Dissertation on

A MULTIFACTORIAL ANALYSIS OF 50 CASES OF PARS PLANITIS

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CERTIFICATE

This is to certify that the dissertation entitled,"A MULTIFACTORIAL ANALYSIS OF 50 CASES OF PARS PLANITIS

" Submitted by DR.Vidhya.E, in partial fulfillment for the award of the degree of Master of Surgery in Ophthalmology by the Tamilnadu Dr. M.G.R Medical University, Chennai is a bonafide record of work done by her in the Regional Institute of Ophthalmology,Government Ophthalmic Hospital, Egmore, Chennai, during the academic year 2008 – 2011.

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PART I

INTRODUCTION

DEFINITION

The term pars planitis should be used only for that subset ofIntermediate Uveitis where there is snow bank or snowball formation occurring in the absence of an associated infection or systemic disease (that is, "idiopathic"). If there is an associated infection or systemic disease, then the term Intermediate Uveitis should be used. According to the diagnostic criteria of the Standardization of Uveitis Nomenclature (SUN) Working Group, pars planitis is defined as the idiopathic Intermediate Uveitis associated with the presence of snow-banking and/or snowballs

ANATOMY

Uveal tract, the vascular middle layer of the eye comprises of iris anteriorly, ciliary body in the middle and choroid posteriorly. Ciliary body extends for 6mm from the root of iris to ora serrata. The anterior portion of the ciliary body is the pars plicata and consists of 70 – 80 ciliary processes. The posterior portion of the ciliary body is the pars plana which is a flat extension from the posterior aspect of ciliary process to the ora serrata. The area is about 3.5 – 4 mm in length. The pars plana has a smooth black surface marked only by faint striae running on from the teeth of ora serrata.

The epithelium lining the parsplana consists of an outer pigmented layer continuous with the pigmented layer of retina. The inner nonpigmented layer is columnar, becoming cuboidal over the ciliary process and flattens as they approach the ora serrata. Blood supply is by the long posterior and anterior ciliary arteries.

Vitreous base is a zone 3 – 4 mm wide which straddles the ora serrata. As a result of strong adhesions of cortical vitreous at the vitreous base in an eye with PVD, the posterior hyaloid surface remains attached to the posterior border of vitreous base.

HISTORY

Intermediate uveitis (para planitis) was first described as chronic cyclitis by Fuch`s in 1908 (Kimura, et. al., Peripheral Retinitis and ChronicCyclitis in Children Trans Ophthal soc UK85: 39-52,1965). Schepens (in 1950) who is the pioneer of modern indirect ophthalmoscope and examination of peripheral retina by scleral depression, described them as peripheral uveitis. This was characterized by inflammation centered around the retinal periphery. Kimura presented the name “Chronic Anterior Uveitis”, to involve the intraocular inflammation primarily affecting ciliary body, anterior vitreous associated with optic disc changes and macular

edema. Nussenblatt in 1988 described pars planitis as an inflammation of the ciliary body, vitreous base and peripheral retina.

CLASSIFICATION

1. The classification scheme recommended by the International uveitisstudy group is based on anatomical location. Intermediate uveitis formerly known as pars planitis is classified into

   a. posterior cyclitis

   b. Hyalitis

   c. Basal retinochoroiditis

2. TERSLER (classification and symptoms and signs of uveitis :Duane) has classified intermediate uveitis into

   a. Cyclitis

   b. Vitritis

   c. Pars planitis

3. Based on clinical presentation BOKE classified pars planitis into

   a. Diffuse inflammatory type : characterized by dust like opacities in vitreous, most prominent inferiorly called snow ball opacities.

   b. Exudative type: with presence of heavy confluent exudate over pars plana and ora serrata. It is often associated with PVD.

c. Vasoproliferative type : characterized by sheathing, perivascular cuffing and partial occlusion of peripheral vessels. This is more prone for developing disc edema, optic atrophy and optic disc neovascularization. Chronic poorly controlled intermediate uveitis often had total retinal detachment, proliferative vitreoretinopathy or cyclitic membrane with ciliary body traction and hypotony (Pederson).Slovak further divided the diffuse inflammatory type of parsplanitis into

   a. Serous type in which pars plana is not grossly involved.
   b. Infiltrative type which presents with small white spots involving the pars plana.

Both the serous and infiltrative type can progress to exudative type.

3. BROCKHURST in 1960 classified 5 possible types of intermediate uveitis:

i. Benign course in which inflammation subsided without any intervention. This type comprised of 28 – 31 % of patients with parsplanitis

ii. Patients who developed choroidal and serous retinal detachments. They accounted for 12% of patients with pars planitis.

iii. Those who developed vascularized high snow banks that eventually formed cyclitic membranes leading to retinal detachments or glaucoma. They formed 6% of group with pars planitis.

iv. Those who developed significant vascular obliteration that led to visual field loss and optic atrophy , were around 8%.

v. 46% of patient had chronic smouldering course.

BROCKHURST further condensed his classification into four groups in 1980 as[30]:

a. Benign : 31%

b. Mild chronic : 49%

c. Severe chronic : 15%

d. Relentlessly progressive : 10%

4. KIMURA classified pars planitis into

a. Mild : characterized by absence of keratic precipitates

: faint or absent flare, cells less than 5

: Anterior vitreous floaters 1 + or 2+

: Slight edema of posterior retina and no exudates

b. Moderate : had no keratic precipitates

: Faint or no flare

: cells 5 – 10 (1+)

: Vitreous floaters: scattered fine and coarse Opacities obscuring fundus

: Moderate edema of posterior retina and exudate over the inferior pars plana

c. Severe : characterized by small to medium sized white KP`s, with a few mutton fat KP`s on gonioscopy.

: Moderate to marked flare, 10 – 20 cells per field.

: Anterior vitreous cells were dense enough to preventa view of the fundus (3 – 4 +)

: marked posterior retinal edema, peripheral vascular sheathing and pigmentary changes in peripheral retina.

5. Standardization of uveitis nomenclature (SUN) working group anatomic classification of uveitis classified intermediate uveitis into

a. Pars planitis

b. Posterior cyclitis

c. Hyalitis

EPIDEMIOLOGY

Pars planitis accounts for 4 – 15 % of uveitis patients in referral practice and constitutes about 91 % of intermediate uveitis

AGE

It primarily affects children and young adults. There is a bimodal distribution with one peak in the second decade and another peak in the third or fourth decade.

In the pediatric age group, intermediate uveitis is associated with a worse presenting visual acuity. Poorer outcomes may be related to delayed presentation/diagnosis, the inherent difficulties of immunosuppression in children, or a more aggressive disease.

SEX AND RACE

There is no sex or race predisposition. A common immunogenetic predisposition to multiple sclerosis and pars planitis was found to be associated with the HLA-DR15 allele. In a prospective study of 53 patients with pars planitis by Raja et al, an association was found with the HLA-DR15, a subtype of HLA-DR2. Other associations include HLA-A28, HLA –DR51, HLA-DR17, HLA-B8, and HLA-B51. In addition, TT homozygotes

for the interferon-gamma (INF-gamma) gene may be at a higher risk of disease development and may also run a more severe course.

CLINICAL MANIFESTATION

Pars planitis is one of the most underdiagnosed uveitic entity. This is due to the lack of expertise in indirect ophthalmoscopy on patients with anterior uveitis and vitreous floaters. There is also lack of awareness and misconception about this entity. Most often it is labeled as cyclitis, posterior uveitis and vitreous haemorrhage.

PATIENT`S SYMPTOMATOLOGY

Patients usually have very few complaints initially. Most of them present with blurring of vision and floaters. In pars planitis cases unlike other uveitic conditions the eye rarely becomes red and painful. There is usually no gross reduction in vision.

70 – 80% of patients have bilateral signs at the time of presentation, even though most of them are usually symptomatic only in one eye. One third with unilateral disease will subsequently develop bilateral involvement.

ANTERIOR SEGMENT

Anterior segment inflammation is mild and is more common in children and patients with multiple sclerosis. Occasionally patients present with pain and photophobia and later develop signs of intermediate uveitis.

Aqueous flare is usually minimal. They rarely exceed 20 cells per field, and moderate flare. Kimura described the keratic precipitates as small or medium sized and white, even though gonioscopy may reveal a few mutton fat keratic precipitates. Brockhurst noted peripheral anterior synechiae in 24% of cases and posterior synechiae in 18%[31]. Children had band keratopathy. Khadodost demonstrated areas of peripheral corneal oedema with keratic precipitates at border between oedematous and normal cornea in 4 out of 10 patients. Autoimmune endotheliopathy is a rare finding. Anterior uveitis can occur due to spill over of inflammation from the parsplana and is typically non granulomatous. Brockhurst described the early parsplana inflammation to consist of yellowish grey globular exudates on the parsplana, ciliaris, at ora serrata and anterior portion of retina. These exudates are also seen at the filtration angle. They may hide the normal landmarks of the angle and ora serrata.

Chronic cyclitis as described by kimura showed varying number of finedust like opacities in the anterior vitreous, coarser opacities were observed later when vitreous degeneration also occurs.

FUNDUS

Fundus examination showed the presence of vitreous cells in almost all cases of active pars planitis. The vitreous opacities which consist of vitreous cells and debris with macrophages typically are seen as multiple snow ball opacities in the peripheral fundus.

Ahens and Sound macher described yellowish grey exudates in the inferotemporal ora serrata and parsplana. Brockhurst noted that these exudates coalesce to form the material commonly described as snowbanking. Snow banking is usually seen in the parsplana region and ora serrata inferiorly. They are usually discontinuous and form a thin or broad band extending into the peripheral retina. Even though this material has been often termed exudates, Nussenblat considers them to be most probably fibroglial scars[32]. Neovascularization associated with it can predispose the patient to vitreous haemorrhage. Pars plana exudates commonly described as snow banking is considered to be the hall markof the disease. Pars plana exudates are not mandatory for the diagnosis of pars planitis. Henderly, et. al, have found that 24% of patients with pars planitis can have vitreous cells

in the other eye which is probably the early sign of this disease. The presence of snow bank has been found to correlate with the severity of the disease. An increased incidence of cystoids macular oedema has been reported in eyes of pars planitis with snow banks.

Abnormality of Retinal Vessels

Peripheral retinal vascular abnormalities characterized by sheathing and obliteration of small peripheral venules can be seen. Periarteritis is less common. Peripheral neovascularization can evolve into a vascular cyclitic membrane. Vitreous haemorrhage is rare sequelae. Retinal venous dilatation predominantly involving the inferior branch is also associated with edema of posterior retinal oedema. It is more common in children[5].

Pigment clumps may be seen in peripheral fundus surrounding a depigmented zone. An active chorioretinitis is rare in these cases, even though Welch has mentioned of its existence. A careful examination of macula is important in patients with parsplana exudates and paravenous oedema.

Macula

Macular changes are quite commonly seen in these patients. The incidence is as varied as from 20-80%. Malinowswki reported macular

changes in 28% of cases (Malinowwski, SM, Pulido, JS, Long-term Visual Outcome and Complications Associated with Pars Planitis Ophthalmology 1993: 100: 818-825). Some reports 24.8% with varied macular pathologies, most of them being cystoid macular oedema. Hogan and associates reported incidence of 50% with macular oedema (Hogan, MJ, Kimura, SJ, Peripheral Retinitis and Chronic Cyclitis in Children Trans Ophthalmol Soc: UK 85: 39-52, 1965). Early macular oedema has loss of foveal reflex with a wet appearance to posterior pole and numerous glistening highlights reflected from the irregularly thickened oedematous retina. Kimura also descried macular oedema in 50% of cases. Severity of macular oedema depends on the severity of pars planitis characterized by heavy vitreous reaction and the presence of parsplana exudates throughout the peripheral retina and ora serrata.

Long standing macular oedema can lead to epithelial stippling in macula and may be a subtle sign of previous oedema in chronic cases of intermediate uveitis. Unfortunately this polycystoid macular degeneration can be the cause of severe visual loss in these patients[5].Loss of visual acuity is usually not present in the milder form of this disease but early profound loss of visual acuity is most commonly associated with cystoid macular edema. 74% patients with CME had poor visual acuity. The severity of reduction of visual acuity depends on the duration and extent of cystoid

macular edema. Disc edema was found in one third of cases. Disc edema was always associated with macular edema in pars planitis according to kimura. Fluorescein Angiography shows late staining of dye around the FAZ, and reduction of FAZ in macular edema. Fundus Angiography showed petalloid hyperfluorescence in cystoid macular edema[5].

Rhegmatogenous retinal detachment associated with vitreous traction is not uncommonly found in pars planitis. The incidence reported varies from 3% by Brockhurst to 22% by Smith, et. al, Nussenblat is of the opinion that the presence of retinal detachment may reflect the positive effect of therapy with corticosteroids. He also is of opinion that the varied range of incidence of retinal detachment is because of the predominantly retinal practice by Brockhurst.

Optic disc edema, optic atrophy and optic disc neovascularization has been reported in patients with intermediate uveitis. Chronic poorly controlled pars planitis often times had total retinal detachment and proliferative vitreoretinopathy due to the formation of cyclitic membrane and subsequent ciliary body traction. Pederson reported the presence of hypotony in these patients.

Brockhurst presented 2 varied type of presentation of pars planitis. 95% patients of 30 years of age predominantly had annular involvement of

parsplana ciliaris. The chief finding was a peripheral choroidal detachment on all quadrants.

Malignant progressive form of peripheral uveitis was present in patients who were under 15 years of age and was characterized by development of massive yellowish grey exudate and new vessels from ciliary body, but not from retina and forming a cyclitic membrane. Associated with this, later the cyclitic cicatrizing membrane adherent to retinal periphery gives rise to retinal detachment. Further cicatrization pulls the lens-iris diaphragm forward causing secondary glaucoma.

Even though inflammatory signs shows partial remission most studies gives very low incidence of permanent remission. Smith found only 5%remission rate when he followed the patients from 4-6 years. Hogan reported only one remission from 56 cases which he followed from 1-9 year. Aaberg states that only very rarely does one observe a permanent resolution of the disease.

SYSTEMIC ASSOCIATIONS

Pars planitis can exist as an isolated idiopathic disorder or can be associated with other diseases. Zieshut found that 9.7% had associated sarcoidosis (Henderly, DE, Changing Patterns of uveitis, Am.j. ophthalmol). In India 13 cases of pars planitis with sarcoidosis was reported from

Sankara Nethralaya. Majority of the patients had associated granulomatous uveitis. Recent reports indicate that 14.8% of patients with pars planitis develop multiple sclerosis over 7 years follow up.

Lyme disease caused by Borrelia burgdorferi and transmitted by Ixodestick seems to be associated with pars planitis. Friedman reported 5 cases with lyme disease.

Boskovich noted systemic disorders in 26 out of 83 patients with pars planitis.

Out of 26, 10 had presumed sarcoidosis, 6 had multiple sclerosis, 2 had optic neuritis, 2 had inflammatory bowel disease, 4 had thyroid abnormalities, 2 EBV infection.

DIFFERENTIAL DIAGNOSIS

1. VITREOUS OPACITY

Posterior uveitis

Endophthalmitis

Amyloidosis

Reticulum cell sarcoma

2. PERIVASCULITIS

Eales disease

Sarcoidosis

Behcet disease

3. PERIPHERAL GRANULOMA

Toxocara

4. SPILLOVER ANTERIOR UVEITIS

Non granulomatous anterior uveitis

PATHOLOGY

Gross examination reveals extension scars over the pars plana region. Microscopy of snow ball opacities shows epitheloid cell granulomas.

Peripheral snow banking reveals, thick connective tissues consisting of glial cells including fibrous astrocytes and scattered inflammatory cells predominantly lymphocytes. The paucity of inflammatory cells indicate that the presence of snow bank is rather suggestive of a chronic inflammation with reactive changes than an acute inflammation[32].

Immunopathologic study of snow bank over pars plana showed a ratio of helper/inducer cells to suppressor/cytotoxic cells to be 10:122. This pattern of CD4:CD8 is also seen in multiple sclerosis. Most of these lymphocytes were found to bear class II HLA antigen.

Muller cells proliferate in response to factors produced by activated lymphocytes, suggesting a means of snow bank formation. In tissues muller cells express MHC class II molecules and present to T cells, contributing to inflammation. Type IV collagen and laminin were the major collagen glycoproteins in this snow bank. High ratio of suppressor/helper cells suggests an active T cell mediated process in the eye.

Aetiopathogenesis

The cause of pars planitis has not been elucidated. The inferior location of snow banking is attributed to gravity. Gartner suggested that it may be due to the more number of vitreous cells normally present in the regions inferiority.

Davis proposed two stage disease process of which stage(1) is immunologically mediated and stage (2) is non-specific breakdown of intraocular regulatory mechanism resulting in chronic inflammatory disease (Nozik, Uveitis: A Clinical Approach in Diagnosis and Management 2nd Edition 166-170). Descheren attributes a diffuse phlebitis and subsequent

breakdown of blood ocular barrier. This causes release of inflammatory cells, cytokines and other inflammatory mediators that settle inferiorly.

Khadodaust had hypothesized that intermediate uveitis is primarily vitreal inflammation which starts adjacent to involved vascular tissue.

Injection of hyaluronic acid into the vitreous of monkey produced a clinical picture of pars planitis.

Gartner demonstrated that the vitreous cellular remnants near the vitreous base may be the antigenic stimulus in pars planitis.

Kaplan noted a protein P36 in the serum of patients with active pars planitis that may be a marker of the disease. Several other studies have revealed several immunological abnormalities in the serum with pars planitis. Rahi, et. al, found decreased levels of complement C3 in the serum of pars planitis patients Kaplan noted that the normal T cell regulation of B cell function is deranged, because they observed a relatively increased number of B cells and null cells in comparison to T cells in the aqueous and vitreous of patients with pars planitis. Retinal S antigen which is located in the plasma membrane of the photoreceptors of retina and having uveitogenic property is increased in patients with pars planitis. Intercellular adhesion molecular (ICAM-1) was raised in a considerable number of patients with pars planitis. Increased serum antibody to Retinal S Antigen,

serum immunoglobulin and circulatory immune complexes associated with HLA-DR2 antigen and presence of familial incidence that a complex immunological process is involved in the pathogenesis of this disorder. The specific cause of this disease is most probably multifactorial and probably differs from case to case.

Treatment

As no specific etiology is found, the treatment of this condition remains non specific. The disease is by nature slowly progressive. The role of treating mild vitritis, mild periphlebitis and early mild cystoids macular edema with good vision, with aggressive anti-inflammatory therapy is unknown.

Kaplan advised treatment of patients with

1. Visual Acuity of less than or equal to 6/12.
2. Eye with extensive neovascularization and snow banking
3. With substantial vasculitis.

Medical Treatment

Topical steroids are not effective except in aphakic patients with mildvitritis. Periocular steroids with depot Triamcianalone is tried twice or

thrice weekly over a 6-8 week period. They are effective in treating uniocular disease.

Systemic steroids of Prednisolone at 1mg/kg/day is given for[5]

1. Bilateral disease
2. Ocular disease resistant to periocular steroids.

Immunosuppressives

Cyclosporine is used as a steroid sparing agent for

1. Resistant cases to steroids or
2. Patients with long term side effects to steroids

Nussenblat described these indications and found out that cyclosporine is effective in the treatment of pars planitis.

Major side effect of cyclosporin is the renal toxicity and hypertension (Nussenblat, uveitis: Fundamentals and Clinical Practise 286-287).

Azathioprine at a starting dose of 50mg thrice daily with gradual tapering is effective in patients who are resistant to corticosteroids.

Various other immunosuppressives like cyclophosphamide, chlorambucil and methotrexate have been tried.

Cryotherapy

Cryotherapy applied to the parsplana exudates is thought to be effective in treatment of Pars planitis. Aaberg reported success with cryotherapy on steroid resistant cases in 1973. Aaberg believes that cryo application to the exudate destroys the vascular component of peripheral retinitis or vitrits and eliminates the entrance sites of inflammatory mediators into the eye (Asberg, TM, Cesarz, et. al., Treatment of Peripheral Uveitis by Cryotherapy Am.J.Ophthalmol 1973, 75: 685-688). Devenyi is of opinion that it is necessary to ablate the new vessel and periphlebitis, rather than areas of exudation, this destroys the neovascularization and the ischaemic tissues, nussenblat is of the opinion that the effect is evident within several weeks and lasts for 2-6 months. A second treatment may be given if the 1st treatment was effective. Devenyi reported the use of cryotherapy in 27 eyes in 18 patients with steroid resistant pars planitis and claimed that vitritis was eliminated in 75% with a follow up of 2-12 years. In 90% of patients steroids were not needed after cryo application was done. 67% of patients demonstrated improvement in visual acuity on an average of 3 lines. 85% needed only 1 treatment, 11% needed 2 sittings and 4% needed 3 sittings before regression of neovascularization was achieved. Aaberg treated patients who were not responding to 80-100mg of prednisolone and 80mg depot Medrol posterior subtenons every 2-4 weeks

for 6 months, and these patients not undergoing remission in 6 months with cryo application. End point was the appearance of an iceball which engulfed exudative focus over the probe tip. After brief thawing the same area was immediately refrozen to the same degree. Further all areas of inflammatory response were also treated. 35% of patients remained inactive for 14 months. 57% of patients had marked decrease in activity requiring no corticosteroid therapy. Complication includes transient increase in vitreal inflammation, decreased accommodation, cataract and hyphaema. There had been reported increase of post treatment retinal detachment.

After ruling out infectious causes of IU, the following (modified Kaplan's) algorithm is suggested

Step 1. Periocular steroids administered by local injection of depot Corticosteroids may be repeated every 4 weeks until 3-4 injections have been administered. Generally the inflammation responds and the CME improves. (Intravitreal triamcinolone (IVTA) may be an alternative to periocular injections in refractory cases.). The procedure followed is described by Nozit. The cotton tipped applicator soaked in 4% lignocaine hydrochloride is applied over the upper outer quadrant (superotemporal). Using a 26 gauge 5/8 inch needle with the bevel facing up, the needle is advanced posteriorly with a broad side to side slow posterior movement

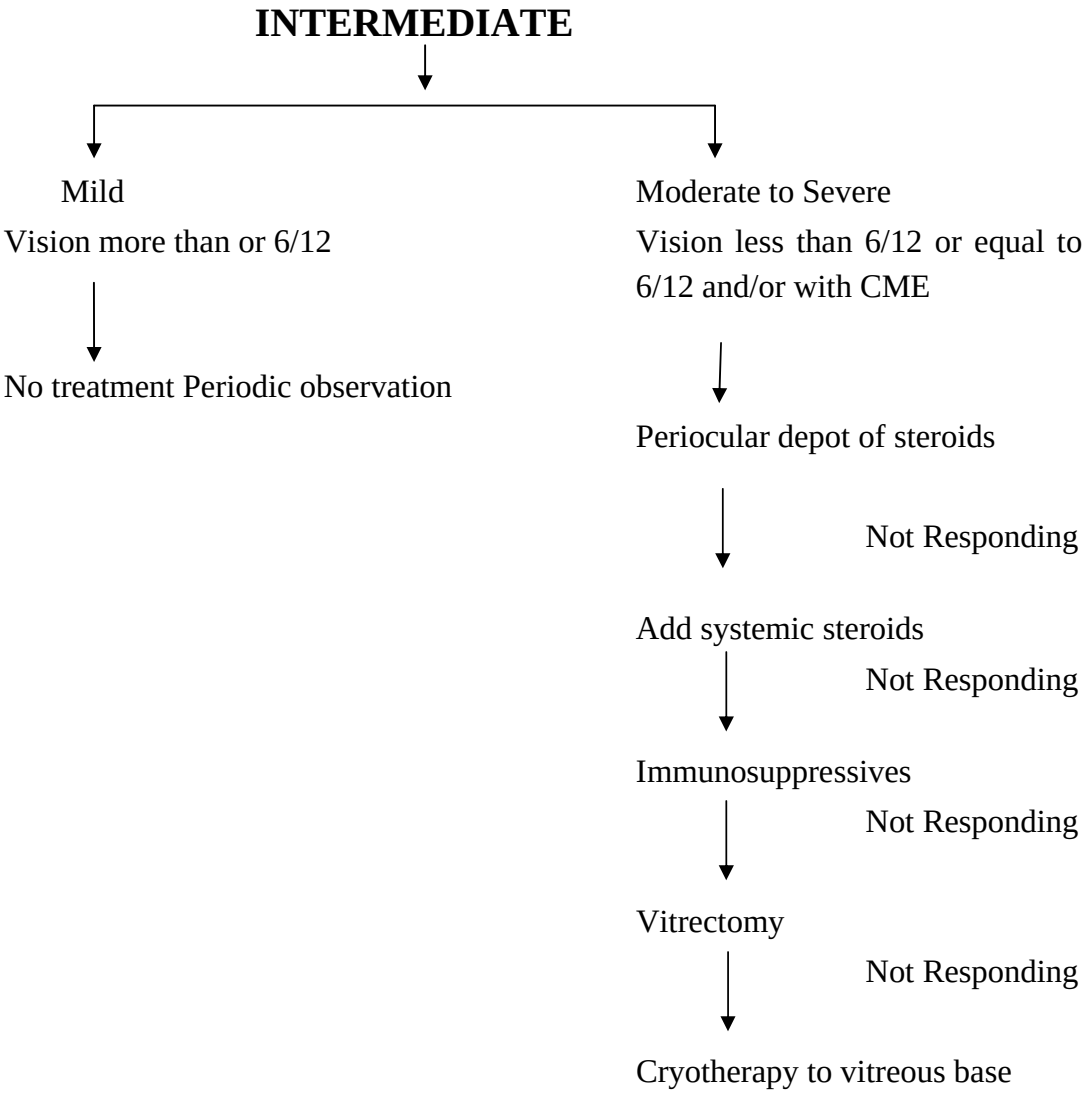
following the curve of the globe, taking care that the globe does not move with the needle. The bevel end is rotated downwards and 0.75 – 1ml of steroids is injected as posteriorly as possible to reduce the risk of steroid induced glaucoma.

Step 2. If local therapy is not effective or bilateral severe disease is seen at presentation oral corticosteroids prednisolone 60-80mg/day are indicated.

Step 3. Systemic immunomodulatory therapy is indicated in the treatment of bilateral disease, and can be considered if corticosteroids fail, are not tolerated or contraindicated

Step 4. If corticosteroids fail, or if corticosteroids and immunomodulatory therapy are contraindicated, and if pars plana snowbanks are present, peripheral ablation with cryotherapy or indirect laser photocoagulation to the peripheral retina can be done.

Step 5. If all the treatment modalities fail to control inflammation, parsplana vitrectomy with induction of posterior hyaloidal separation and peripheral laser photocoagulation to parsplana snowbank may be performed, along with immunomodulatory therapy.



CATARACT EXTRACTION IN PARS PLANITIS

The eye should be quiet for at least 3 months prior to surgery. Preoperative systemic steroids of 40 – 60 mg per day and posterior subtenon injection of depot steroid 3 – 4 days prior to surgery is preferred.

Phacoemulsification with IOL implantation in eyes with pars planitis is reported to be safe leading to good visual outcomes in most cases[11]. The factors in surgical success were control of inflammation, meticulous surgery, in-the-bag IOL implantation, and vigilant postoperative care.The frequent cause of poor visual recovery was CME, submacular fibrosis, and epiretinal membrane. Absolute control of inflammation in patients with pars planitis through a stepladder approach may reduce the incidence of cataract development, and can certainly improve visual rehabilitation after cataract extraction. Implantation of a posterior chamber lens can be well tolerated in selected cases. Careful patient selection, coupled with the use of an appropriate surgical technique, appear to be of major importance.

PART II

AIM OF THE STUDY

1. To find out the prevalence, the clinical profile and response to therapy in patients with pars planitis.

2. To identify different modes of presentation and a comparison was made with other studies.

METHODS AND MATERIALS

Fifty cases of pars planitis who attended the uvea clinic of the Regional Institute of Ophthalmology from March 2009 to September 2010, were taken up for this study.

The main criteria of diagnosis were

1. Presence of vitreous cells and / or vitreous opacities in the vitreous base adjoining the pars plana and peripheral retina.

2. Absence of predominant anterior segment inflammation characterized by cells of more than 2+ and KP`s of more than 2+.

3. Presence of snow ball opacities and snow banking with peripheral vasculitis signs, like periphlebitis and paravenous oedema were considered to be the hallmark of pars planitis, eventhough they were not considered essential for the diagnosis of parsplanitis.

Patients with following conditions were excluded

1. Anterior segment predominantly involved with more than 2+ cells and 2+ KP`s.

2. Active posterior segment inflammation like retinochoroiditis in the posterior pole. But patients with central retinal leaks alone on FFA were not excluded from the study.

3. Patients with signs of active tuberculous choroiditis and other signs of granulomatous uveitis, were excluded from the study.

4. Patients who showed peripheral tubercles and lesions suggestive of Toxocariasis and Toxoplasmosis were excluded from the study.

The presenting complaints of the patients were sought for and the laterality of the symptoms was noted. Patients were encouraged to quantify the loss of vision and the hazy vision. Patients who complained of vitreous floaters were asked for the laterality of the symptoms and history of flashes of light, or trauma before the onset of floaters.

A negative history of symptoms of anterior uveitis like redness, pain and photophobia were asked for. A careful history of systemic manifestations of sarcoidosis like wheezing, rashes and arthritis was asked for. A family history of similar complaints was asked. A diligent search was made to find out any etiological factor or aggravating factor in the history of the disease.

PERSONAL HISTORY

1. Ocular Toxoplasmosis and Toxocara were suspected in patients coming into close association with pets.
    1. Dietary history was taken.
    2. Prevalence of Hypothyroidism, Diabetes mellitus and Hypertension were asked for.
    3. Family history of Glaucoma was asked before the patient was started on steroids.
    4. Before considering FFA the females were asked about the menstrual habits and pregnancy.

DEMOGRAPHIC FACTORS

Age and sex of the patients were noted.

CLINICAL SIGNS

The patients who were suspected to have pars planitis based on the complaints of blurring of vision with floaters with very rare complaints of redness, watering and photophobia were subjected to clinical examination to rule out pars planitis.

Slit lamp examination was done and flare and cells were graded as per the grading by the Standardization Of Uveitis (SUN) working group.

I. FLARE

Flare	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)

II. CELLS

Grade	Cells in field
0	<1
0.5+	1 – 5
1+	6 – 15
2+	16 – 25
3+	26 – 50
4+	>50

III. KERATIC PRECIPITATES

Keratic precipitates were categorized on the basis of size (as small, medium and large), shape, colours (fresh - white and round, old – shrunken and pigmented) and appearance (mutton fat KP`s signifying granulomatous uveitis and white round KP`s non granulomatous).

They were further graded as

	Per field
1 +	5 - 1 0
2 +	11 - 20
3 +	21 – 50
4 +	> 50

Patients who presented with signs of predominant anterior segment involvement i.e. ., cells and KP`s over 2+ and flare more than 3+ were excluded from the study, as it was presumed that the intermediate uveitis seen along with the anterior uveitis, is due to the spill over from iridocyclitis. Further granulomatous uveitis without signs of systemic sarcoidosis was also excluded from the study.

Gonioscopy was done on all patients to rule out the presence of KP`s at the Angle and Peripheral anterior synechiae. The angle was graded on all patients with raised intraocular tension by Shaffer system as

Grade 4: 35 - 45°	-	Ciliary body can be visualized
Grade 3: 20 - 35°	-	Scleral spur can be identified.
	-	It is incapable of closure
Grade 2: 20°	-	Trabeculum can be identified
	-	Moderately narrow angle
	-	Angle closure is unlikely
Grade 1: 10°	-	Only schwalbe`s line visible
	-	Angle closure is not inevitable even though risk is high
Slit angle	-	No obvious iridocorneal contact
	-	No angle structure identified
	-	Angle has greatest danger of imminent closure
Grade 0	-	Closed angle resulting from iridocorneal contact

Patients with IOP of over 20 mm Hg by schiotz were subjected to detailed evaluation for glaucoma at the glaucoma clinic of Regional Institute of Ophthalmology.

IV. VITREOUS CELLS

Vitreous cells were graded with Hruby lens examination by retroillumination, cells appear as back dots, debris is often pigmented and forms clumps.

GRADE	VITREOUS CELLS
4+	>250 cells
3+	101 – 250
2+	51 – 100
1+	21 – 50
Trace	2 – 20
0	0 – 1

The presence of vitreous cells or opacities was considered necessary to make the diagnosis of active pars planitis, and to be included in the study.

Fundus examination was done by dilating the pupil with tropicamide and 10% phenylephrine. Detailed fundus examination nincluded direct ophthalmoscopy, indirect ophthalmoscopy and confirmation of the diagnosis by Goldmann 3 mirror examination and the site of abnormalities carefully noted. The findings were confirmed independently by a uveitis specialist.

Vitreous opacities were graded as by Nussenblatt`s classification as

GRADE	DESCRIPTION
0	Clear vitreous
1+	Few scattered opacities, Fundus view unimpaired
2+	Moderate vitreous opacities, Fundus view somewhat impaired
3+	Many opacities, marked blurring of fundus details
4+	Dense opacities. No fundus view

The location of whitish gray snow ball opacities in the peripheral retina was noted. Exudates characteristically present as snow banking was considered Hallmark of the disease. The site and extent of the snow banking was noted.The character of the retinal vessels both in periphery and inthe posterior pole was observed by indirect ophthalmoscopy and 3 mirror examination. Para venous edema and periphlebitis and vasculitis were localized. Macular changes were looked for, and early and suspected macular edema was subjected to fundus fluorescein angiography, Goldmann

3 mirror examination was done to look for macular hole and epiretinal membrane formation.

A detailed and careful examination of the fundus was done to rule out any retinochoroiditis. Active and multifocal retinochoroiditis was considered to be posterior uveitis with intermediate spill-over and were excluded from the study. Patients with peripheral few retinochoroiditis were not excluded from the study.

Patient’s visual acuity was measured by Snellen`s charts and refraction with improvement of vision with glasses were recorded at the time of inclusion into the study. The patients’ visual acuity was followed up at the periodic review of the patient.

Amsler grid was examined at the time of inclusion. Patients were followed up for alteration in the image size and shape at every periodic review. Pupillary reflex was carefully noted and swinging flash light done to exclude optic neuritis.

Presence of complications like peripheral anterior synechiae, posterior synechiae, glaucoma, posterior capsular cataract was looked for.

Macular degeneration was looked for presence of retinal detachment, vitreous haemorrhage were examined.

INVESTIGATIONS

Routine investigations were done on all patients. They included total, differential count, urine analysis, serum VDRL and chest X-ray. Mantoux was done on all patients to rule out tuberculosis and to get the baseline before the patients were put on steroids. Anergy to PPD was also looked for along with the clinical examination and chest X-ray to rule out sarcoidosis. Patients who had a positive mantoux test were referred to TB clinic forruling out primary pulmonarytuberculosis.

Associated Rheumatological disorders were ruled out by looking for rheumatoid factor on patients who were suspected to have clinical symptoms and signs of collagen vascular disease.

Fluorescein Angiography was done mainly

1. To assess the presence and extent of cystoid macular edema in suspected eyes.

2. To examine the retinal vasculature for signs of perivasculitis which included staining of vessel wall and vascular leakage of dye.

Cystoid macular edema was classified as

a. Mild : with increase thickness of macula

: decrease in size of Foveal Avascular Zone

b. Moderate : Pooling of dye in paramacular region. It is often associated with late leakage

c. Severe : Flower petal pattern due to leakage of dye from the perifoveal capillaries.

OCT was done in four patients with clinical evidence of CME.

TREATMENT AND FOLLOW UP

After detailed examination of the patients and recording of findings, and result of investigations, and opinion from dental, otolaryngology and rheumatology for ruling out focal sepsis and collagen vascular disease, the patients were categorized for medication.

TREATMENT PROTOCOL

Patients observed :

a. Vision of more than 6/12

b. Absence of CME

c. Absence of confluent exudates or vasculitis

d. Symptoms which did not hinder their routine daily activities,

provided they were not started on steroids from the referral units.

Patients started on Posterior Subtenon Dexamethasone

a. Presence of definite CME

b. Vision of less than or equal to 6/12

c. Patients who had bilateral disease, but were willing for daily injection

d. Patients who had vision of more than 6/12, but had symptoms like floaters, haziness of vision which hampered their daily activities.

Patients started on systemic steroids

a. Eyes with severe CME

b. Bilateral signs of pars planitis

c. Patients not responding to subtenons for 2 weeks or worsening during treatment or not showing compliance for posterior subtenon injections.

d. Confluent pars plana exudates and vitreous opacities causing defective vision.

Patients started on Immunosuppressives

a. Not responsive to oral and subtenon`s steroids for 2 weeks

b. Not tolerant to steroids.

Patients who had cryopexy

a. Were not regular for follow up.

b. Had confluent snow banking and vasculitis.

c. Not responsive to oral and subtenon`s steroids for 2 weeks and not tolerant or fit for immunomodulators and for worsening on treatment without a vitreous haemorrhage.

Treatment was indicated when

1. Visual acuity was 6/12 or less.

2. Patients with presence of CME and

3. Patients with extensive pars plana exudates and vitreous opacity, who were symptomatic even though the visual acuity was maintained.

Most of these patients presented with hazy vision, and floaters which prevented them from doing their routine activities were considered for treatment if their complaints did not improve after 2 weeks on placebo treatment.

4. Patients who were classified as exudative type of pars planitis and vasoproliferative type of pars planitis were considered for more radical treatment which included systemic steroids and Cryotherapy.

5. Patients who developed complications like glaucoma, while on steroids were considered for cryotherapy.

6. Patients who had anterior segment inflammation which included cells in anterior chamber and fresh KP`s were started on treatment of anterior uveitis with topical steroids and patients were Atropinised to prevent complication of anterior uveitis.

7. Treatment for cataracts, with pars planitis were considered when the gross decrease in vision was due to the lens opacity

Once the patients were decided to be started on therapy, with steroid, routine mantoux, blood sugar 2 hours post prandial and IOP were noted. Those who had unilateral disease, or predominantly unilateral disease, mostly of the serous and infiltrative type were started on posterior

subtenon`s injection of Dexamethasone by Nussenblatt`s method in the superotemporal quadrant alternate days for 2 weeks. The findings were reviewed after 2 weeks. Subjective improvement of quantity of visionwas also sought for. Exudative type of pars planitis was also similarly given a therapeutic trial with subtenon injection of dexamethasone of 4mg alternate days for 2 weeks.

Patients who had equivocal macular edema and clinical findings not consistent with the loss of vision and subjective symptoms were subjected to fundus fluorescein angiography by the zeiss camera and further peripheral leaks were noted.

Those patients who had not responded to posterior subtenons steroids were started on systemic steroids. Patients who had also visual acuity less than 6/12 even after subtenons injection of steroids after 2 weeks were considered for systemic steroids. Patients who had bilateral disease were considered for systemic steroids especially when they had CME. Patients who were not cooperative for subtenon injection and were willing to come on atleast fortnightly follow up were also put on systemic steroids.

Tablet prednisolone was given at 1mg/kg/day in a single dose. This was continued for 1 week and improvement of visual acuity and subjective symptoms were asked for. Clinical examination was also done to correlate

the complaints. Severe inflammation and CME with gross reduction of vision were put on 1.5mg/kg/day of prednisolone.

The patient was reassessed after 2 weeks and those patients who had improved visual acuity, complaints and clinical findings were considered for tapering of steroids.

Those patients who did not respond to steroids even after 2 weeks were considered for immunosuppressives. Patients who were not willing or not tolerant to immunosuppressives were subjected to Cryotherapy.

Cryotherapy was considered for patients with extensive exudative disease and vasoproliferative type of pars planitis. Modified Aaberg`s method of Cryotherapy was done without peritomy. Areas of extensive pars plana exudate was treated with freeze thaw freeze technique. Areas of abnormal vasculature were treated, most of which were associated with exudates. Patients, who were on systemic steroids, were continued at same dose for 1 week. The patient’s visual acuity and subjective response was asked for on review after 1 week and decision to taper the steroids or continue them was taken. If there was deterioration of visual acuity of more than 2 lines, patients were started on steroids. Further examination was done after 1 month.

Patients were followed up on maintenance dose of steroids and on observation once every month for possible complications and exacerbation of disease. Remainders were sent once in 4 months for those patients who did not come for follow up.

Patients who needed cataract surgery were taken up for phacoemulsification with PCIOL under cover of systemic and subtenon dexamethasone 2 days before surgery with subtenon on the day of surgery. Patients were also started on T.prednisolone at 0.5mg/kg/day. Patients were followed up once a day for 4 days and subsequently once a week for development of pars planitis for one month.

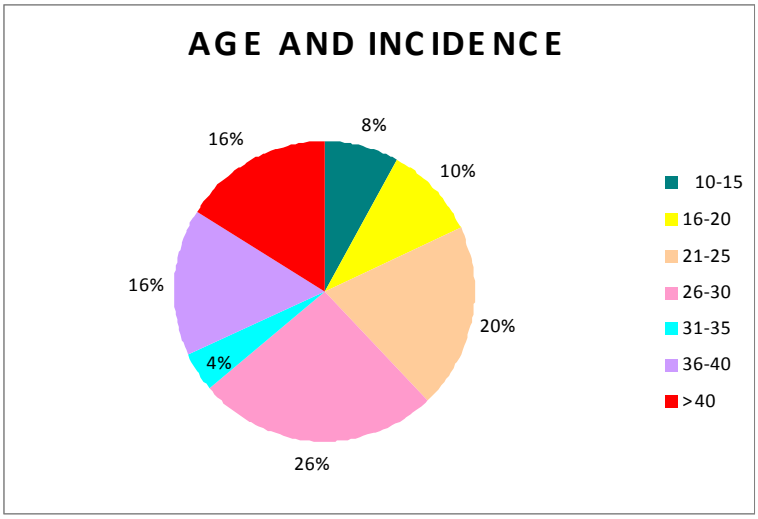
RESULTS AND ANALYSIS

A. DEMOGRAPHY AND SYMPTOMS

Total no. of cases included in study: 50 patients

I. AGE : INCIDENCE

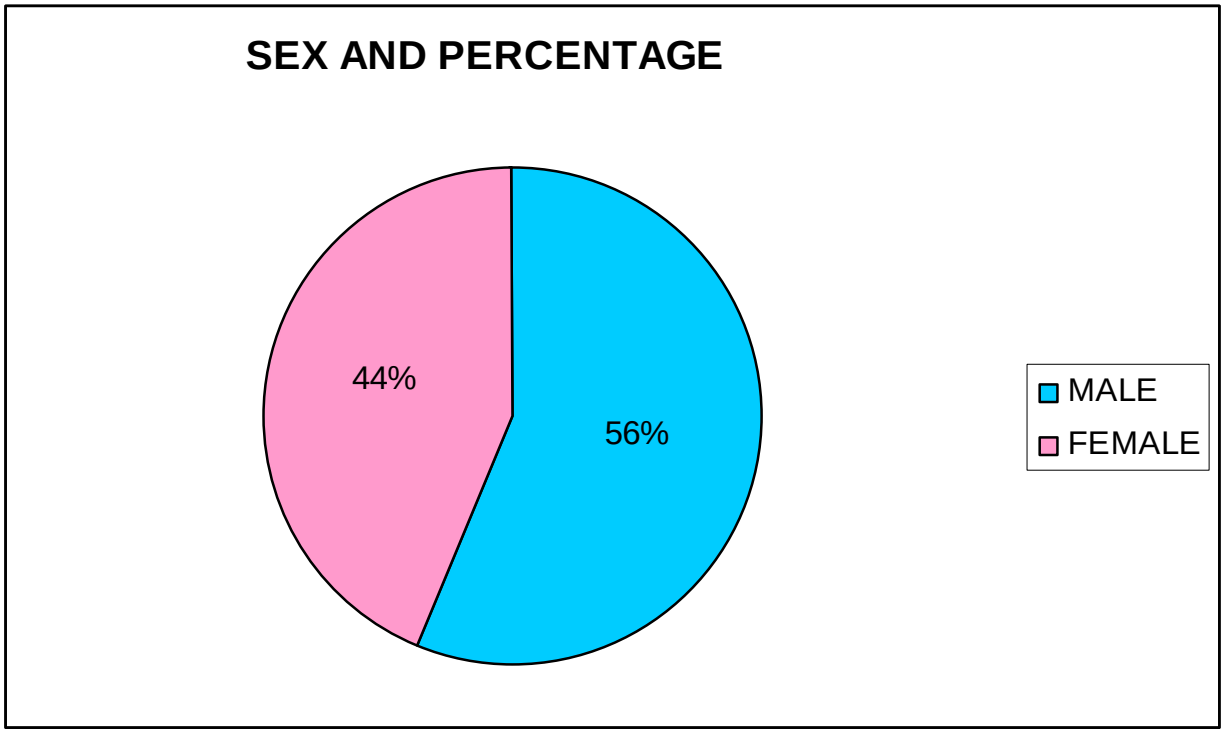
YEARS	NO. OF CASES	PERCENTAGE
10-15	4	8
16-20	5	10
21-25	10	20
26-30	13	26
31-35	2	4
36-40	8	16
>40	8	16



46% of patients were between the age groups of 20-30 years.

II. SEX

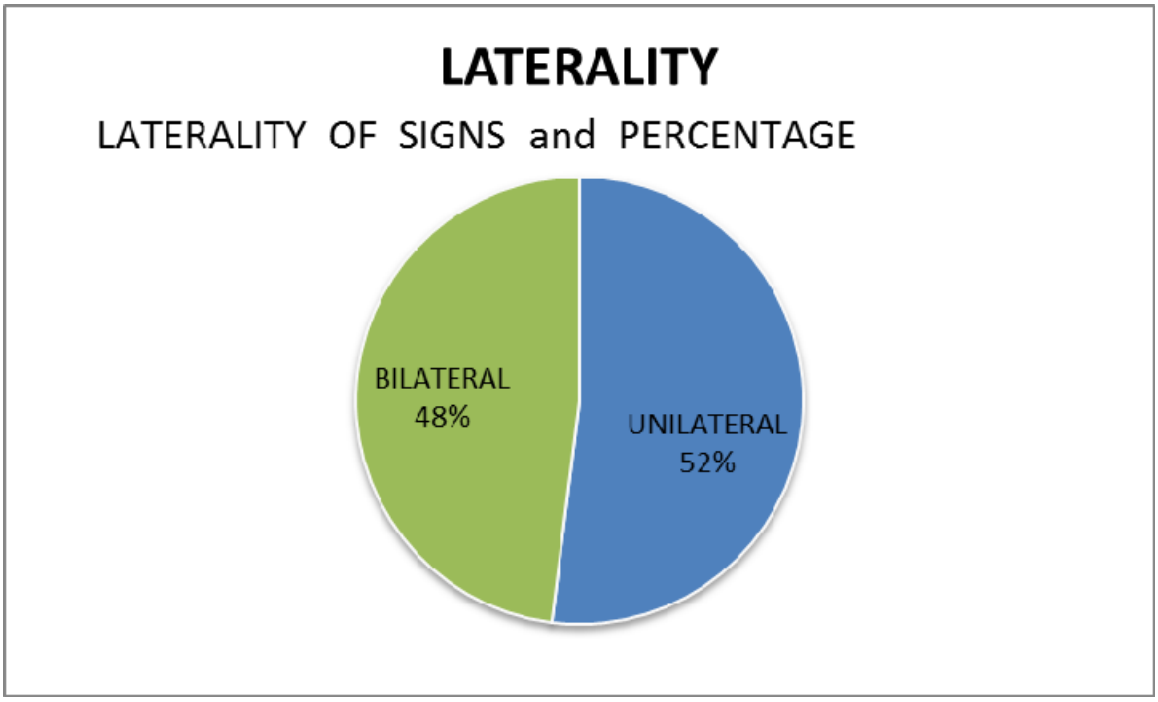
SEX	NO. OF CASES	PERCENTAGE
MALE	28	56
FEMALE	22	44



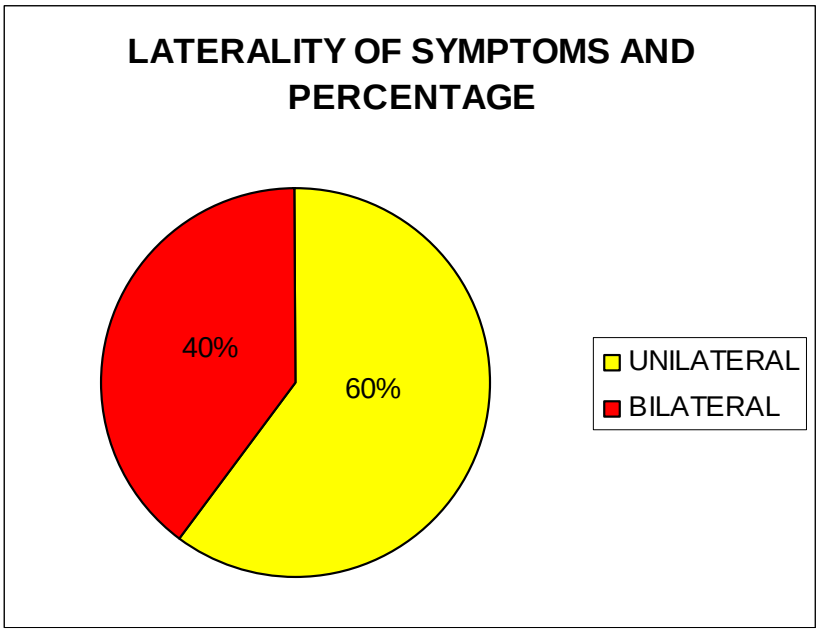
There was no sex predilection. Percentages were 56 male patients and 44 female patients.

III. LATERALITY

LATERALITY OF SIGNS	NO. OF CASES	PERCENTAGE
UNILATERAL	26	52
BILATERAL	24	48



LATERALITY OF SYMPTOMS	NO. OF CASES	PERCENTAGE
UNILATERAL	30	60
BILATERAL	20	40



60% of patients came with unilateral symptoms. 48% of patients had signs of bilateral disease. Total no. of eyes affected was 74.

IV. ASSOCIATED DISORDERS

S. No	ASSOCIATED DISORDERS	NO. OF CASES
1	HEALED CHORIORETINITIS	6
2	HIGH MYOPIA	2
3	PRIMARY COMPLEX	1

B. SIGNS

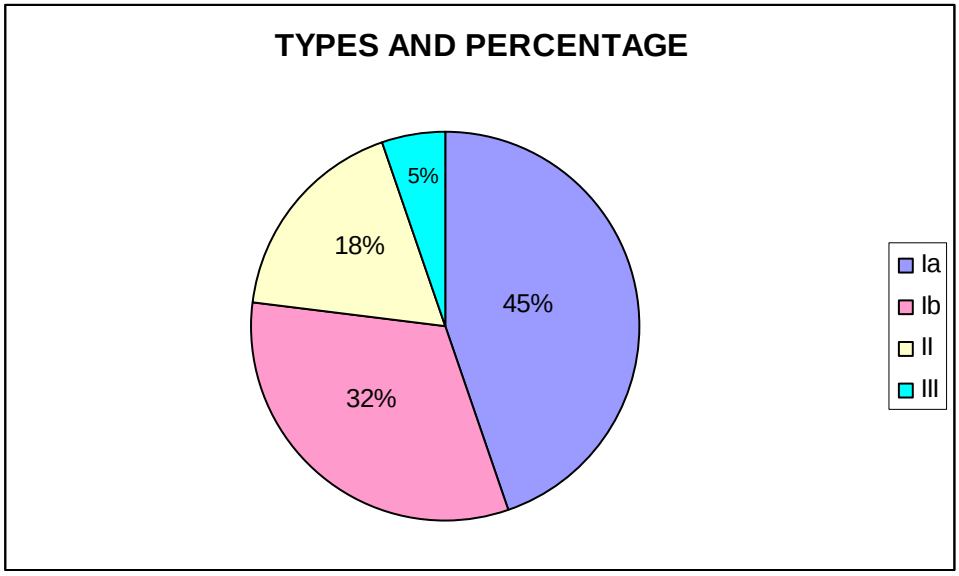
I. a. ANTERIOR SEGMENT INVOLVEMENT

SIGNS	NO. OF EYES
KP`S	18
FLARE`	25
CELLS	19

25 out of 74 eyes had signs of anterior segment spill over. All thepatients with anterior segment signs had mild degree of flare. KP`s were present in 24% of patients with pars planitis. 26% of patients had cells in anterior chamber.

b. **TYPE OF PARS PLANITIS (BOKE`S CLASSIFICATION WITH SLOVAK MODIFICATION)**

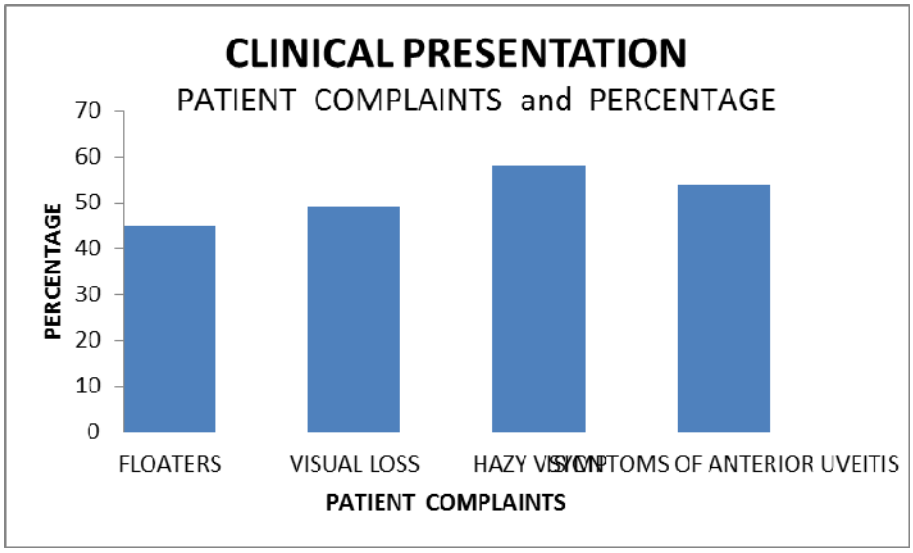
TYPE	NO. OF EYES	% n = 74
Ia	33	44.6
Ib	24	32.4
II	13	17.6
III	4	5.4



77% of the eyes involved belonged to the diffuse inflammatory type (typeI).

II. CLINICAL PRESENTATION

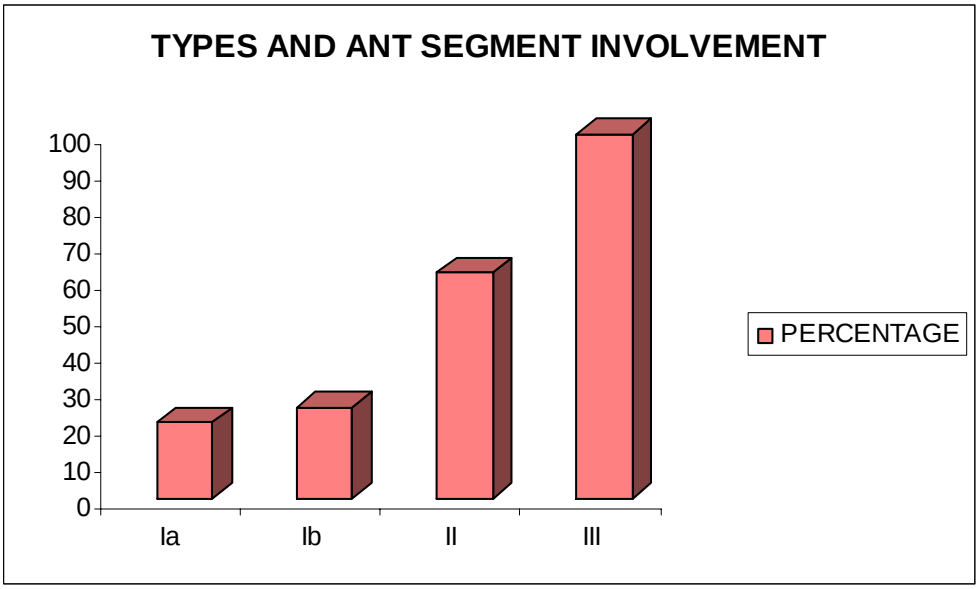
PATIENT COMPLAINTS	NO. OF EYES	PERCENTAGE
FLOATERS	33	45%
VISUAL LOSS	36	49%
HAZY VISION	43	58%
SYMPTOMS OF ANTERIOR UVEITIS	40	54%



Haziness of vision was the chief complaint in 58% of patients.54% of them had symptoms of anterior uveitis like redness, photophobia and watering.

III. CORRELATION WITH TYPE AND ANTERIOR SEGMENT INVOLVEMENT

TYPE	NO. OF EYES	EYES WITH ANTERIOR SEGMENT INVOLVEMENT	PERCENTAGE
Ia	33	7	21%
Ib	24	6	25%
II	13	8	62%
III	4	4	100%



More than half of the eyes with exudative type had anterior segment spill over. All cases of type III had spill over anterior uveitis.

IV. POSTERIOR SEGMENT INVOLVEMENT

A.CORRELATION BETWEEN VITREOUS CELLS AND VISUAL ACUITY AT PRESENTATION

VITREOUS CELLS	VISION AT PRESENTATION				
	6/6-6/9	6/12-6/18	6/24-6/36	6/60 & >	TOTAL
0	27	2	4	0	33
1+	3	5	5	3	16
2+	1	11	9	17	38
3+	0	2	3	7	12
4+	0	0	0	0	0

Eyes with visual loss of more than 6/24 on snellen`s chart were more likelyto have vitreous cells. The loss of vision is directly proportional to theintensity of inflammation of vitreous.

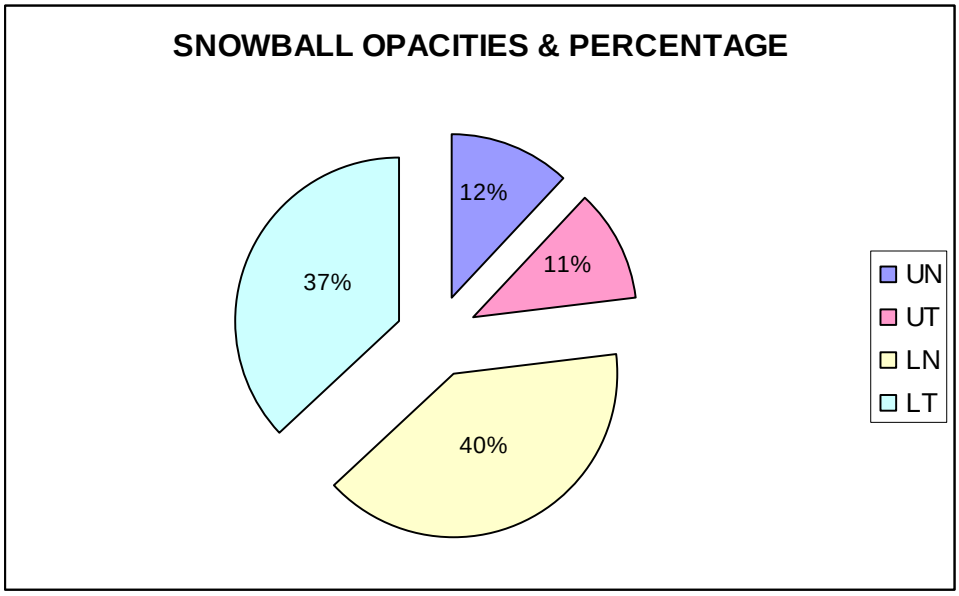
B. VITREOUS OPACITY AND VISUAL ACUITY AT PRESENTATION

VITREOUS OPACITY	VISION AT PRESENTATION				
	6/6-6/9	6/12-6/18	6/24-6/36	6/60&>	TOTAL
0	22	3	2	0	27
1+	6	7	7	14	34
2+	3	9	11	8	31
3+	0	1	1	2	4
4+	0	0	0	2	2

34% of patients had vitreous opacities of 1+. Eyes with vitreous opacities of 4+ at presentation had considerable loss in visual acuity. No direct correlation was found between the visual loss and amount of vitreous opacities.

C. SITE OF SNOWBALL OPACITIES

SITE OF SNOW BALL	NO. OF EYES
UN	12
UT	11
LN	40
LT	37

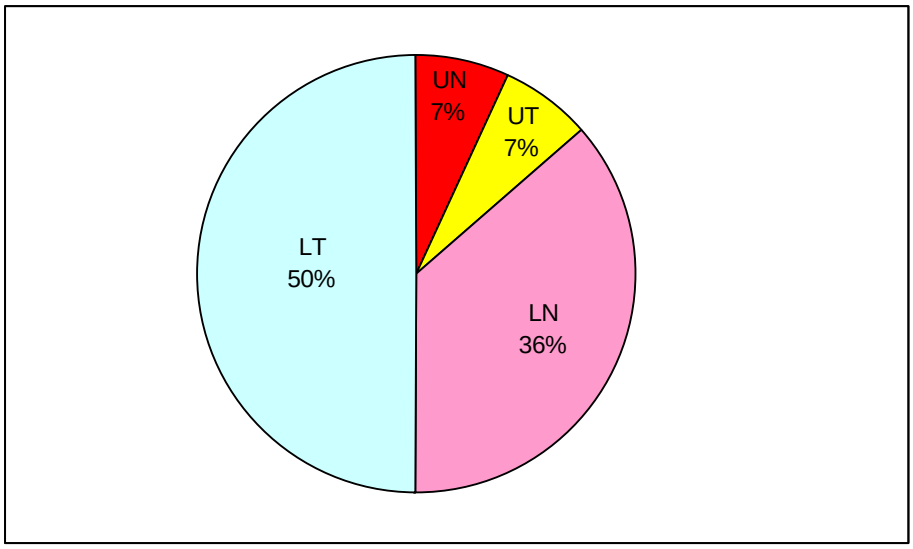


Snow ball opacities are more common in the inferior half of the vitreous.

40% of eyes had involvement of the lower nasal quadrant.

D. SITE OF SNOW BANKING

SITE OF SNOW BANKING	NO. OF EYES
UN	3
UT	3
LN	16
LT	22



50% of eyes with snow banking had it in the lower temporal quadrant. 86% of the snow banking was in the lower half of retina.

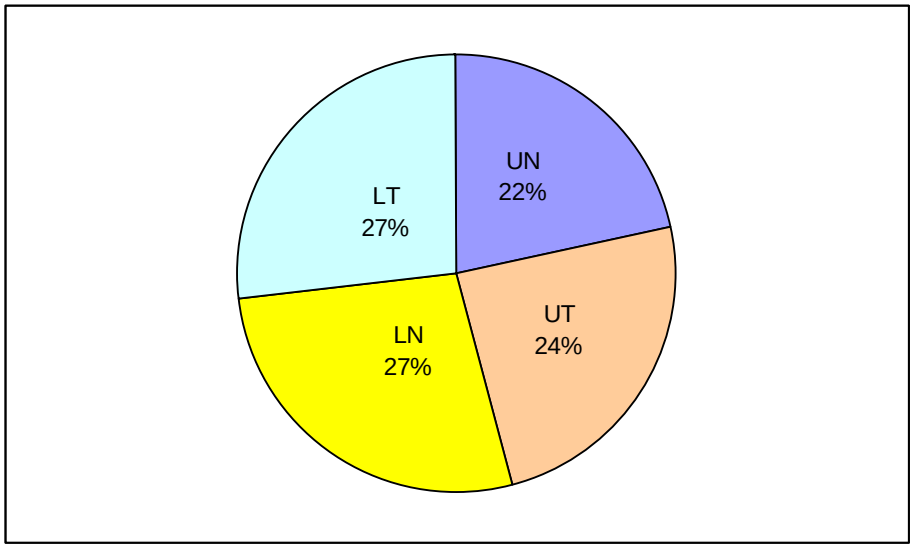
E. CORRELATION BETWEEN EXTENT OF SNOWBANKING AND VISUAL LOSS

NO. OF QUADRANTS INVOLVED	6/6-6/9	6/12-6/18	6/24-6/36	6/60&>	T
1Q	1	2	2	3	
2Q	-	1	4	7	12
3Q	-	-	1	1	2
4Q	-	-	1	-	1

Snow banking was present in 23 eyes with pars planitis. Eyes with more than 2 quadrant of snow banking had considerable visual loss.

F. SITE OF PERIVASCULITIS

SITE OF PERIVASCULITIS	NO. OF EYES
UN	8
UT	9
LN	10
LT	10



Lower half of retina had 54% of the vasculitis. But there was no direct correlation between vasculitis and snow banking even though most of the vasculitic vessels had snow banking near them.

G. EXTENT OF VASCULITIS AND THEIR CORRELATION WITH REDUCTION IN VISUAL ACUITY

EXTENT	6/6-6/9	6/12-6/18	6/24-6/36	6/60&>	T
1Q	4	1	2	-	7
2Q	-	2	1	2	5
`3Q	-	1	2	-	3
4Q	-	-	-	3	3

Gross reduction of vision was seen in patients with all 4 quadrants with vasculitis. More than 50% of patients with one quadrant of vasculitis had good visual acuity.

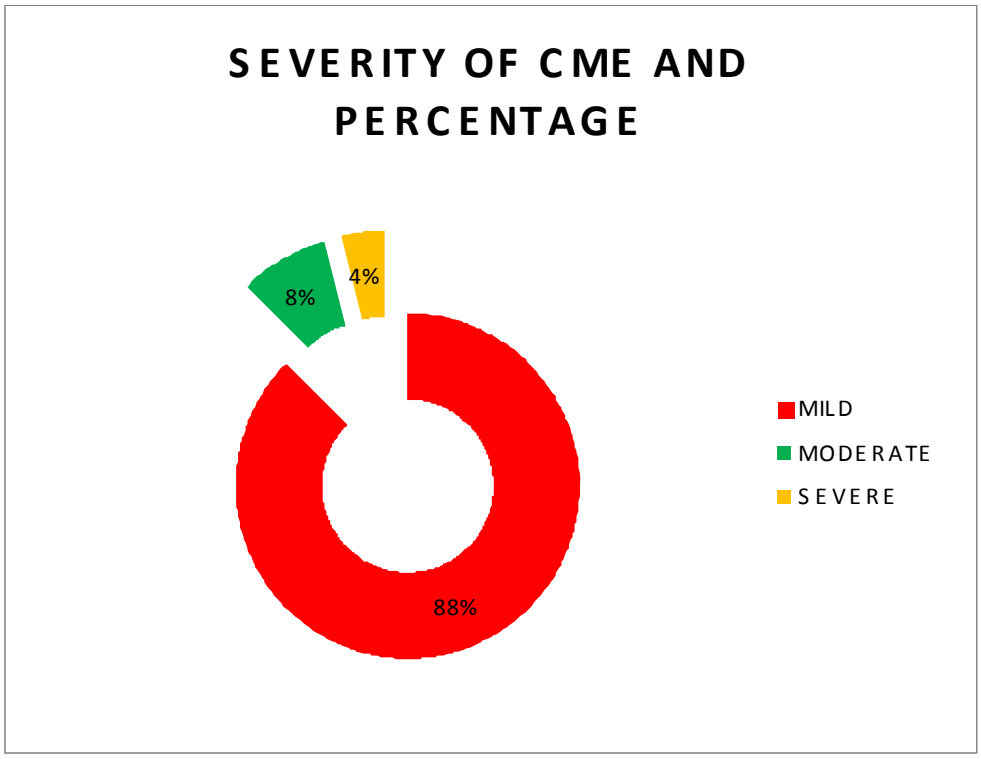
H. VISUAL LOSS WITH CLINICAL EVIDENCE OF CYSTOID MACULAR OEDEMA

SEVERITY OF CME	6/6-6/9	6/12-6/18	6/24-6/36	6/60&>	T
MILD	1	5	10	6	22
MODERATE	-	1	-	1	2
SEVERE	-	-	-	1	1

25 eyes had evidence of CME (34%) out of 74 eyes with pars planitis. 88% of patients had mild CME. Moderate CME was associated with gross reduction in visual acuity.

I. FUNDUS FLUORESCEIN ANGIOGRAPHY

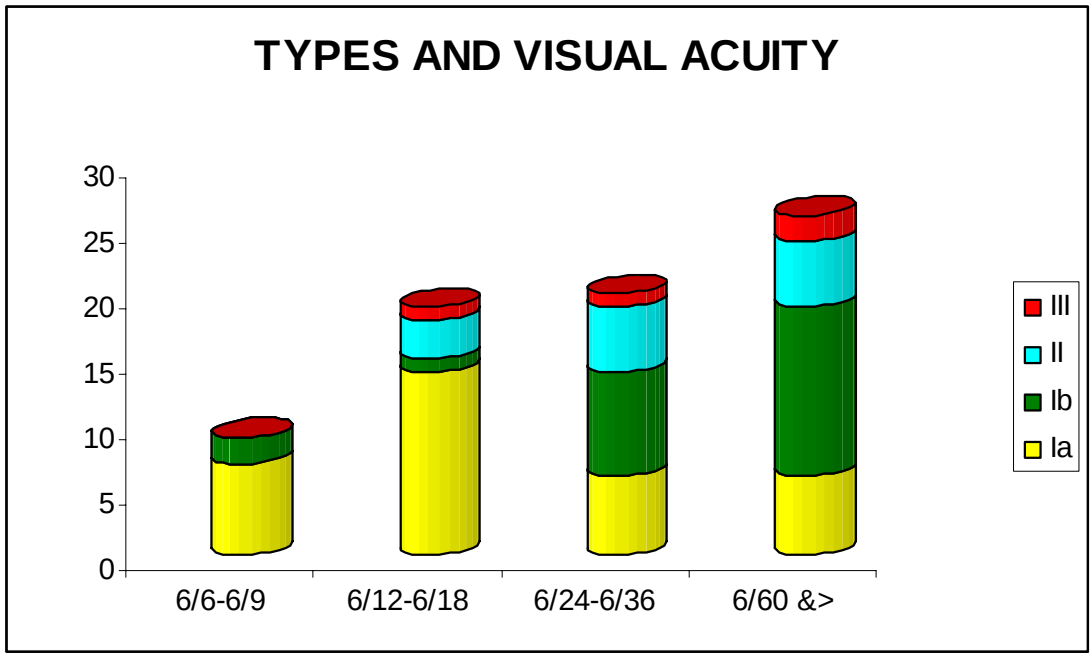
SEVERITY OF CME	NO. OF EYES
MILD	21
MODERATE	2
SEVERE	1



88% of eyes had mild form of CME.

J.TYPE OF PARS PLANITIS AND THEIR SIGNIFICANCE ON VISUAL ACUITY

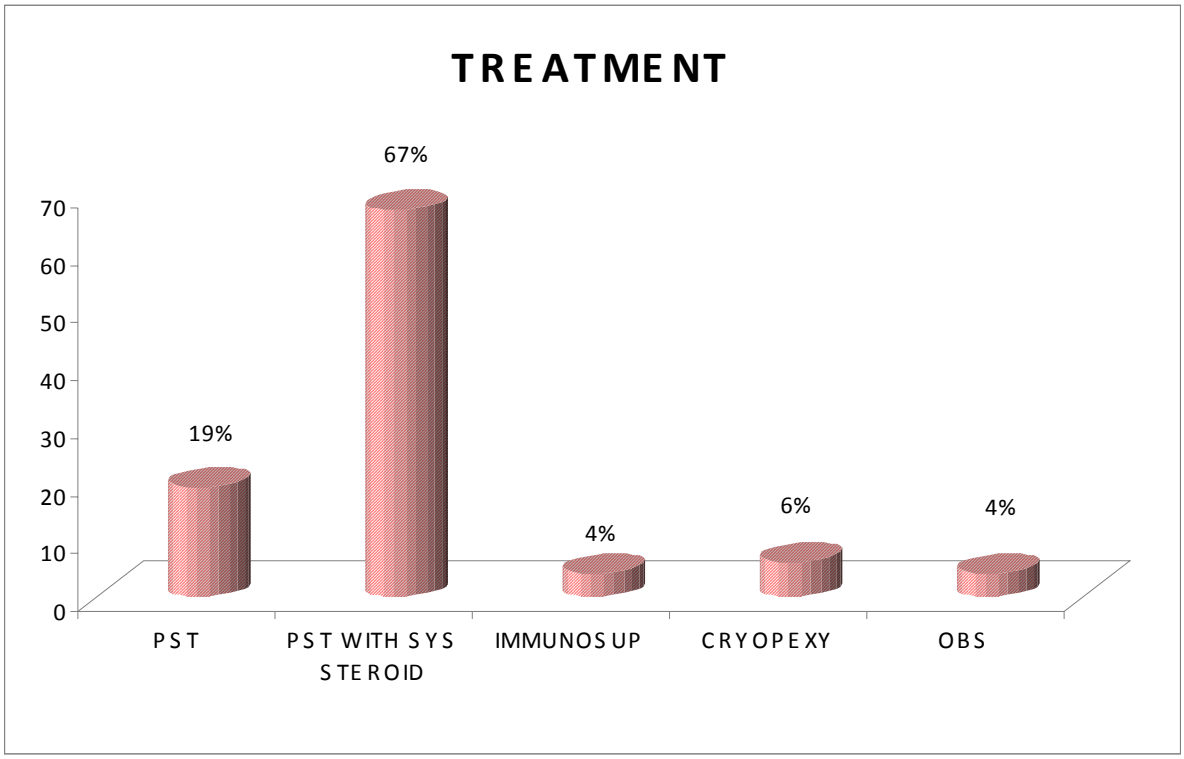
TYPE	6/6-6/9	6/12-6/18	6/24-6/36	6/60&>	T
Ia	7	14	6	6	33
Ib	2	1	8	13	24
II	-	3	5	5	13
III	-	1	1	2	4



Type Ib, type II and type III ie.,infitrative, exudative and vasculitic type of pars planitis had gross defective vision (less than 6/18). While in serous type 21% had normal visual acuity.

V.A. TREATMENT

Modality of Treatment	Stable	Improve 1 line	Improve 2 lines	Improve 3 or more lines	Deterioration	T
SUBTENONS STEROIDS	1	4	6	4	-	15
SYSTEMIC STEROID WITH POSTERIOR SUBTENONS	7	12	14	20	-	53
IMMUNOSUPPRESSIVE	-	1	1	1	-	3
WITH CRYOPEXY	-	1	3	1	-	5
OBSERVATION ONLY	-	1	2	-	-	3
TOTAL	8	19	24	26	-	79



Most of our patients (67%) were treated with systemic and subtenon steroids. 90% of patients had improvement of visual acuity on treatment

B. SUBJECTIVE IMPROVEMENT OF SYMPTOMS

	Ia	Ib	II	III	TOTAL
WORSENING	1	1	4	-	6
STATIC	3	5	2	1	11
IMPROVED	30	15	6	3	54

C. COMPLICATIONS

COMPLICATIONS	NO. OF PATIENTS
RAISED INTRAOCULAR PRESSURE	3
VITREOUS HAEMORRHAGE	1
POSTERIOR SUBCAPSULAR CATARACT	8

Posterior subcapsular cataract was the most common complication noted.Out of 8 patients, 3 patients underwent phacoemulsification with PCIOL implantation after observation of quiescence of eye for 3 months during the follow up period.

DISCUSSION

In our study of 50 patients (74 eyes) with pars planitis it was observed that 30% of patients were in the age group of 15-25 years and 32% of patients in fourth and fifth decade. This shows the bimodal age distribution with peaks in second and fifth decade.

Fourty four percentages of patients were females. There was no sex predilection as reported in other studies as well. (Nussenblatt uveitis, fundamentals and clinical practice).

Fifty two percent of patients had unilateral signs of pars planitis. This did not correlate with studies done by Kimura which showed 71% being bilateral and 75% bilaterality reported by Stewart Duke Elder.

Aaberg had noticed minimal anterior segment inflammation in many of his patients, 34% of our patients had minimal anterior segment signs. But none of our patients had either peripheral anterior synechiae, or posterior synechiae as reported by Brockhurst in 24 and 18 percent ofhis patients respectively. 3 patients had increased intraocular pressure and there were no patients who had glaucoma. But Kimura reported that 8% of his 182 eyes developed glaucoma of which 67% of them were related to corticosteroid use and only 2% had disease induced glaucoma.

Boke`s classification modified by Slovak was used to classify pars planitis because it was a purely clinical method of classification. There has been no reported study which has correlated the clinical presentation based on classification by Boke. Smith in 1977 had reported 43% with mild, 46% with moderate severity of pars planitis while he had 11% who had severe type of pars planitis. Our study showed that 77% had diffuse inflammatory type of which 44.6% were of the serous type and 32.4% had infiltration type of pars planitis. The most severe vasculitic type was present in only 5.4% of our patients. Gross defective vision was present in 77% of patients with type II (exudative type) and 75% of patients with type III (vasoproliferative) pars planitis.

Brockhurst had noted that snow banking and snow ball opacities wasmostly located inferiorly. It was true in our study where 86% and 77% ofpatients had inferior location of snow banking and snow ball opacities.

There was no direct correlation between the presence of vitreous celland vitreous opacities with visual loss in our patients, even though only 17% of patients with vitreous cells of 3+ and 25% of patients with vitreous opacities of 3+ had normal visual acuity. 67% of patients with moderate CME were associated with gross defective vision (Aaberg, TM., The Enigma of Pars planitis Am.J.Ophthal 103: 828-830, 1987).

Schlaegel had found that 67% of eyes treated with corticosteroids maintain visual acuity of more than 6/12. 67% of our patients responded to subtenons steroids while 64% of the patients responded to addition of systemic steroids along with subtenon`s injection of steroids. In our study, out of 50 patients, 5 patients underwent Cryotherapy. Visual acuity improvement was seen in 80% of the patients who were subjected to Cryotherapy. This correlates well with the study by Devenyi in which he reported elimination of vitritis with improvement in visual acuity in 78% of cases.

Cataract extraction by phacoemulsification with posterior chamber intraocular lens implantation was done in 3 of our patients after 3 months of quiescence of the eye. Two out of three patients had more than 6/12 visual acuity post operatively. Michelson has reported 51% of eyes with more than 6/12 vision after cataract surgery. Sudha.k.ganesh in her study analyzed that phacoemulsification with IOL implantation in eyes with pars planitis was safe and led to good visual outcomes in most cases. The factors in surgical success were control of inflammation, meticulous surgery, in-the-bag IOL implantation, and vigilant postoperative care.

CONCLUSION

1. The demographic pattern of our pars planitis compares well with the other studies with the bimodal age distribution with peaks in second and fifth decade and with no sex predilection.

2. Though the commonest type of pars planitis is serous or infiltrative type (Type Ia&Ib), Patients who belonged to BOKE`s classification Type II and Type III ie., exudative and vasculitic type had a more severe form of pars planitis and requires early aggressive treatment.

3. Fundus fluorescein angiography was required to ascertain whether the patient had CME. It not only picks up early CME, but also helps inidentifying patients who will develop more aggressive disease. Optical Coherence Tomography has become a better tool to diagnose and follow up patients with CME.

4. Severity of vitreous cells, extent of snow banking and CME are prognostic indicators. Patients with increased amount of vitreous cells, with more than 3 quadrant of snow banking and with moderate CME warrant intensive treatment.

5. There is a high incidence of snow banking in the inferior half of retina, gravity being a determinant as comparable with other studies.

6. Treatment with a combination of systemic with subtenon`s administration of steroids gives good visual outcome for patients with pars planitis. Patients refractory to this line of management responds well to immunomodulators and Cryotherapy.

7. Cataract extraction on a quiescent eye under cover of steroids helps in visual rehabilitation of patients who develop significant loss of visual acuity due to the same.

PART III

PROFORMA

A MULTIFACTORIAL ANALYSIS OF 50 CASES OF PARS PLANITIS

NAME: ADDRESS:

AGE:

SEX:

SYMPTOMS

	UNILATERAL		BILATERAL	
	RE		LE	
I. Laterality				
Duration				
II. Defective vision				
Hazy vision				
Signs of anterior uveitis				
Pain				
Redness				
Watering				
Photophobia				
III. History suggestive of				
Rheumatological disorder				
Collagen vascular disease				

Vasculitic disorder

Infective: Tuberculosis

Others

Demyelinating diseases

Focal sepsis: ENT, Dental, Gynac (in females)

Pets

Diet

IV. Previous Episode

Age at onset

No. of episodes

Response to treatment

V. History of

Diabetes

Hypertension

Smoking

Alcohol intake

Exposure to pulmonary tuberculosis patient

VI. Family history of similar illness

SIGNS

1. Laterality	Unilateral	Bilateral
	RE	LE
	At onset/On review	At onset/On review

2. Anterior segment spillover

a. Circumciliary congestion

b. Keratic precipitates (1+, 2+, 3+, 4+)

c. Flare (0, ±, 1+, 2+, 3+, 4+)

d. Cells (0, ±, 1+, 2+, 3+, 4+)

3. Intermediate Uveitis

a. Vitreous cells (0, ±, 1+, 2+, 3+, 4+)

b. Vitreous opacity (0, ±, 1+, 2+, 3+, 4+)

c. Snow ball opacity (UN LN LT UT)

d. snow banking (UN LN LT UT)

e. Pars plana vascularization (UN LN LT UT)

f. Retinal periphlebitis (UN LN LT UT)

g. Vitreous haemorrhage

h. Retinal detachment

i. Cystoid macular edema No

Yes- mild/moderate/severe

4. Presence of complications- at onset/on review

a. Peripheral anterior synechiae

b. Posterior synechiae

c. Glaucoma

d. Complicated cataracts

e. Macular degeneration

5. Presence of optic neuritis

a. Visual acuity

b. Visual fields

c. Relative afferent pupillary defect

d. Colour vision

e. ONH swelling

f. Amslers grid

DIAGNOSTIC TYPE

1. Diffuse inflammatory
   a. Serous
   b. Infiltrative
2. Exudative
3. Vasoproliferative

INVESTIGATIONS

1. Total blood count
2. ESR

3. Mantoux

4. VDRL

5. Rheumatoid factor

6. TORCH screening

7. Antinuclear antibody

8. Chest X-ray

FUNDUS FLUORESCEIN ANGIOGRAPHY

Normal

Abnormal:

Disc

Presence of vasculitis

Cystoid macular edema- mild/moderate/severe

Others:

TREATMENT

1. Posterior subtenon`s steroids: +/-

2. Systemic steroids: +/-

3. Immunosuppressives: +/-

4. Cryotherapy:

    a. Quadrants covered (UN LN LT UT)

    b. Visual acuity at 1 week, 2 weeks, 1 month

OUTCOME

a. Visual outcome

b. Amsler`s grid

c. Subjective response- static/improve/worsen

ABBREVIATIONS

M	MALE
F	FEMALE
RE	RIGHT EYE
LE	LEFT EYE
UL	UNILATERAL
BL	BILATERAL
R	REDNESS
Ph	PHOTOPHOBIA
W	WATERING
P	PAIN
CT	CARIES TOOTH
CR	CHORIORETINITIS

HM	HIGH MYOPIA
Pt	PTHYSIS
PC	PRIMARY COMPLEX
KP	KERATIC PRECIPITATE
UT	UPPER TEMPORAL
UN	UPPER NASAL
LT	LOWER TEMPORAL
LN	LOWER NASAL
CME	CYSTOID MACULAR EDEMA
PVD	POSTERIOR VITREOUS DETACHMENT
ERM	EPIRETINAL MEMBRANE
DE	DISC EDEMA
HMD	HYPEREMIA OF DISC
Mi	MILD

Mo	MODERATE
Se	SEVERE
FFA	FUNDUS FLUORESCEIN ANGIOGRAPHY
C V/A-Ad	CORRECTED VISUAL ACUITY AT THE TIME OF ADMISSION
PSCC	POSTERIOR SUBCAPSULAR CATARACT
IOP	INTRAOCULAR PRESSURE
VitHmg	VITREOUS HAEMORRHAGE
HM	HIGH MYOPIA
O	OBSERVATION
S/T S	SUBTENONS STEROIDS
SS	SYSTEMIC STEROIDS
IS	IMMUNOSUPPRESSIVES
CRYO	CRYOTHERAPY

V/A EF	VISUAL ACUITY AT THE END OF FOLLOW UP
N	NORMAL
MH	MILD HAZINESS
St	STABLE
MoH	MODERATE HAZINESS
HV	HAZY VISION

LIST OF SURGERIES PERFORMED

1	SUNDARI	35	F	652301	RE-CHALAZION	RE-INCISION AND CURETTAGE
2	SELVI	26	F	281766	RE-PTERYGIUM	RE-PTERYGIUM EXCISION WITH AUTOGRAFT
3	RAJAN	45	M	622091	LE-PANOPHTHALMITIS	LE-EVISCERATION
4	VELU	68	M	521907	RE-CHRONIC DACRYOCYSTITIS	RE-DACRYOCYSTECTOMY
5	PONNAMMAL	42	F	646111	LE-CHRONIC DACRYOCYSTITIS	LE-DACRYOCYSTORHINOSTOMY
6	MOORTHY	34	M	523012	LE-TRAUMATIC ENDOPHTHALMITIS	LE-INTRAVITREAL CEFTAZIDIME AND VANCOMYCIN
7	RAMESH	20	M	644432	RE-TRICHIASIS	RE-EPILATION
8	MUNIAMMA	63	F	679438	RE-ABSOLUTE GLAUCOMA	RE-CYCLOCRYOTHERAPY
9	CHELLAM	57	M	612406	RE-UPPER LID LACERATION	RE-SUTURING OF UPPER LID
10	RAMASAMY	70	M	676450	RE-PROPTOSIS WITH EXPOSURE	RE-CENTRAL TARSORRHAPHY
11	BALAMMA	43	F	633218	LE-CHRONIC DACRYOCYSTITIS	LE-DACRYOCYSTORHINOSTOMY
12	GOVINDARAJ	55	M	679032	LE-POSTOPERATIVE ENDOPHTHALMITIS	LE-INTRAVITREAL CEFTAZIDIME AND VANCOMYCIN
13	VELANKANNI	62	F	632456	RE-MATURE CATARACT	RE-ECCE WITH PCIOL
14	RAJESHWARI	55	F	680933	LE-IMMATURE CATARACT	LE-SICS WITH PCIOL
15	RAMANI	58	M	600092	RE-MATURE CATARACT	RE-ECCE WITH PCIOL
16	BASHA	23	M	657490	LE-PTERYGIUM	LE-PTERYGIUM EXCISION WITH AMNIOTIC MEMBRANE GRAFT
17	KANMANI	56	F	612900	RE-MATURE CATARACT	RE-SICS WITH PCIOL
18	NITHYANANDAM	13	M	654739	LE-CHALAZION	LE-INCISION AND CURETTAGE
19	RAMACHANDRAN	68	M	699435	RE-MATURE CATARACT	RE-ECCE WITH PCIOL
20	SELVAM	72	M	621213	LE-IMMATURE CATARACT	LE-SICS WITH PCIOL

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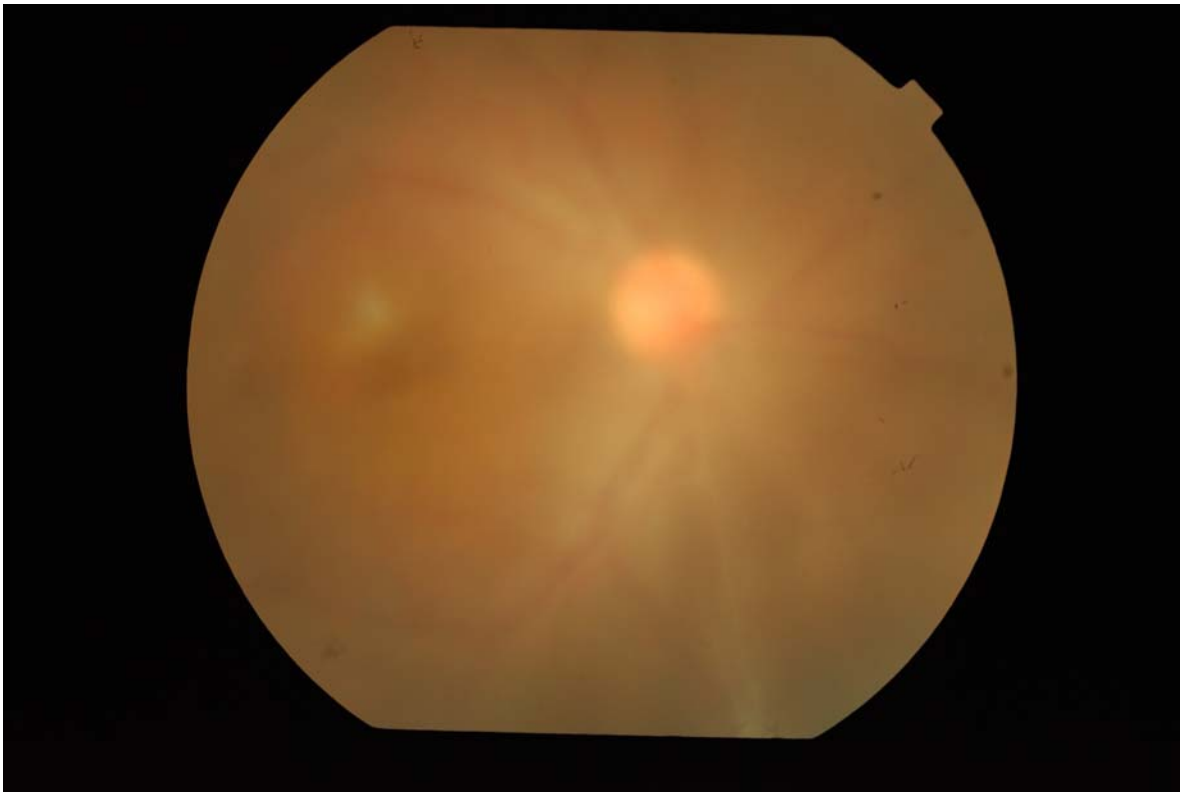
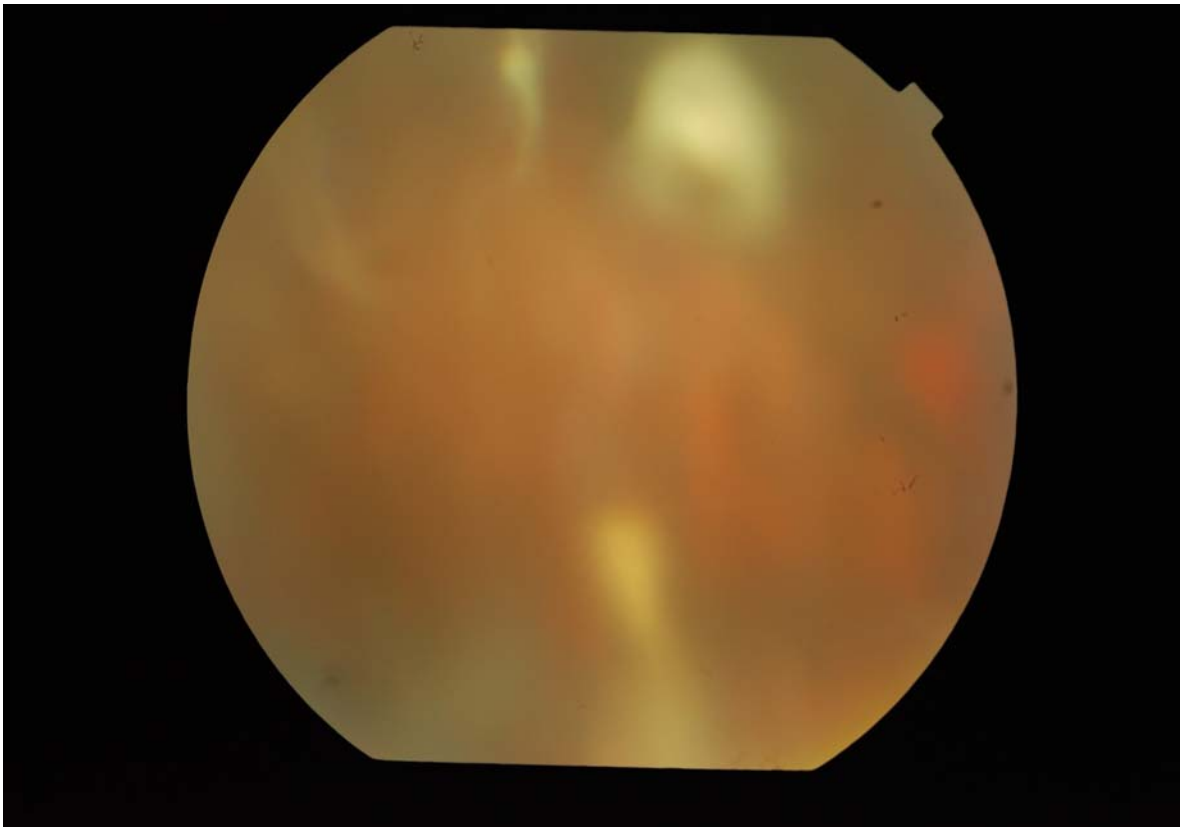
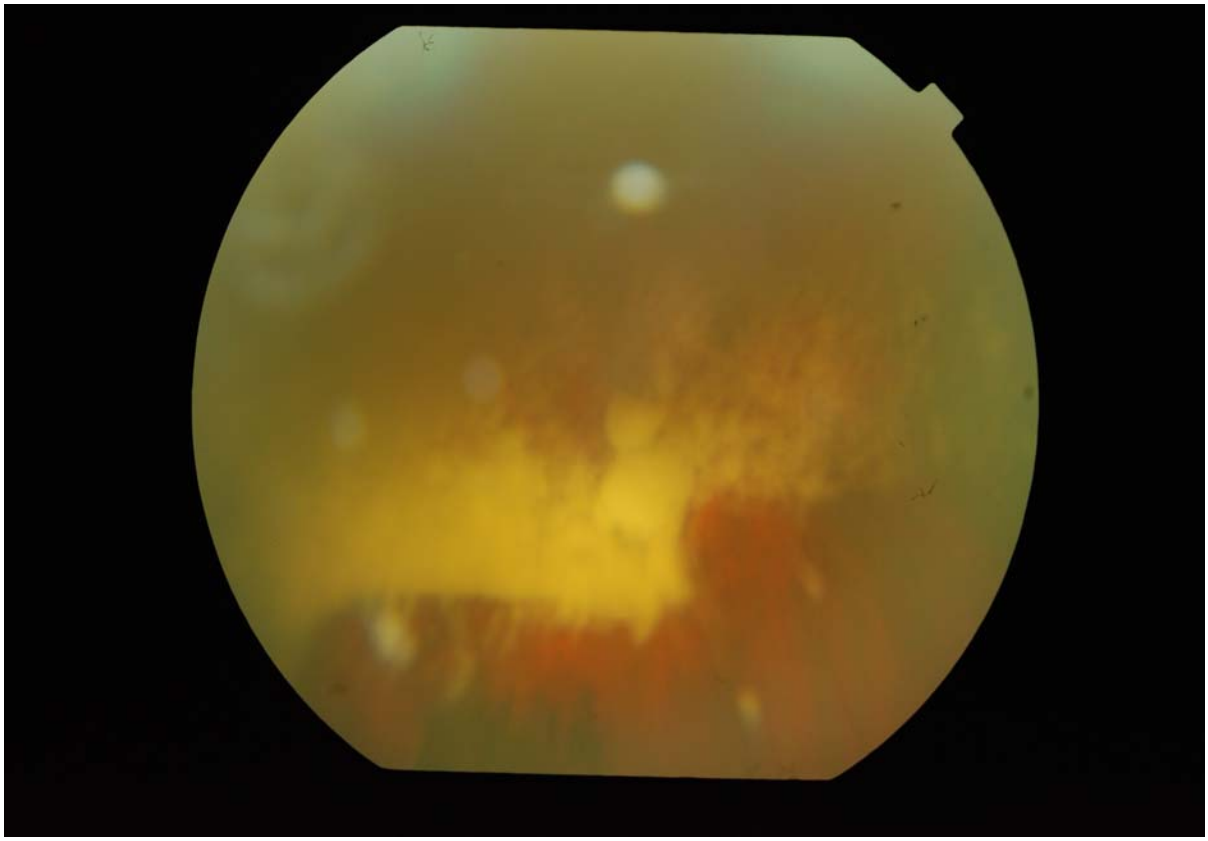


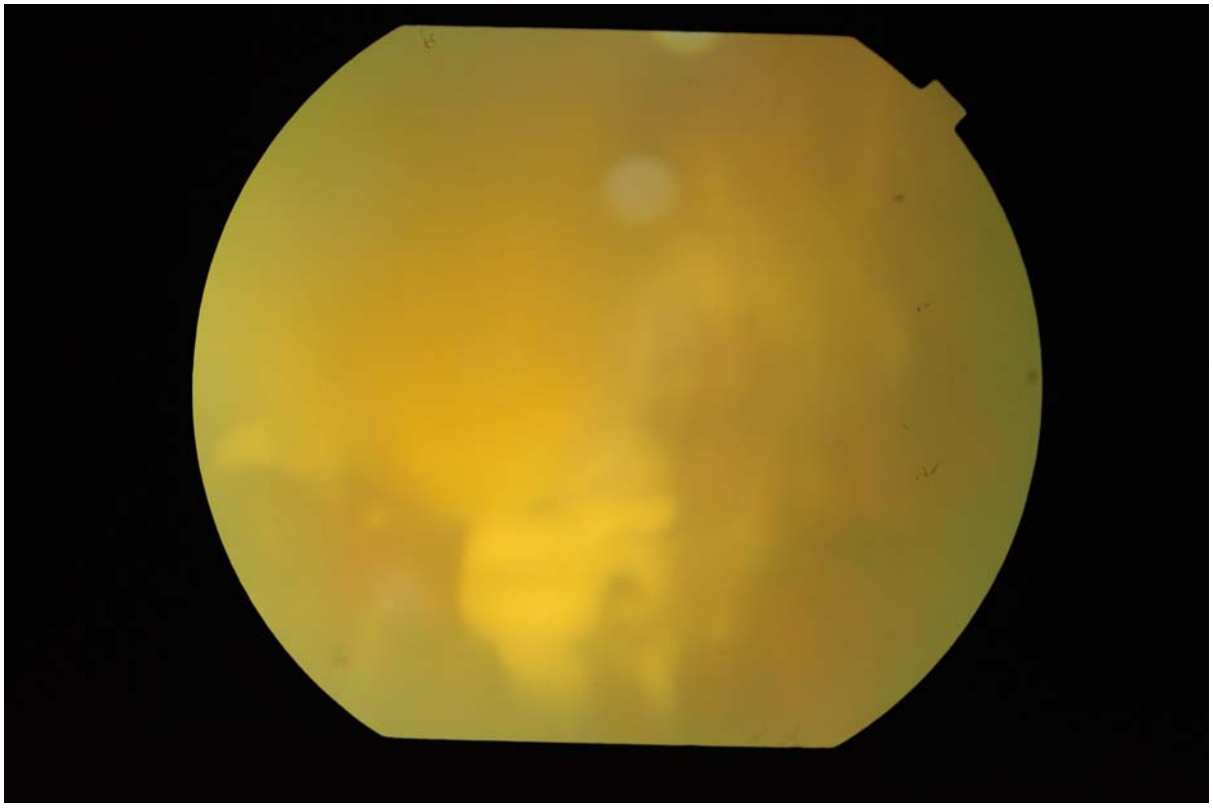
FUNDUS PICTURE OF A PATIENT SHOWING VITRITIS

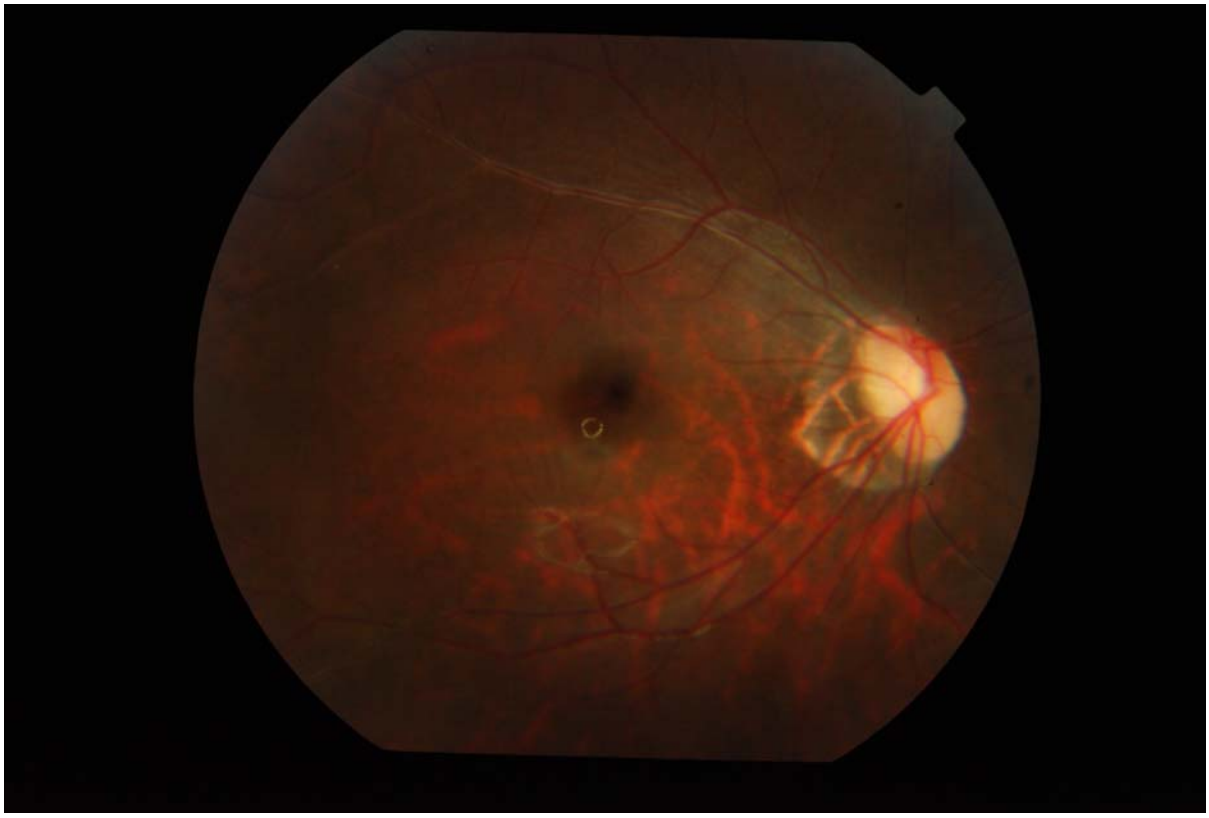


FUNDUS PICTURE SHOWS SNOWBALL OPACITIES

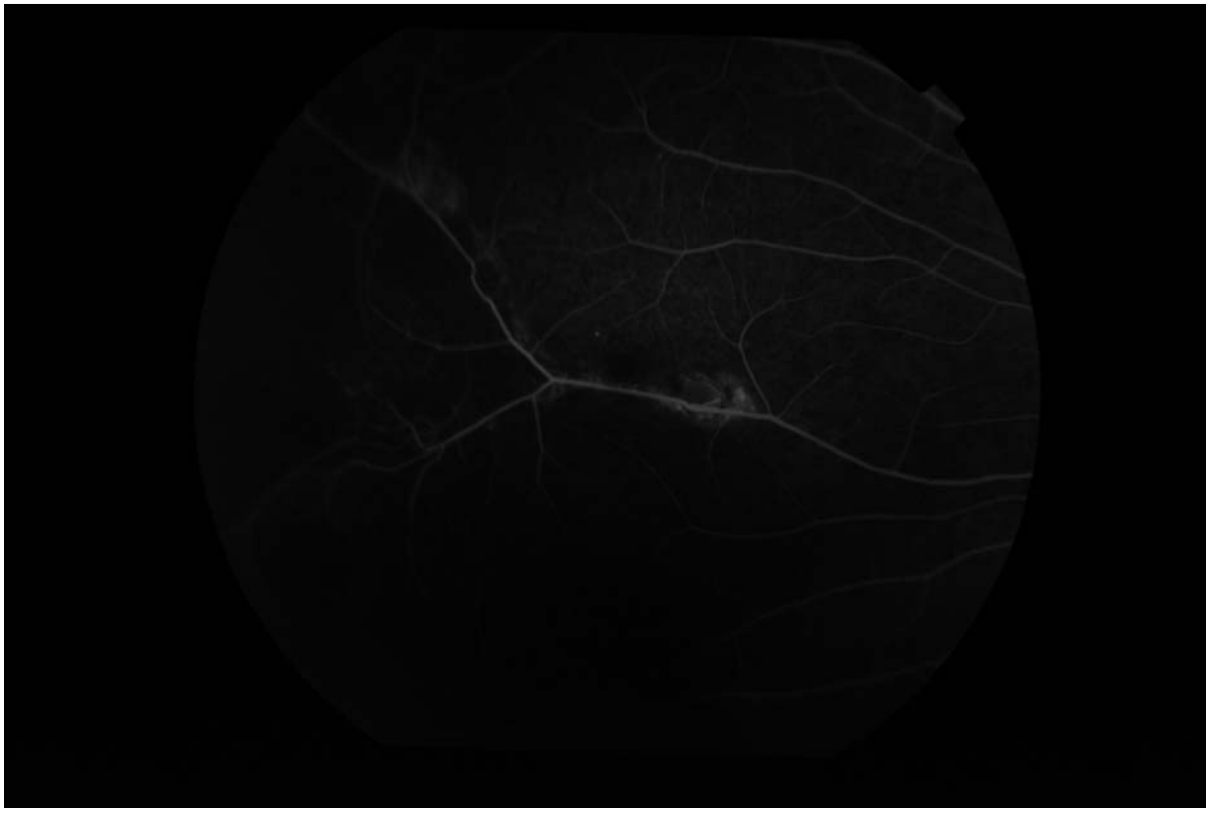


FUNDUS PICTURE OF A PATIENT WITH SNOWBANKING

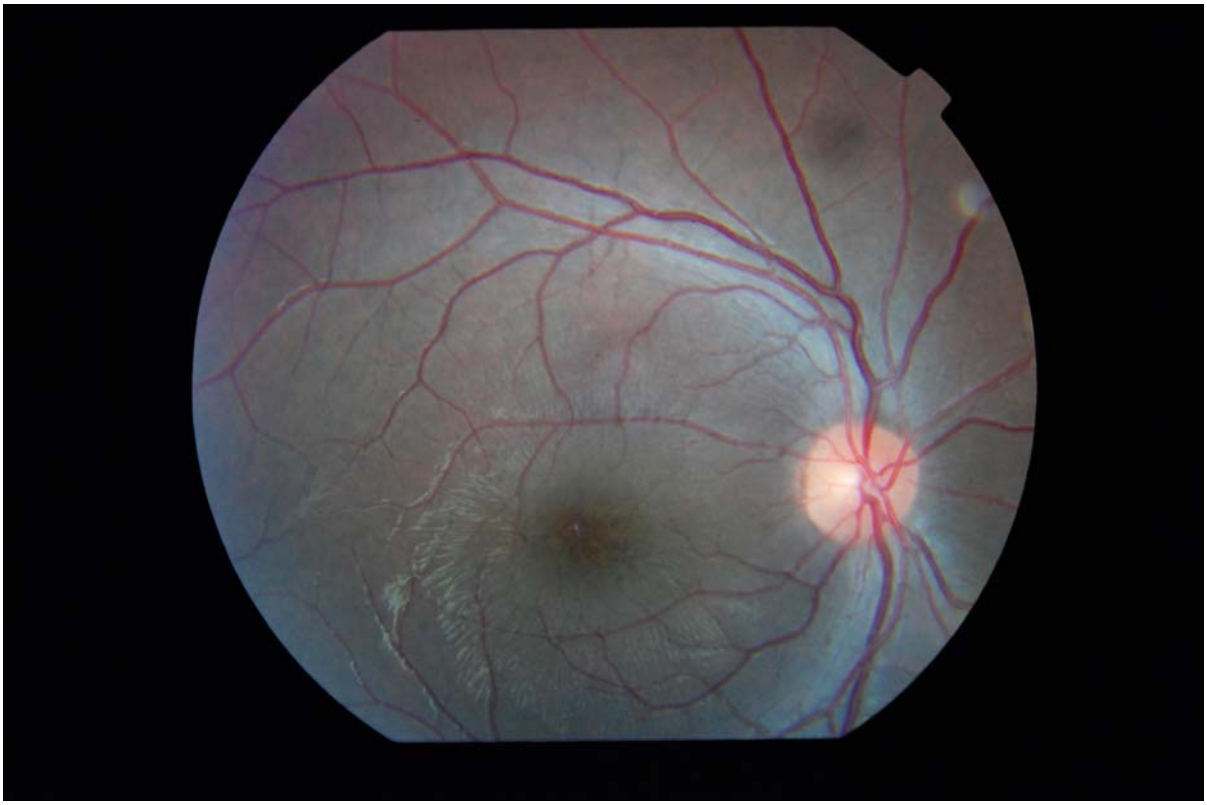




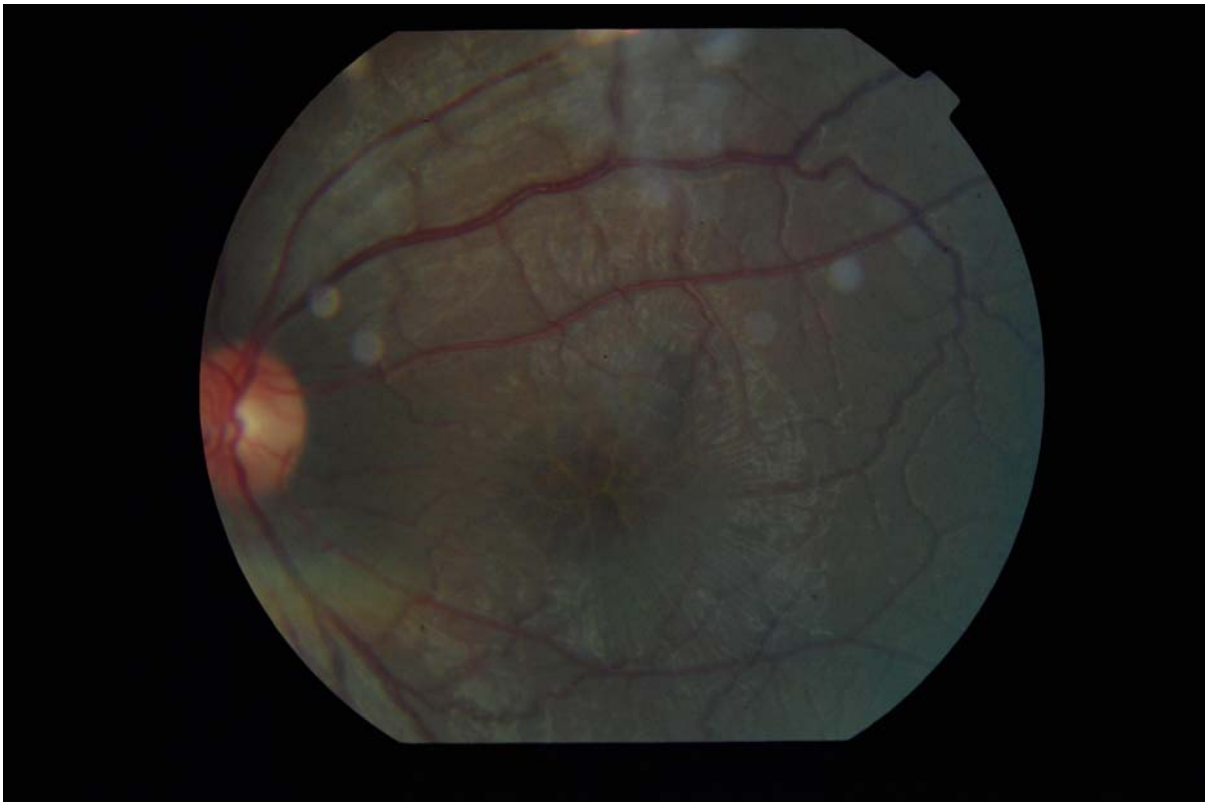
VASCULAR SHEATHING SEEN IN A CASE OF PARSPLANITIS WITH VASCULITIS

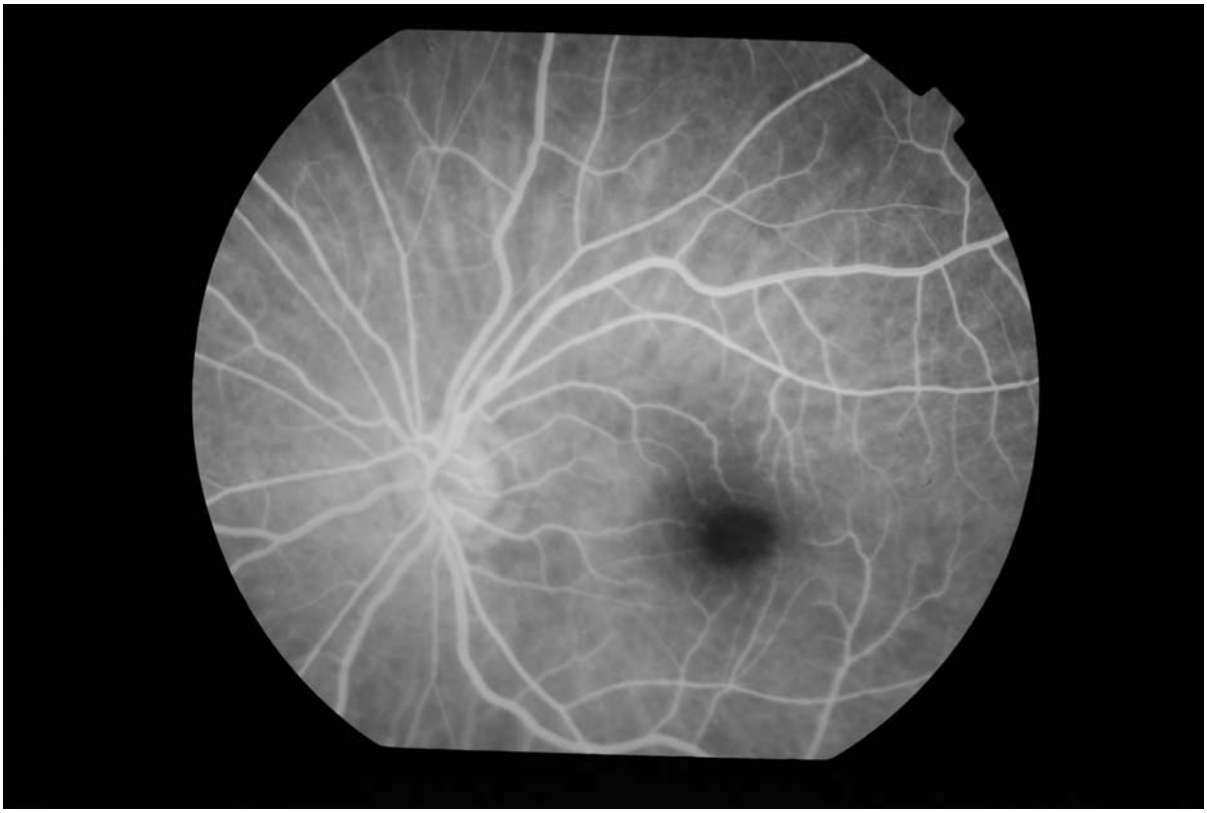


FLUORESCEIN ANGIOGRAM SHOWING LATE STAINING OF VESSEL IN A PATIENT OF PARS PLANITIS WITH VASCULITIS

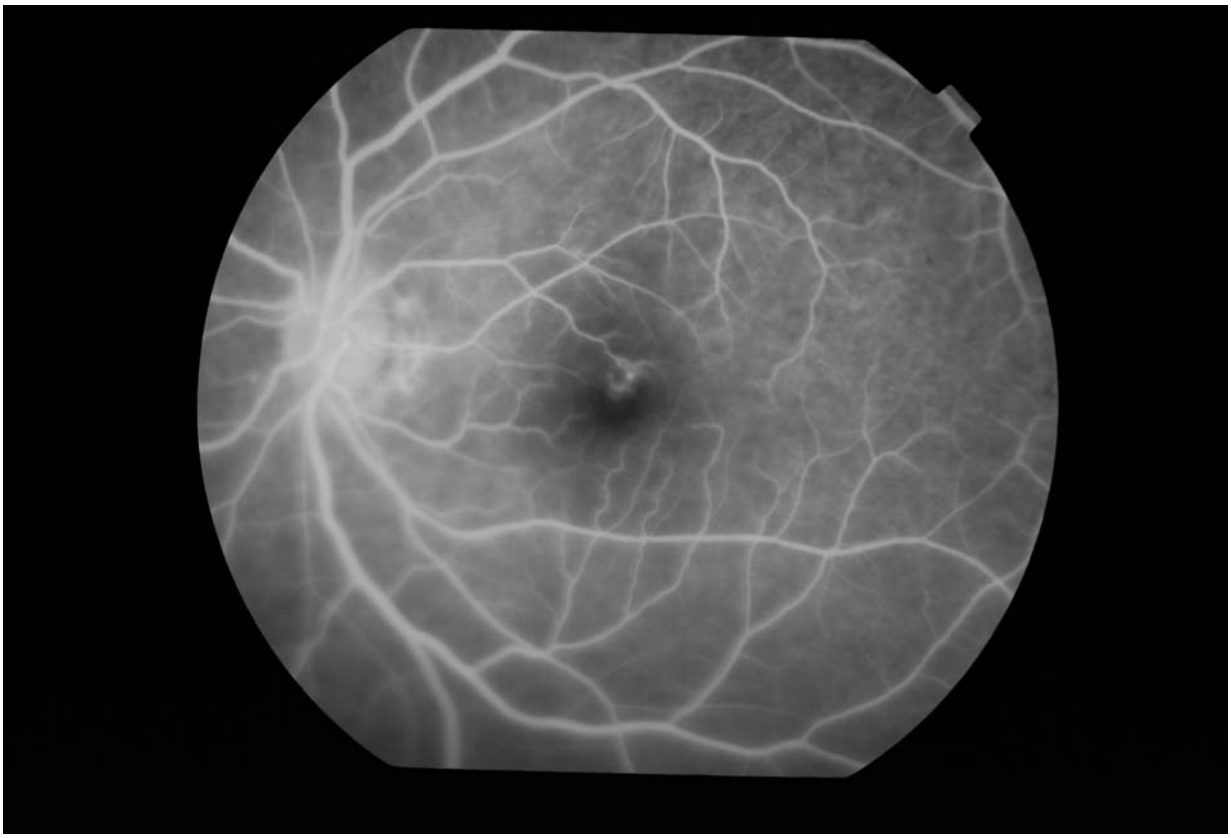


FUNDUS PICTURE OF A PATIENT WITH BILATERAL CME

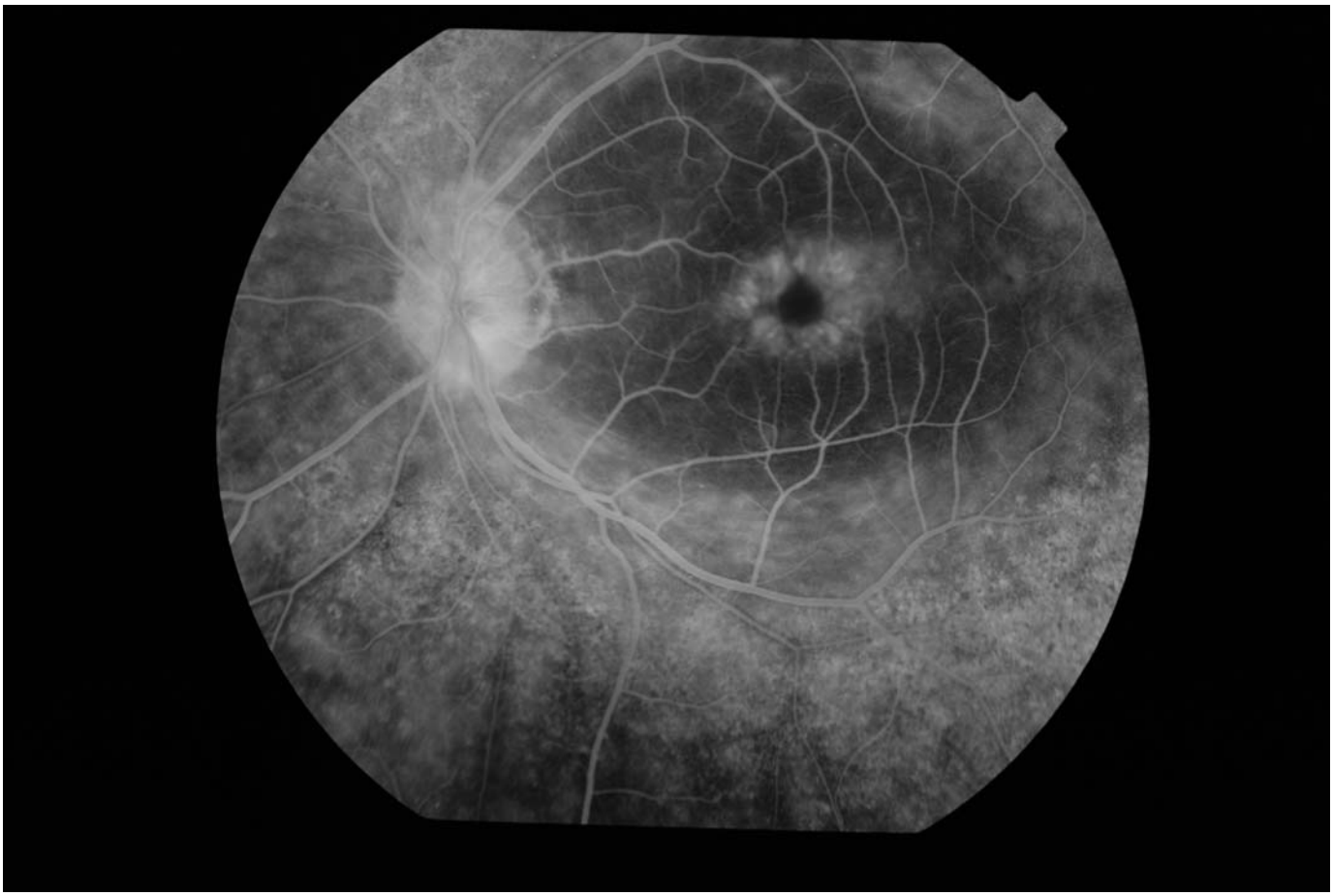




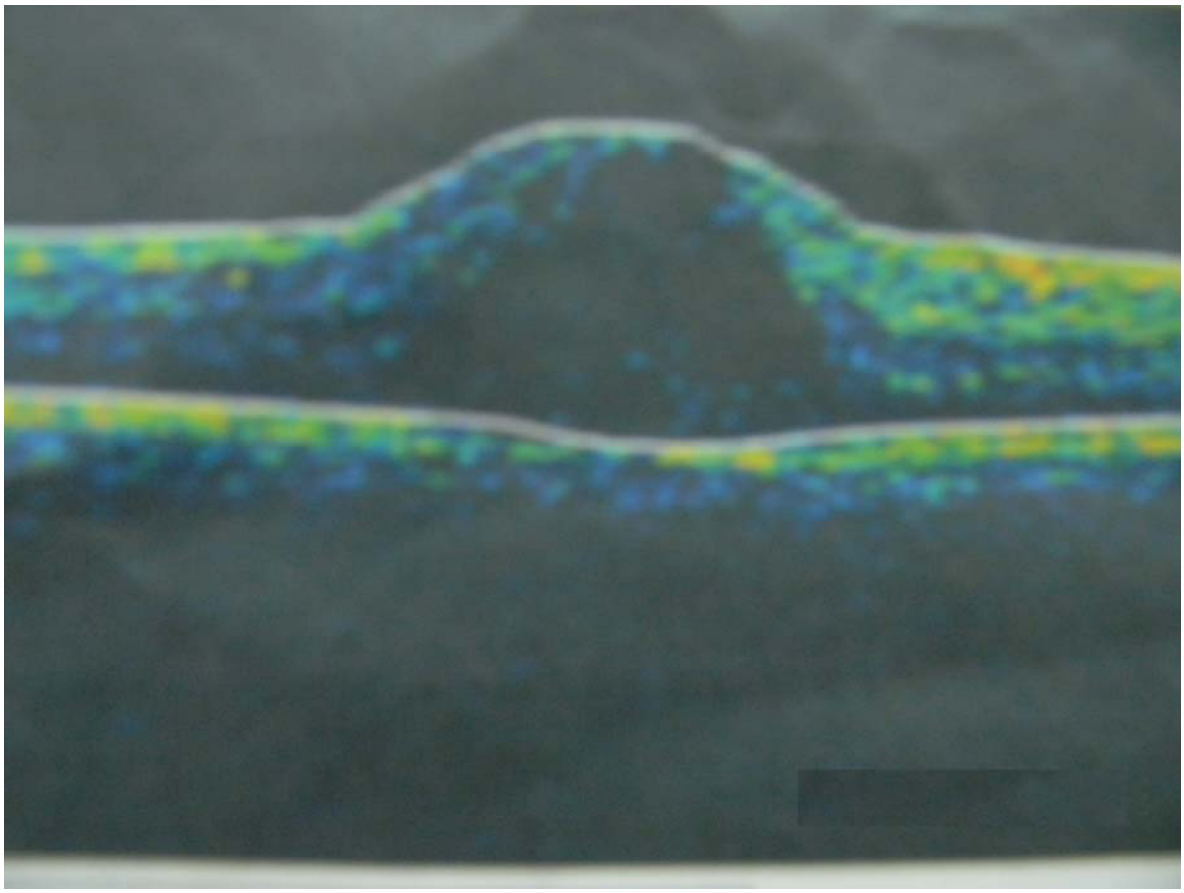
FLUORESCEIN ANGIOGRAM OF A PATIENT WITH MILD CME – DECREASE IN FOVEAL AVASCULAR ZONE



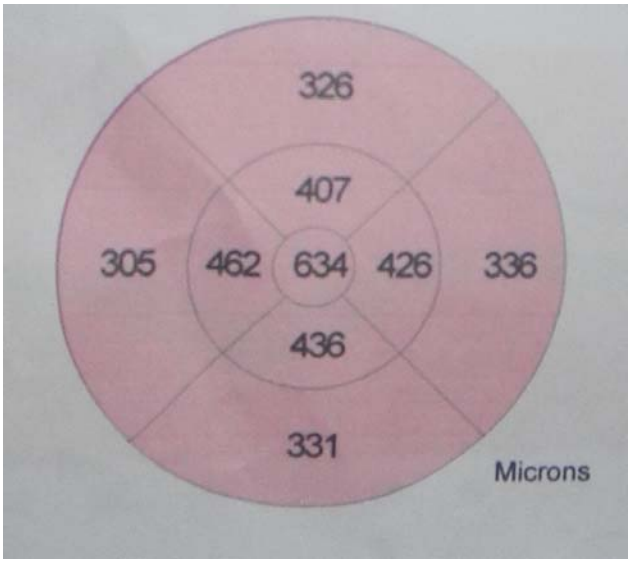
FLUORESCEIN ANGIOGRAM OF A PATIENT WITH MODERATE CME



FLUORESCEIN ANGIOGRAM OF A PATIENT WITH SEVERE CME FLOWER PETAL PATTERN



OCT SHOWS CYSTOID MACULAR EDEMA WITH CENTRAL MACULAR THICKNESS OF 634µ



MASTER CHART

S.NO.	NAME	AGE	SEX	SYMPTOMS							ANTERIOR UVEITIS	ASSO. DISORDERS	ANTERIOR SEGMENT					ANTERIOR SEGM	
				UL/BL	FLOATERS		VISUAL LOSS		HAZY VISION				UL/BL	RE				LE	
					RE	LE	RE	LE	RE	LE				KP s	CELLS	FLARE	S.NO.	KPs	CELLS
1	MANIKANDAN	17	M	UL	-	-	-	-	-	+	R,P,Ph(LE)	-	UL	-	-	-	1	-	-
2	SELVARANI	28	F	UL	+	-	-	-	+	+	Ph (RE)	-	BL	-	-	-	2	-	-
3	MALLIKA	24	F	BL	-	-	+	+	-	-	CCC (BE)	-	BL	+	-	-	3	+	2+
4	PRABAKARAN	13	M	UL	-	-	-	-	-	+	Ph(LE)	-	UL	-	-	-	4	-	-
5	CHANDRAN	47	M	BL	+	+	+	+	-	-	R (BE)	CT	BL	-	-	-	5	-	-
6	DHAMODARAN	28	M	UL	-	-	-	+	-	-	-	-	UL	-	-	-	6	-	1+
7	SELVARAJ	14	M	UL	-	-	+	-	-	-	-	-	UL	-	-	-	7	-	-
8	PARTHIBAN	35	M	UL	-	+	-	+	-	+	Ph(LE)	-	UL	-	-	-	8	-	-
9	SIVARANJANI	23	F	UL	-	-	+	-	-	-	R (RE)	-	UL	-	-	-	9	-	-
10	MOHAN	45	M	BL	-	-	-	-	+	+	-	-	BL	-	1+	1+	10	-	-
11	GEETHA	30	F	UL	-	-	+	-	-	-	-	CR (RE)	UL	-	-	-	11	-	-
12	SELVI	20	F	UL	-	-	-	+	-	-	-	-	UL	-	-	-	12	-	-
13	JEEVA	50	F	BL	+	+	+	+	-	-	Ph(BE)	CR (RE)	BL	-	1+	2+	13	-	1+
14	SANGEETHA	11	F	UL	-	-	-	-	+	-	R,Ph(RE)	Asthmatic	UL	-	-	-	14	-	-
15	SARASWATHY	43	F	BL	+	+	+	+	-	-	P,W(BE)	-	BL	+	1+	1+	15	-	-
16	KEERTHANA	8	F	UL	-	-	-	+	-	+	Ph(LE)	CR(LE)	UL	-	-	-	16	-	-
17	SRIKUMARAN	28	M	UL	-	-	-	-	-	+	R,P,Ph(LE)	-	UL	-	-	-	17	-	-
18	KARTHICK	30	M	UL	+	-	+	-	+	-	-	-	UL	-	-	-	18	-	-
19	RAVI	37	M	BL	+	-	+	+	-	-	-	-	BL	-	-	-	19	-	-
20	MEENAKSHI	50	F	BL	+	+	-	-	+	+	-	CR (RE)	BL	-	-	-	20	-	-
21	MANI	40	M	UL	-	-	+	-	+	-	-	-	UL	-	-	-	21	-	-
22	PARAMASIVAN	28	M	BL	+	+	+	+	-	-	R(BE)	-	BL	-	-	-	22	+	-
23	SAMPATH	45	M	BL	+	+	-	-	+	+	Ph(BE)	HM(BE)	BL	-	-	-	23	+	1+
24	SARAVANAMOORTHY	36	M	BL	+	+	-	-	+	+	-	-	BL	+	1+	1+	24	+	1+
25	RAVUTHAR	22	M	UL	-	-	-	-	-	+	P,R(LE)	-	UL	-	-	-	25	+	-
26	KUMAR	30	M	UL	+	-	-	-	+	-	Ph(RE)	-	UL	-	1+	1+	26	-	-
27	SHANTHI	40	F	BL	-	-	-	-	+	+	-	CR(RE)	BL	-	-	-	27	-	-
28	JAYANTHI	24	F	UL	-	-	-	-	-	+	P,W(LE)	CT	UL	-	-	-	28	-	-
29	JAYA	40	F	BL	+	+	+	-	+	+	Ph(BE)	HM(BE)	BL	-	1+	1+	29	-	1+
30	MANIMARAN	30	M	UL	+	-	-	-	+	-	-	-	UL	-	-	-	30	-	-
31	VINOTH	22	M	UL	-	-	+	-	+	-	R(RE)	CT	UL	+	2+	2+	31	-	-
32	SUJATHA	30	F	BL	-	-	-	-	+	+	R,Ph(BE)	-	BL	+	1+	1+	32	+	1+
33	ARUNACHALAM	42	M	BL	+	+	+	+	-	-	R(BE)	-	BL	-	-	-	33	-	2+
34	SELVAKUMAR	21	M	UL	-	-	+	-	+	-	R(RE)	-	UL	+	1+	1+	34	-	-
35	ASHOKAN	39	M	BL	-	-	-	-	+	+	R,Ph(BE)	-	BL	+	1+	1+	35	+	-
36	PERUMAL	22	M	UL	-	+	-	-	-	-	R(LE)	Pt(RE)	UL	-	-	-	36	-	-
37	AMSABAI	54	F	UL	-	-	-	-	+	-	R,Ph(RE)	-	UL	-	-	-	37	-	-
38	SULEKA	38	F	UL	-	+	-	-	-	+	Ph(LE)	-	UL	-	-	-	38	-	-
39	UDHAYAKUMAR	20	M	UL	-	-	+	-	-	-	R (RE)	-	UL	-	-	-	39	-	-
40	MADHANKUMAR	28	M	UL	-	-	-	-	-	+	R,P,Ph(LE)	-	UL	-	-	-	40	-	-
41	RAJESH	26	M	UL	+	-	+	-	+	-	-	-	UL	-	-	-	41	-	-
42	NATARAJAN	22	M	BL	-	-	+	+	-	-	Ph,R(LE)	-	BL	-	-	-	42	+	1+
43	NAGALAKSHMI	16	F	BL	-	-	+	+	-	-	-	PC	BL	-	-	-	43	-	-
44	ETHIRAJ	30	M	UL	-	-	+	-	+	-	-	CR(RE)	UL	-	-	-	44	-	-
45	LALITHA	23	F	BL	+	+	-	-	+	+	-	-	BL	-	-	+	45	-	-
46	RANI	26	F	UL	-	+	-	+	-	+	-	-	BL	-	-	-	46	-	1+
47	MANOJ	23	M	UL	-	-	+	-	+	-	-	-	BL	-	-	-	47	-	-
48	KAMALA	36	F	BL	-	-	-	-	+	+	-	-	BL	-	-	-	48	-	-
49	VIJAYAKUMARI	20	F	BL	+	+	-	-	-	-	-	CT	BL	-	-	-	49	-	-
50	DEVAKI	33	F	UL	-	+	-	+	-	-	R(LE)	-	BL	-	-	-	50	-	-

IENT	VITREOUS CELLS		VITREOUS OPACITY		SNOWBALL OPACITY								SNOW BANKING					SNOW BANKING			
					RE				LE				RE					LE			
FLARE	RE	LE	RE	LE	UT	UN	LN	LT	UT	UN	LN	LT	UT	UN	LN	LT	S.NO.	UT	UN	LN	LT
-	-	2+	-	2+	-	-	-	-	-	-	+	+	-	-	-	-	1	-	-	-	-
-	3+	2+	2+	2+	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-
1+	2+	2+	1+	1+	-	-	-	-	-	-	-	-	-	-	+	+	3	-	-	+	+
-	-	1+	-	1+	-	-	-	-	-	-	+	+	-	-	-	-	4	-	-	-	-
1+	1+	1+	1+	1+	-	-	-	-	-	-	-	-	-	-	-	+	5	-	-	-	-
1+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	6	-	-	-	-
-	1+	-	1+	-	-	+	-	-	-	-	-	-	-	-	-	-	7	-	-	-	-
-	-	2+	-	1+	-	-	-	-	-	-	-	-	-	-	-	-	8	-	-	+	+
-	2+	-	2+	-	-	-	+	+	-	-	-	-	-	-	-	-	9	-	-	-	-
-	3+	3+	2+	2+	-	-	-	-	-	-	-	-	-	-	-	-	10	-	-	+	+
-	2+	-	1+	-	-	-	+	+	-	-	-	-	-	-	-	-	11	-	-	-	-
1+	-	2+	-	1+	-	-	-	-	-	-	+	+	-	-	-	-	12	-	-	-	-
2+	2+	2+	1+	2+	-	-	+	+	-	-	+	+	-	-	-	-	13	-	-	-	-
-	2+	-	1+	-	-	-	+	+	-	-	-	-	-	-	+	+	14	-	-	-	-
-	1+	-	1+	1+	-	+	+	-	-	-	+	+	-	-	-	-	15	-	-	-	-
1+	-	2+	-	2+	-	-	-	-	-	-	-	-	-	-	-	-	16	+	-	-	+
-	-	2+	-	2+	-	-	-	-	-	-	+	+	-	-	-	-	17	-	-	-	-
-	2+	-	4+	-	-	-	-	-	-	-	-	-	-	-	-	-	18	-	-	-	-
-	1+	1+	1+	1+	-	-	-	-	-	-	-	-	-	-	+	+	19	-	-	-	-
-	1+	1+	1+	1+	-	-	-	-	-	-	-	-	-	-	-	-	20	-	-	+	+
-	2+	-	1+	-	-	-	+	-	-	-	-	-	-	-	-	-	21	-	-	-	-
-	1+	2+	1+	1+	+	-	+	-	+	+	+	+	-	-	-	-	22	-	-	-	-
1+	2+	3+	1+	2+	-	-	+	+	-	-	+	+	-	-	-	+	23	-	-	+	+
1+	3+	2+	2+	1+	-	-	-	-	-	-	-	-	-	-	-	+	24	-	-	-	+
-	-	1+	-	1+	-	-	-	-	-	-	+	+	-	-	-	-	25	-	-	-	-
-	2+	-	2+	-	-	-	+	-	-	-	-	-	-	-	-	-	26	-	-	-	-
-	2+	2+	1+	2+	-	-	-	-	-	-	+	+	-	-	-	-	27	-	-	-	-
-	-	3+	-	2+	-	-	-	-	-	-	-	+	-	-	-	-	28	-	-	-	-
1+	2+	2+	2+	1+	-	-	+	+	-	-	-	+	-	-	-	-	29	-	-	-	-
-	2+	-	1+	-	-	-	-	-	-	-	-	+	-	-	-	-	30	-	-	-	-
-	2+	-	1+	1+	-	-	+	+	-	-	-	-	-	-	-	-	31	-	-	-	-
1+	3+	3+	1+	2+	-	-	-	-	-	-	-	-	-	-	+	-	32	-	-	-	-
2+	1+	2+	1+	1+	+	-	+	-	+	+	+	+	-	-	-	-	33	-	-	-	-
-	1+	-	1+	-	-	-	+	+	-	-	-	-	-	-	-	-	34	-	-	-	-
-	1+	1+	1+	1+	-	-	+	+	-	-	-	+	-	-	-	-	35	-	-	-	-
1+	-	2+	-	3+	-	-	-	-	-	+	+	+	-	-	-	-	36	-	-	-	-
-	2+	-	2+	-	-	-	+	+	-	-	-	-	-	-	+	+	37	-	-	-	-
-	-	3+	-	3+	-	-	-	-	+	+	+	+	-	-	-	-	38	-	-	-	-
-	2+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	39	-	-	-	-
-	-	2+	-	2+	-	-	-	-	-	-	+	+	-	-	-	-	40	-	-	-	-
-	2+	-	4+	-	-	-	-	-	-	-	-	-	-	-	-	-	41	-	-	-	-
1+	2+	2+	2+	2+	+	+	+	+	-	-	-	-	-	-	-	-	42	+	+	+	+
-	-	-	2+	2+	-	-	-	-	-	-	-	-	-	-	-	+	43	-	-	-	-
-	2+	-	2+	-	-	-	+	+	-	-	-	-	-	-	+	+	44	-	-	-	-
-	3+	3+	3+	1+	+	-	-	+	-	-	-	+	+	-	+	+	45	-	-	-	+
1+	-	2+	1+	2+	-	+	-	-	-	+	+	-	-	-	-	-	46	-	+	+	+
-	2+	3+	2+	3+	-	-	+	+	+	-	-	+	-	-	-	+	47	-	-	+	+
-	-	2+	2+	2+	-	+	+	+	+	-	-	-	-	-	-	-	48	-	-	-	-
+	-	-	1+	2+	-	-	+	-	-	+	+	-	-	-	-	-	49	-	+	+	-
-	1+	2+	2+	1+	+	-	+	-	+	+	+	+	-	-	-	-	50	-	-	-	-

VASCULITIS								CME			
RE				LE						DISC & VITREOUS	
UT	UN	LN	LT	UT	UN	LN	LT	RE	LE		
-	-	-	-	-	-	-	-	-	+	-	
-	-	+	+	-	-	-	-	-	-	PVD (BE) , DE (RE)	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	+	-	PVD(LE), ERM (LE)	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	+	-	-	
-	-	-	-	-	-	-	-	-	-	PVD (BE)	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	+	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	+	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	+	-	-	+	-	+	-	
-	-	-	-	-	-	-	-	-	+	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	+	-	-	
+	-	-	-	+	+	+	+	-	+	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	+	PVD(LE)	
+	+	+	+	-	-	-	-	+	-	HMD(RE)	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	+	-	
-	-	-	-	-	-	-	-	+	+	PVD(BE)	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
+	-	-	-	+	+	+	+	-	Mi	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	+	-	-	-	-	-	
-	-	-	-	-	-	-	-	+	-	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	-	-	-	-	-	-	+	-	-	
-	-	-	-	-	-	-	-	-	+	-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	+	-	+	+	+	+	-	Mi	-	
-	-	-	-	-	-	-	+	Mi		-	
-	-	-	-	-	-	-	-	-	-	-	
-	-	+	+	-	-	-	-	-	Mi	-	
-	-	-	-	-	+	+	-	-	Mo	-	
-	-	-	-	-	-	+	+	Mi	Mo	-	
-	-	-	-	-	-	-	-	Mi	-	-	
-	-	-	-	-	+	-	-	-	Mi	-	
+	-	-	-	+	+	+	+	-	Mi	-	

		FFA				VASCULITIS	
		RE				L	
S.NO.	CME	UT	UN	LN	LT	UT	UN
1	Mi(LE)	-	-	-	-	-	-
2	-	-	-	+	+	-	-
3	-	-	-	-	-	-	-
4	-	-	-	-	-	-	-
5	Mo(RE)	-	-	-	-	-	-
6	-	-	-	-	-	-	-
7	-	-	-	-	-	-	-
8	-	-	-	-	-	-	-
9	Mi(RE)	-	-	-	-	-	-
10	-	-	-	-	-	-	-
11	-	-	-	-	-	-	-
12	Mi(LE)	-	-	-	-	-	-
13	-	-	-	-	-	-	-
14	Mi(RE)	-	-	-	-	-	-
15	-	-	-	-	-	-	-
16	Mo(LE)	-	-	-	-	+	-
17	Mi(LE)	-	-	-	-	-	-
18	N	+	-	+	+	-	-
19	-	-	-	-	-	-	-
20	-	-	-	-	-	-	-
21	Mi(RE)	-	-	-	-	-	-
22	Mi(LE)	-	-	-	-	+	+
23	-	-	-	-	-	-	-
24	-	-	-	-	-	-	-
25	Mi(LE)	-	-	-	-	-	-
26	Mi(RE)	+	+	+	+	-	-
27	-	-	-	-	-	-	-
28	Mi(LE)	-	-	-	-	-	-
29	Mi(BE)	-	-	-	-	-	-
30	-	-	-	-	-	-	-
31	-	-	-	-	-	-	-
32	-	-	-	-	-	-	-
33	-	-	-	-	-	+	+
34	-	-	-	-	-	-	-
35	-	-	-	-	-	-	-
36	-	-	-	-	-	-	-
37	Mi(RE)	-	-	-	-	-	-
38	-	-	-	-	-	-	-
39	Mi(RE)	-	-	-	-	-	-
40	Mi(LE)	-	-	-	-	-	-
41	N	+	-	+	+	-	-
42	Mi(LE)	-	-	-	-	-	-
43	Mi(RE)	-	-	-	+	-	-
44	-	-	-	-	-	-	-
45	Mi(RE)	-	-	+	-	-	-
46	Se(LE)	-	-	-	-	-	-
47	Mi(BE)	-	-	-	-	-	-
48	Mi(RE)	-	-	-	-	-	-
49	Mi(LE)	-	-	-	-	-	-
50	-	-	-	-	-	+	+

E		C V/A - Ad		COMMENTS		O	S/T S	SS	IS
LN	LT	RE	LE						
-	-	6/6	6/12			-	+	-	-
-	-	6/18	6/12			-	+	+	-
-	-	6/60	6/36			-	+	-	-
-	-	6/6	6/18			-	+	+	-
-	-	6/60	6/24	PSCC(BE),↑IOP		-	+	+	-
-	-	6/6	6/24	PSCC(LE)		-	+	+	-
-	-	6/18	6/6	↑IOP,HM		-	+	-	-
-	-	6/6	6/60	-		-	+	+	-
-	-	6/12	6/6	-		-	+	+	-
-	-	1/60	6/24	↑IOP, Vit Hmg		-	+	+	+
-	-	6/60	6/12	-	-	-	+	-	-
-	-	6/6	6/36	-		-	+	+	-
-	-	5/60	2/60	PSCC(BE)		-	+	+	-
-	-	6/60	6/6	-		-	+	-	-
-	-	6/36	6/36	PSCC(BE)		-	+	+	-
-	+	6/6	6/18	-		-	+	+	-
-	-	6/6	6/36	-		-	+	-	-
-	-	3/60	6/6	-		-	+	+	-
-	-	6/24	6/24	PSCC(BE)		-	+	+	-
-	-	5/60	2/60	-		-	+	+	-
-	-	6/18	6/6	-		-	+	+	-
+	+	6/6	4/60	PSCC(BE)		-	+	+	-
-	-	1/60	6/60	-		-	+	+	-
-	-	6/18	6/12	-		-	+	+	-
-	-	6/6	6/36	-		-	+	+	-
-	-	6/36	6/18	-		-	+	+	-
-	-	6/12	6/18			-	+	-	-
-	-	6/6	6/60	-		-	+	+	-
-	-	1/60	6/60	-		-	+	+	-
-	-	6/9	6/6	-		+	-	-	-
-	-	6/60	6/6	PSCC(RE)		-	+	-	-
-	-	1/60	1/60	-		-	+	+	-
+	+	6/6	4/60	PSCC(BE)		-	+	+	-
-	-	6/12	6/6	-		-	+	-	-
-	-	6/12	6/18	-		-	+	+	-
-	-	NO PL	6/12	-		+	-	-	+
-	-	6/36	6/6			-	+	+	-
-	-	6/6	6/60	-		-	+	+	-
-	-	6/12	6/6	-		-	+	+	-
-	-	6/6	6/18	-		-	+	-	-
-	-	3/60	6/6	-		-	+	+	-
-	-	6/24	6/36	-		-	+	+	-
-	-	6/12	6/9	-		-	+	+	-
-	-	6/36	6/6	-		-	+	+	-
-	-	6/24	6/36	-		-	+	+	-
+	-	6/9	6/60	-		-	+	+	-
-	-	6/36	6/60	-		-	+	+	-
-	-	6/36	6/60	-		-	+	+	-
-	-	6/6	6/24	-		-	+	-	-
+	+	6/6	6/60			-	+	+	-

S.NO.	CRYO		V/A AFTER CRYO			V/A EF			SUBJECTIVE E.F	
			1 WEEK	2 WEEKS	1 MONTH	RE	LE	D-Months	RE	LE
1	-		-	-	-	6/6	6/9	4	N	N
2	-		-	-	-	6/9	6/6	10	MH	N
3	-		-	-	-	6/24	6/12	8	MH	MH
4	-		-	-	-	6/6	6/9	6	N	N
5	-		-	-	-	6/36	6/18	12	HV	St
6	-		-	-	-	6/6	6/12	8	N	MH
7	-		-	-	-	6/12	6/6	5	MH	N
8	-		-	-	-	6/6	6/36	6	N	MoH
9	-		-	-	-	6/12	6/6	3	MoH	N
10	-		-	-	-	6/36	6/18	10	St	MH
11	-		-	-	-	6/24	6/9	5	MH	N
12	-		-	-	-	6/6	6/12	4	N	MH
13	-		-	-	-	6/60	5/60	3	HV	HV
14	-		-	-	-	6/18	6/6	3	MH	N
15	-		-	-	-	6/18	6/9	4	MH	N
16	-		-	-	-	6/6	6/9	8	N	N
17	-		-	-	-	6/6	6/12	4	N	N
18	-	-	-	-	-	6/36	6/6	10	MH	N
19	-		-	-	-	6/12	6/12	6	MH	MH
20	-		-	-	-	6/18	6/36	10	N	N
21	-		-	-	-	6/9	6/6	8	N	N
22	360°(LE)		5/60	6/60	6/60	6/6	6/36	4	N	MH
23	-		-	-	-	6/60	6/36	3	HV	HV
24	-		-	-	-	6/12	6/9	6	MH	N
25	-		-	-	-	6/6	6/12	8	N	N
26	-		-	-	-	6/36	6/18	4	HV	HV
27	-		-	-	-	6/12	6/12	5	MH	HV
28	-		-	-	-	6/6	6/12	3	N	N
29	-		-	-	-	6/36	6/24	6	MH	MH
30	-		-	-	-	6/6	6/6	4	N	N
31	-		-	-	-	6/12	6/6	6	MH	N
32	-		-	-	-	6/18	6/36	4	N	MH
33	360°(LE)		6/60	6/60	6/36	6/6	6/36	4	N	MH
34	-		-	-	-	6/6	6/6	3	N	N
35	-		-	-	-	6/9	6/9	7	N	N
36	-		-	-	-	NO PL	6/6	24	-	N
37	-		-	-	-	6/9	6/6	6	N	N
38	360°(LE)		6/60	6/36	6/36	6/6	6/24	10	N	MH
39	-		-	-	-	6/9	6/6	3	N	N
40	-		-	-	-	6/6	6/9	4	N	N
41	-	-	-	-	-	6/6	6/6	10	N	N
42	360°(RE)LN(LE)		5/60|6/36	6/36(BE)	6/36(BE)	6/18	6/18	12	St	St
43	-		-	-	-	6/9	6/6	18	N	N
44	-		-	-	-	6/36	6/6	5	St	-
45	-		-	-	-	6/12	6/18	3	HV	HV
46	UNLNLT(LE)		6/60	6/60	6/36	6/6	6/36	4	LFU	-
47	-		-	-	-	6/36	6/60	1	LFU	-
48	-		-	-	-	6/12	6/12	8	N	N
49	-		-	-	-	6/6	6/6	16	N	N
50	-		-	-	-	6/6	6/12	4	N	MH

CLINICAL RESPONSE TO TREATMENT									CLINICAL RESPONSE TO TREATMENT					
VASCUITIS		VITREOUS CELLS		VITREOUS OPACITY		SNOWBALL			SNOWBANK		CME		TYPES	
RE	LE	RE	LE	RE	LE	RE	LE	S.NO.	RE	LE	RE	LE	RE	LE
-	-	-	-	-	-	-	-	1	-	+	-	-	-	Ia
-	-	-	-	-	-	-	-	2	-	-	-	-	II	II
-	-	-	-	-	-	-	-	3	-	-	-	-	Ib	Ib
-	-	-	-	-	-	-	-	4	-	-	-	-	-	Ia
-	-	-	-	-	-	-	-	5	-	-	Mi	-	Ia	II
-	-	-	-	-	-	-	-	6	-	-	-	-	-	Ia
-	-	-	-	-	-	-	-	7	-	-	-	-	Ia	-
-	-	-	-	-	-	-	-	8	-	+	-	-	-	Ib
-	-	-	-	-	-	-	-	9	-	-	-	-	Ia	-
-	-	-	1+	-	1+	-	-	10	-	+	-	-	II	II
-	-	-	-	-	-	+	-	11	-	-	-	-	Ia	-
-	-	-	-	-	-	-	-	12	-	-	-	Mi	-	Ia
-	-	-	-	1+	1+	+	+	13	-	-	-	-	II	II
-	-	-	-	1+	-	-	-	14	-	-	-	-	Ib	-
-	-	-	-	1+	-	-	-	15	-	-	-	-	Ib	Ib
-	+	-	-	-	1+	-	-	16	-	-	-	-	-	III
-	-	-	-	-	-	-	-	17	-	+	-	-	-	Ia
-	-	-	-	2+	-	-	-	18	-	-	-	-	Ib	-
-	-	-	1+	-	-	-	-	19	-	-	-	-	Ia	Ia
-	-	-	-	-	-	-	-	20	-	-	-	-	Ia	Ib
-	-	-	-	-	-	-	-	21	-	-	-	-	Ia	-
-	+	1+	2+	1+	1+	-	+	22	-	-	-	Mi	Ia	III
-	-	-	-	-	-	-	-	23	+	+	-	-	Ib	Ib
-	-	-	-	-	-	-	-	24	-	-	-	-	Ia	Ia
-	-	-	-	-	-	-	-	25	-	-	-	-	-	II
+	-	+	-	+	-	-	-	26	-	-	Mi	-	III	-
-	-	-	-	-	1+	-	-	27	-	-	-	-	Ia	Ia
-	-	-	-	-	+	-	-	28	-	-	-	-	-	Ia
-	-	-	-	1+	1+	-	-	29	-	-	Mi	-	II	II
-	-	-	-	-	-	-	-	30	-	-	-	-	Ia	-
-	-	1+	-	-	-	-	-	31	-	-	-	-	Ia	-
-	-	-	-	1+	1+	-	-	32	-	-	-	-	Ib	Ib
-	+	1+	2+	1+	1+	-	+	33	-	-	-	Mi	Ia	III
-	-	-	-	-	-	-	-	34	-	-	-	-	Ia	-
-	-	-	-	-	-	-	-	35	-	-	-	-	Ia	Ia
-	-	-	-	-	-	-	-	36	-	-	-	-	-	Ib
-	-	-	-	-	-	-	-	37	-	-	-	-	Ia	-
-	-	-	-	-	+	-	-	38	-	-	-	-	-	Ib
-	-	-	-	-	-	+	-	39	-	-	-	-	Ia	-
-	-	-	-	-	-	-	-	40	-	+	-	-	-	Ia
-	-	-	-	2+	-	-	-	41	-	-	-	-	Ib	-
-	-	-	-	+	-	-	-	42	-	-	-		Ib	II
-	-	-	-	-	-	-	-	43	-	-	-	-	Ib	Ia
-	-	-	-	-	-	-	-	44	-	-	-	-	II	-
-	-	+	+	+	+	+	+	45	-	-	Mi	-	Ib	Ib
-	-	+	2+	2+	-	-	-	46	-	-	Mo	-	Ib	II
-	-	-	-	-	-	-	-	47	-	-	-	-	Ib	Ib
-	-	-	-	-	+	-	-	48	-	-	-	-	Ia	Ia
-	-	-	-	-	+	-	-	49	-	-	-	-	Ia	Ib
-	+	1+	2+	1+	2+	-	+	50	-	-	-	Mi	Ia	Ib