Dissertation on

A CLINICAL STUDY ON RETINAL VASCULITIS

Submitted in partial fulfillment of requirements of

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CHENNAI

APRIL 2012

CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL STUDY ON RETINAL VASCULITIS"** is a bonafide record of the research work done by **Dr.S.PREMA.,** Post graduate in Regional Institute of Ophthalmology, Madras Medical College and Research Institute, Government General Hospital,Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr.M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2009-2012.

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled, **"THE CLINICAL STUDY ON RETINAL VASCULITIS"** is a bonafide and genuine research work conducted by me under the guidance of **Prof. Dr. R. Ravikumar, M.S., D.O.,** Professor Department of Uvea / Retina services, Regional institute of ophthalmology. Government Ophthalmic hospital. Chennai-600008.

Date **Dr. S. Prema**

Place

CONTENTS

PART ONE

ABBREVATION

NVD - Neovascularistion of disc

NVE - Neovascularisation elsewhere

SLE - Systemic Lupus Erythematosus

PAN - Polyarteritis Nodosa

ESR - Erythrocyte Sedimentation Rate

CRP - C-Reactive protein

HLA - Human leukocyte antigen

CMV - Cytomegalovirus

AIDS - Acquired immunodeficiency syndrome

ACE - Angiotensin converting enzyme

TORCH- Toxoplasmosis, Rubella, Cytomegalovius, Herpes virus

INTRODUCTION

Retinal vasculitis is considered to be Type 3 hypersensitivity reaction of retinal vasculature affecting veins, arteries or both. The pathological evidence indicates that the basic pathology is perivascular. The disease is characterized by occurrence of hemorrhages in the retina, vitreous, or with recurrence of haemorrhages. The disease can progress and lead to fibovascular membrane and tractional retinal detachment, complicated cataract and secondary glaucoma.

The first attempt to discuss in detail the clinical syndrome of recurrent haemorhages was made by HENRY EALES (1880-82).[1]

PERLS(1873) and ANGELUCCI(1878) studied the histological appearance of periphlebitis.

WADSWORTH (1887) was the first to describe the clinical picture of perivasculitis.

AXENFELD and STOCK (1909-11) drew attention to the etiological significance of tuberculosis.

CLASSIFICATION OF RETINAL VASCULITIS

DUKE ELDER'S CLASSIFICATION[1]

1. Vasculitis secondary to uveitis
2. Vasculitis secondary to systemic disease
3. Apparently primary vasculitis

COGAN'S CLASSIFICATION[2]

1. Mild papillophlebitis,
2. Moderate vasculitis-bilateral-arterioles and/or veins affected,
3. Severe vasculitis – arterioles involved more than veins.

RECENT CLASSIFICATION[3]

1. Primary vasculitis,
2. Vasculitis secondary to systemic diseases,
3. Vasculitis secondary to ocular disease.

CLINICAL CHARACTERISTICS OF RETINAL VASCULITIS

SYMPTOMS[3]

Painless decrease/marked loss of vision,

Floaters,

Large areas of scotomata,

Asymptomatic if the lesions are in the peripheral retina.

SIGNS[3]

Sheathing of vessels,

Obliteration and perivascular edema,

Occlusive retinopathy characterized by large areas of capillary drop outs,

Vascular architectural alteration manifesting as

arteriolar/venule anastamosis and crossing of vessels

over horizontal raphe,

Recurrent vitreous hemorrhage,

Cystoid macular edema.

PRIMARY VASCULITIS

Eales Disease,

Idiopathic Retinal Vasculitis, Aneurysms And Neuroretinitis

(IRVAN)

Frosted Branch Angiitis,

Scleritis.

EALES DISEASES

Eales disease is a idiopathic bilateral, asymmetric condition that manifest as an obliterative perivasculitis[4] (particularly involving the venous side) affecting retina in multiple quadrant, starting at or anterior to the equator and progressing posteriorly.

The disease is named after Henry Eales, an ophthalmologist in 1880, he described as a syndrome of recurrent vitreous hemorrhage in young men with epistaxis and constipation.[5] He termed the condition as primary recurrent retinal hemorrhage. With the advent of ophthalmoscope he was able to document abnormal retinal veins and areas in the peripheral retina that were free of capillaries.

In 1887, Wadsworth described the association of inflammation and neovascularization with the disease.

Eales disease affects healthy young adults with average age of onset at 15-45 years, with male predominance[6] and poor socioeconomic class.

CLINICAL FEATURES

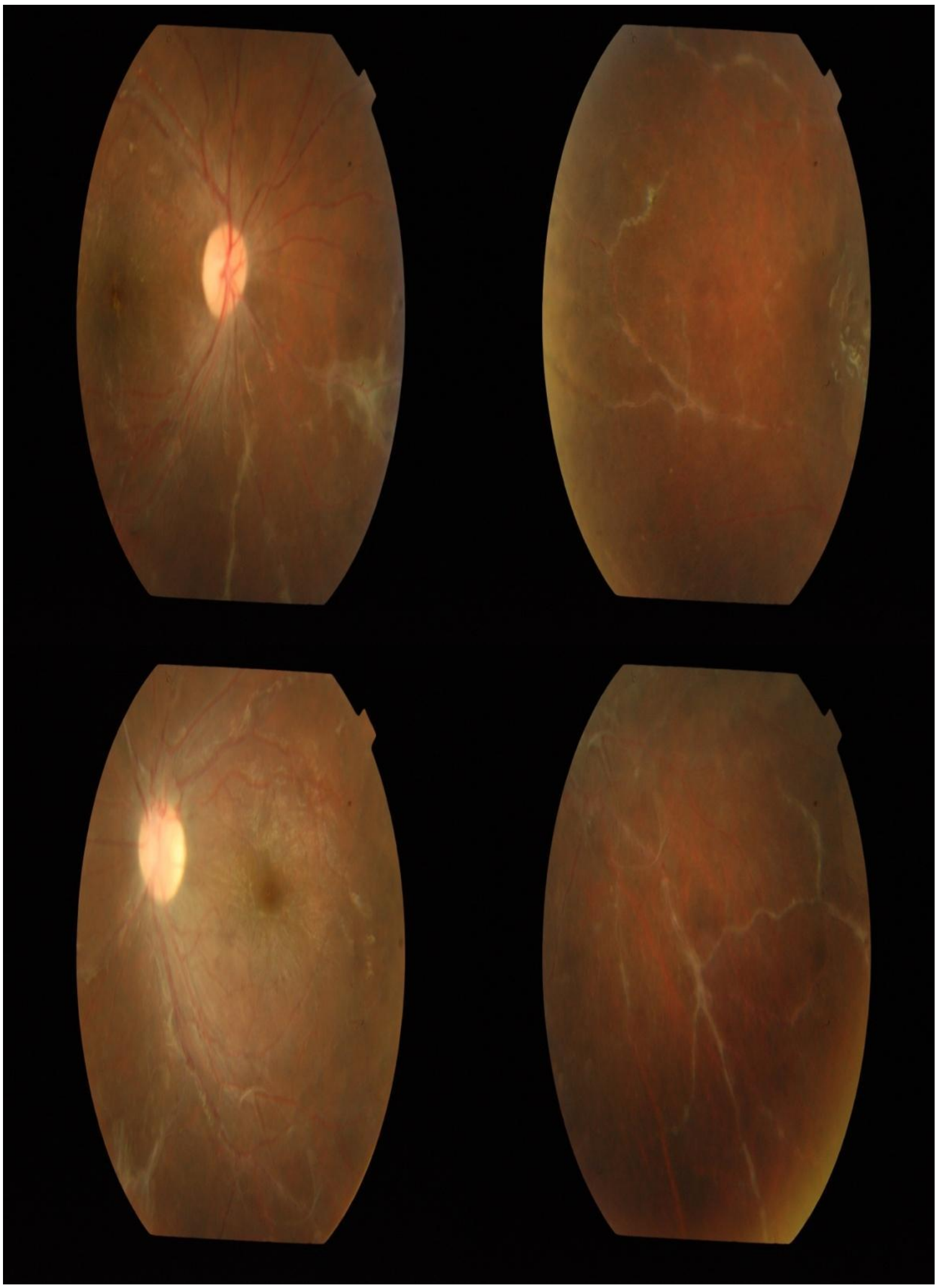
Eales disease is an idiopathic, peripheral, bilateral, retinal vasculitis resulting in peripheral non-perfusion and neovascularization in healthy male population.

Presenting symptoms in most patients are floaters and painless diminution of vision.

Anterior segment is normal in patients with Eales, presence of cells and flare in these patient is the first indicator of development of rubeosis iridis.

Clinically the various features of Eales'disease can be divided into signs of Inflammation, signs of Ischemia, signs of Neovascularization and its sequelae.[7]

FIG-1 BILATERAL EALES



INFLAMMATION

Ocular inflammation is the commonest manifestation of Eales disease. Vascular sheathing occurs in 80% of patients with involvement ranging from thin, contiuous white lines, to thick exudates which are segmental.[8]

Keratic precipitates, anterior chamber cells and flare, vitreous cells. Cystoid macular edema occurs in eyes with extensive sheathing. Fundus fluorescein angiography demonstrates hyperfluorescence in the areas of vascular sheathing and staining in the active stage of disease.(Figure 2,3,4 &5)

EALES DISEASE
FIGURE-2-STAGE OF INFLAMMATION

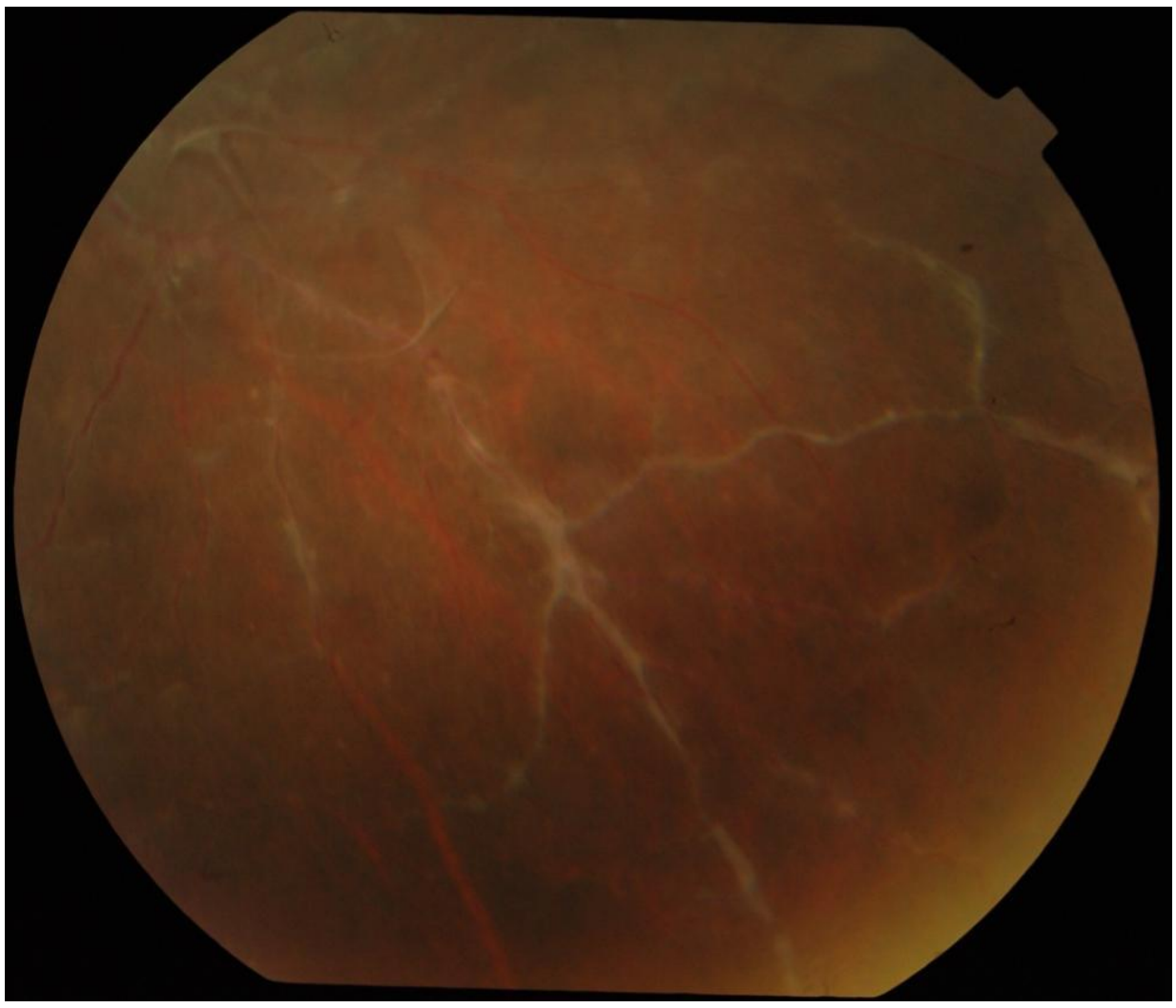


FIGURE-3-SHEATHING OF VESSELS

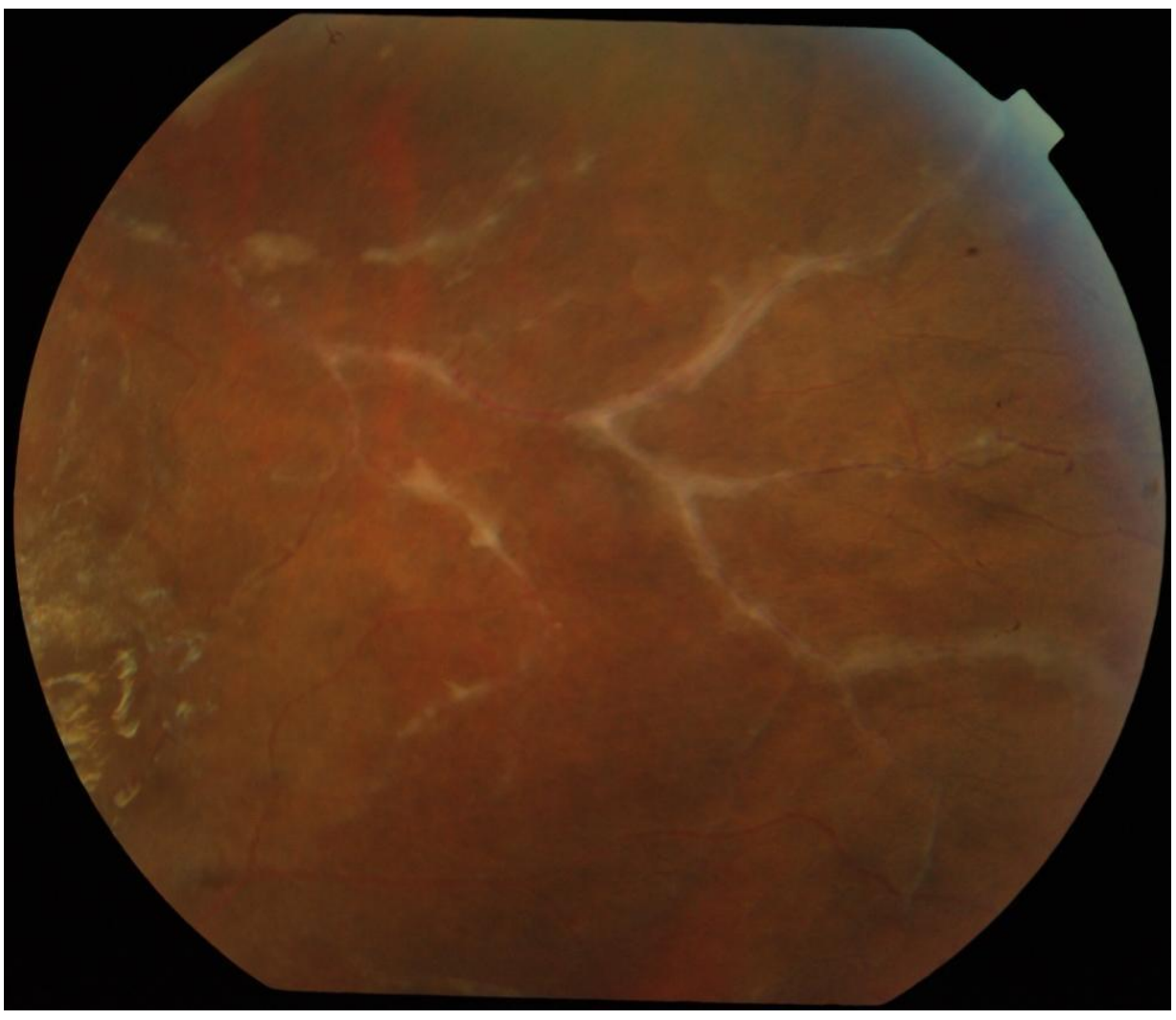
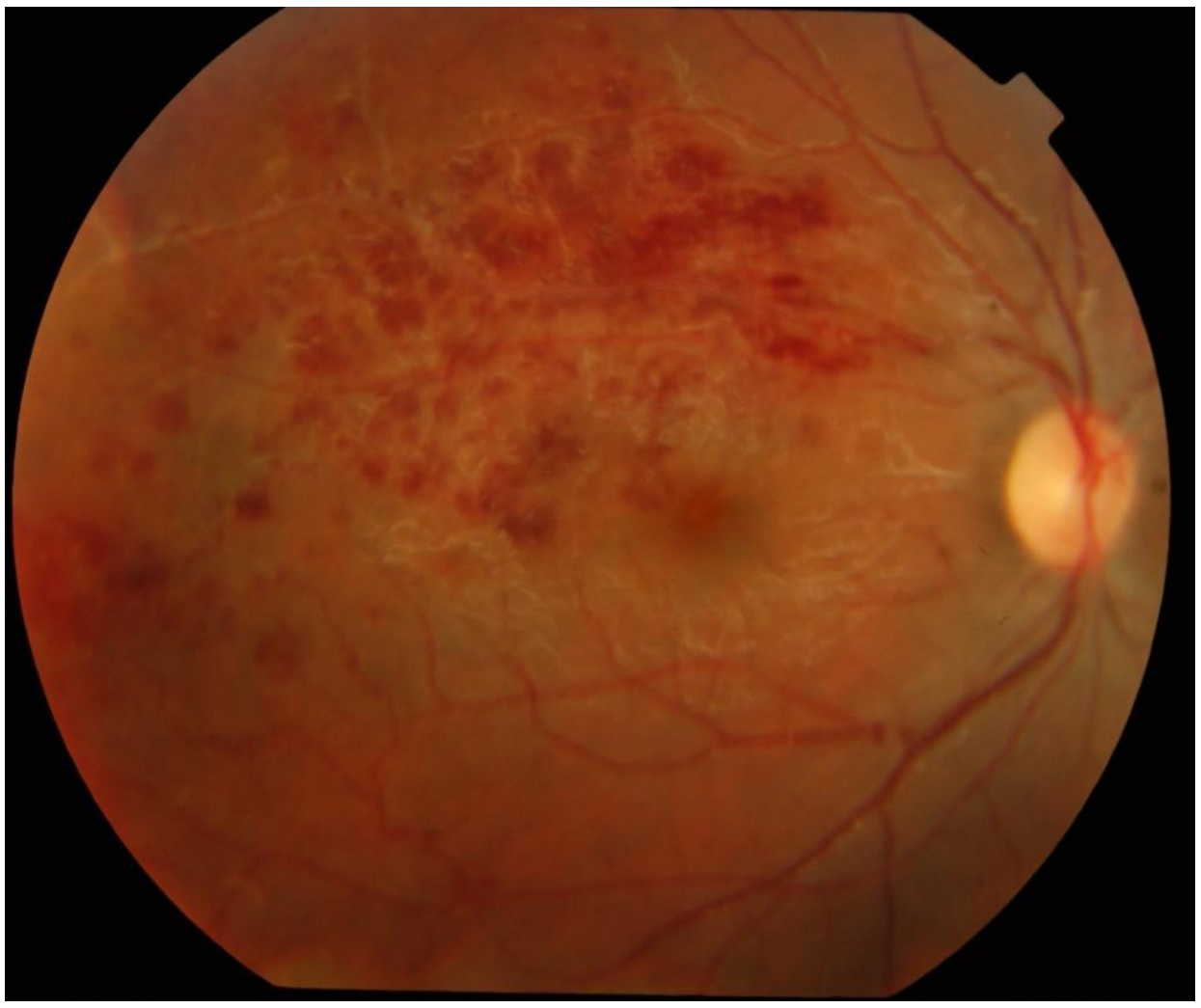
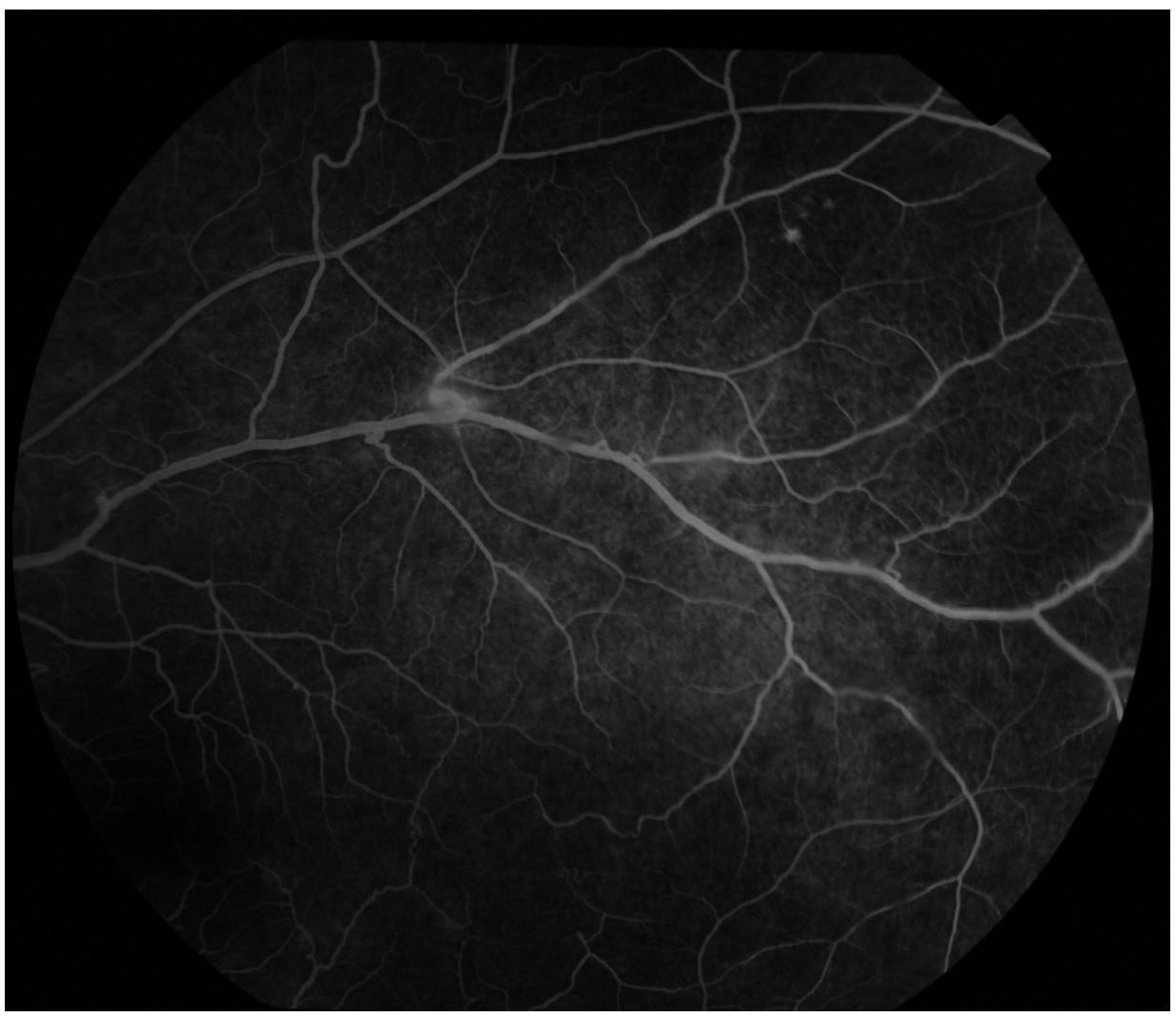


FIGURE-4-SHEATHING WITH HAEMORRHAGE



FUNDUS FLUORESCEIN ANGIOGRAPHY
FIGURE-5-STAINING OF VESSELS



NONPERFUSION OR ISCHEMIA

Peripheral retinal nonperfusion is present in all the patients, which extends from small to massive areas.

Intraretinal hemorrhages appears first in the affected area, followed by increase in vascular tortuosity with collateral formation around the occluded vessels, with microaneurysm, arteriovenous shunt and venous beading seen at the junction.[9]

Patients with Eales disease experience branch retinal vein occlusion, can be single or multiple associated with peripheral retinal involvement not respecting the horizontal midline.(Figure-6,7)

STAGE OF ISCHEMIA
Figure-6 AREAS OF CAPILLARY NON-PERFUSION

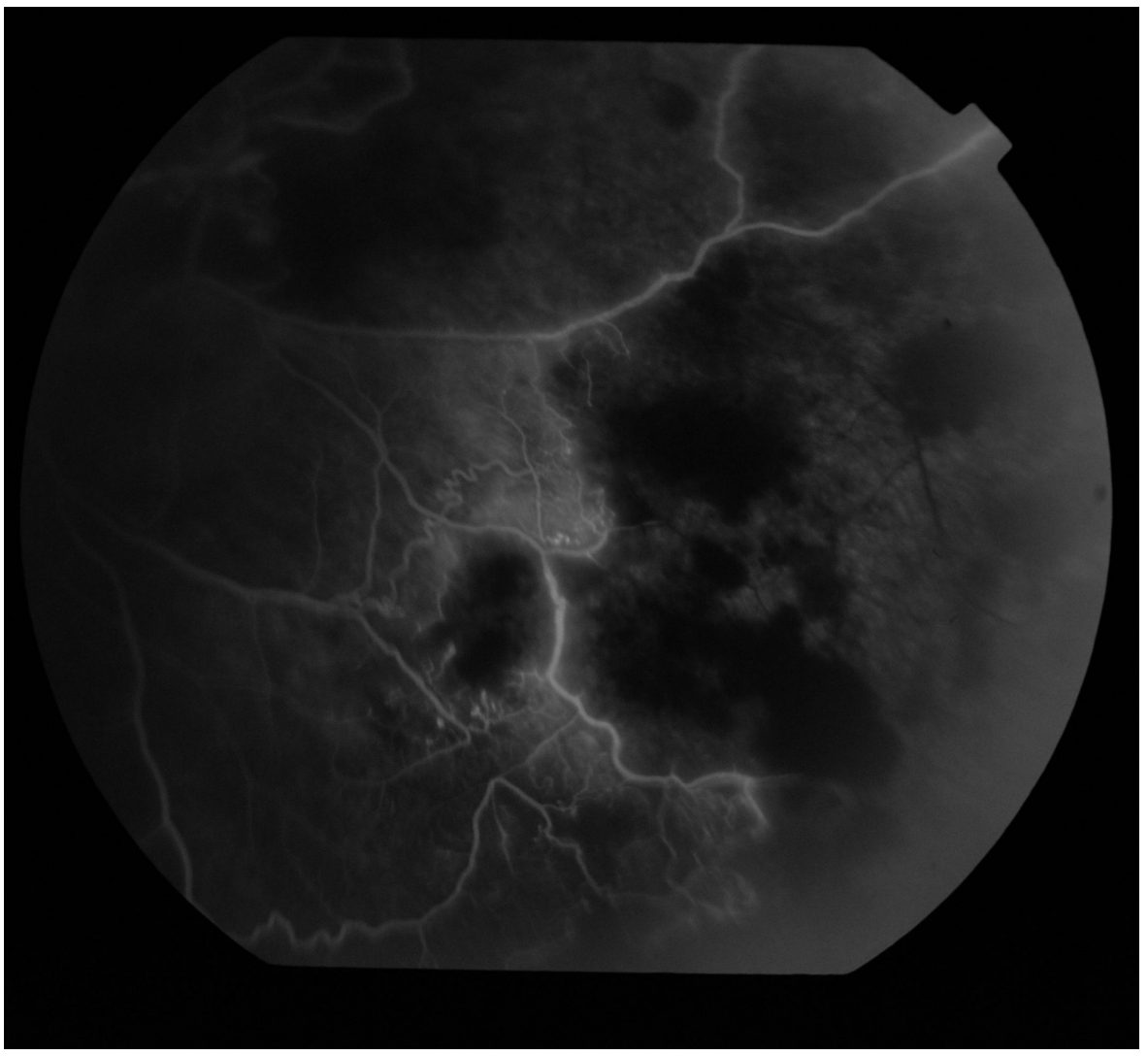
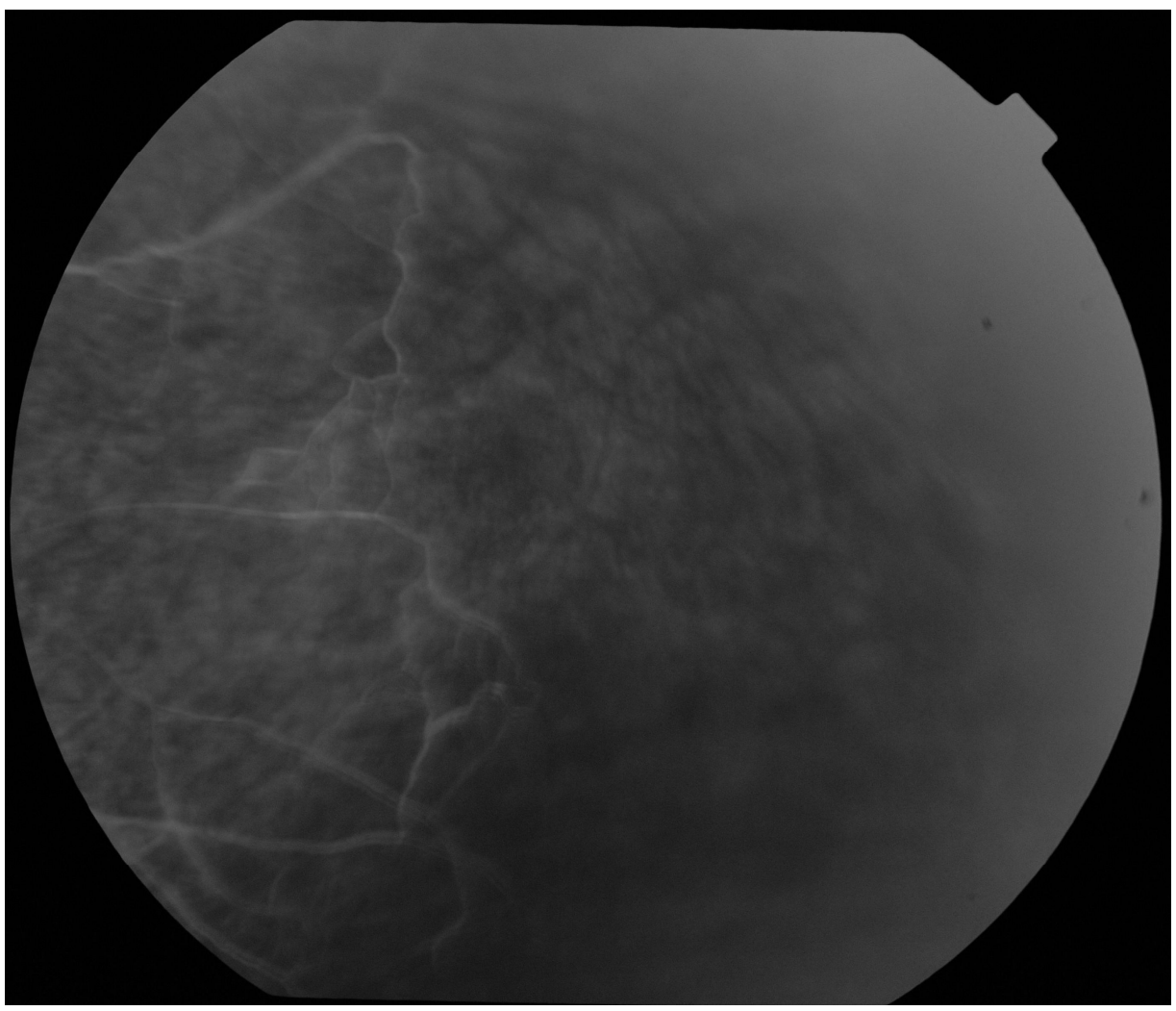


FIGURE-7



NEOVASCULARIZATION

The retinal nonperfusion leads to eventual development of new vessels. Neovascularisation occurs over disc, retina or iris. These abnormal blood vessels frequently bleed and are major cause of visual loss.

About 80% of the patient present with vitreous hemorrhage due to neovascularization. New vessels occur in the intraretinal, preretinal and intravitreal locations. In advanced disease due to global ischemia NVD develops.

The commonest sequelae of neovascularization is vitreous hemorrhage which can lead to tractional retinal detachment. Since in Eales the posterior pole is not involved, visual loss is minimal. But as the disease progresses tear develops in the retina due to traction, producing combined detachment**.**[9,10]**(Figure 8,9)**

Figure-8 STAGE OF NEOVASCULARISATION

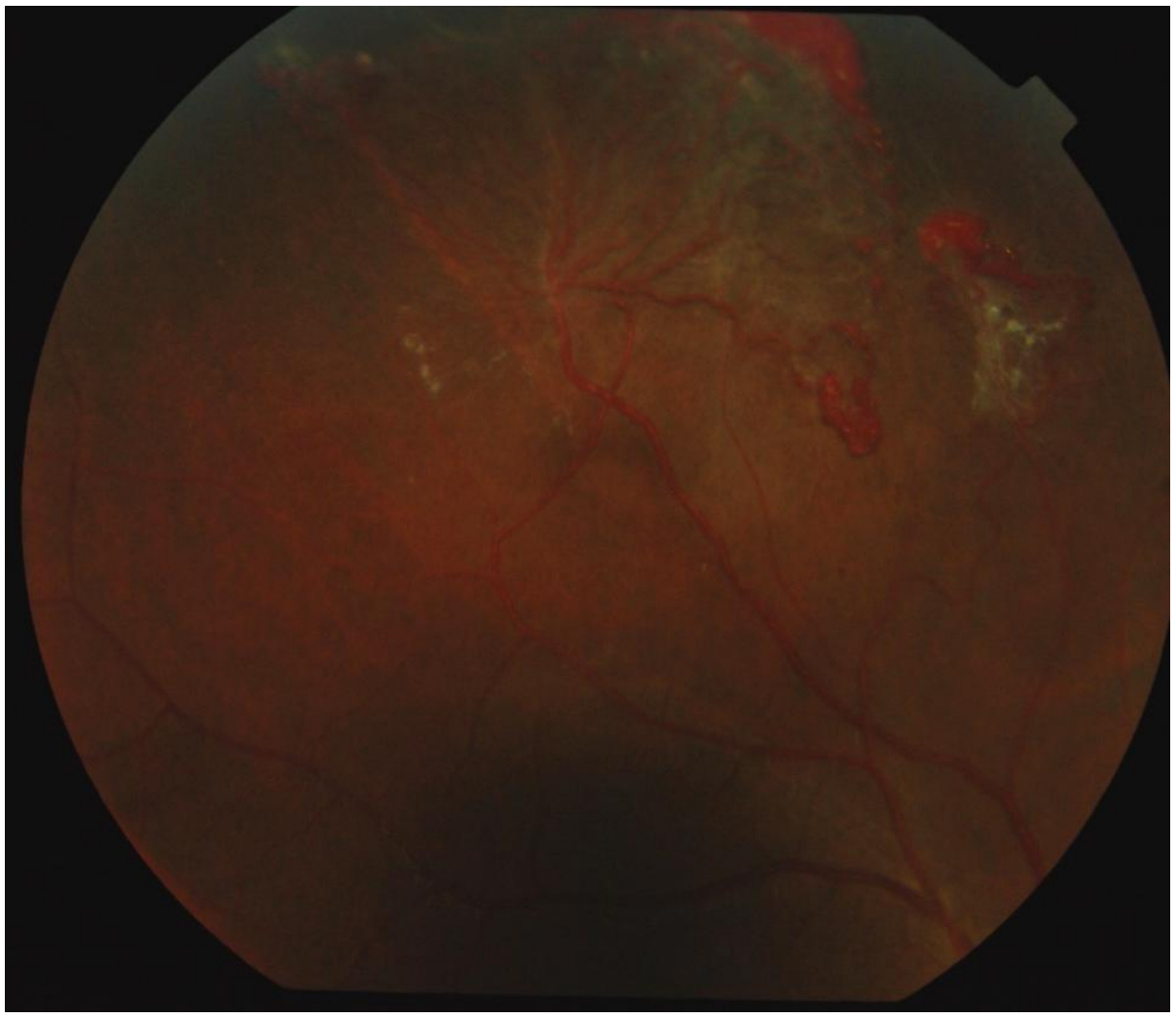
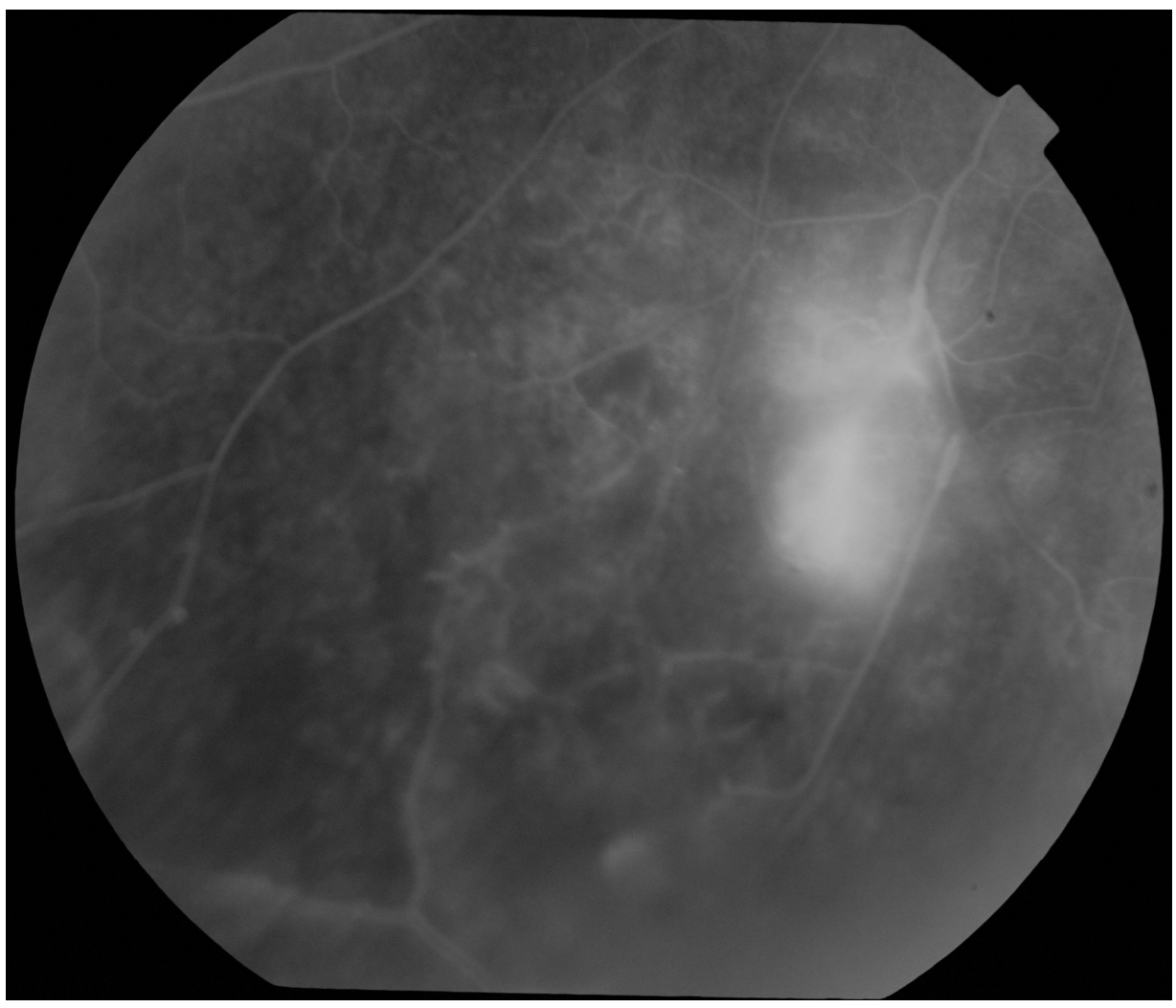


Figure-9 FFA-MID-PHASE SHOWING INTENSE LEAKAGE SUGGESTIVE OF NEW VESSEL



CENTRAL EALES

The prevalence of central Eales is 6%. This presents as a papillophlebitis or non-ischemic central retinal vein occlusion. Prognosis is good in this case.[9]

CLASSIFICATION OF EALES

Classification is based on the evolution and progress of the disease.

The classification divided the disease into four stages

Stage 1

Mild periphlebitis of peripheral retinal capillaries

Stage 2

Wide spread periphlebitis of the venous system

Stage 3

New vessel formation and vitreous hemorrhage

Stage 4

End result of multiple hemorrhages –Retinitis proliferans.

NEW SYSTEM OF CLASSIFICATION

This new system of classification divides the disease into central and peripheral, and peripheral type is further subdivided as

STAGE 1a

Periphlebitis of small caliber vessels with superficial retinal hemorrhages.

Stage 1b

Periphlebitis of large caliber vessels with superficial hemorrhages.

Stage 2a

Areas of capillary non-perfusion.

Stage 2b

Neovascularization of disc and elsewhere.

Stage 3a

Fibrovascular Proliferation

Stage 3b

Vitreous hemorrhage

STAGE -4a

Traction Or Combined Retinal detachment

Stage-4b

Rubeosis iridis, Neovascular glaucoma, cataract and optic atrophy.[9]

ETIOPATHOLOGY

Multiple pathological, immunological, molecular biological and biochemical studies have indicated the role of HLA antigen, autoimmunity, mycobacterial genome and oxidative stress mechanism in the pathogenesis of vasculitis.[11]

Higher phenotypic association of HLA B5, DR1 and DR4 was seen in patients with retinal vasculitis.[12]

Polymerase chain reaction using IS 6110 primers was done to detect the bacterial genome in vitreous fluid, Epiretinal membrane and was statistically found in Eales patients.

Biochemical studies show that the protein carbonyl group content increases with severity of Eales disease, indicating the role of oxidative stress in Eales disease.[13]

DIFFERENTIAL DIAGNOSIS

Eales disease has to be differentiated from other causes of Retinal vasculitis or Retinitis.

Cytomegalovirus Retinitis

Behcet’s Disease

Sarcoidosis

Leukemia

Syphilis

Tuberculosis

Multiple sclerosis

Parsplanitis

Toxoplasma

Toxocara

Systemic lupus erythematosus

Lyme borreliosis

SYSTEMIC ASSOCIATION OF EALES[14]

Acute/subacute myelopathy

Multifocal white matter abnormality

Ischemic or infarction of brain and manifests as, focal neurological signs and demyelination

Abnormalities in the peripheral vestibular system

Bilateral sensorineural hearing loss

Hemiplegia and paraparesis

Internuclear ophthalmoplegia

Psychosis

MANAGEMENT

MEDICAL TREATMENT

Corticosteroids are given to the patients systemically 2mg/kg during the phase of inflammation and tapered over a period of 6-8 weeks.

Subtenon injections of triamcinalone are administered in the dose of 0.5-1ml(40mg/ml).

Antimetabolite, low dose oral methotrexate (12.5mg/wk) for 12 weeks are indicated for one eyed and patients with central Eales disease.

Patients with excessive exudative sheathing and nodule formation, strongly positive mantoux were started on antitubercular drugs, Isoniazid (300mg) and Rifampicin (450mg) for a period of 9 months.[14]

LASER TEATMENT

Retinal laser treatment is performed for the regression of existing new vessels by photocoagulating the areas of ischemia. Panretinal photocoagulation performed in the presence of NVD or when extensive areas of ischemia exists.

Photocoagulation is done to the extreme periphery using laser indirect ophthalmoscope.

Retinal anchoring treatment, in this procedure laser is applied to the base of the fibrovascular traction to prevent combined detachment.[15]

PARS PLANA VITRECTOMY

INDICATIONS

Media opacities

Fibrous and fibrovascular areas of adhesion of the posterios vitreous

Nonresolving vitreous hemorrhage (>6-8 weeks)

Combined retinal detachment

Tractional detachment threatening the macula [16]

IDIOPATHIC RETINAL VASCULITIS,

ANEURYSMS, NEURORETINITIS (IRVAN syndrome)

IRVAN is a disease of young age with features of multiple saccular and fusiform aneurysm involving large arterioles combined with peripheral vascular non-perfusion and uveitis, neuroretinitis, retinal neovascularization, optic nerve head swelling and anterior uveitis associated with this disorder.

The disease has been seen in pregnant women in a hypercoagulable state and peripheral venule and arteriolar occlusion is noted

Retinal laser photocoagulation performed as early as possible to the areas of non-perfusion.[17]

FROSTED BRANCH ANGIITIS

As per Walker et al, Frosted branch angiitis is a florid translucent retinal perivascular sheathing. It is generally noted in young individuals. There is severe drop in visual acuity during acute period. Both arteries and veins can be involved mostly venules are affected more. Most of the cases are bilateral, there is no gender predilection.

Frosted branch angiitis may be

Idiopathic

CMV retinitis

Tuberculosis

Syphilis

Herpes simplex type 2

Aseptic meningitis.

Lymphoma

Leukemia

Sarcoidosis

Multiple sclerosis

Pars planitis

Systemic lupus erythematosis

MASUDA and COWORKERS reported a patient with frosted branch angiitis with multiple placoid lesion suggestive of occlusion of choriocapillaries.

Neovascularisation of disc and retina were reported in some patient in whom retinal ischemia was associated.

Steroids and laser photocoagulation to ischemic areas had good visual outcome.[11]

SCLERITIS

The vasculature near the scleritis is integrally involved in inflammatory and pathological process. The inflammatory reaction in anterior scleritis with spill over leading to anterior or intermediate uveitis.

Posterior scleritis patients present with severe ocular pain with minimal anterior segment changes, minimal vitreous changes, choroidal folds and retinal thickening. Ultrasound examination helps in the diagnosis. Corticosteroid therapy helps to control the disease. But recurrences do occur. Whether Posterior scleritis is associated with a systemic disorder must be ruled out.[18]

OCULAR VASCULITIS DUE TO SYSTEMIC DISEASES

Behcet’s Disease

Sarcoidosis

Systemic Lupus Erythematosus

Postvaccination

Multiple Sclerosis

Wegener’s Granulomatosis

Takayasu’s Disease

Buerger’s Disease

Polyarteritis Nodosa

Polymyositis

Dermatomyositis

Whipple’sdisease

Crohn’s Disease

Sjogren’s A Antigen

Kikuchi-Fujimoto Disease

Susac’s Syndrome

SYSTEMIC DISORDER

Retinal vasculitis is associated with collagen vascular group of disorder. Patients usually present with malaise, weight loss, arthritis, fever and rash. Also present with mononeuritis multiplex, a disease which progress to affect multiple nerve roots especially in the lower extremities.

SARCOIDOSIS

Sarcoidosis causes myriad of ocular manifestations including severe involvement of retinal vasculature. It affects commonly the age group less than 40 yrs.

Unknown antigen or abnormal defense mechanism triggered by various insults responsible for T-cell activation is the prime pathogenesis in sarcoidosis. Increased levels of serum VEGF has been observed in sarcoidosis.

PATHOLOGY

Histopathology findings are multiple non-caseating, epitheloid granulomas. The centre of granulomas consist of histiocytes, epitheloid cells and multinucleated giant cells that may contain intracytoplasmic inclusions such as asteroid and schaumann bodies.[19]

OCULAR SARCOIDOSIS

It’s the third common manifestation of sarcoidosis preceded by pulmonary disease and hilar lymphadenopathy. Ocular disease may be the presenting feature in 9% cases. Two peaks in age incidence for ocular sarcoid include 20-30yrs and 50-60yrs.

Sarcoid related uveitis occur in five ways

Acute anterior uveitis

Chronic anterior uveitis

Intermediate uveitis.

Posterior uveitis

Panuveitis

Retinal vasculitis constitute one of the cardinal features of posterior segment involvement. It is characterized by perivenous sheathing in the equatorial retina, which is usally segmental. Vasculitis may result in leakage or occlusion of the lumen of vein leading to ischemia, exudation and edema. Neovascularization is due to ischemia resulting from inflammation and vein occlusion.[19](Fig-10,11)

FIG-10SARCOIDOSIS

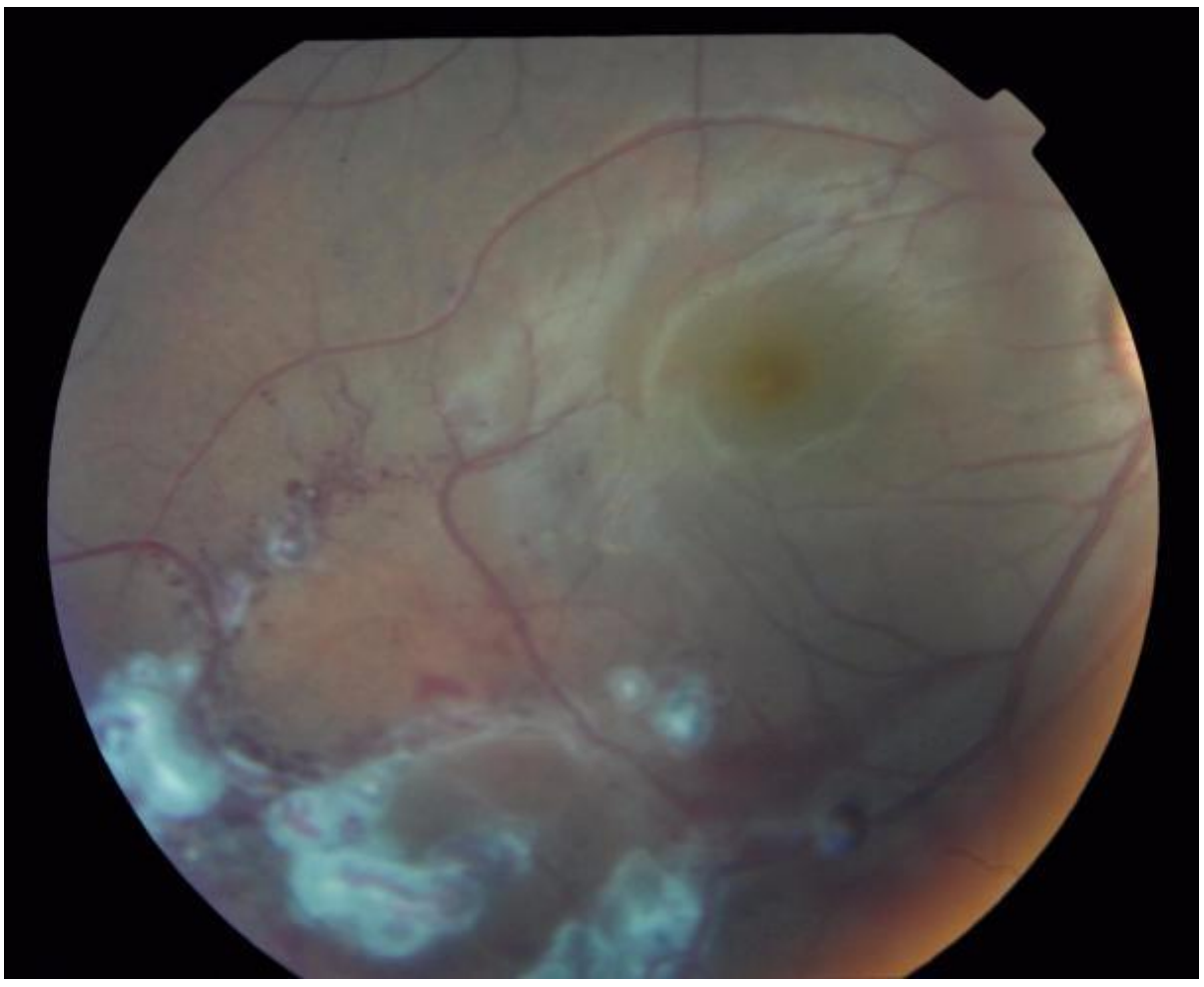
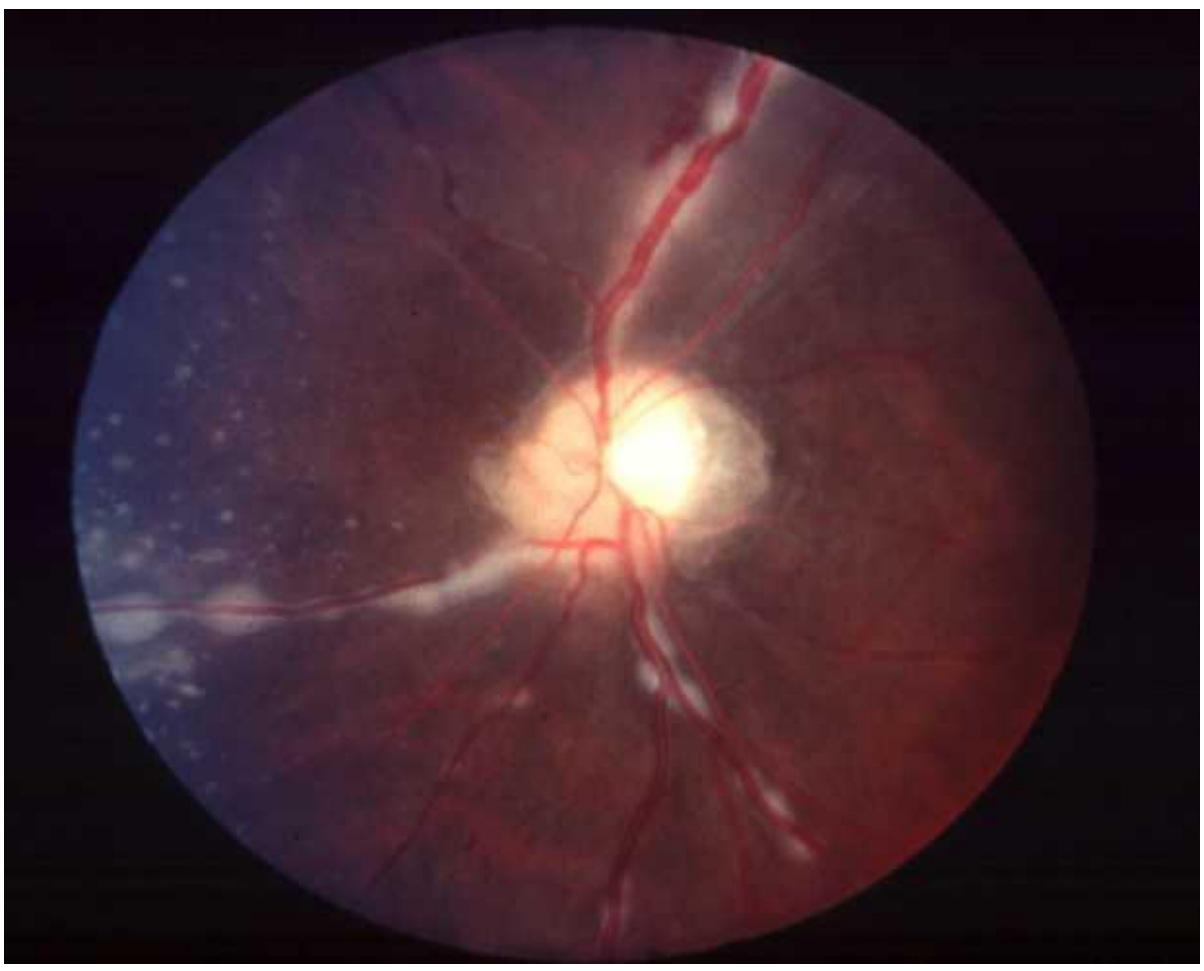


FIG-11 CANDLE WAX DRIPPING



VASCULITIS IN MULTIPLE SCLEROSIS

Multiple sclerosis is a chronic inflammatory demyelinating disease of central nervous system mostly affecting young adults. ocular manifestations include Opticneuritis, Extraocularmuscle disturbance and intraocular inflammation.

It was reported by Rucker that 20% of patient with Multiple sclerosis have vascular sheathing as their association.[20]

Retinal vasculitis in multiple sclerosis patient is exclusively periphlebitis, [21]Retinal venous sheathing. It encompasses both the active and chronic form. The chronic form of sheathing appears typically as dense white strips over several branches of vascular tree.

Activity of periphlebitis is not correlated with Optic neuritis, systemic exacerbation or severity of disease. It can lead to occlusive vasculitis with subsequent formation of neovascularisation.[22]

BEHCET'S DISEASE

Ocular Behcet's is characterized by nongranulomatous uveitis with retinal vasculitis. The reported frequency of ocular manifestation is 83-95% in males and 67-73% in females.

Retinal vasculitis is of occlusive nature characterized by retinal edema, yellowish white exudates and hemorrhages. Behcet's disease is the only systemic vasculitis that affects both arteries and veins and this feature is pathognomic of Behcet's vasculitis. Occlusive nature of vasculitis will lead to neovascularistion.[23]

NECROTIZING VASCULITIS

Disorder with pathogenesis being linked to antineutrophilic cytoplasmic antibodies plus environmental and genetic factors.

POLYARTERITS NODOSA AND MICROSCOPIC POLYANGIITIS

SYSTEMIC COURSE OF PAN

Neuropathy

Nephropathy

Cutaneous ulcers

Gastrointestinal thrombosis and infarction

Musculoskeletal pain

Coronary arteritis

CNS involvement

OCULAR MANIFESTATION OF PAN

Microangiopathy with cotton wool patches

Serous Retinal Detachment

Retinal Vasculitis

Artery occlusion

Ischemic Optic Neuropathy.[24]

CHURG STRAUSS SYNDROME

ACR criteria for diagnosis of Churg Strauss Syndrome

Asthma, adult onset

Eosinopilia >10% in the blood

Sinusitis

Pulmonary infiltrates

Neuropathy

Biopsy with vasculitis and Eosinophils

OCULAR MANIFESTATION

Retinal vascular disorders like

Ischemic Optic Neuropathy

CRAO

BRAO

CRVO

Retinal Vasculitis

WEGENER GRANULOMATOSIS

It’s a triad of Systemic necrotizing vasculitis(87%), necrotizing granulomatous involvement of respiratory tract and necrotizing glomerulonephritis.

ACR criteria for diagnosis

2 of 4

Inflammation in the nose and mouth

Abnormal urinary sediment

Abnormal chest x-ray

Granulomatous inflammation of arteries

OCULAR MANIFESTATION

Orbit (13%)

Eyelid and Nasolacrimal duct (13%)

Episcleritis and Scleritis (11%)

Keratitis (8%)

Optic neuropathy (6%)

Conjunctivitis (4%)

Retinal vasculopathy (5%)(Figure-12,13)

Uveitis (3%).[24]

Fig-12PERIARTERITIS AND PERIPHLEBITIS IN WEGENERS GRANULOMATOSIS

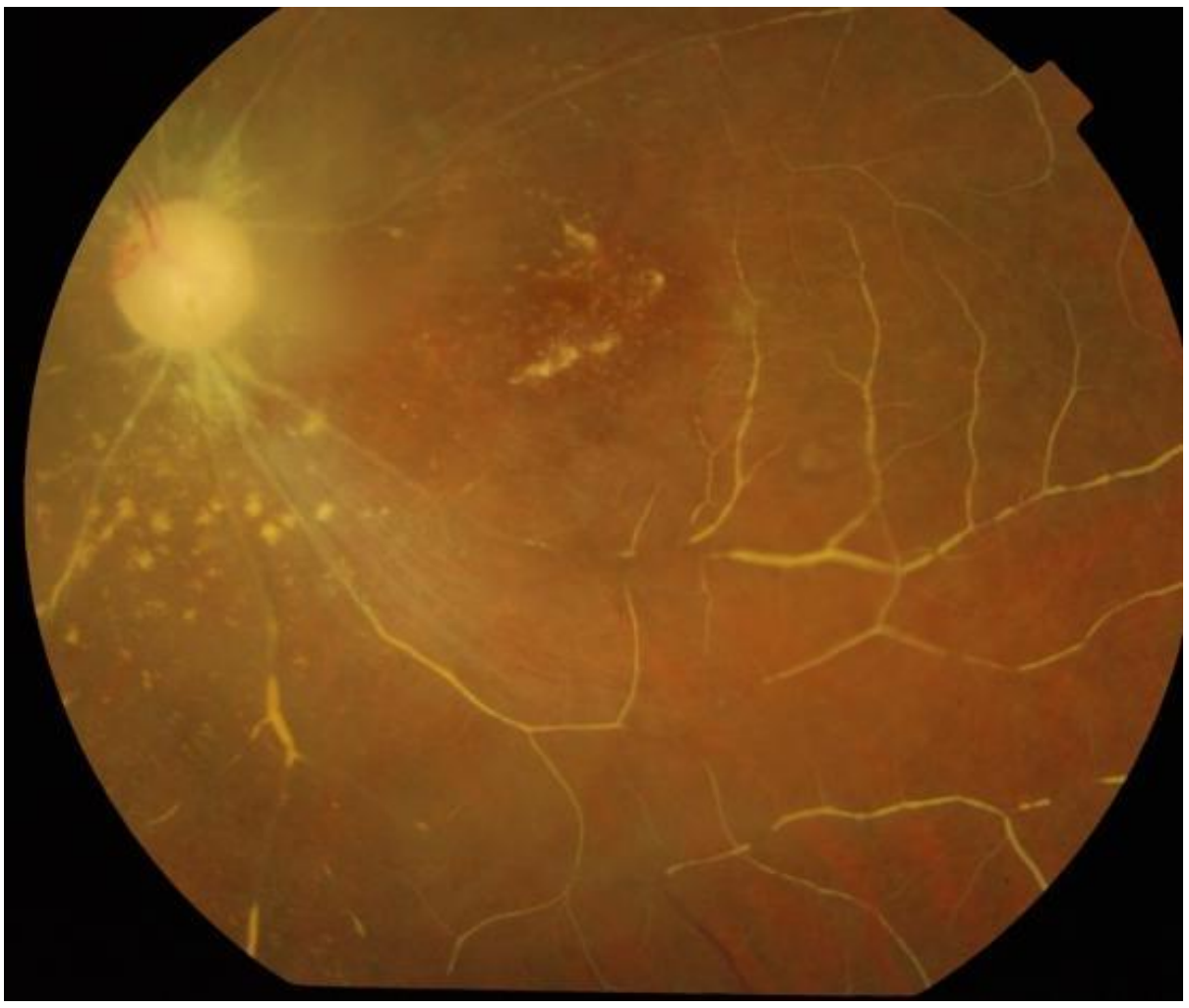
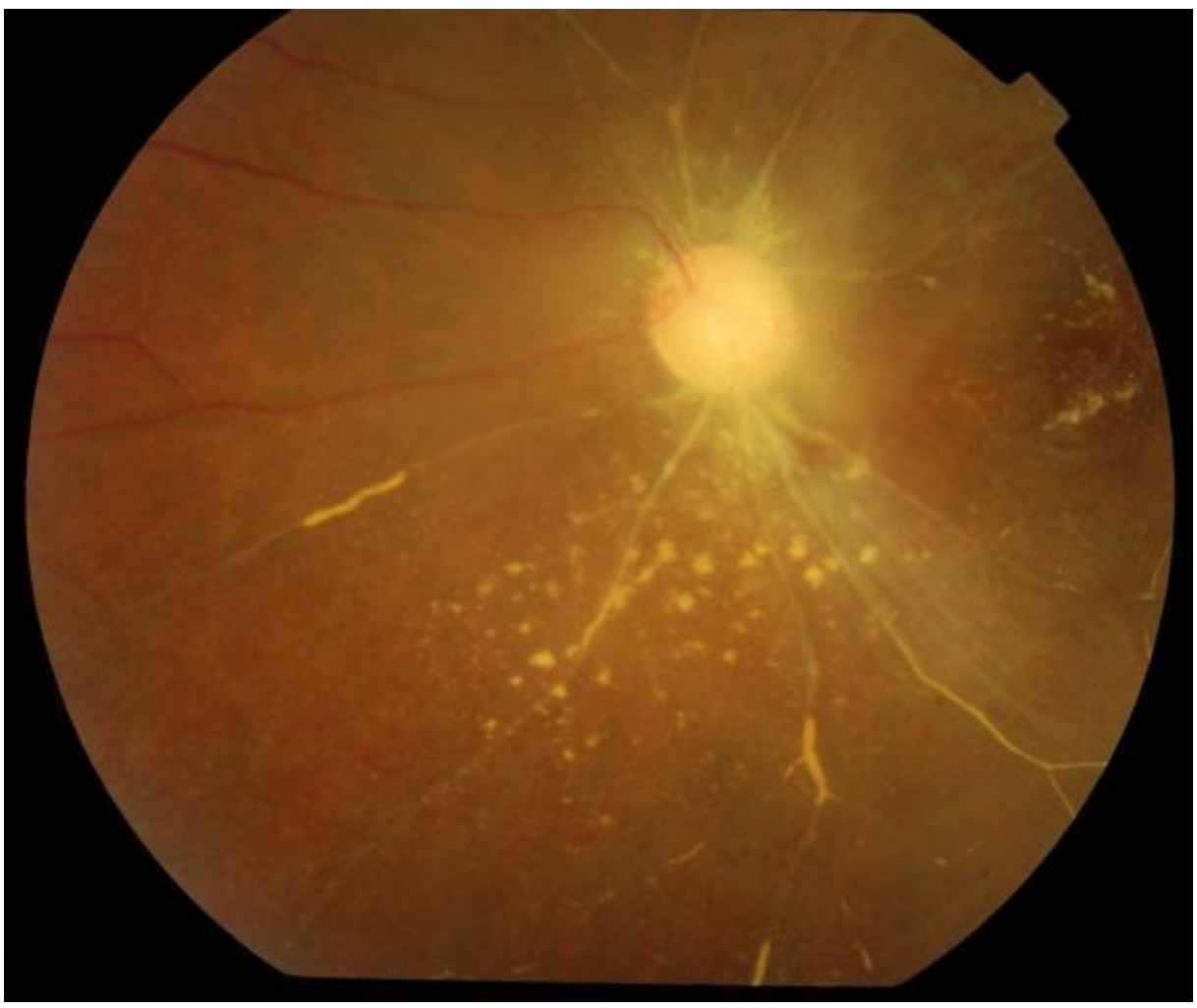


FIG-13



SYSTEMIC LUPUS ERYTHEMATOSUS

It’s an immune complex disorder, which affects the eye with severe intraocular complication .Ocular involvement is common in the form of dryness.

In 7.5% of the patients with SLE retinal vasculopathy is mostly as microangiopathic changes with cotton wool spots.

Cotton wool spots and retinal hemorrhages correlate with disease activity and are of negative prognostic sign.

SLE patients with anti-phospholipid syndrome have increased risk for retinal vasoocclusive disease.

The pathogenesis of SLE are complex and partially understood, include triggering agent such as infection might lead to activation of autoreactive T and B cells

Treatment of microangiopathic changes should be chosen in the context of systemic disease. Remission is induced with intravenous cyclophosphamide and maintained with other immunosuppresants like azathioprine, Mycophenolate mofetil and Rituximab are emerging therapies for SLE.(FIG-14,15 &16)

FIG-14 SLE PERIARTERITIS-OD

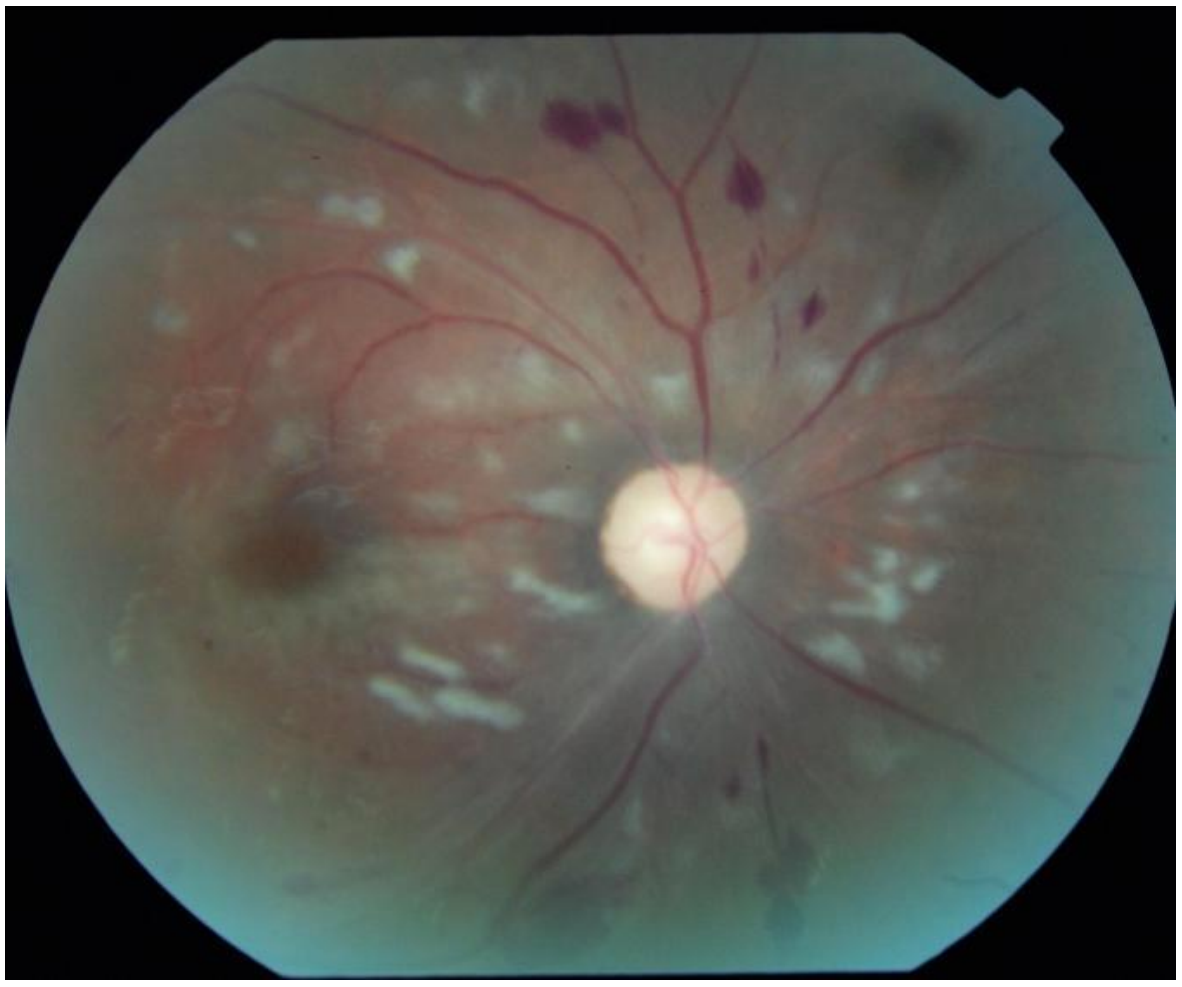


FIG-15 SLE PERIARTERITIS –OS

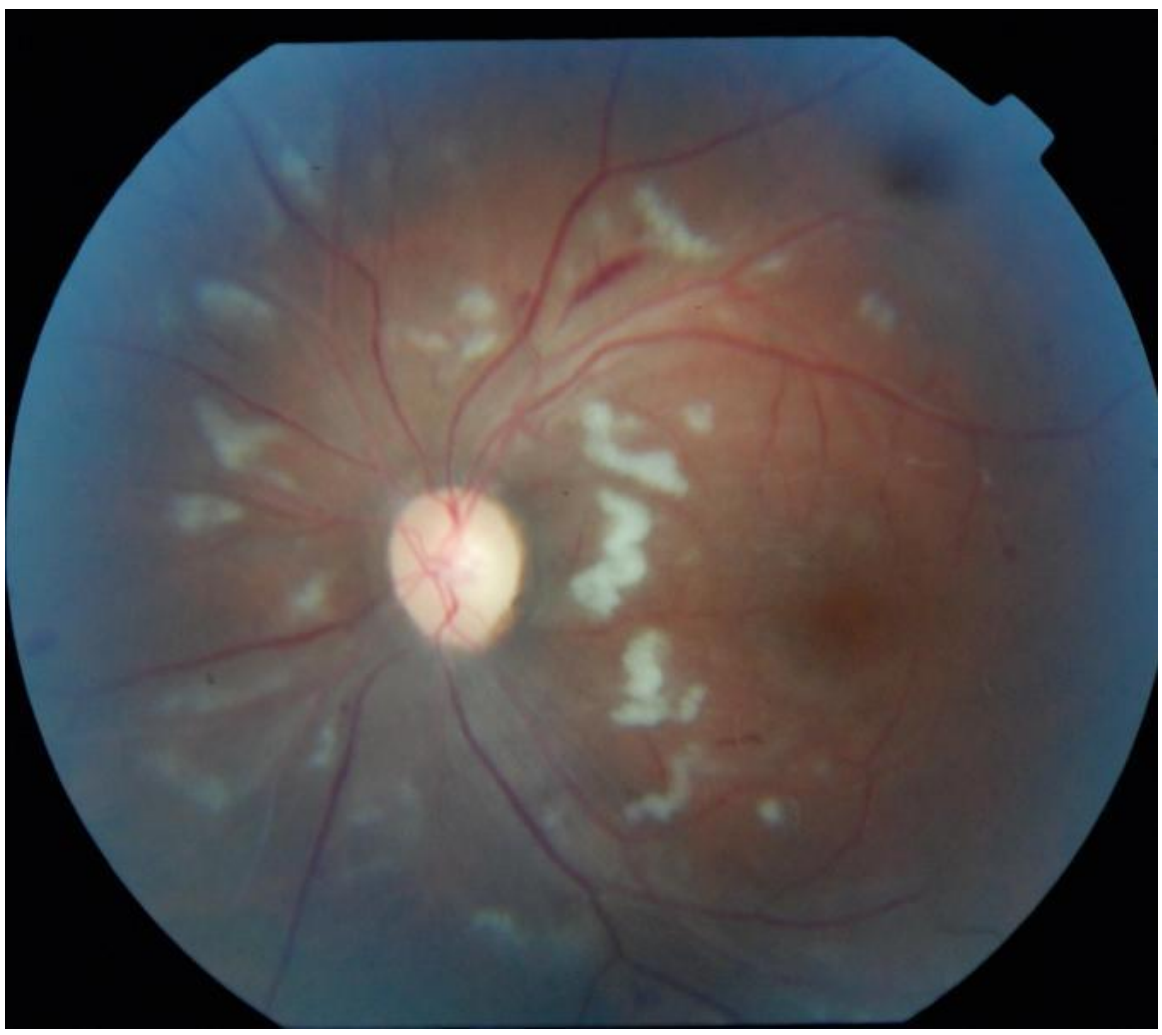
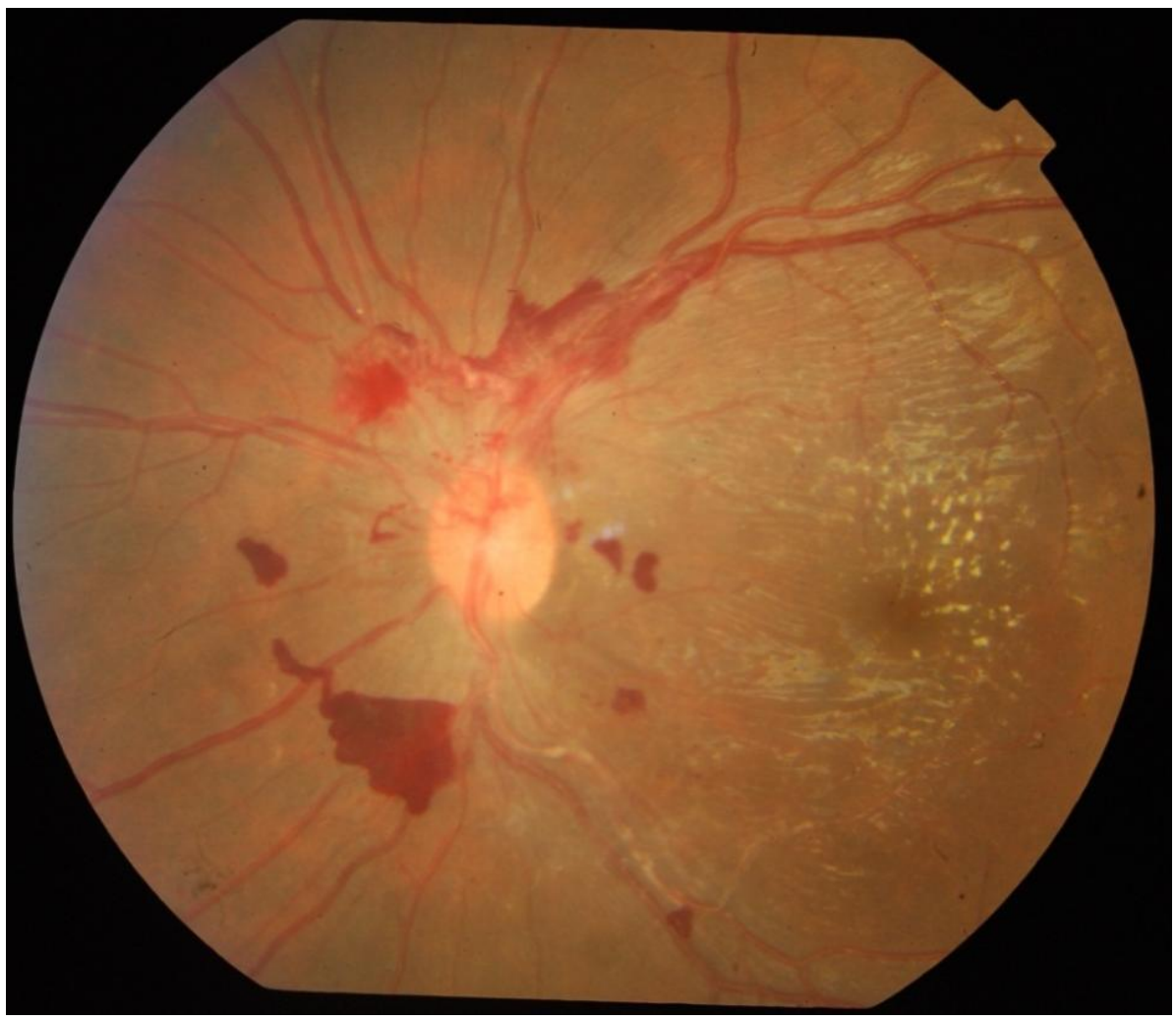


FIG-16 VASCULITIS DUE TO SYSTEMIC LUPUS ERYTHEMATOSUS



RETINAL VASCULITIS DUE TO INFECTIOUS DISORDER

Syphilis

Hepatitis C

Chickenpox

Toxoplasmosis

Toxocariasis

Coccidiomycosis

Tuberculosis (Fig-17)

Cytomegalovirus infection (Fig-18,19)

Herpes simplex

Herpes zoster

Acute retinal necrosis

Candidiasis

Leptospiosis

Rickettsia

Brucellosis

Amoebiasis

Mononucleosis

Lyme’s disease

Hepatitis B

FIG-17 POST-INFECTIVE VASCULITIS

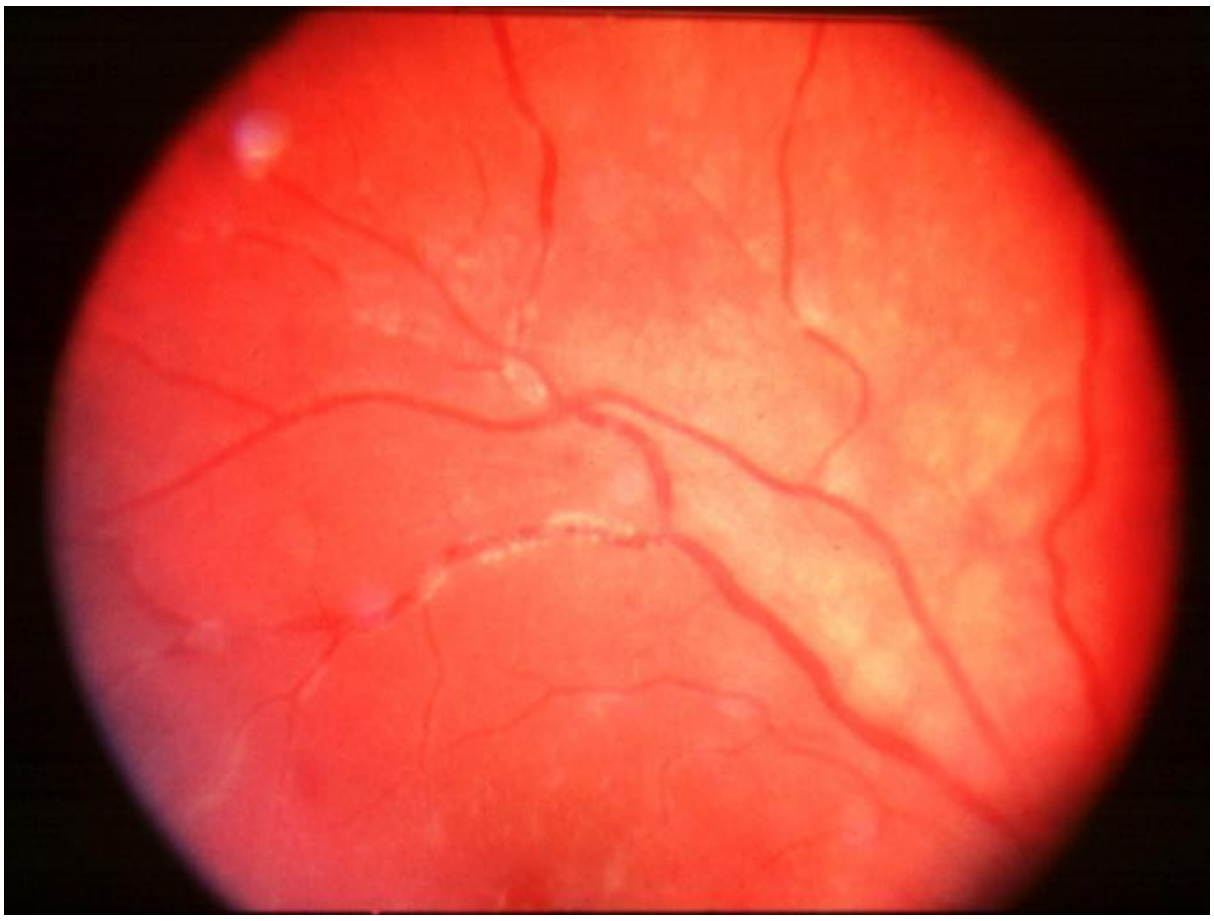


FIG-18 CMV RETINITIS

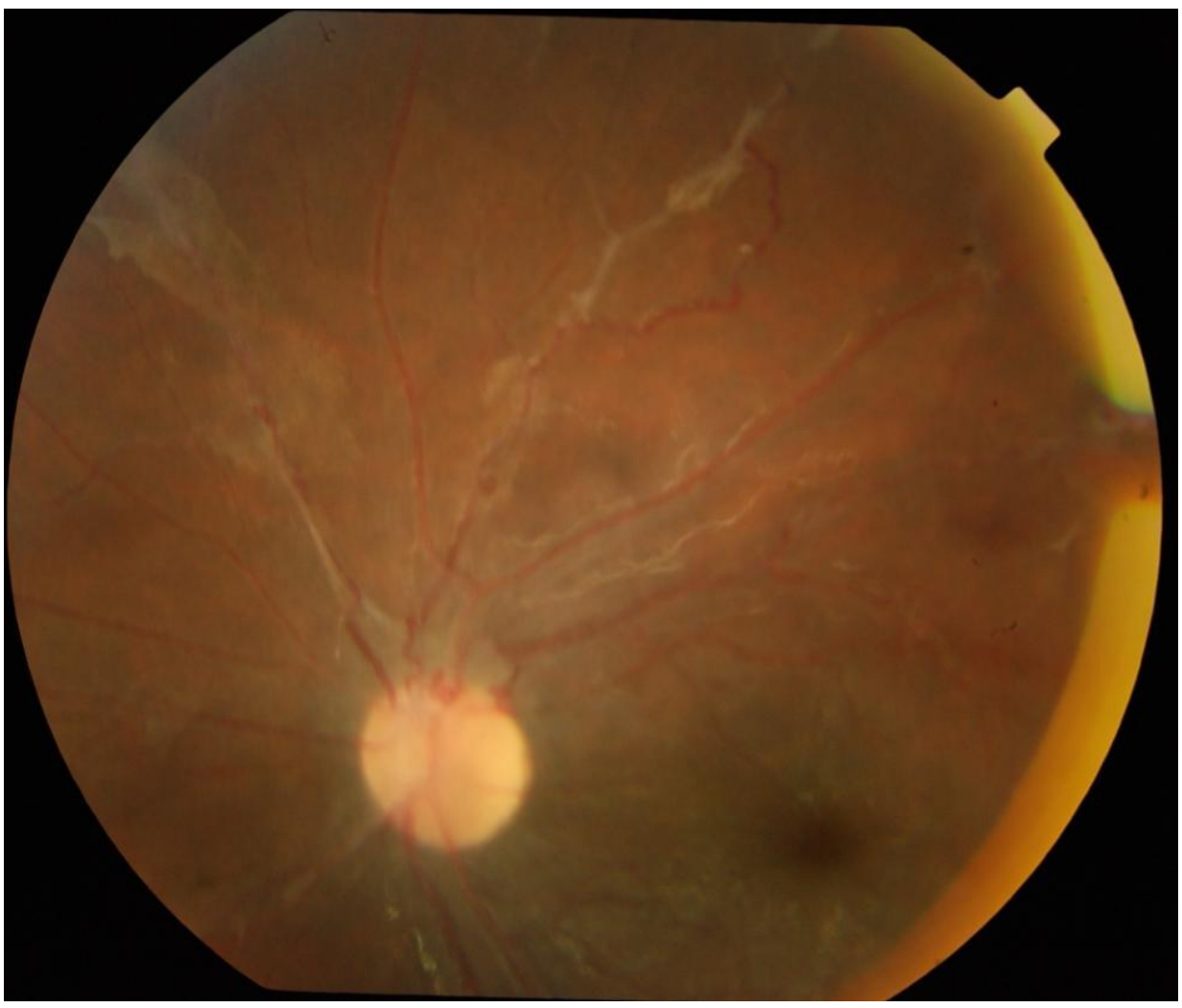
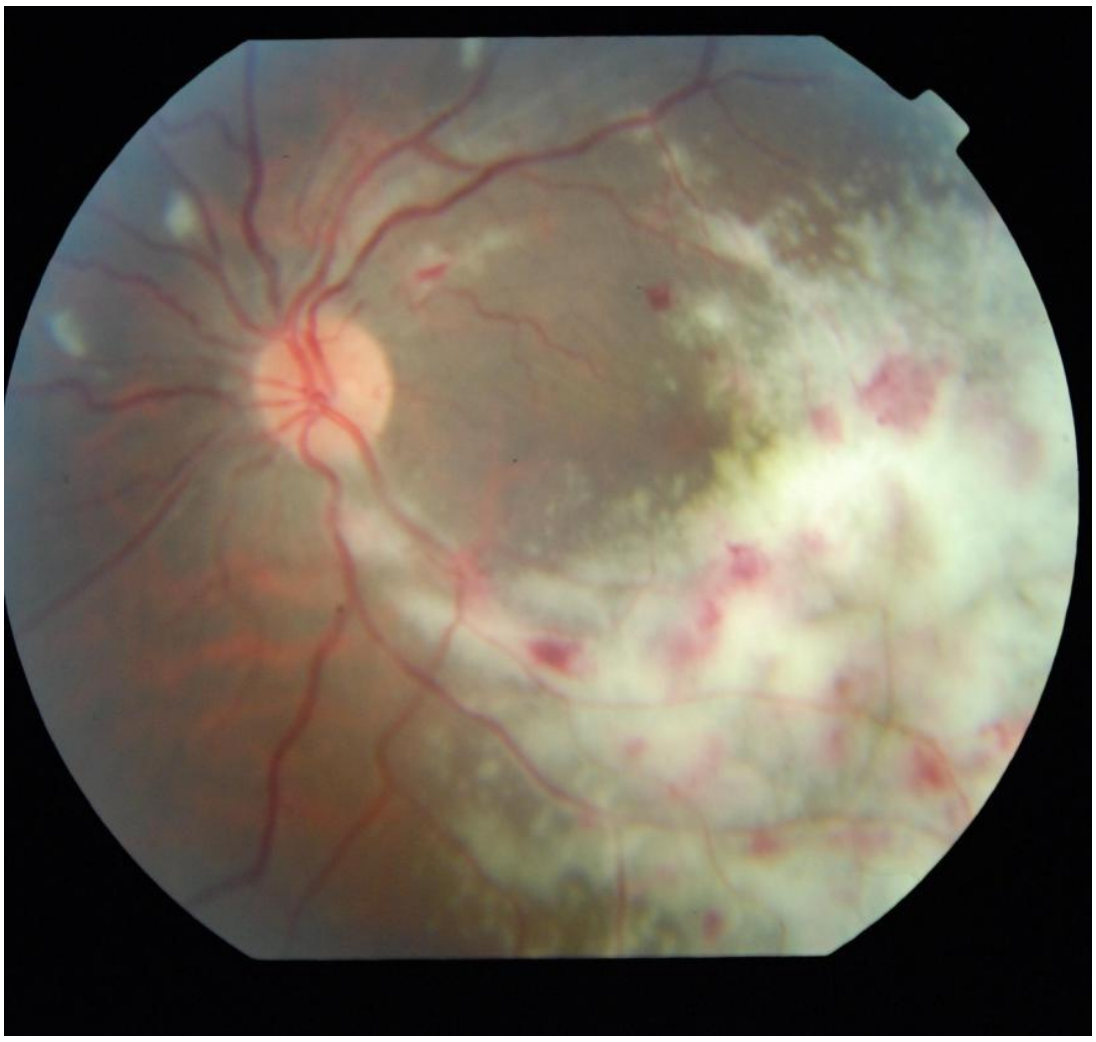


FIG-19 CMV RETINITIS



VASCULITIS DUE TO OCULAR DISEASES

Pars planitis

Choroiditis

Posterior uveitis

Behcet’s retina sine systemic disease

Birdshot retinochoroidopathy

Acute multifocal hemorrhagic retinal vasculitis[25]

DIAGNOSTIC STUDIES IN PATIENTS WITH RETINAL VASCULITIS

LABORATORY TESTS

Complete blood count with differential count

Erythrocyte sedimentation rate

C-reactive protein

Serum chemistry panel with tests for retinal and liver function

Bloodsugar

Urinanalysis

VDRL and FTA-ABS

Tuberculin skin testing

TORCH Screening

Serum ACE

Rheumatoid factor

Antinuclear antibody

Antineutrophil cytoplasmic antibody

Antiphospholipid antibody

Serum protein electrophoresis

Serum cryoglobulins

Human leukocyte antigen testing

Vitreous biopsy

CSF Cytology and cell count

IMAGING

Fluorescein angiography

Optical Coherence Tomography

Ultrasonography

Chest X-ray

Magnetic Resonance Imaging

Gallium scan

Sacroiliac joint x-ray.[26]

TREATMENT MODALITIES

ANTI-INFLAMMATORY

CORTICOSTEROIDS

Systemic route (oral or intravenous administration)

Sub-tenon’s injection

Intravitreal Triamcinalone

Intravitreal implants

IMMUNOMODULATORS

Alkylating agents

Anti-metabolite

T-lympocytes modulators

Biological response modifiers.

LASER THERAPHY

Pan-retinal photocoagulation

SURGICAL THERAPHY

Pars plana vitrectomy

CORTICOSTEROIDS

INDICATIONS

Treatment of active inflammation in the eye

Prevention and treatment of complication

Reduction of inflammatory infiltrates of retina, choroid or optic nerve.

SYSTEMIC THERAPY

Prednisalone is commonly used drug 1-2mg/kg/day of oral prednisalone given for period of 6-8wks and gradually tapered.

In cases of severe non-infectious posterior uveitis and panuveitis pulse dose of steroids 1gm/day infused over 1 hour administered for 3 days followed by gradual taper of oral steroids.

Complications of oral steroids

FLUIDS,ELECTROLYTES

Sodium retention, potassium loss

Fluid retention

Hypokalemic alkalosis

Hyperosmolar coma

MUSCULOSKELETAL

Muscle weakness

Steroid myopathy

Osteoporosis

Aseptic necrosis of femoral and humeral head

Tendon rupture

GASTROINTESTINAL

Nausea

Increased appetite

Peptic ulcer

Perforation of small and large bowel

Pancreatitis

DERMATOLOGIC

Poor wound healing

Easy brusiability

NEUROLOGICAL

Convulsion

Headache

Hyperexcitability

Moodiness

Psychosis

ENDOCRINE

Menstrual irregularities

Cushingoid state

Suppression of adrenocortical pituitary axis

Diabetes

OPHTHALMIC

Cataract

Glaucoma

Central serous retinopathy

Activation of herpes

OTHERS

Weight gain

Thromboembolism

SUB-TENON INJECTION

INDICATIONS

Posterior uveitis

Active stage of Retinal vasculitis

Non-complaint

Poor response to topical /systemic administration

METHOD OF INJECTION

Under topical anesthesia, using 25 gauge, 5/8 inch needle injection given in the superotemporal or inferotemporal quadrant.

COMPLICATION

Upperlid ptosis

Globe perforation

Periorbital hemorrhage.

Glaucoma

Cataract.

Newer approach of local steroid injection include Trans Tenon steroid injection.[27]

PART TWO

AIM OF THE STUDY

PRIMARY OBJECTIVE

- To classify the RETINAL VASCULITIS
- Compare the efficacy of Posterior sub tenon steroids and oral steroids in patients with active vasculitis.
- To assess the final visual outcome in the above groups after complete treatment with (Immunosuppresant/laser as per indication.)

SECONDARY OBJECTIVE

- To determine epidemiological factors(age/sex), mode of presentation

MATERIALS AND METHODS

This study was conducted in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Egmore, Chennai from November 2009 to November 2011 for a period of 2 years.Its a single blind randomised prospective and retrospective interventional clinical study.

INCLUSION CRITERIA:

All cases of retinal vasculitis associated with ocular or systemic disease Criteria for diagnosis of retinal vasculitis

One of following

1. Sheathing,
2. Para-vascular edema,
3. Peri-vascular inflammation,
4. Staining of vessel wall in FFA.

EXCLUSION CRITERIA:

1. Patients with contraindication for steroids,
2. Patient with scleral necrosis,
3. Patient with history of glaucoma,

SCREENING PROCEDURE

1. History,
2. Visual acuity,
3. Intra-ocular pressure,

4. Slit lamp examination,
5. Fundus examination with90D & IDO,
6. FFA ,
7. complete hemogram,ESR,blood and urine sugar,mantoux,
8. VDRL,ELISA for HIV,
9. serum ace,chest x-ray,
10. anti-nuclear antibody,RF,anti-DNA,C-reactive protein,
11. TORCH titre
12. c-ANCA/p-ANCA

DATA COLLECTION AND METHODS

All new and review cases diagnosed as retinal vasculitis were subjected to above mentioned investigations and disease entity was classified based on the results of detailed evaluation.Total study population included 50 patients.Of which 24 patients were classified as primary vasculitis assigned for intervention study as Subgroups by randomization.

- Patients with primary vasculitis with acute disease were randomly divided into two subgroups
- Subgroup 1 treated with oral steroids(1mg/kg/day) for a period of 8 weeks. .

- Subgroup 2 with posterior sub-tenon steroids(20mg/0.5ml) given 2 wks once for 8 weeks
- Best corrected visual acuity treatment (snellen's chart) . Media clarity using indirect ophthalmoscope, Fundus fluorescein angiography if hemorrhages and media view was possible was perfomed every week upto 8 weeks period
- Immunosuppressants (Azathioprine 2mg/kg/day) planned for resistant cases (not responding to steroids treatment for period of 8 weeks)in the above two groups,
- Patient treated with laser therapy pan retinal photocoagulation for those associated with neovascularistaion and extensive capillary non -perfusion
- Follow up was done every week upto 8^{th} , 12^{th} and 16^{th} weeks of treatment .Results analysed at end of 16^{th} week.

Assessment of parameters during follow up visits

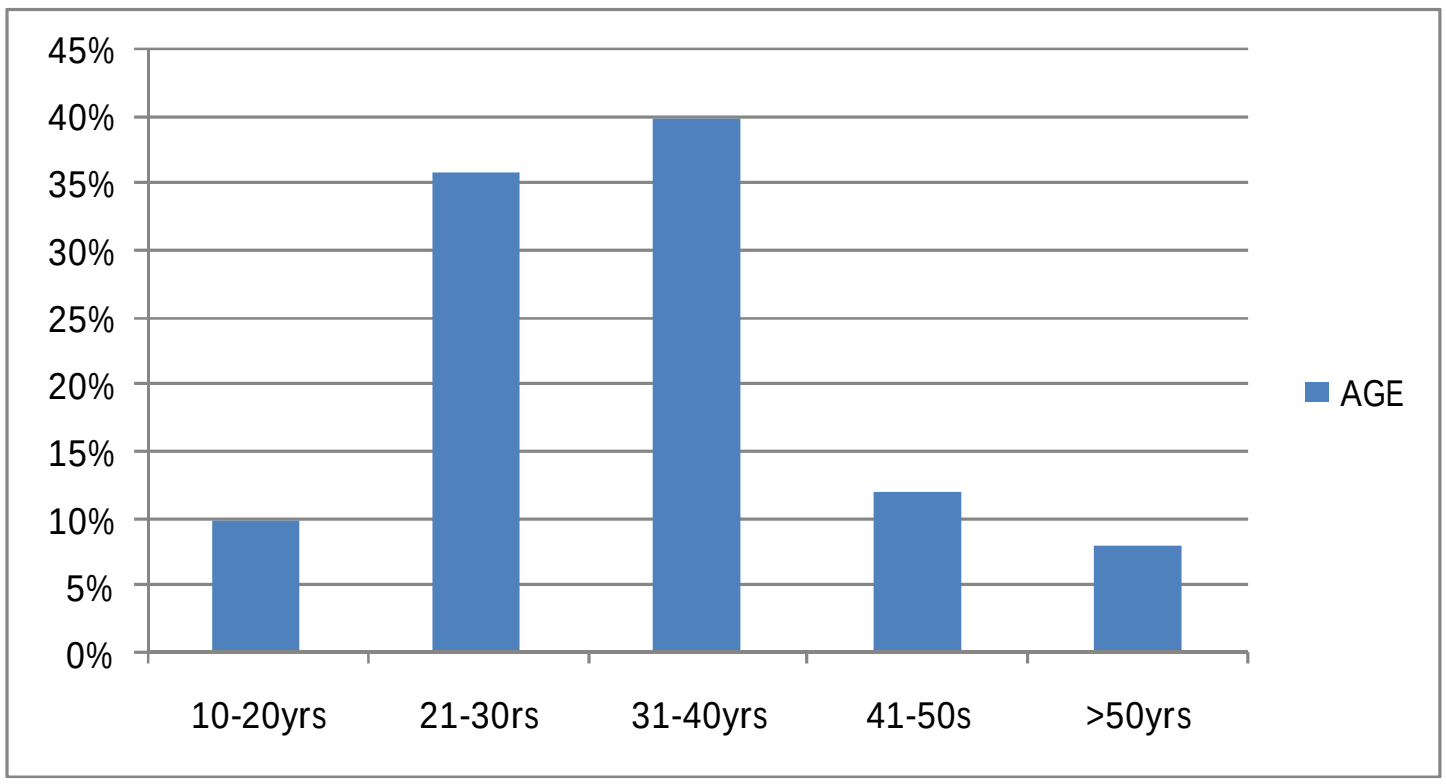
1. Best corrected visual acuity before & after treatment (snellen's chart) .
2. Media clarity using indirect ophthalmoscope
3 Fundus fluorescein angiography if hemorrhages and media view was possible

OBSERVATION AND ANALYSIS

1. AGE DISTRIBUTION

TABLE-1

Age Group (Yrs)	No. of Cases	Percentage
10-20	2	10%
21 – 30	18	36%
31 – 40	20	40%
41 – 50	6	12%
>50	4	8%

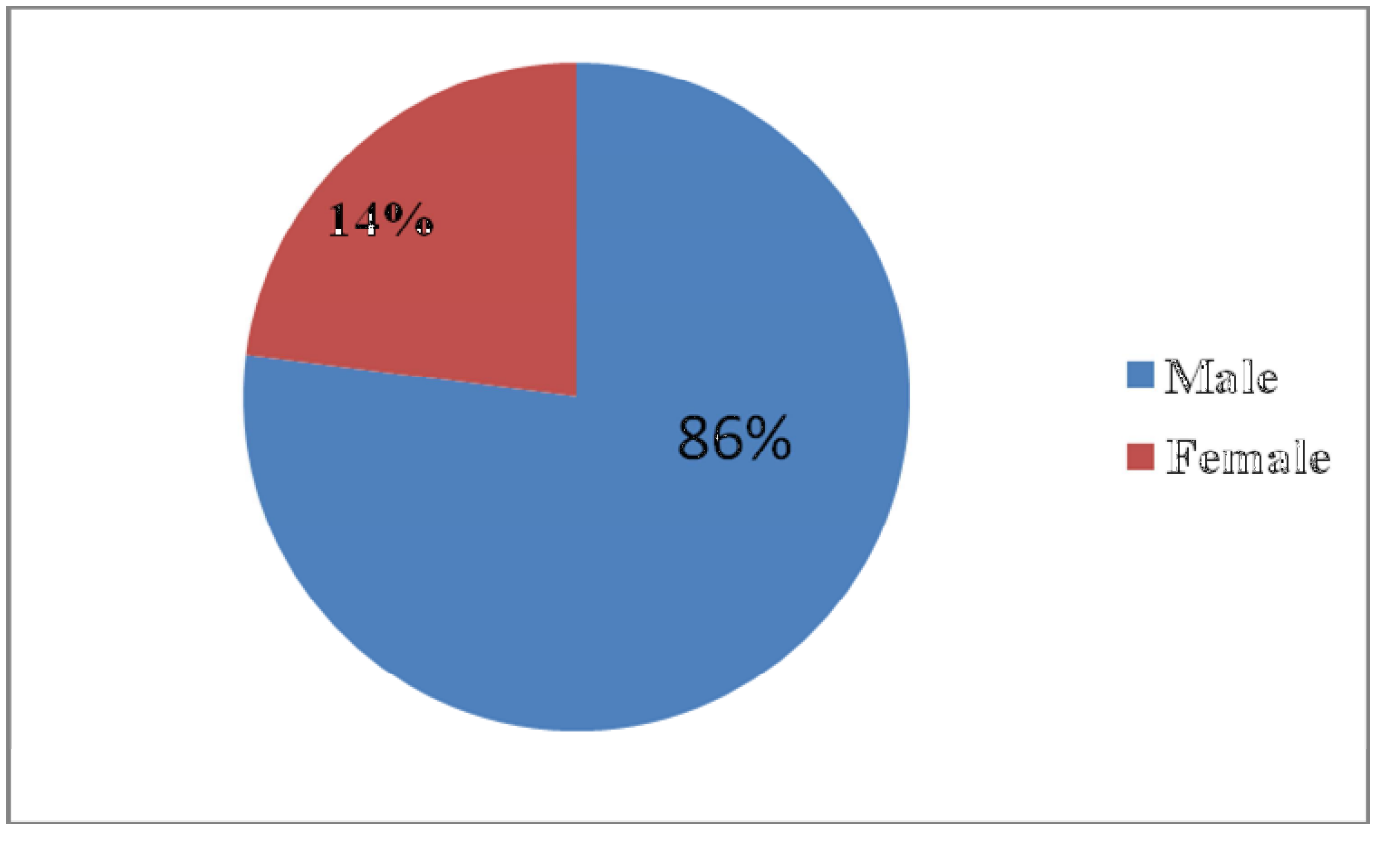


Incidence of Retinal vasculitis is more in the age group of 31-40 years of age (40%) followed by age group of 21 to 30 (36%).

2. SEX DISTRIBUTION

TABLE-2

Sex	No. of Cases	Percentage
Male	43	86%
Female	7	14%

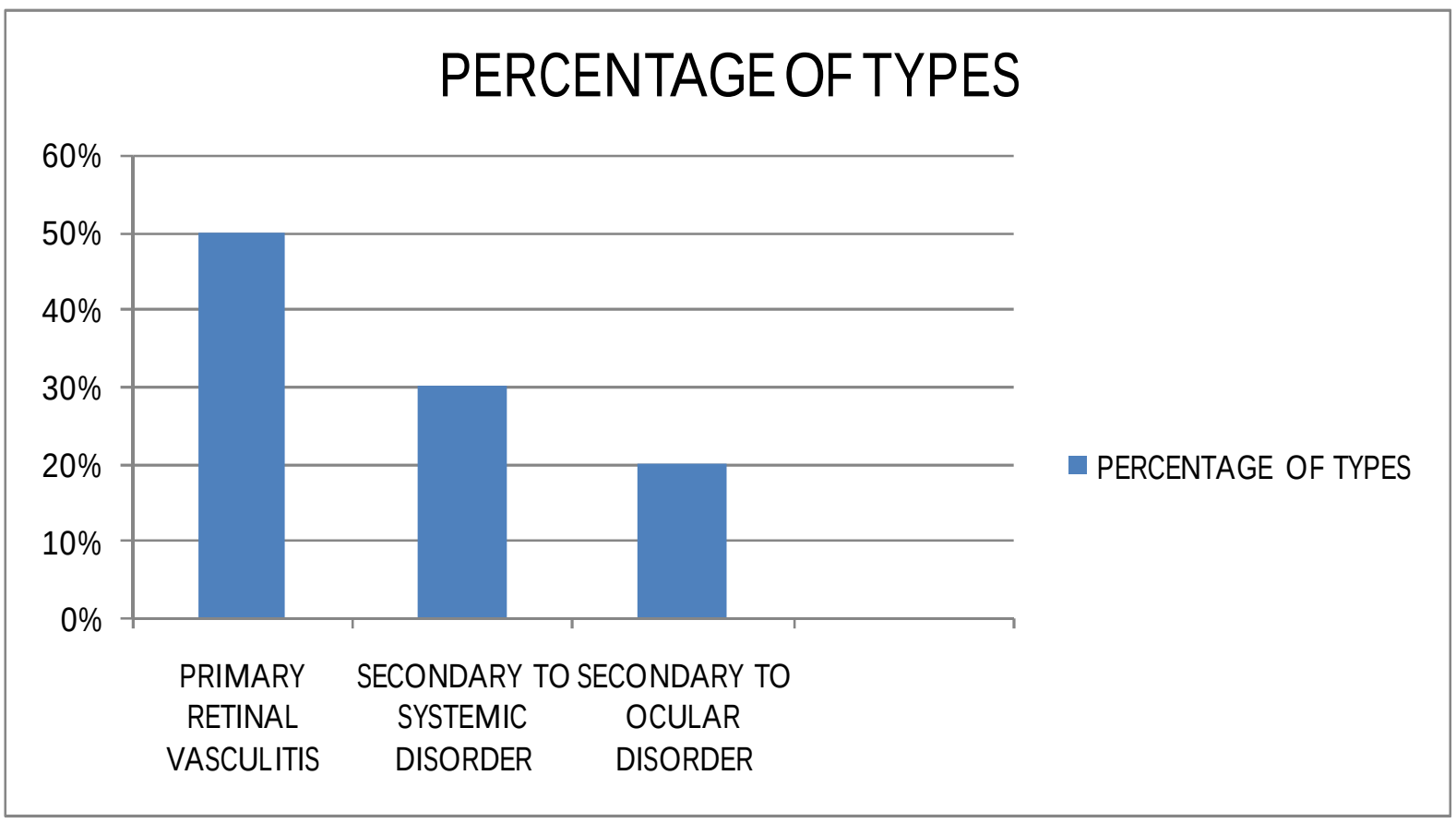


In our study, males were predominantly affected (86%).All Eales patient were Male. In patients with systemic disease 5 patient among the 15 were female and remaining 10 were male. All 10 patients with ocular disease were male.

3. CLASSIFICATION OF RETINAL VASCULITIS

TABLE-3

DISEASE	NO	PERCENTAGE
PRIMARY RETINAL VASCULITIS	25	50%
VASCULITIS SECONDARY TO OCULAR DISEASE	10	20%
VASCULITIS SECONDARY TO SYSTEMIC DISEASE	15	30%

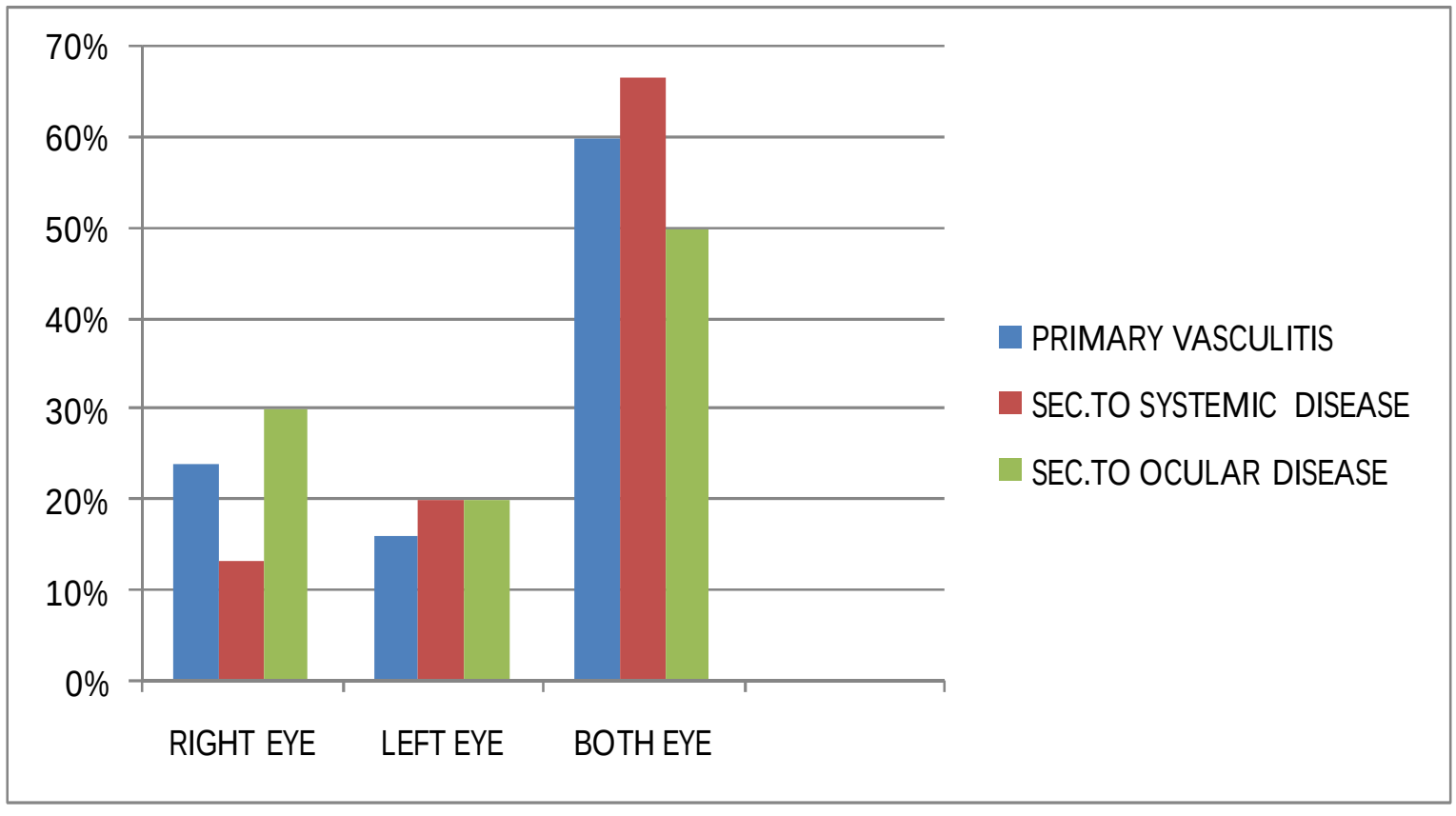


In our study primary vasculitis was predominant, this was followed by vasculitis secondary to systemic disease followed by vasculitis due to ocular disease.

4. LATERALITY

TABLE-4

LATERALITY	PRIMARY VASCULITIS	SEC.TO SYSTEMIC DISEASE	SEC.TO OCULAR DISEASE
RIGHT EYE	6(24%)	2(13.33%)	3(30%)
LEFT EYE	4(16%)	3(20%)	2(20%)
BOTH EYE	15(60%)	10(66.66%)	5(50%)

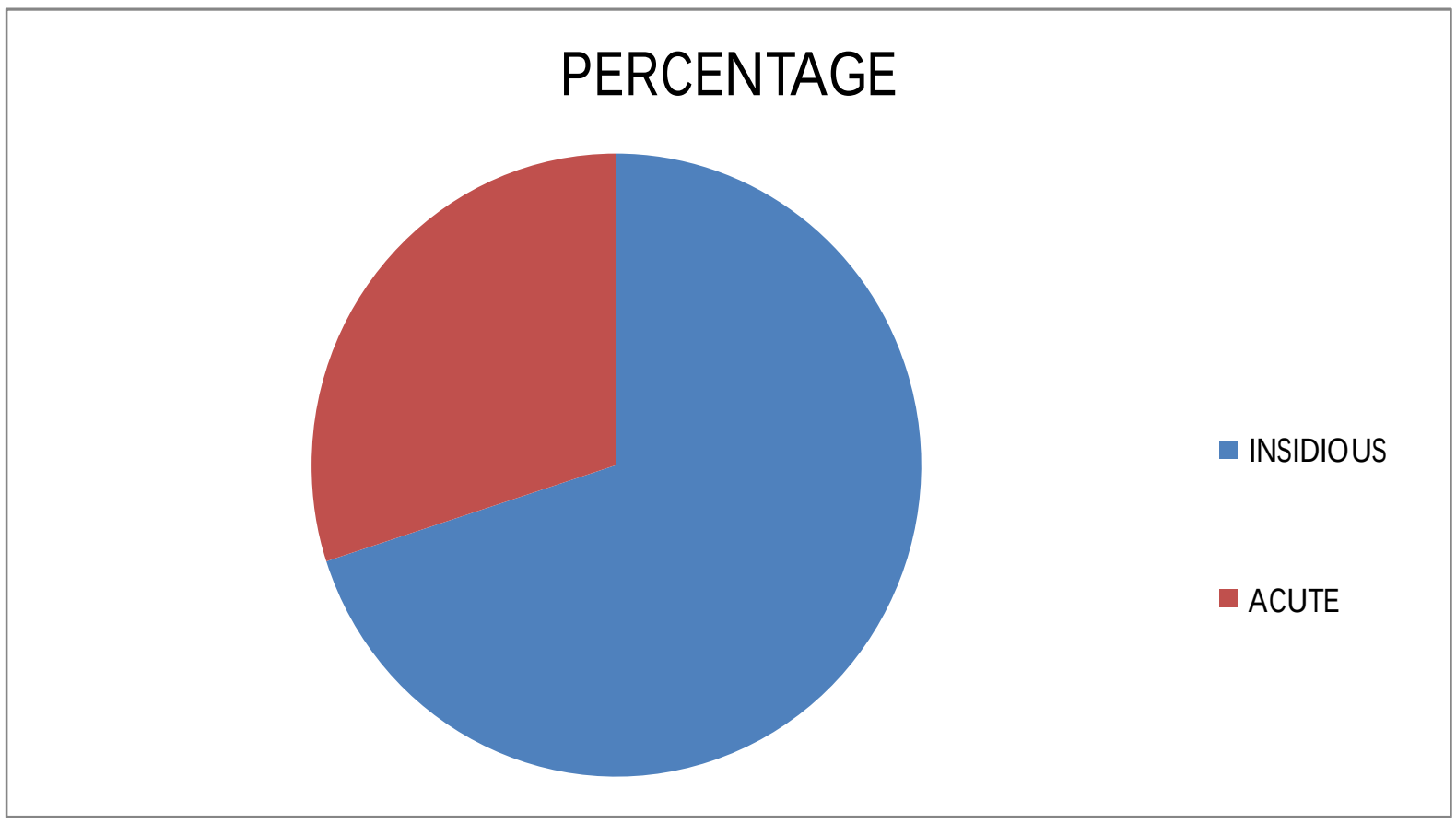


In our study patients with eales and vasculitis due to systemic disease had predominant bilateral involvement.Patient with ocular disease had equal unilateral and bilateral involvement.

5. MODE OF PRESENTATION

TABLE-5

MODE OF PRESENTATION	NO	PERCENTAGE
ACUTE	15	30%
CHRONIC	35	70%

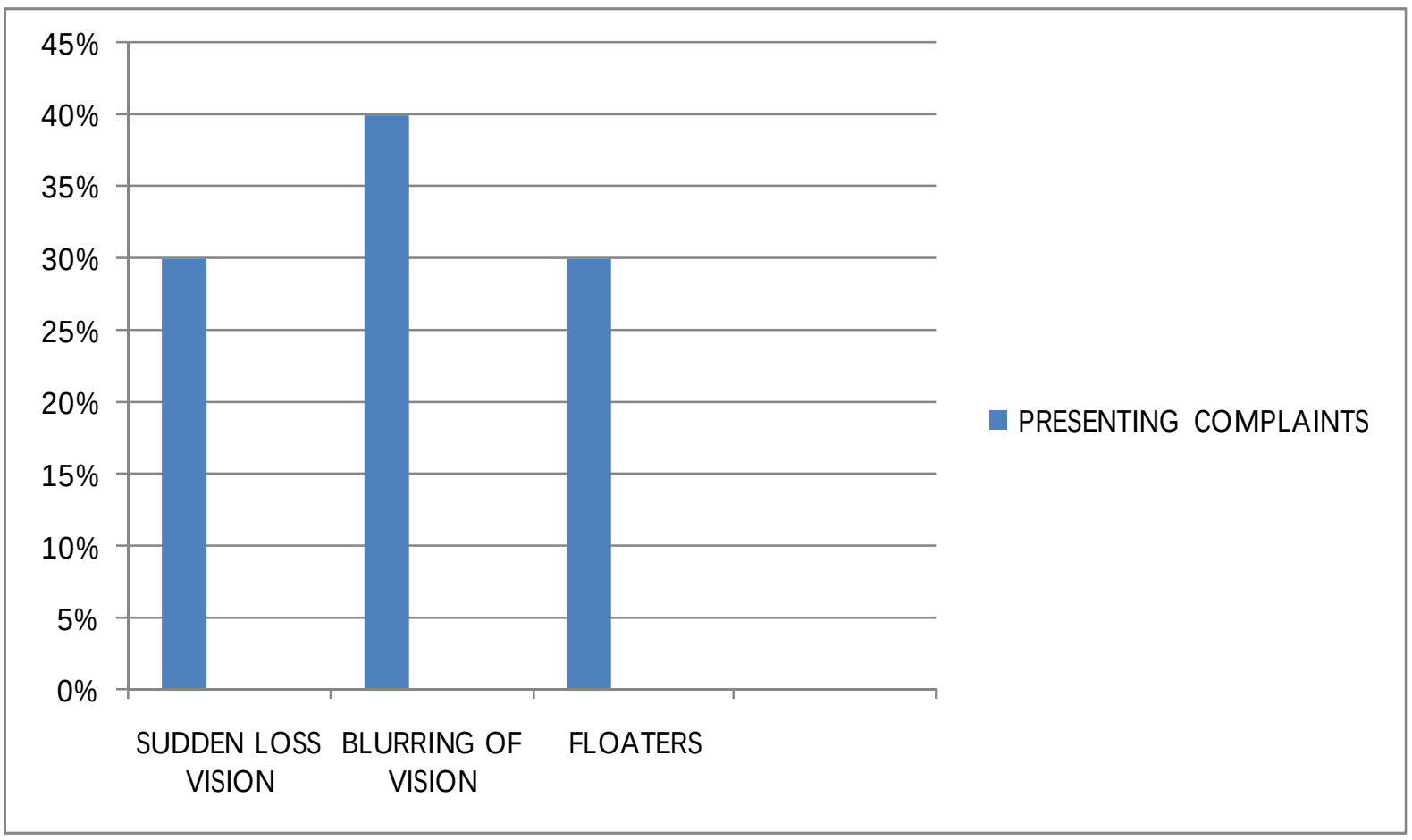


Acute onset of visual loss was common in patients with Eales disease due to vitreous hemorrhage

6. PRESENTING COMPLAINTS

.

PRESENTING COMPLAINTS	NO	PERCENTAGE
SUDDEN LOSS OF VISION	15	30%
BLURRING OF VISION WITH FLOATERS	20	40%
FLOATERS	15	30%

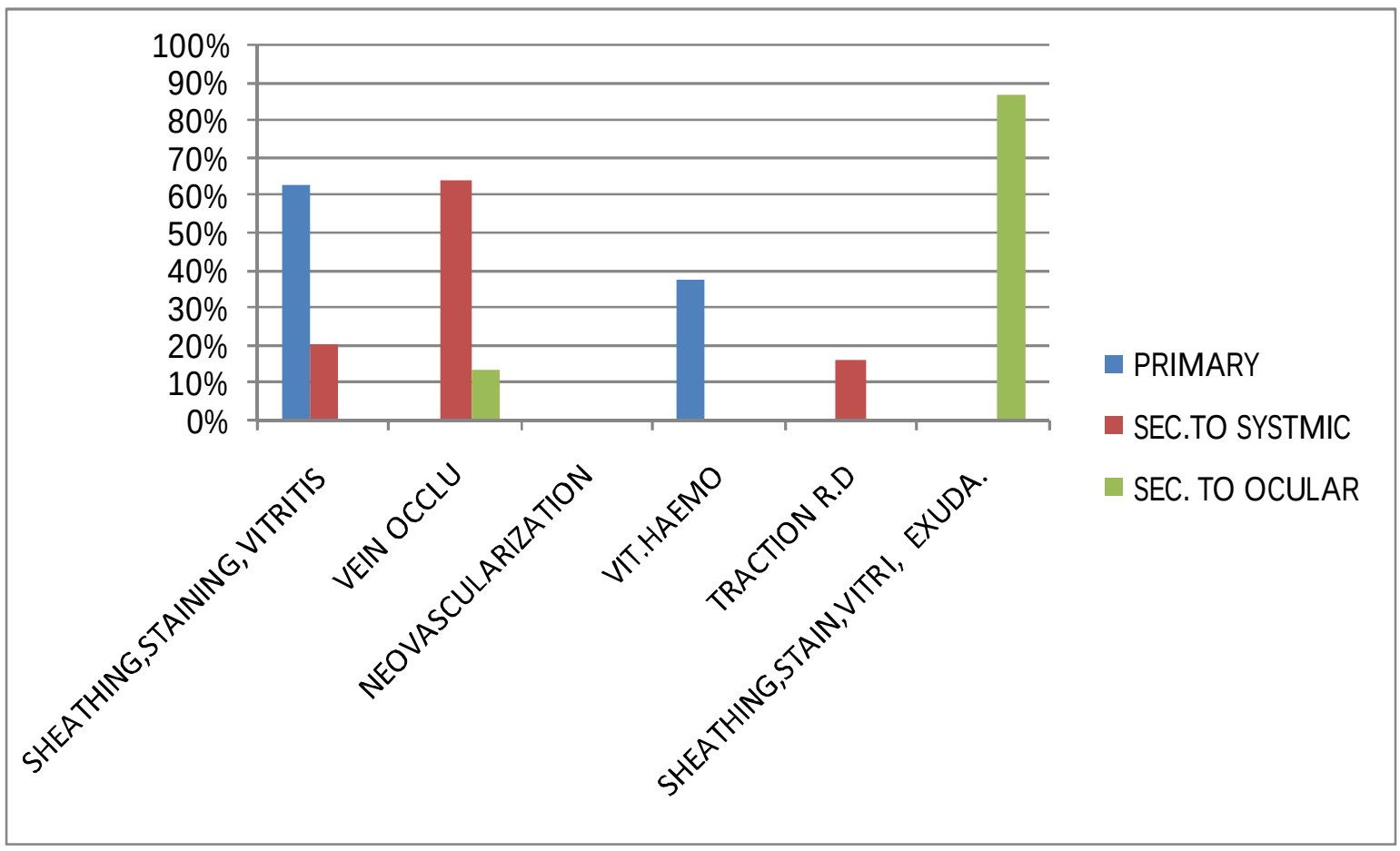


Blurring of vision was the predominant symptom and was associated with floaters. Sudden loss of vision was seen in patients with Eales with vitreous hemorrhage.

7. CLINICAL SIGNS

TABLE-7

CLINICAL SIGNS	PRIMARY VASCULITIS	SEC.TO SYSTEMIC DISORDERS	SEC.TO OCULAR DISORDERS
Sheathing of vessels with staining and vitritis	25 EYES (62.50%)	5 EYES (20%)	
Vein occulsion		16 EYES (64%)	2 EYES (13.33%)
Neovascularisation	0		
Vitreous hemorrhage	15 EYES (37.5%)		
Tractional retinal detachment		4 EYES (16%0	
Sheathing with exudation and vitritis			13 EYES (86.66%)

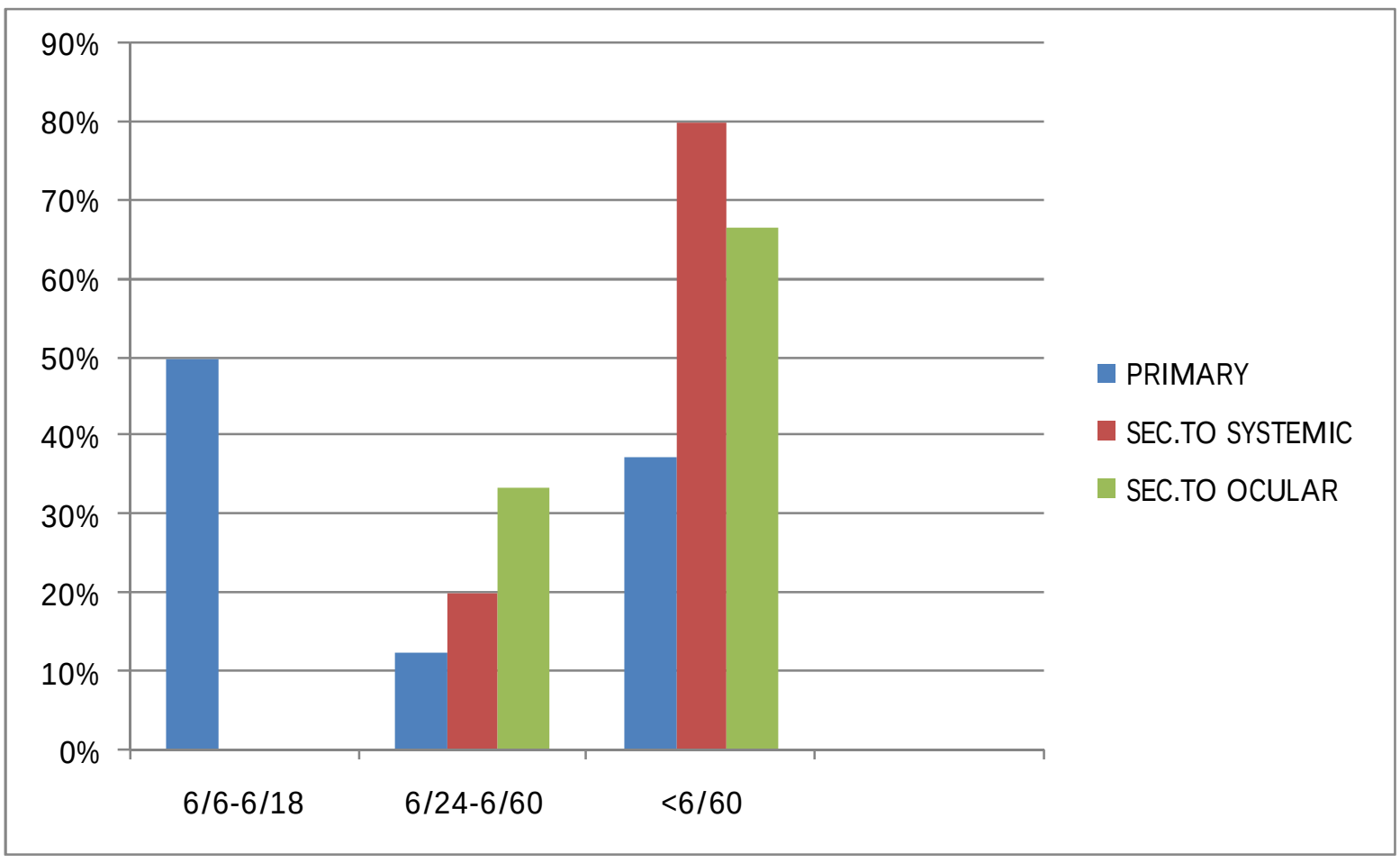


Sheathing of vessels with vitritis and staining was seen in all patients with acute disease due to Eales and ocular disease like posterior uveitis.Vitreous hemorrhage and Neovascularisation was present in patients with Eales disease.Vein occlusion present in patients with systemic disease.Tractional Retinal Detachment was noted in 2 patients with AIDS.

8. BEST CORRECTED VISUAL ACUITY

TABLE-8

VISUAL ACUITY	PRIMARY VASCULITIS	SEC.TO SSTEMIC DISEASE	SEC.TO OCULAR DISEASE
6/6-6/18	20 EYES (50%)		
6/24-6/60	5 EYES (12.5%)	5 EYES (20%)	5 EYES (33.33%)
<6/60	15 EYES (37.5%)	20 EYES (80%)	10 EYES (66.66%)



Visual acuity of <6/60 was seen in patients with vitreous hemorrhage (among Eales patient),with AIDS (among patients with vasculitis due to systemic disease), In other patient with systemic disease visual acuity ranged from 6/24-6/60 .6/6-6/18 was noted in patients with Eales .

MANTOUX RESPONSE IN PATIENT WITH EALES

Of the 25 cases of Eales 5 cases had mantoux positivity,of these patient screened for pulmonary tuberculosis one patient was positive for pulmonary tuberculosis and was excluded from the intervention

TREATMENT

24 Cases (24 eyes with active inflammation) of Eales patient randomly divided into 2 Subgroups.

Subgroup1 subjected to systemic steroids of 1mg / kg / day for 8week,

Subgroup2 subjected to posterior sub-tenons 20mg / 0.5ml once every 2 wks for 8weeks,

Both groups observed at 4week and 8 week,12th week and 16th week.

Parameters observed on follow up

Visual acuity

Vitritis grading (media clarity)

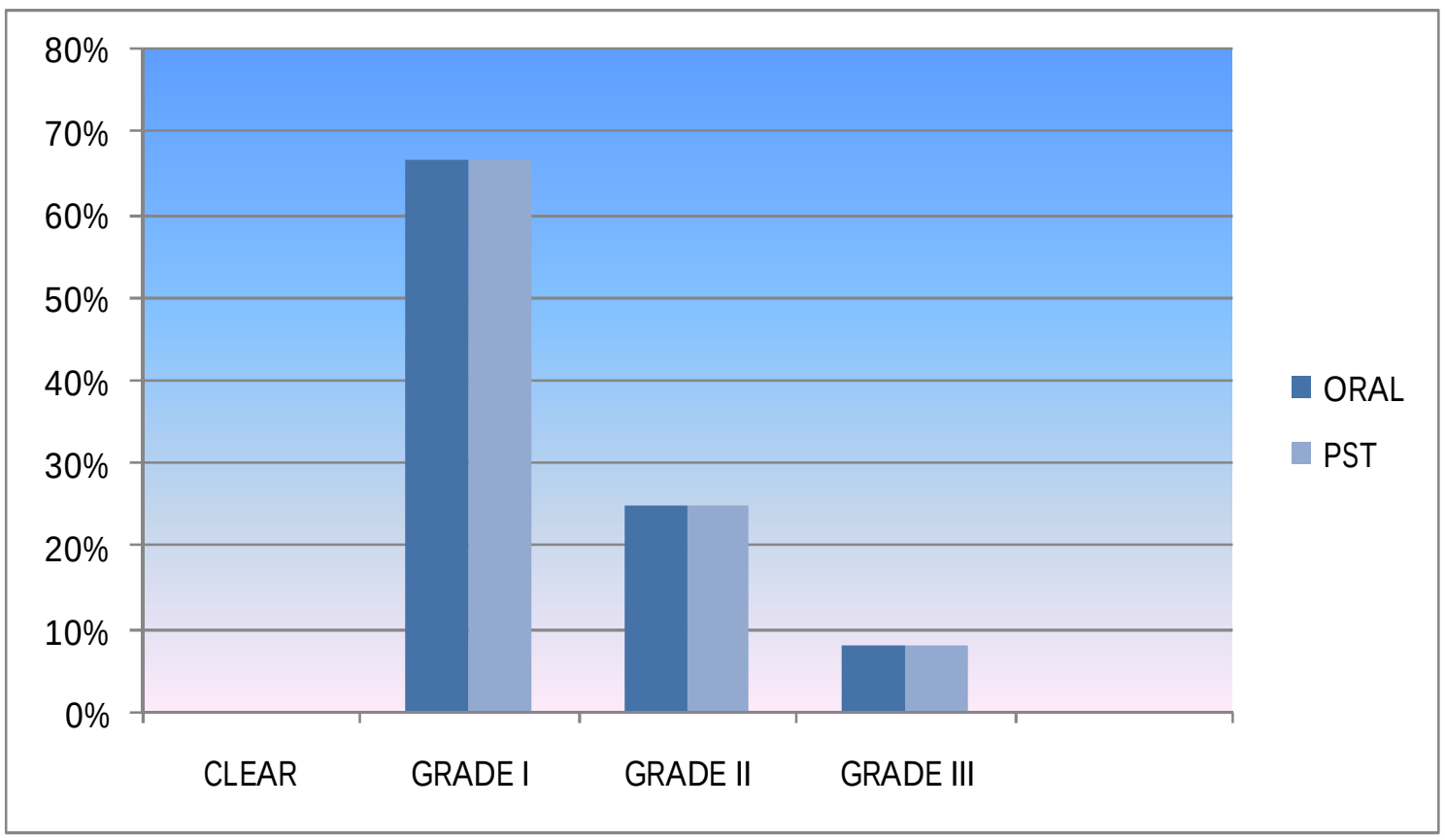
Fundus fluorescein angiography.

SUB-GROUP ANALYSIS

9. MEDIA CLARITY (VITRITIS) AT INITIATION OF TREATMENT

TABLE-9

MEDIA CLARITY VITRITIS(GRADE)	SUB-GROUP I ORAL	SUB-GROUP-II POST.SUB-TENON
0	0	0
I	8(66.67%)	8(66.67%)
II	4(25%)	4(25%)
III	0	0

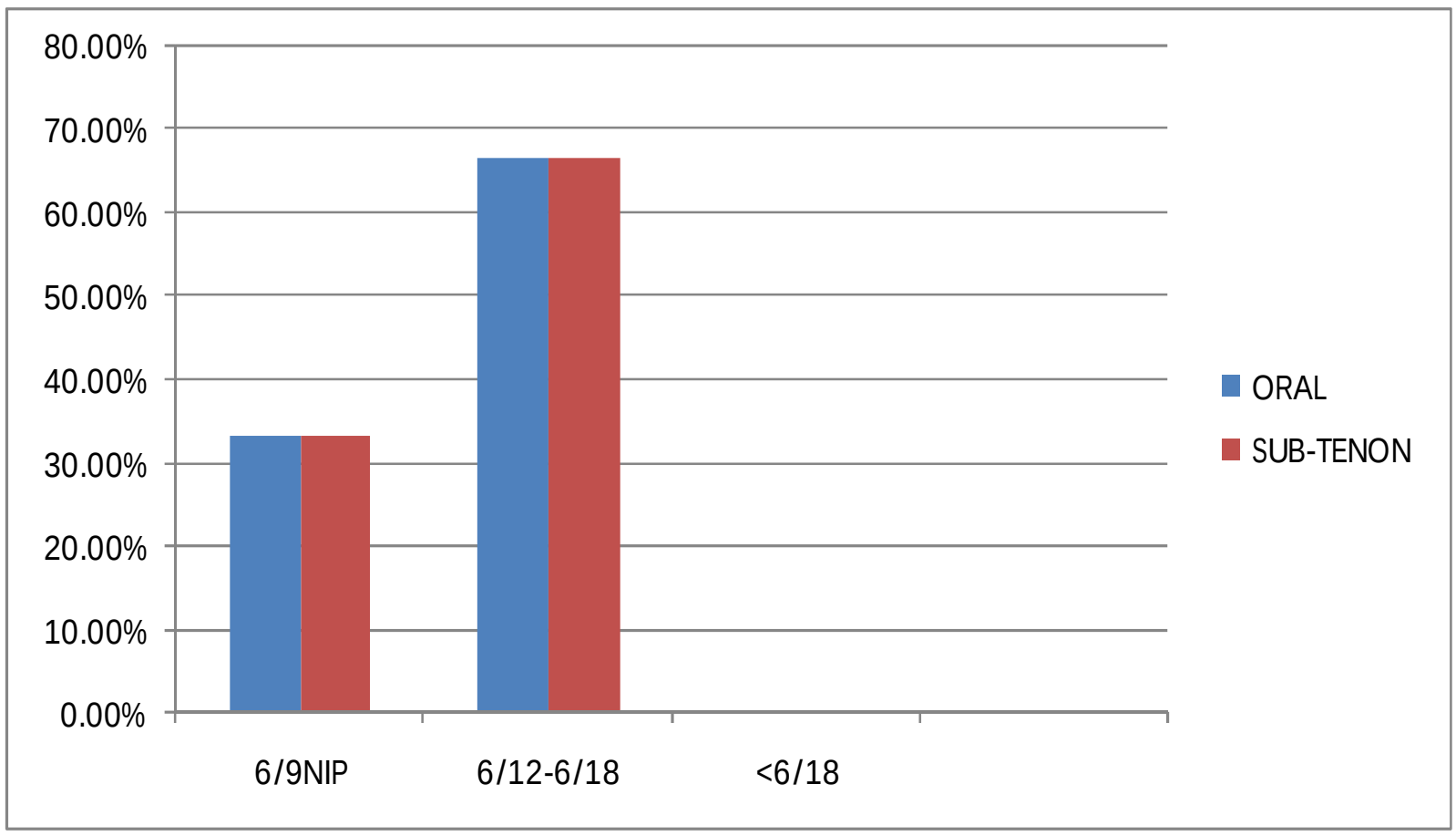


66.667% was had grade 1 vitritis at initiation of treatment in both the sub group.

10. VISUAL ACUITY AT INITIATION OF TREATMENT

TABLE-10

BCVA	SUB-GROUP- I ORAL	SUB-GROUP-II SUB-TENON
6/9NIP	4(33.33%)	4(33.33%)
6/12-6/18	8(66.67%)	8(66.67%)
<6/18	0	0

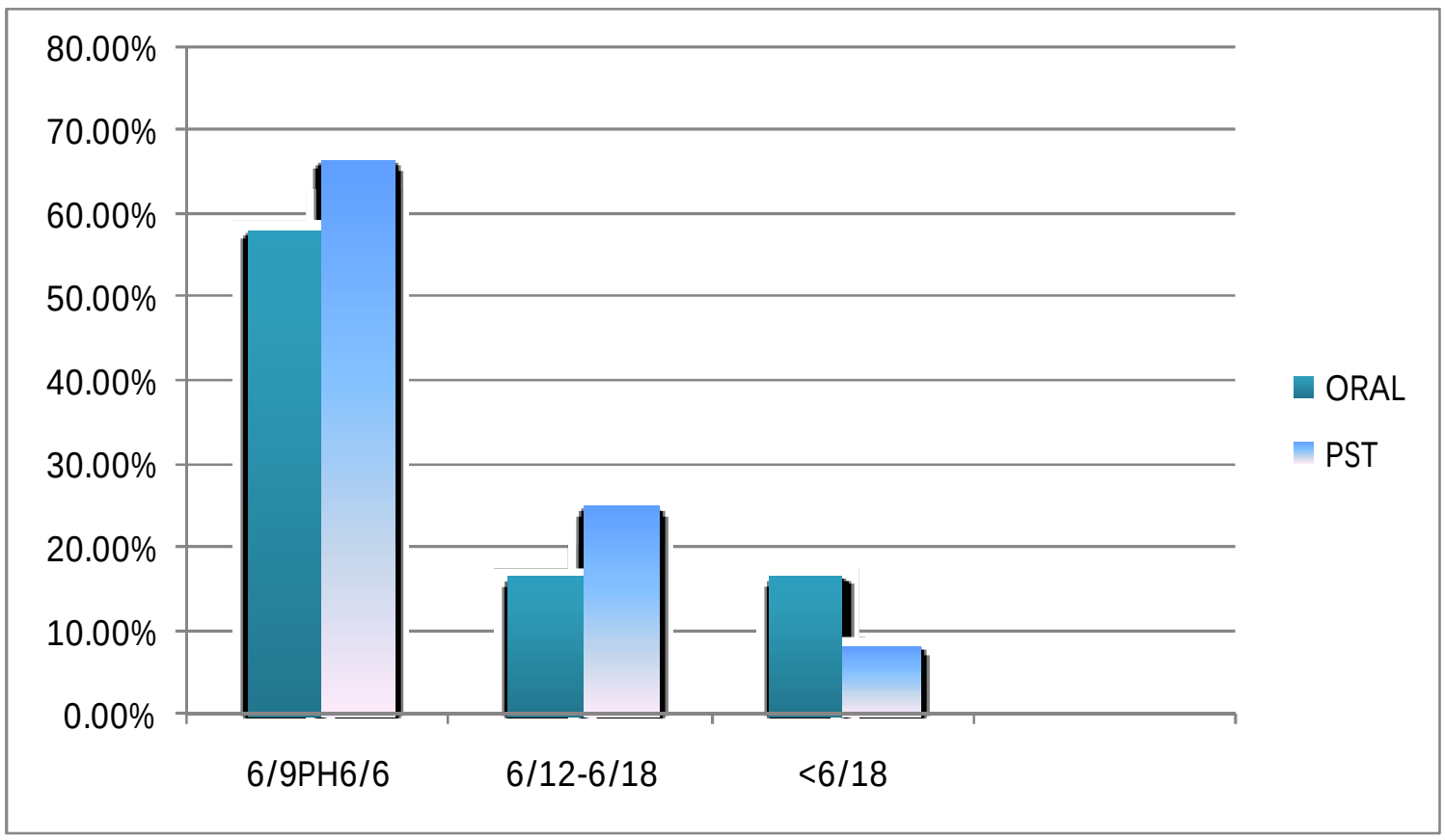


Best corrected visual acuity was 6/9NIP in 33.33%,In 66.67% visual acuity was between 6/12-6/18

11. VISUAL ACUITY AT 8 WEEKS

TABLE-11

BCVA	SUB-GROUP-I ORAL STEROIDS	SUB-GROUP-II POST.SUB TENON
6/9PH6/6	8(58.33%)	8(66.67%)
6/12-6/18	2(16.67%)	3(25%)
<6/18	2(16.67%)	1(8.33%)

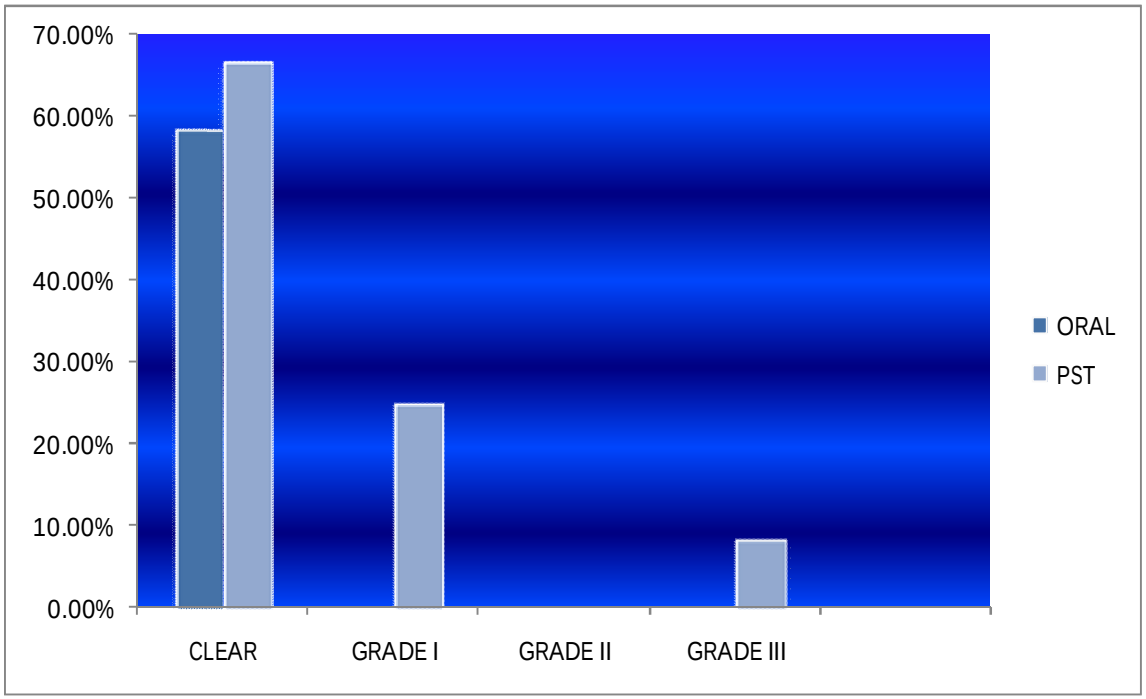


58.33% had BCVA of 6/6 at the end of 8 weeks in subgroup 1 and 66.67% had 6/6 in subgroup 2. In 16.67% of subgroup1 and 8.33% of subgroup 2 the visual acuity was persistently low <6/18

12. MEDIA CLARITY (VITRITIS) AT 8 WEEKS

TABLE-12

MEDIA CLARITY VITRITIS	SUB-GROUP-I ORAL	SUB-GROUP-II POST.SUB-TENON
CLEAR	7(58.33%)	8(66.66%)
I	3(25%)	3(25%)
II	2(16.67%)	1(8.33%)
III	0	0

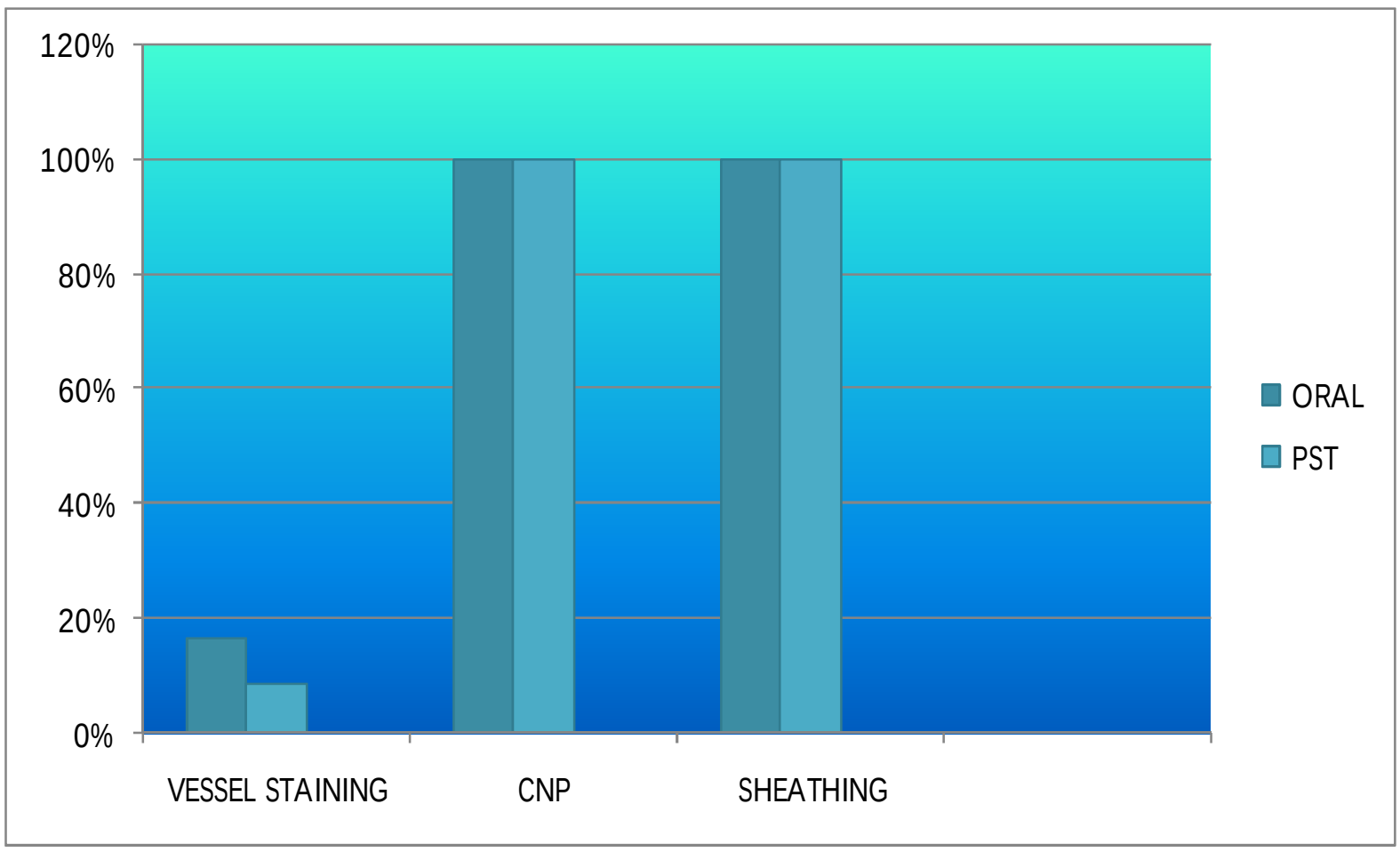


In 58.33% patient in the subgroup 1 media cleared at end of 8th week in subgroup 2 66.66% had clear view at end of 8th week. 16.67% of patient in subgroup 1 and 8.33% of patient in subgroup 2 were resistant to steroids and started on immunosuppressants.

13. FUNDUS FLUORESCEIN ANGIOGRAPHY AT 8TH WEEK

TABLE-13

FFA	SUB-GROUP-I ORAL	SUB-GROUPII POST.SUB-TENON
Vessel staining	2 (16.67%)	1 (8.33%)
Areas capillary non-perfusion	12 (100%)	12 (100%)
Sheathing	12 (100%)	12 (100%)

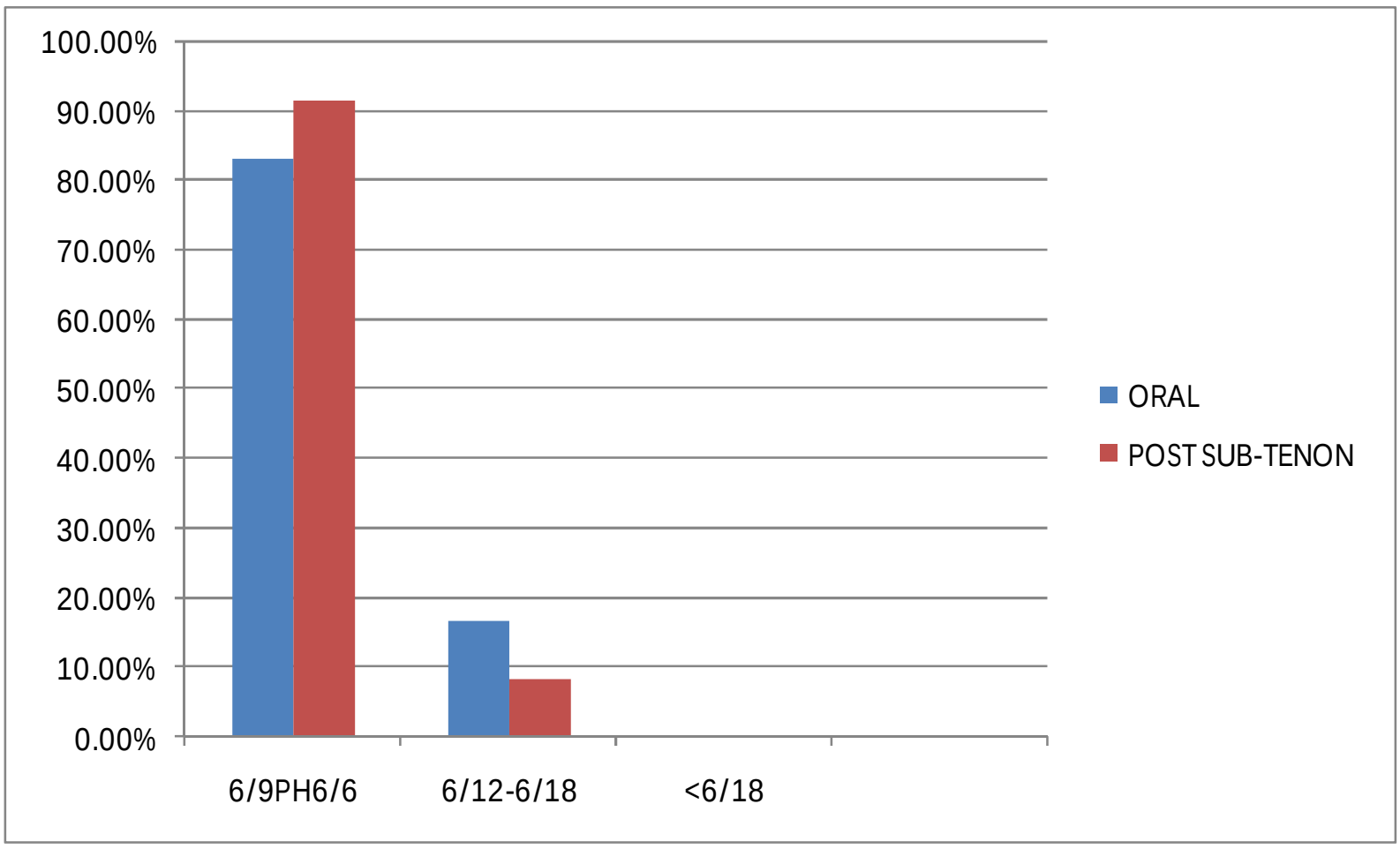


In FFA it was found that 16.67% in the subgroup 1and 8.33% in subgroup 2 showed active disease.

14. VISUAL ACUITY AT 16 WEEKS

TABLE-14

BCVA	SUB-GROUP-I ORAL	SUB-GROUP-II POST.SUB-TENON
6/9PH6/6	10(83.33%)	11(91.67%)
6/12-6/18	2(16.67%)	1(8.33%)
<6/18	0	0

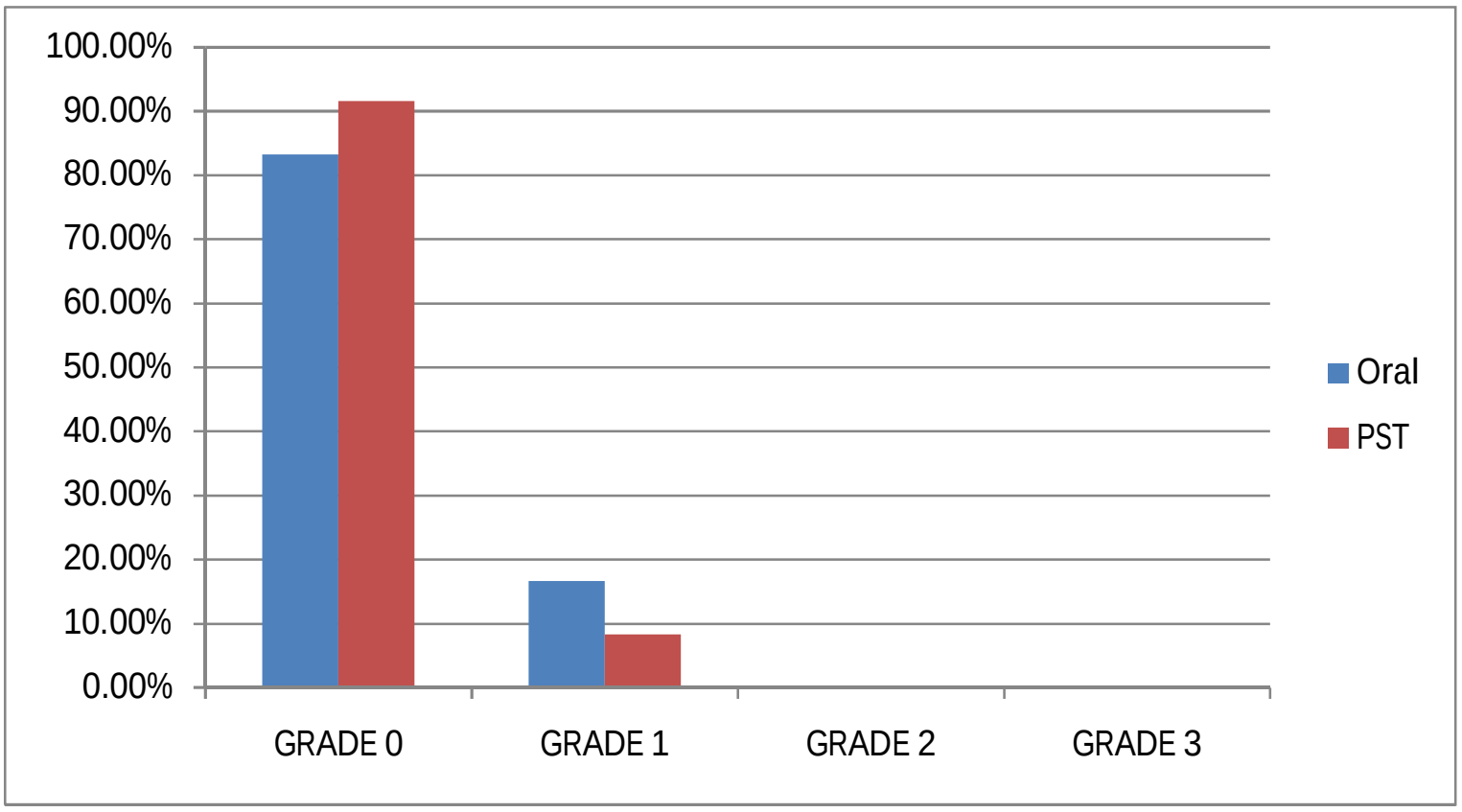


The Best corrected Visual acuity improved to 6/6 in 83.33% of oral steroids group and in subgroup treated with posterior sub-tenon 91.67% had BCVA of 6/6 at the end of 16weeks.

15. MEDIA CLARITY AT 16TH WEEK

TABLE-15

MEDIA CLARITY (GRADE)	SUB-GROUP-I ORAL	SUB-GROUP-II POST.SUB-TENON
0	10 (83.33%)	11 (91.67%)
I	2 (16.67%)	1 (8.33%)
II	0	0
III	0	0

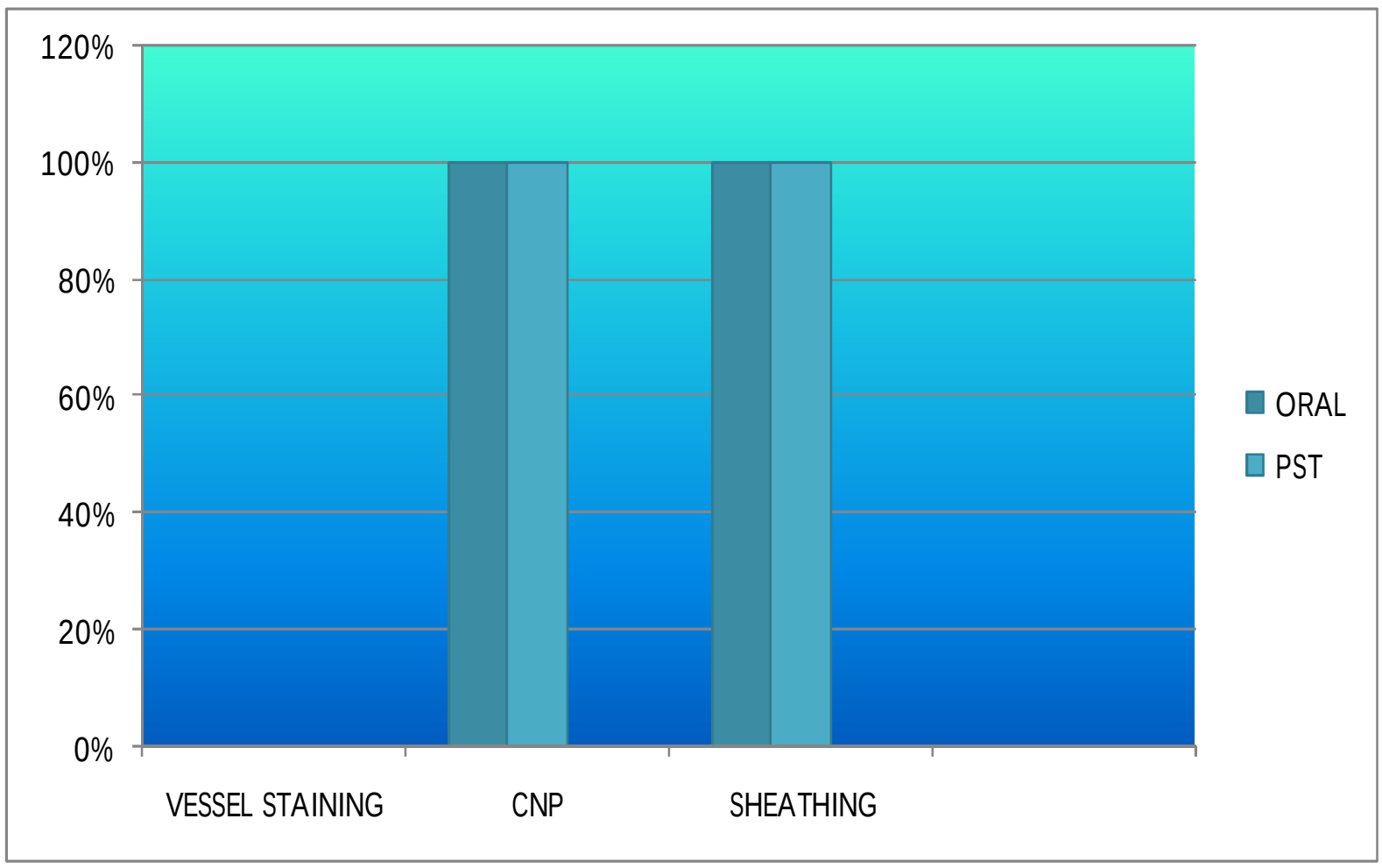


91.67% of patient with posterior sub tenon and 83.33% of patient with oral steroids had clear media at end of 16 weeks .Only 8.33% in the subgroup 2 and 16.67% in the subgroup 1 had minimal activity indicate resolution of disease

16. FUNDUS FLUORESCEIN ANGIOGRAPHY AT 16TH WEEK

TABLE-16

FFA	SUB-GROUP-I ORAL	SUB-GROUP-II POST.SUB-TENON
VESSEL STAINING	0	0
AREAS CAPILLARY NON-PERFUSION	12(100%)	12(100%)
SHEATHING	12(100%)	12(100%)

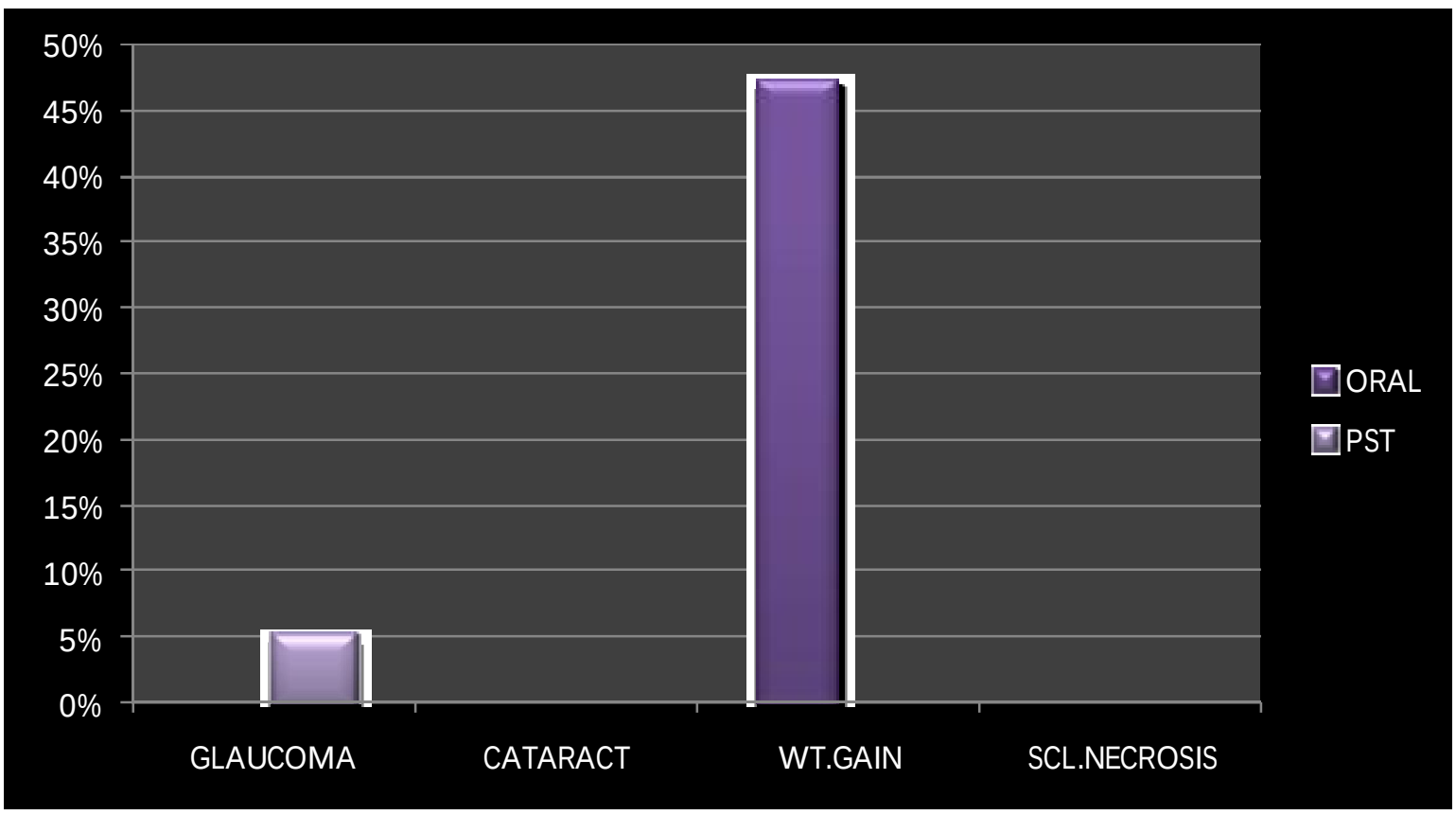


All Patients in the two group had capillary non-perfusion and sheathing of vessels, no evidence of activity at the end of 16wks.

17. POST INTERVENTIONAL COMPLICATIONS

TABLE-17

COMPLICATIONS	ORAL	POST.SUB-TENON
GLAUCOMA	0%	1(5.26%)
CATARACT	0%	0%
WEIGHT GAIN	9(47.37%)	0%
GASTRITIS	2((16.67%)	0%



WEIGHT GAIN was the predominant adverse effect(47.37%) among the oral steroid group only 16.67% reported, Gastritis problem in the oral steroid group.

DISCUSSION AND RESULTS

In this study of 50 cases of retinal vasculitis it was found that 25 cases (50%) were due to Eales disease and 10 cases(20%) were secondary to ocular disease and 15 cases (30%) were secondary to systemic disease.

Among the 50 cases studied 43(86%) were males and 7 (14%) were females.

In our study all Eales patients were Male.In patients with systemic disease 7 patient among the 15 were females and remaining 8 were males.All 10 patients with ocular disease were males.

This male predilection was supported by Elliot AJ:Thirty year observation of patients with Eales disease,in his study 93% were Male.[6]

Out of the 50 cases studied 20 cases (40%) were in the age group of 31-40 yrs.18 cases(36%) in the age group of 21-30yrs.6 cases(12%) in the age group of 41-50 yrs. 4(8%) were >50 yrs of age. 2(4%) in the age group of 10-20yrs.

Majority of patients with Eales were in the age group of 30-40 yrs.In a study by Donders in 1958 the average age for men was 28 years and for women it was 30 years.

Among the 25 cases with Eales 15(60%) cases were bilateral ,10 cases(40%) were unilateral , of the 15 patient with systemic disease 10 cases were bilateral and 5 were unilateral. In patients with ocular disease 5 were bilateral and 5 were unilateral.

Duke Elder had reported that as a rule Eales disease would be bilateral in 90% of the cases if they are observed over a period of atleast 5 years.[1]

Among the 25 patients with Eales, 15 patients had an acute onset, remaining patients in Eales disease and those with systemic and Ocular disease had insidious onset.

Blurring of vision was the predominant symptom in about 20 cases (40%)

Sudden loss of vision in 15 cases (30%), floaters were noted in both group.

Among the clinical signs in patients with Eales, sheathing of vessels noted in 20 eyes(50%), 15 eye (37.5%) had vitreous hemorrhage and neovascularisation was seen in 5 eyes(12.5%)

Among the clinical sign in patients with vasculitis secondary to systemic disease sheathing noted in 5 eyes (20%), vein occlusion in 16 eyes (64%),

4 eyes (16%) had tractional retinal detachment.

Sheathing with exudation and vitritis noted in 13 eyes(86.66%) of patients with vasculitis secondary to ocular disease and vein occlusion noted in 2 eyes.(13.33%).

Visual acuity of 6/6-6/12 was noted in 20 eyes (50%) of patients with Eales, 6/24-6/60 seen in 5 eyes(12.5%) of patients and 5/60-PL seen in 15 eyes(37.5%).

5 eyes (20%) of patient with vasculitis due to systemic disease had visual acuity of 6/24-6/60, 20eyes(80%) had visual acuity of <5/60 .

5 eye (33.33%) of patients with vasculitis due to ocular disease had visual acuity of 6/24-6/60, 10 eye (66.66%) of these patient had visual acuity of <5/60.

Of the 25 cases of Eales, 5 cases had mantoux positivity. These patient were referred to chest physician and one patient was stated on Antitubercular treatment as advised and was excluded from sub-group analysis.

SUB-GROUP ANALYSIS

Management

24 cases of Eales patient were randomly divided into 2 subgroups of 12 cases each,

Sub group1 subjected to oral prednisalone (1mg/kg/day)

Subgroup 2 subjected to posterior sub tenon triamcinalone

Visual acuity and media clarity of the two groups observed after 8th, 12th, 16th week.

At the end of 8 weeks

58.33% had BCVA of 6/6 in subgroup 1 and 66.67% had 6/6 in subgroup 2.In 16.67% of subgroup1 and 8.33% of subgroup 2 the visual acuity was persistently low <6/18.

At the end of 8weeks

In 58.33% patient in the subgroup 1and 66.66% in subgroup 2 had clear view of the media.16.67% of patient in subgroup 1 and 8.33% of patient in subgroup 2 had grade II vitritis and were resistant to steroids and started on immunosuppressants

The patients were reviewed at 12th and 16th week

At the end of 16th week

The Best corrected Visual acuity improved to 6/6 in 83.33% of oral steroids group(sub-group 1) and in subgroup 2(treated with posterior sub-tenon) 91.67% had BCVA of 6/6. In 8.33% of subgroup2 and 16.67% in the subgroup 1 had visual acuity of 6/12-6/18. The **chi square test showed a p=0.361**, showed no significant difference in the efficiency of the two subgroup. 91.67% of patient with posterior sub tenon and 83.33% of patient with oral steroids had clear media at end of 16 weeks .Only 8.33% in the subgroup2 and 16.67% in the subgroup 1 had minimal activity which indicate resolution of disease following treatment with immunosuppressants.

15 eye of Eales patient with vitreous hemorrhage were kept under observation and the hemorrhage was found to be resolving during the follow up period.

Complications

Oral steroids were associated with systemic complication of weight gain in 9 out of 12 cases

Gastritis was observed in 2 out of 12 cases treated with oral steroids. Posterior sub-tenon was associated with glaucoma in 1 of 12 cases

CONCLUSION

Primary vasculitis is predominant (50%)among the vasculitis in our study.

From our intervention it was clear that steroid in any mode of administration was efficacious in controlling the inflammation in acute disease .

2 patients (16.67%) in the oral steroid group and 1 patient (8.33%) in posterior sub-tenon group required immunosuppressants.

No patients in our study required laser theraphy for neovascularisation during the follow up period.

The final visual outcome in

Subgroup 1(ORAL STEROIDS) at end of 16 weeks was 6/6 in 83.33%,16.67% had visual acuity of 6/12-6/18

Subgroup 2(POSTERIOR SUB-TENON) at the end of 8 weeks was 6/6 in 91.67%, 8.33% had visual acuity of 6/12-6/18.

No activity(grade-0 vitritis) was seen in 83.33% of cases in the subgroup 1 and 91.67% of subgroup 2.

Male predliction was noted in our study

30-40yrs is the predominant age group affected in our study

Bilaterality was predominant among patient with Eales disease and vasculitis due to Systemic disease.

In patients with Primary vasculitis local steroid administered with safety and skill is efficacious in controlling active disease with minimal complication compared to systemic administration.

Although systemic steroids are equally efficacious in controlling active disease and easily administered, adverse systemic complications is a matter of concern.

PART THREE

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PROFORMA

NAME AGE/SEX

OP.NO/IP.NO: DATE

ADDRESS

OCCUPATION

PRESENTING COMPLAINTS

Defective Vision

Distance/Near

Total Loss Of Vision

Floaters

Positive Scotoma

Micopsia/Macropsia/Metamorphopsia

Field Defect

Pain And Redness

OCULAR EXAMINATION

	RE	LE
Visual Acuity		
Lids/Conjunctiva		
Cornea		
Anterior chamber		

Iris

Pupil

Lens

Vitreous

Tension

Retinoscopy

Subject

Myopia/hyperopia

Normal

Corrected vision

Fundus Examination

Direct Ophthalmoscopy

RE LE

Media

Disc

Vessels – Phlebitis peripheral

Central

Arteritis - Peripheral

Central

Combined arteritis/phlebitis

Neovascularisation

NVD -

NVE -

Indirect Ophthalmoscopy

Goldman 3 mirror

INVESTIGATION

Total count

Differential count

Peripheral smear

ESR

Bleeding time/Clotting time

Bloodsugar

Urinanalysis

VDRL and FTA-ABS

Tuberculin skin testing

TORCH Screening

Serum ACE

Rheumatoid factor

Antinuclear antibody

Antineutrophil cytoplasmic antibody

Fundus fluorescein angiography

1.New vessels

2.Capillary non-perfusion

OTHER SPECIALITIES OPINION:

TREATMENT

Oral

Posterior Sub-Tenon

Laser

Pan-Retinal Photocoagulation

Vitrectomy.

FOLLOW-UP

4th week-

Visual acuity

Vitritis grade.

8th week

Visual acuity

Vitritis grade.

12th week

Visual acuity

Vitritis grade.

16th week

Visual acuity

Vitritis grade.

KEY TO MASTER CHART

M	-	Male
F	-	Female
BE	-	Both eye
RE	-	Right eye
LE	-	Left eye

SYMPTOMS

LOV	-	Loss of vision
F	-	Floaters
DV	-	Defective vision

M.O.P-MODE OF PRESENTATION

SO	-	Sudden onset
IO	-	Insidious onset
V.A	-	Visual Acuity
NIP	-	No improvement with pin hole
PL	-	Perception of light
HM	-	Hand movements
CFCF	-	Counting fingers close to face

C.O.R.V-CLASSIFICATION OF RETINAL VASCULITIS

Primary

Secondary to systemic disease

Secondary to ocular disease

CLINICAL SIGNS

S/V	-	Sheathing of vessels/vitritis
V.O	-	Vein of Occlusion
Neo	-	Neovascularization
T.R.D	-	Tractional Retinal Detachment
S/V/E	-	Sheathing of vessel/vitritis/ Exudation

MEDIA CLARITY

M.N.V	-	Media no view
CL	-	Clear

NAME	SEX	AGE	EYE AFFECTED	M.O.P	ONSET	B.C.V.A		C.O.R.V			CLINICAL SIGNS						VITRITIS GRADING		SUB GROUP	V.A.AT 8 WEEKS		VITRITIS AT 8 WEEKS		V.A.AT 16 WEEKS		VITRITIS AT 16 WEEKS		FFA AFTER 8 WEEKS				FFA AFTER 16 WEEKS				POST INTERVENTIONAL COMPLICATION			
						RE	LE	PRIMARY	SYSTEMIC.D	OCULAR.D	S/V	V.O	NEO	V.H	T.R.D	S/E/V	RE	LE		RE	LE	RE	LE	RE	LE	RE	LE	VES.STAIN	CNP	NEO	SHEATHING	VES.STAIN	CNP	NEO	SHEATHING	GLAUCO	CATRAC	WT.GAIN	GASTRITIS
KUMAR	M	32	BE	LOV	SO	HM	6/12NIP	YES			LE			RE			MNV	I	1	1/60NIP	6/9PH6/6	MNV	CL	4/6OPH6/60	6/9PH6/6	III	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	YES	NO
SASIKUMAR	M	38	BE	LOV/F	SO	6/9NIP	HM	YES			RE			LE			I	MNV	1	6/9PH6/6	2/60NIP	CL	MNV	6/9PH6/6	3/60NIP	CL	III	NO	RE	NO	RE	NO	RE	NO	RE	NO	NO	YES	YES
ARUNKUMAR	M	36	BE	LOV/F	SO	6/12NIP	HM	YES			RE			LE			II	MNV	2	6/9NIP	1/60NIP	I	MNV	6/9PH6/6	3/60PH6/60	CL	III	NO	RE	NO	RE	NO	RE	NO	RE	NO	NO	NO	NO
SRINIVASAN	M	31	BE	LOV	SO	HM	6/12NIP	YES			LE			RE			MNV	II	2	1/2/60NIP	6/9NIP	MNV	I	3/60PH6/60	6/9PH6/6	III	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	NO	NO
VIMALKUMAR	M	47	BE	DV	IO	2/60NIP	3/60NIP		AIDS			BE					CL	CL																				
SRINIVASAN	M	32	BE	LOV/F	SO	6/9NIP	1/2/60NIP	YES			RE			LE			I	MNV	1	6/9NIP	2/60NIP	I	MNV	6/9PH6/6	4/60PH6/36	CL	III	NO	RE	NO	RE	NO	RE	NO	RE	NO	NO	YES	NO
SAKTHIVEL	M	31	BE	LOV	SO	6/18NIP	HM	YES			RE			LE			II	MNV	1	6/24NIP	2/60NIP	III	MNV	6/12NIP	2/60PH6/60	I	III	RE	**RE**	NO	RE	NO	**RE**	NO	RE	NO	NO	NO	NO
PRIYA	F	10	BE	DV	IO	3/60NIP	4/60PH6/60		SLE			BE					II	II																				
KATHIKA	F	15	BE	DV	IO	6/60NIP	6/60NIP		SLE			BE					II	II																				
SADAIYAPPAN	M	42	RE	DV	IO	6/36PH6/24	6/24PH6/12		AIDS			RE					CL	CL																				
KRISHNAMOORTHY	M	27	BE	LOV/F	SO	I/2/60NIP	6/9NIP	YES			LE			RE			MNV	I	2	2/60NIP	6/9PH6/6	MNV	CL	6/60NIP	6/9PH6/6	III	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	NO	NO
FRANCINA	F	51	BE	DV	IO	PL	PL		AIDS						BE		CL	CL																				
POOJA	F	30	BE	DV	IO	6/36NIP	6/36NIP		APS								II	II																				
AMULRAJ	M	40	BE	LOV/F	SO	6/12NIP	HM	YES			RE			LE			I	MNV	2	6/9PH6/6	1/60NIP	CL	MNV	6/9PH6/6	5/60PH6/60	CL	III	NO	RE	NO	RE	NO	RE	NO	RE	NO	NO	NO	NO
RAJA	M	40	BE	LOV/F	SO	6/18NIP	CFCF	YES			RE			LE			II	MNV	2	6/24NIP	2/6ONIP	II	MNV	6/12NIP	5/60PH6/60	I	III	RE	RE	NO	RE	NO	RE	NO	RE	YES	NO	NO	NO
RAVUKUMAR	M	52	BE	DV	IO	PL	PL		AIDS						BE		CL	CL																				
RAJESH	M	40	BE	LOV/F	SO	1/60NIP	6/18NIP	YES			LE			RE			MNV	II	1	2/60NIP	6/12NIP	MNV	I	4/60PH6/36	6/9PH6/6	III	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO		NO
RAMESH	M	37	BE	LOV/F	SO	1/2/60NIP	6/9NIP	YES			LE			RE			MNV	I	1	2/60NIP	6/9PH6/6	MNV	CL	6/60NIP	6/9PH6/6	III	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO		NO
KASIAMMAL	F	25	BE	DV	IO	1/60NIP	3/60NIP		SLE			BE					II	II																				
RAJENDRAN	M	35	BE	DV/F	IO	4/60NIP	HM			BP						BE	III	III																				
KUMAR	M	48	LE	DV/F	IO	6/9NIP	4/60NIP		AIDS		LE						CL	CL																				
KILLIVALLAVAN	M	27	BE	DV/F	IO	2/60NIP	2/60NIP			BPAN						BE	III	III																				
SURESH	M	26	LE	DV/F	IO	6/9PH 6/6	6/12NIP	YES			LE						CL	II	1	6/9PH6/6	6/9PH6/6	CL	CL	6/9PH6/6	6/9PH6/6	CL	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO		NO
RAVI	M	30	LE	BV/F	IO	6/9PH6/6	6/12NIP	YES			LE						CL	I	1	6/9PH6/6	6/9PH6/6	CL	CL	6/9PH6/6	6/9PH6/6	CL	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	YES	YES
LATHA	F	25	BE	DV	IO	6/36NIP	6/60NIP		SARCOID			BE					II	III																				
SENTHIL	M	39	BE	LOV/F	SO	6/18NIP	HM	YES			RE			LE			II	MNV	1	6/24NIP	2/60NIP	II	MNV	6/12NIP	6/60NIP	I	III	RE	RE	NO	RE	NO	RE	NO	RE	NO	NO	YES	NO
SHIVAKUMAR	M	40	BE	LOV/F	SO	CFCF	6/12NIP	YES			LE			RE			MNV	II	2	2/60NIP	6/12NIP	MNV	I	6/60NIP	6/9PH6/6	III	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	NO	NO
RAMESH	M	40	BE	LOV/F	SO	6/9NIP	1/2/60NIP	YES			RE			LE			I	MNV	2	6/9PH6/6	2/60NIP	CL	MNV	6/9PH6/6	6/60PH 6/36	CL	III	NO	RE	NO	RE	NO	RE	NO	RE	NO	NO	YES	NO
MURUGAN	M	37	LE	DV	IO	6/12PH6/6	5/60NIP			LP						LE	CL	III																				
BABU	M	35	RE	DV	IO	4/60NIP	6/12PH6/6			RP						RE	III	CL																				
DEVI	F	46	BE	DV	IO	1/2/60NIP	1/60NIP		AIDS			BE					CL	CL																				
SETHU	M	27	RE	BV/F	IO	6/9NIP	6/9PH6/6	YES			RE						I	CL	2	6/9PH6/6	6/9PH6/6	CL	CL	6/9PH6/6	6/9PH6/6	CL	CL	NO	RE	NO	RE	NO	RE	NO	RE	NO	NO	NO	NO
RAJA	M	21	LE	BV/F	IO	6/12PH6/6	6/12NIP	YES			LE						CL	I	2	6/9PH6/6	6/9PH6/6	CL	CL	6/9PH6/6	6/9PH6/6	CL	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	NO	NO
VINOTH	M	28	BE	DV/F	IO	4/60NIP	4/60NIP			BP						BE	III	III																				
SURYA	M	30	BE	DV/F	IO	3/60NIP	4/60NIP			BP						BE	III	III																				
MUTHUKUMAR	M	43	LE	DV	IO	6/24PH6/6	6/36NIP		AIDS		LE						CL	CL																				
VELMURUGAN	M	42	LE	DV	IO	6/18PH6/6	6/60NIP		AIDS		LE						CL	CL																				
VIJAY	M	24	RE	DV/F	IO	4/60NIP	6/12PH6/6			RP						RE	III	CL																				
VEERA	M	26	LE	DV/F	IO	6/9PH6/6	3/60NIP			LP						LE	CL	III																				
RAJESHKRISHNA	M	30	RE	DV/F	IO	4/60NIP	6/24NIP			RP						RE	III	I																				
MANIAJ	M	51	BE	DV	IO	1/60NIP	2/60NIP		AIDS			BE					CL	CL																				
DHARSAN	M	24	BE	BV/F	SO	6/9NIP	HM	YES			RE			LE			I	MNV																				
SATHYAMOORTHY	M	27	BE	DV/F	IO	4/60NIP	3/60NIP			BP							III	III																				
SENTHILMURUGAN	M	30	LE	BV/F	IO	6/9PH 6/6	6/12NIP	YES			LE						CL	I	1	6/9PH6/6	6/9NIP	CL	CL	6/9PH6/6	69PH6/6	CL	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	YES	NO
BALAJI	M	51	RE	DV	IO	5/60NIP	6/12NIP		AIDS		RE						CL	CL																				
SANTHOSH	M	37	RE	BV/F	IO	6/12NIP	6/9PH6/6	YES			RE						I	CL	2	6/12NIP	6/9PH6/6	I	CL	6/9PH6/6	6/9PH6/6	CL	CL	NO	RE	NO	RE	NO	RE	NO	RE	NO	YES	NO	NO
SUNDAR	M	30	LE	BV/F	IO	6/9PH6/6	6/9NIP	YES			LE						CL	I	1	6/9PH6/6	6/9PH6/6	CL	CL	6/9PH6/6	6/9PH6/6	CL	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO		YES	NO
KAESAVAN	M	40	LE	BV/F	IO	6/9PH6/6	6/9NIP	YES			LE						CL	I	2	6/9PH6/6	6/12NIP	CL	I	6/9PH6/6	6/9PH6/6	CL	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	NO	NO	NO
MADHAVAN	M	38	RE	BV/F	IO	6/12NIP	6/9PH6/6	YES			RE						I	CL	2	6/9PH6/6	6/9PH6/6	CL	CL	6/9PH6/6	6/9PH6/6	CL	CL	NO	RE	NO	RE	NO	RE	NO	RE	NO	NO	NO	NO
SHRIRAM	M	40	LE	BV/F	IO	6/9PH6/6	6/12NIP	YES			LE						CL	II	1	6/9PH6/6	6/12NIP	CL	I	6/9PH6/6	6/9PH6/6	CL	CL	NO	LE	NO	LE	NO	LE	NO	LE	NO	YES	NO	NO