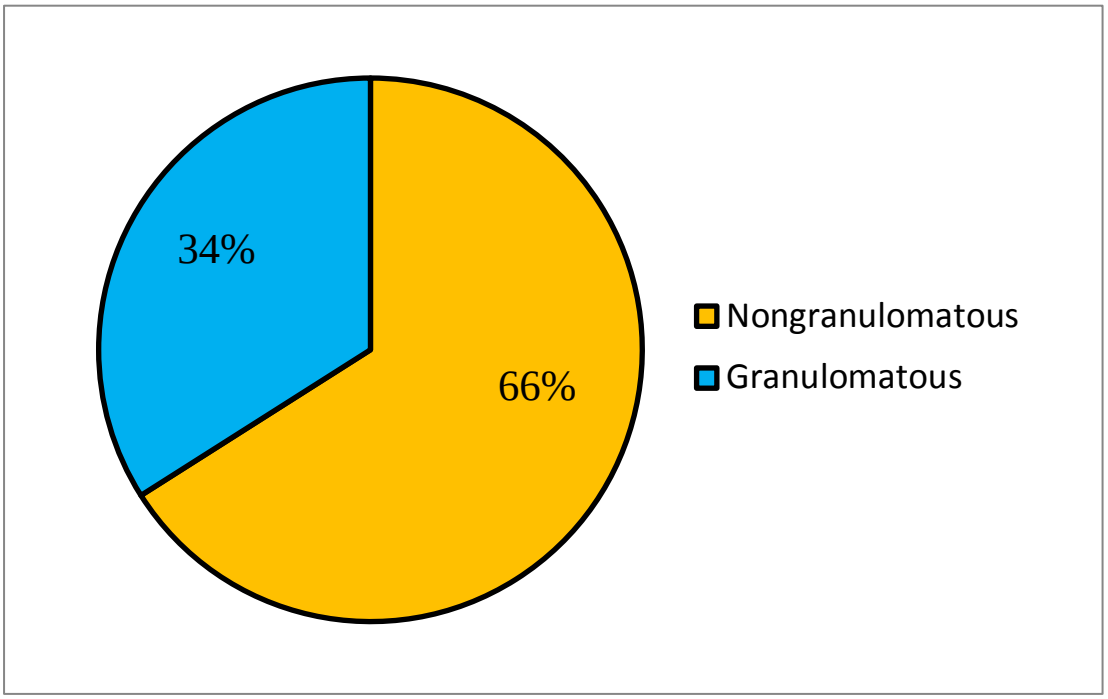
In our study Nongranulomatous uveitis (66%) occurred more frequently than granulomatous uveitis (34%). In granulomatous inflammation clinical presentation was chronic in most of the patients. Granulomatous type inflammation was observed in patients of tuberculosis, herpes zoster and idiopathic anterior uveitis.

TABLE 14: ETIOLOGICAL TYPES

Sl. No.	Type	Number	Percentage (%)
1	Non infectious	38	76
2	Infectious	12	24
Total		50	100

Majority of the cases in our study were found out to be non- infectious in etiology (76%). Of the 12 patients suspected to have infectious etiology, tuberculosis was the commonest infection (7 patients; 58.3%); followed by Herpes zoster (2 patients, 16.7%); Syphilis, Leprosy and HIV retinopathy (1 patient each 8.3%).

GRAPH 5: TYPE OF INFLAMMATION



GRAPH 6: ETIOLOGICAL TYPES

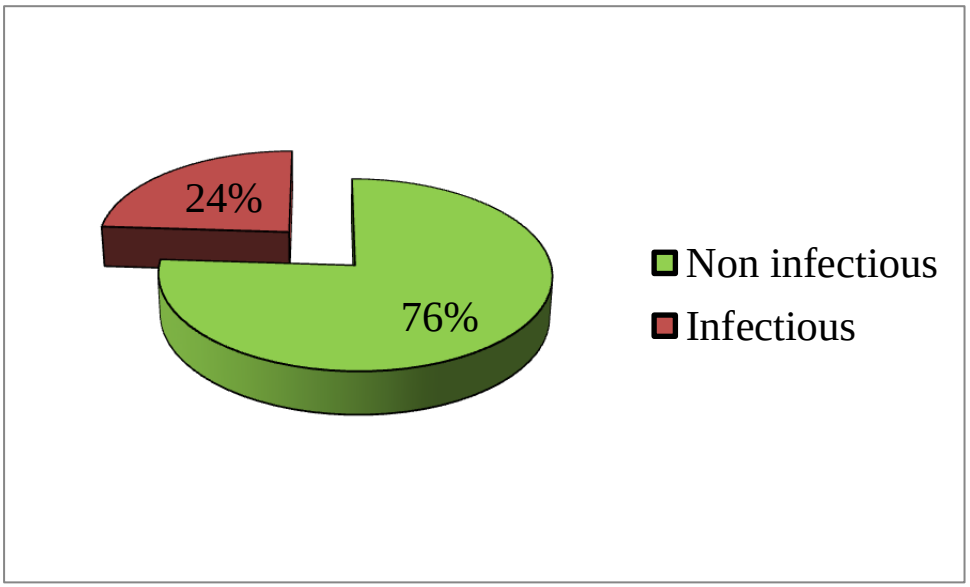


TABLE 15: ANATOMICAL PATTERN OF UVEITIS

Sl. No.	Type	Number	Percentage (%)
1	Anterior Uveitis	24	48
2	Intermediate Uveitis	3	6
3	Posterior Uveitis	18	36
4	Panuveitis	5	10

Anterior uveitis was the most frequent form accounting for (48%) of all patients, followed by posterior uveitis (36%), panuveitis (10%) and intermediate uveitis (6%). There was a significant predominance of bilateral disease among patients with panuveitis. Chronic presentation was predominant in patients with panuveitis (100%) and posterior uveitis (61%).

TABLE 16: ETIOLOGICAL DISTRIBUTION

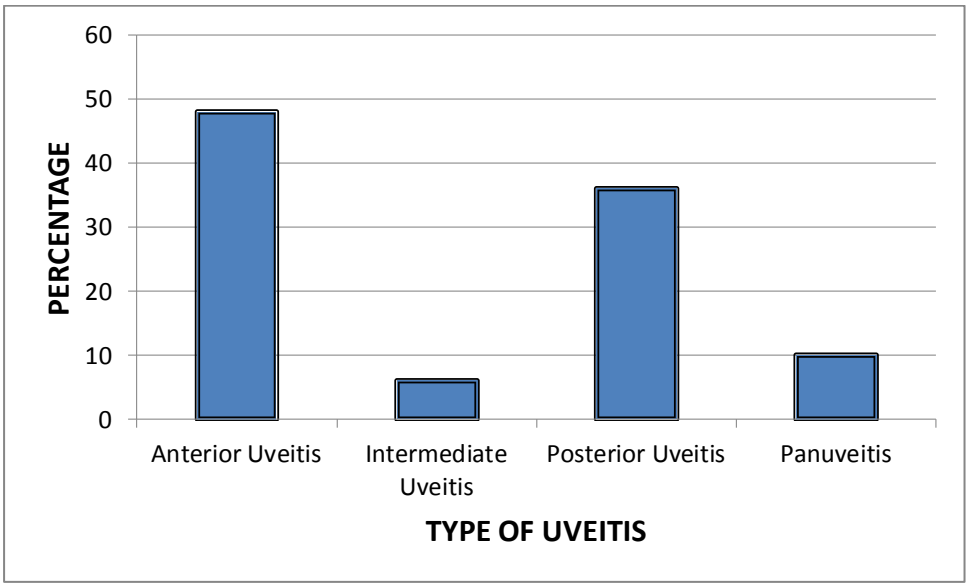
Sl. No.	Etiology	Number	Percentage (%)
1	Tuberculosis	7	14
2	Rheumatoid Arthritis	7	14
3	Pars Planitis	3	6
4	Herpes zoster	2	4
5	Ankylosing Spondylitis	1	2
6	JRA	1	2
7	Syphilis	1	2
8	AIDS	1	2
9	Leprosy	1	2
10	Idiopathic	26	52

Out of 50 patients, 24 patients (48%) had a specific etiology. Of the 24 patients with specific etiology, 12 (50%) were infectious etiology, of which tuberculosis was the commonest infection (7 patients; 58.3%); followed by Herpes zoster (2 patients, 16.7%); Syphilis, Leprosy and HIV retinopathy (1 patient each 8.3%). Among the specific etiology of non-infectious origin (12 patients, 50%), Rheumatoid arthritis was the commonest (7 patients, 58.3%), followed by pars planitis (3 patients, 25%); Ankylosing spondylitis (1 patient, 8.3%); and Juvenile Rheumatoid arthritis (1 patient, 8.3%).

In anterior uveitis, a specific associated disease could be established in 13 patients (54.2%). The most common among them for anterior uveitis was Rheumatoid arthritis in 7 patients (29.1% of anterior uveitis patients) followed by Herpes zoster (2 patients) and Tuberculosis (1 patient). In posterior uveitis, the specific diagnosis could be established in 7 cases (38.9%). Tuberculosis (5 patients, 27.8%) was the most common specific associated disease in posterior uveitis; followed by Syphilis and AIDS (1 patient each).

In intermediate uveitis, no specific cause could be found; these were labeled idiopathic pars planitis. In the panuveitis group, 1 patient (20%) had Tuberculosis. In 4 patients (80%), the etiology could not be determined.

GRAPH 7: ANATOMICAL PATTERN OF UVEITIS



GRAPH 8: ETIOLOGICAL DISTRIBUTION

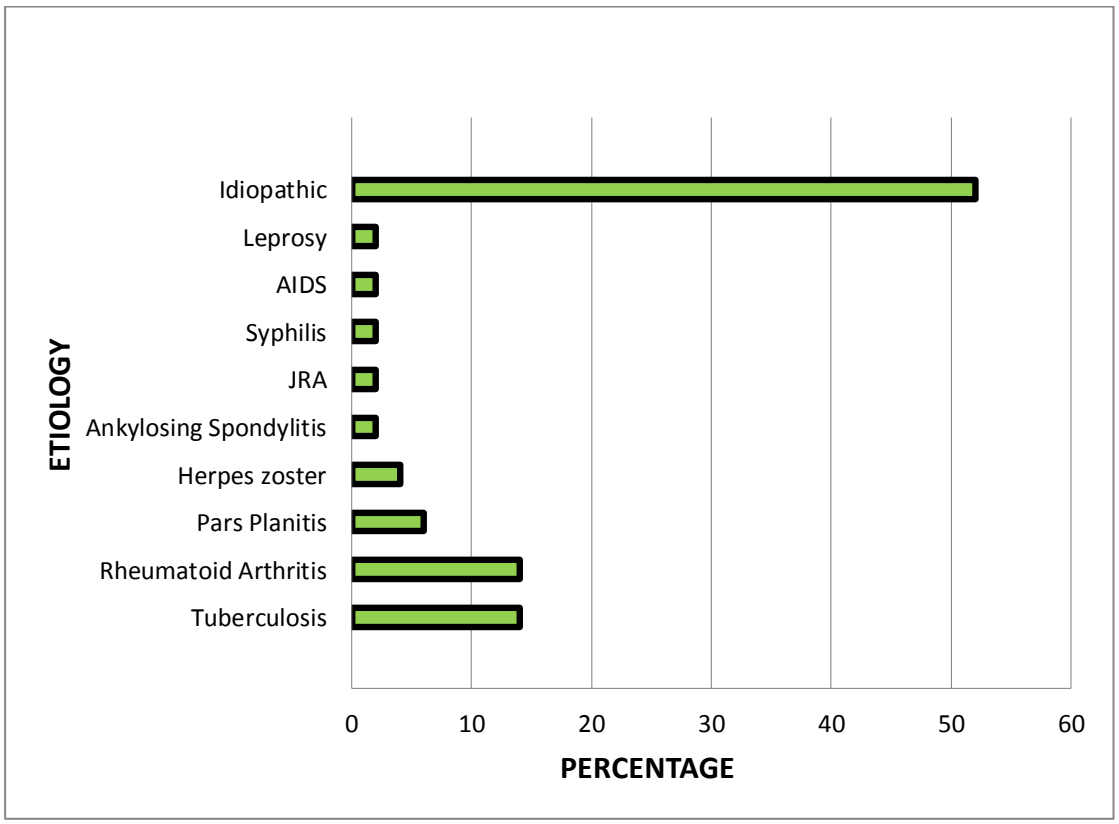
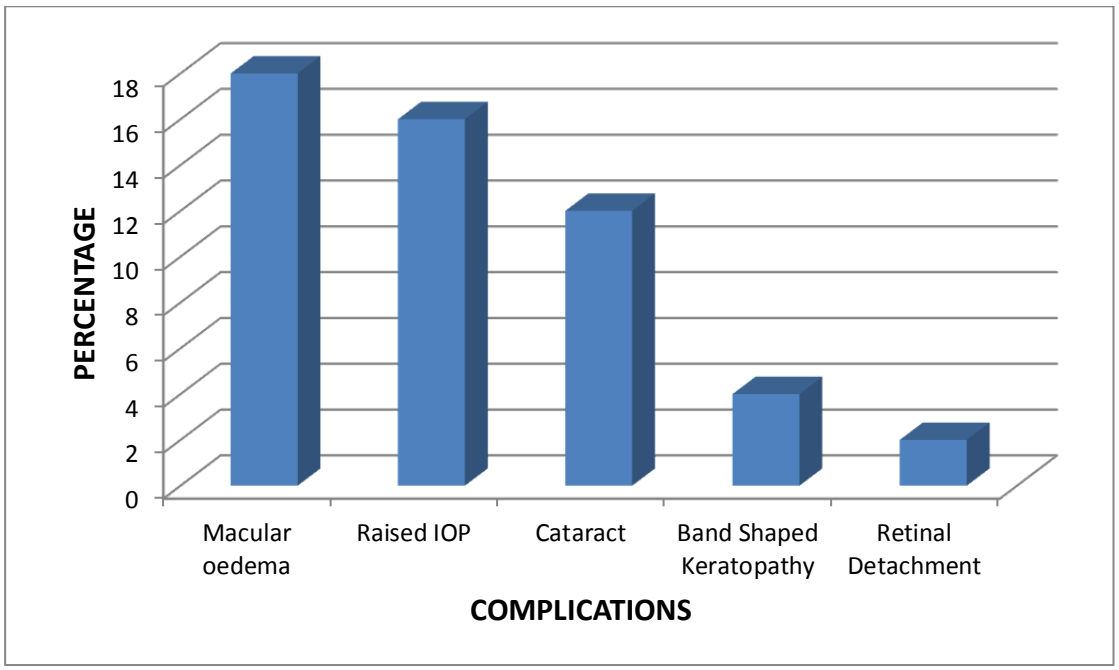


TABLE 17: COMPLICATIONS

Sl. No.	Complications	No. of cases	Percentage (%)
1	Macular oedema	9	18
3	Raised IOP	8	16
4	Cataract	6	12
5	Band Shaped Keratopathy	2	4
6	Retinal Detachment	1	2

The table 17 shows the complications of uveitis in present study. The complications were observed in 26 eyes (52%). The most common complications encountered in our patients were cystoid macular oedema (18%), intraocular pressure elevation (>21mmHg) (16%), and cataract (12%).

GRAPH 9: COMPLICATIONS



DISCUSSION

DISCUSSION

Studies of the distribution of the various types and causes of uveitis are important in aiding the clinician in the appropriate focused approach to investigation and diagnosis as well as management. They also provide the foundation for research into the areas such as etiology and pathogenesis of uveitis.

Limitations of epidemiological studies on uveitis stem from a variety of factors including heterogeneity of diagnostic criteria and workup, lack of uniform classification systems or precise definitions, and the effects of referral and selection bias. These factors render comparison of epidemiological studies from different regions and populations difficult.

The variation in the spectrum of uveitis is largely due to complex geographic, ecological, racial, nutritional and socioeconomic differences**.** Our uveitis study population had fairly homogenous background; all patients were Asian Indians and majority of patients belonged to south India.

To the best of our knowledge, this study is the first to provide data on uveitis from the Kolar region, Karnataka.

Findings from our study, consistent with those from previous reports, showed that males and females were almost equally affected, and that age at onset of uveitis widely varied, with a peak in the third and fourth decades. However, the mean age at presentation in our study (approximately 40 years) appeared to be slightly higher than that found in most previous reports (approximately 35 years).[2,6,49] Children comprised 4% of new uveitis patients in our study, which was similar to most estimates from previous studies. [35,50-52]

Considering different uveitis classification criteria, the most common types of uveitis in our patient population were idiopathic, anterior, chronic, non-granulomatous, and

noninfectious. The fact that chronic uveitis was the most common type (68%) probably reflects the referral character of our clinic and indicates that most patients with acute uveitis were treated by local ophthalmologists and not referred. This may also be due to the fact that most of the episodes of acute anterior uveitis resolve on their own, therefore many patients do not go for evaluation.

Overall, acute uveitis comprised 32% of the patient population and was particularly common in anterior (37.5%) uveitis. Chronic uveitis was particularly predominant among patients with panuveitis (80%), which is similar to other published studies.[53]

Anterior uveitis (48%) was the most common anatomical type of uveitis in our study. This was similar to several series in India[49,54,55] and elsewhere in other countries.[56-58]

In contrast, in the study by Henderly et al[59] posterior uveitis was most common form of uveitis (38.4%) in the USA.

Data from referral centers revealed that anterior uveitis was the most common form of uveitis (27.8–63%), followed by posterior (9.3–38%) or panuveitis (7–38%), and intermediate uveitis (4–17%).[60]

The frequencies for various forms of uveitis in our study were comparable to those of studies from tertiary referral centers.

The results of our study showed that for a large proportion of patients (52%), a definitive or presumed specific diagnosis was established based on history, including a review of medical systems, a comprehensive ophthalmologic examination, and selected laboratory and ancillary tests. The reported frequency of a systemic disease or a specific ocular entity underlying uveitis varies from 47.1 to 69.7%.[61]

The proportion of idiopathic uveitis cases in our series varied, depending on the site of inflammation, from 45% in patients with posterior uveitis to all the patients in patients with intermediate uveitis. Similarly, results of most previous studies showed

that the great majority of intermediate uveitis was idiopathic. There was however a large discrepancy between different studies in the proportion of idiopathic cases among other forms of uveitis.[60]

The most common underlying cause for non infective anterior uveitis was Rheumatoid arthritis (7 patients, 29.1%), which is in contrast to trends observed in other studies previously.[49,54] Amongst the infective etiologies of anterior uveitis, tuberculosis constituted 7.7% of anterior uveitis patients. A somewhat similar incidence has been reported in a study from North India[49] and Italy[62] where 7.9% and 6.31% of the anterior uveitis patients had a tubercular origin respectively.

Herpes zoster infection, presenting as anterior uveitis or keratouveitis, diagnosed based on clinical features, was found to be the most common infective cause of anterior uveitis in our series. The rate of herpetic uveitis in our patients (15.4%) is high compared to those found in previous reports. [49,54] It is unclear if this discrepancy is due to differences in the circulating virus's virulence, background immunity, or prevalence of other predisposing genetic or acquired conditions in the affected populations.

Intermediate uveitis is most often idiopathic. However, specific systemic disorders recently have been associated with this form. In our study, no specific cause was attributed to the cases of intermediate uveitis therefore were classified as idiopathic pars planitis. This observation was in accordance with Henderly et al[59] who reported all cases of intermediate uveitis (pars planitis, classical form or variant form) as idiopathic. Tuberculosis, Sarcoidosis, Multiple sclerosis, Lyme disease, systemic lupus erythromatoses and Juvenile rheumatoid arthritis have been reported as a cause of intermediate uveitis in many studies[53,63] but were not seen in our series.

Majority of the cases of posterior uveitis were idiopathic (61%) in nature in our study. Tuberculosis was the most common underlying infective cause (5 patients, 27.8%) of posterior uveitis, which was significantly higher than other reports.[49,62]

In other studies toxoplasma was the most common cause of posterior uveitis.[53,54,59]

Other infective causes of posterior uveitis in our study were HIV retinopathy and syphilis (5.5% each).

Tuberculosis was most common cause of panuveitis among specific etiology diagnosed (20% of panuveitis patients). This was similar to study carried out by Singh et al[49] (26%), which was significantly higher than other reports by Rodriguez et al[53] (2.0% of panuveitis patients), Biswas et al[54] (2.16% of panuveitis patients), and Mercanti et al[62] (5.8% of panuveitis patients).

Early recognition of uveitis patients is very important as chronicity of the disease leads to increase in the rate of complications affecting vision. Accurate diagnosis and timely treatment decreases the chances of recurrence and has good visual prognosis and therefore is key in management of uveitis.

The common complications encountered in our study were cystoid macular oedema (18%), cataract (12%) followed by raised intraocular pressure, band shaped keratopathy. These findings were consistent with various other studies done elsewhere.[50,64,65] These complications were treated accordingly.

Our study institute being a rural referral centre, more patients with posterior uveitis and panuveitis could have been referred and hence the total incidence quoted may not truly reflect the actual incidence in the population. Rural setup, Health insurance disparities and laboratory costs are the primary reasons for the absence of an accurate diagnosis in many of our patients. Despite these limitations, our results still convey, to a large extent, the general uveitis pattern in Kolar.

CONCLUSION

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Uveitis, irrespective of its type and presentation, is a chronic progressive disease with potentially blinding consequences. The etiology is varied and remains undetermined in most cases.

The challenge in a case uveitis is to develop tailored laboratory investigations that will facilitate a diagnosis.

Ordering a standard battery of tests for uveitis patients leads to delay in diagnosis and excessive expense. A more fruitful method is to first consider which diagnosis are more likely considering the factors like, the patients' age, sex, history, ocular examination findings and then performing tailored laboratory evaluation.

In all cases of uveitis a thorough ocular examination should be done, including posterior segment. A thorough systemic examination should be done to rule out systemic disease, as it may be an early manifestation of systemic disease.

Chronicity increases the risk of complications as does delay in receiving appropriate therapy, but early recognition and treatment of patient who are prone to recurrences can improve their outcome.

In our study anterior uveitis was most common type followed by posterior uveitis. The most common specific etiology was tuberculosis; however most of the cases were idiopathic. There was no significant sex predisposition seen.

Availability of specific investigation facilities at the rural level will help in deriving a region specific list of differential diagnosis which in turn will help in early treatment and taking preventive steps towards visual complications. Adoption of a universal classification systems and population-based studies in all countries may provide more reliable data for comparisons among different regions.

SUMMARY

- In the present study, 50 patients with uveitis, attending R L JALAPPA hospital and research center, attached to Sri Devaraj Urs medical college, Tamaka, Kolar were included. The average age of presentation these patients was around 40 years with no significant gender preponderance.

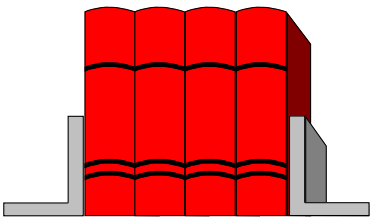
- All patients underwent a through systemic and ocular examination. A tailored laboratory investigation was done in each case to facilitate diagnosis.

- Most common type of uveitis in our study was anterior uveitis which was chronic and non-infectious in nature.
- Diagnosis was made with respect to the type and etiology of uveitis.

- Etiology remained undetermined in majority of cases. Most common cause in present study was Tuberculosis and Rheumatoid arthritis.

- Most common complications were macular oedema and raised intraocular pressure.

- Majority of patients responded well to treatment.



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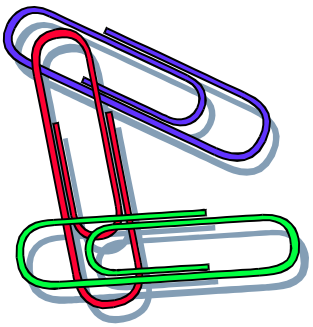

ANNEXURES

PROFORMA

NAME:

Age: Sex: M/F Hosp. No.:

Occupation:

Address:

- Chief complaints : Headache / pain / photophobia / redness / watering / decreased vision.
- H/O present illness:

  Mode of onset:

  Duration:

  No. of attacks

  Associated complaints: Fever / Joint pains / backache / skin lesions / oral ulcers.

  Treatment taken:
- Past History:
- Personal history:
- Family history:

GENERAL PHYSICAL EXAMINATION:

SYSTEMIC EXAMINATION:

LOCAL AND OCULAR EXAMINATION

		RE	LE
Lids			
Lacrimal Apparatus			
Conjuctiva			
Cornea	Size & Shape		
	Surface		
	Transparency		
	KPs		
Anterior Chamber	Depth		
	Flare		
	Cells		
	Other contents		

Iris	Colour & Pattern		
	Atrophic Patches		
	Nodules		
Pupil	Size & Shape		
	Reaction		
Lens			
Fundus			
Vision			

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS:

Complete haemogram Hb TC

ESR DC

Peripheral smear:

Urine: Albumin Sugar Microscopy

Stool: Ova Cyst

X-ray: Chest Paranasal sinuses Sacroiliac joint

Mantoux test:

Serological test

- VDRL
- ELISA for HIV
- Others :

ANCILLARY EXAMINATION:

- Dental examination :
- ENT examination :

FINAL DIAGNOSIS:

MASTER CHART

S.no	Hosp no.	Name	Age	Sex	Type	Laterality	C/P	Type of inflammation	Infection	Diagnosis	Complications	BCVA
1	715306	Nagraj	44	M	PU	UL	CHRONIC	G	I	Syphillis	ME	6/6
2	673910	Lal Khan	78	M	AU	BL	CHRONIC	NG	NI	RA	RIOP	6/9
3	720119	Krishnappa	80	M	AU	UL	CHRONIC	G	NI	Idiopathic	Cataract	CF 3m
4	697409	Girijamma	38	F	AU	UL	CHRONIC	NG	NI	Idiopathic	ME	6/36
5	710466	Vimala	23	F	AU	BL	CHRONIC	NG	NI	RA	-	6/6
6	789590	Gangamma	65	F	AU	UL	CHRONIC	NG	NI	Idiopathic	Cataract	CF 3m
7	729502	Narayanappa	43	M	AU	BL	CHRONIC	G	NI	Idiopathic	-	6/6
8	644698	Maqbool Jaan	50	F	PAU	BL	CHRONIC	G	I	TB	RIOP	6/6
9	627567	Lavanya	10	F	AU	BL	CHRONIC	NG	NI	JRA	-	6/6
10	743119	AjayTN	18	M	PU	UL	CHRONIC	NG	NI	Idiopathic	ME	6/60
11	SNR	Ibrahim	65	M	PU	BL	CHRONIC	G	I	TB	ME	CF 5m
12	SNR	Munikrishnappa	65	M	PU	UL	CHRONIC	G	I	TB	ME	6/24
13	653461	Venkatappa	65	M	PU	UL	CHRONIC	G	I	TB	RD	CF 1m
14	707539	Gangamma	60	F	PAU	UL	CHRONIC	NG	NI	Idiopathic	Cataract	6/60
15	709212	Asuma	24	F	PAU	UL	CHRONIC	NG	NI	Idiopathic	ME	6/36
16	SNR	Saddam	27	M	PAU	UL	CHRONIC	NG	NI	Idiopathic	ME	6/24
17	745043	Vijaya	60	F	AU	BL	CHRONIC	G	NI	Idiopathic	Cataract	CF 1m
18	751176	Venkateshappa	65	M	AU	BL	CHRONIC	G	I	TB	-	6/9
19	749922	Seetharamappa	60	M	PU	UL	CHRONIC	G	I	TB	RIOP	6/9
20	782511	Govindappa	50	M	PU	UL	CHRONIC	G	I	HIV	-	6/6
21	762621	Mymunissa	45	F	AU	UL	CHRONIC	G	NI	RA	-	6/6
22	759217	Nayaz Ahmed	38	M	AU	UL	ACUTE	NG	NI	Idiopathic	-	6/6
23	779929	Puttamma	65	F	AU	UL	CHRONIC	G	NI	Idiopathic	Cataract	CF 1m
24	677740	Suma	26	F	PU	UL	CHRONIC	G	NI	Idiopathic	-	6/9
25	748794	Ramesh	35	M	PU	UL	CHRONIC	G	I	TB	-	6/9
26	796102	Gangamma	55	F	AU	UL	CHRONIC	NG	NI	Idiopathic	-	6/6
27	697595	Akeem saab	72	M	AU	BL	CHRONIC	G	I	Leprosy	RIOP	6/6
28	801828	Gopalappa	51	M	AU	UL	ACUTE	NG	NI	Idiopathic	-	6/6
29	797425	Ramachandra	70	M	AU	UL	CHRONIC	NG	NI	RA	-	6/6
30	799112	Venkatlakshmamma	50	F	AU	UL	CHRONIC	NG	NI	RA	-	6/6
31	649598	Kanthamma	50	F	AU	UL	CHRONIC	NG	NI	RA	-	6/6
32	657232	Pushpa	40	F	PU	UL	ACUTE	NG	NI	Idiopathic	-	6/9
33	657234	Sarojamma	68	F	PU	UL	CHRONIC	NG	NI	Idiopathic	BSK	6/60
34	657236	Muniyamma	70	F	PU	UL	CHRONIC	NG	NI	Idiopathic	BSK	CF 3m
35	819992	Shakunthalamma	40	F	IM	UL	ACUTE	NG	NI	Pars Planitis	RIOP	6/12
36	798042	Adavakka	65	F	IM	UL	ACUTE	NG	NI	Pars Planitis	-	6/9
37	794082	Chinakka	52	F	AU	UL	ACUTE	NG	NI	Idiopathic	-	6/6
38	794078	Venkatarayappa	70	M	PU	UL	ACUTE	NG	NI	Idiopathic	RIOP	6/6
39	794103	Munihanumakka	60	F	PU	UL	ACUTE	NG	NI	Idiopathic	-	6/6

40	796071	Chinnappa	60	M	PU	UL	ACUTE	NG	NI	Idiopathic	-	6/6
41	796084	Papamma	55	F	PU	UL	ACUTE	NG	NI	Idiopathic	RIOP	6/6
42	798024	Jai Kumar	53	M	IM	UL	ACUTE	NG	NI	Pars Planitis	-	6/12
43	797451	Parvathamma	40	F	AU	BL	ACUTE	NG	NI	Idiopathic	-	6/6
44	797444	Muniyamma	52	F	PU	UL	ACUTE	NG	NI	Idiopathic	ME	6/60
45	794081	Muniyamma	60	F	PU	UL	ACUTE	NG	NI	Idiopathic	ME	CF 4m
46	829604	Rathnaiah	70	M	PAU	BL	CHRONIC	G	NI	Idiopathic	Cataract	CF 2m
47	833109	Iriyamma	48	F	AU	UL	CHRONIC	NG	NI	RA	RIOP	6/9
48	833188	Gopal	42	M	AU	UL	CHRONIC	NG	NI	AS	-	6/6
49	807869	Gopalappa	63	M	AU	UL	ACUTE	NG	I	HZO	-	6/6
50	815296	Varalakshmi	46	F	AU	UL	ACUTE	NG	I	HZO	-	6/6

KEY TO MASTER CHART

S.No – Serial number

Hosp No – Hospital number

M – Male

F – Female

AU – Anterior Uveitis

IU – Intermediate Uveitis

PU – Posterior Uveitis

PAU – Panuveitis

UL – Unilateral

BL – Bilateral

C/P – Clinical Presentation

G – Granulomatous

NG – Nongranulomatous

I – Infectious

NI – Noninfectious

ME – Macular edema

RIOP – Raised intraocular pressure

RD – Retinal detachment

BSK- Band shapedkeratopathy

CF – Counting fingers

TB – Tuberculosis

RA – Rheumatoid Arthritis

JRA – Juvenile Rheumatoid Arthritis

HIV – Human immunodeficiency virus

AS – Ankylosing Spondylitis

HZO – Herpes zoster ophthalmicus