A Dissertation on

A STUDY ON EVALUATING THE COMMON RISK FACTORS, SYSTEMIC ASSOCIATION AND CLINICAL PROFILE OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI

With partial fulfillment of the regulations for the award of the degree of

M.S. (OPHTHALMOLOGY)
BRANCH – III

MADRAS MEDICAL COLLEGE AND RESEARCH INSTITUTE CHENNAI – 600003

REGISTRATION NUMBER - 221913024

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CERTIFICATE

This is to certify that **Dr.R.SETHUKKARASI,** Post Graduate student in M.S Ophthalmology, at Regional Institute of Ophthalmology and Government Ophthalmic hospital attached to Madras Medical College, Chennai, carried out this dissertation on **" A STUDY ON EVALUATING THE COMMON RISK FACTORS, SYSTEMIC ASSOCIATION AND CLINICAL PROFILE OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS."** under our direct guidance and supervision during the academic years from 2019 to 2022.

This dissertation is submitted to the TamilNadu Dr.MGR Medical University, Chennai for the fulfillment of award of M.S. Degree in Ophthalmology in Branch III.

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CERTIFICATE FROM GUIDE

This is to certify that this dissertation entitled,

" **A STUDY ON EVALUATING THE COMMON RISK FACTORS, SYSTEMIC ASSOCIATION AND CLINICAL PROFILE OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS.** ".is a bonafide record of research work done by Dr.R.SETHUKKARASI, Post Graduate Resident (May 2019 to May 2022) in Department of Ophthalmology, Madras Medical College,Chennai . This dissertation is submitted to the TamilNadu Dr.MGR Medical University, Chennai for the fulfillment of award of M.S. Degree in Ophthalmology in Branch III

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DECLARATION

I, **Dr.R.SETHUKKARASI,** solemnly declare that the dissertation titled“ **A STUDY ON EVALUATING THE COMMON RISK FACTORS, SYSTEMIC ASSOCIATION AND CLINICAL PROFILE OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS** ” has been prepared by me. This is submitted to The Tamil Nadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S. Ophthalmology, degree Examination to be held in May 2020.

Signature of the candidate,

DR.R.SETHUKKARASI

Place: Chennai

Date:

/

CERTIFICATE – II

This is to certify that this dissertation work titled "**A STUDY ON EVALUATING THE COMMON RISK FACTORS,SYSTEMIC ASSOCIATION AND CLINICAL PROFILE OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS "** of the candidate **DR.R.SETHUKKARASI** with Registration **Number 221913024** for the award of **M.S.,DEGREE BRANCH – III (OPHTHALMOLOGY).**I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result 7 **% (SEVEN)** percentage of plagiarism in the dissertation.

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PART-II

PART-III

PART-I

INTRODUCTION:

Retinal vein occlusion (RVO) is the common cause of visual loss in older individuals .It is the second most common retinal vascular disease next to diabetic retinopathy caused by blockage of small or large calibre vessels carrying blood away from retina.It is classified according to site of involvement and extent of perfusion

- According to site of occlusion:
    - CENTRAL RETINAL VEIN OCCLUSION(CRVO)-occlusion is at or proximal to lamina cribrosa of optic nerve.
    - BRANCH RETINAL VEIN OCCLUSION(BRVO)-occlusion at any branch of central retinal vein.It is of 3 types:
        a. MAJOR BRVO-involving first order temporal vessels
        b. MACULAR BRVO-involving only macular branch
        c. PERIPHERAL BRVO-not involving the macular circulation.
    - HEMIRETINAL VEIN OCCLUSION(HRVO)-occlusion at primary superior or inferior branch involving half of retina.

- According to perfusion status:
    - NON-ISCHEMIC-perfused status
    - ISCHEMIC-non perfused status

REVIEW OF LITERATURE

1) The burden of disease of retinal vein occlusion

   M Laouri, E Chen, M Looman, M Gallagher 2011

   Retinal vein occlusion is second most common cause of visual loss due to retinal vascular disease. Population based studies report a prevalence rate of 0.5-2% for branch RVO and 0.1-0.2% for central RVO. The 15-year incidence rate is estimated to be 1.8% in branch RVO and 0.2% for central RVO. Laser photocoagulation is only recommended for branch RVO who have not experienced severe visual loss.

2) Classification of central retinal vein occlusion

   Sohan Singh Hayreh, Ophthalmology 90,1983

   Prospective clinical study of 360 eyes with CRVO have shown that CRVO consists of 2 entities-Non-ischemic which is venous statis and Ischemic which is haemorrhagic retinopathy.

3) Natural history of central retinal vein occlusion-an evidence based Systematic review.

   Rachel L McIntosh , Ophthalmology 117,2010

   Visual acuity was generally poor at baseline and decreased over time.

Upto 34% of eyes with non-ischemic CRVO converted to ischemic type over 3 year period. In ischemic CRVO Neovascular glaucoma occurred in atleast 23% eyes within 15 months. In non-ischemic CRVO macular edema resolved in 30% eyes over time and subsequent NVG was rare.

4) Central retinal vein occlusion in young adults

Andrew CO Fong, Howard Schatz 1993

Inflammation of central retinal vein has been reported as a cause of Occlusion in young adults so called as papillophiebitis.

5) The natural course of central retinal vein occlusion

Patricia M Quinlan, Micheal J Elman 1990

Of 107 nonischemic eyes, 10% converted to ischemic variant.

A final visual acuity of less than 20/200 was found in 93% of ischemic eyes and 50% of nonischemic eyes.

6) Risk factors for central and branch retinal vein occlusion- a metaanalysis of published clinical data

Petr Kolar ,2014

A strong risk factor for RVO is the metabolic syndrome-Hypertension, Hyperlipidemia and diabetes mellitus. Congenital thrombophilic disease Smoking, vasculitis also increase risk of CRVO. Ophthalmic risk factors are ocular hypertension and glaucoma

7) A longitudinal analysis of risk factors associated with CRVO

Maxwell S Stem, 2013

Blacks had 58% increased risk of CRVO compared to whites. Women had 25% decreased risk of CRVO compared with men.A diagnosis of stroke increased the risk of CRVO by 44% & hypercoagulable state with 145% increased CRVO risk.Individuals with hypertension had 92% & Diabetes mellitus 53% increased hazard of CRVO.

8) Thrombophilic risk factors in patient with CRVO.

Rosella Marcucci 2001

At multivariate analysis only hyperhomocysteinemia and elevated PAI-1 Levels in addition to hypertension & hyperlipidemia were independent risk factors for CRVO.

9) Prognostic factors for retinal vein occlusion

Agnes Glacet-Bernard 1996

A prospective study of 175 cases showed the prognostic factors in CRVO are older age, male sex, number of riskfactors which correlated with poor visual outcome & development of retinal ischemia.

10) Neovascular complications after central retinal vein occlusion

K Evans et al,1993

Out of 73 patients studied , ocular neovascularization had occurred in 60%.More specifically 39% developed neovascular glaucoma.

Pan retinal photocoagulation produced regression in only 37% of those

With established neovascularization and was unsuccessful in preventing Neovascularization in five patients treated prophylactically.

Patient with pre-existing Primary open angle glaucoma were Statistically more likely to develop ocular neovascularization (p=0.02) which was also less responsive to laser therapy. Adequate prior Glaucoma therapy did not protect against this enhanced complication rate . It was concluded that POAG is a significant risk factor for developing ocular neovascularization after CRVO which will be refractory to laser therapy.

11) Impending CRVO in patient with COVID-19

Alessandro Invernizzi, Marco Pellegrini , 2020

COVID-19 related pneumonia presented with CRVO.

Retinal circulation should be considered as a potential site for Thromboembolic complications from COVID-19.

12) Ischemic central retinal vein occlusion in the Young.

Amod Gupta, Anita Agarwal 1993

25 patients with CRVO less than 40 years of age was studied.

8 patients had ischemic CRVO while 17 had non-ischemic CRVO.

Systemic disorders associated with ischemic CRVO included end stage Renal disease in 3,& hypercholesterolemia ,hypertension , Beta-thalassemia in 1 each. Patients with ischemic CRVO had severe Disease with a rapid downhill course.

13) Central retinal vein occlusion complicating Systemic Lupus Erythematosus.

Michael Silverman 1978

Retinal lesions occur in 25-30% patients with Systemic lupus Erythematosus. A patient with active SLE has developed central retinal Vein occlusion and blindness during their course.

14) Simultaneous bilateral central retinal vein occlusion as the initial Presentation of acute myeloid leukemia.

Mei-Yu Tseng ,2010

CRVO is common cause of visual impairment in patients less than 40 Years of age with 10-20%.Ocular involvement is common in patient With leukemia.

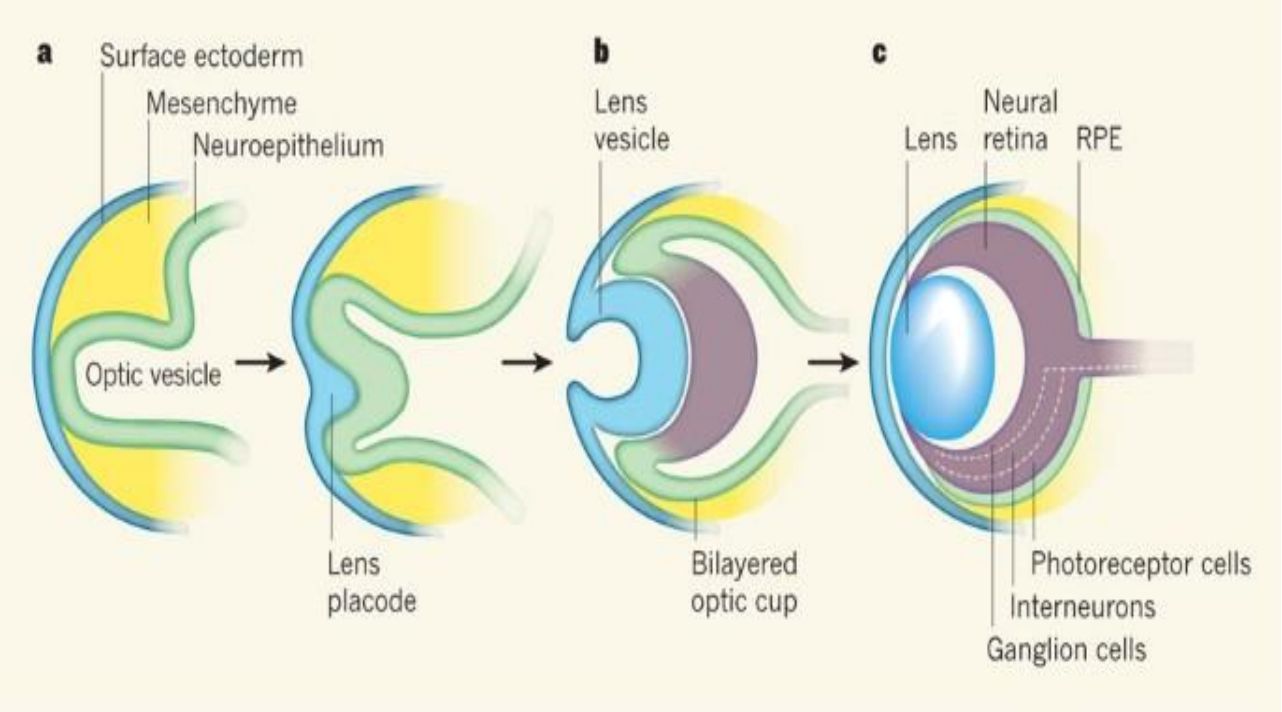
15) Progression of nonischemic central retinal vein obstruction to Ischemic variant.

John Minturn, Gary C Brown 1986

310 patients with CRVO were reviewed. Among these 16 patients who Presented with nonischemic CRVO progressed to an ischemic CRVO. They were associated with severe neovascularization and macular Edema.

DEVELOPMENT AND ANATOMY OF RETINA

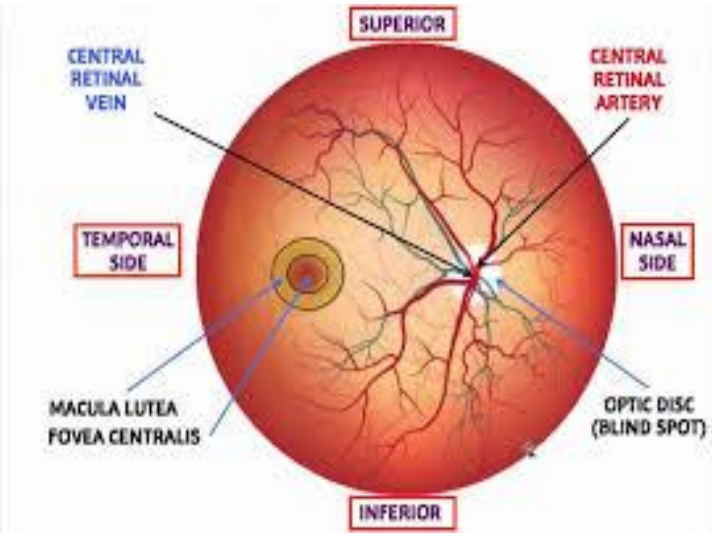
The retina is first recognized as optic pit in anterior neuroectoderm at day 23 of gestation.By day 25 the optic vesicle can be recognized as an evagination from diencephalon of the neural tube.The neural crest cells are interposed between the ectoderm and vesicles.Then the optic vesicle folds on itself becoming optic cup and the lens ectoderm invaginates into the vesicle.Resulting two tissue layers of optic cup are outer presumptive retinal pigment epithelium(RPE) and inner presumptive neural retina with lens vesicle filling much of interior of cup.The optic stalk connecting the developing retina with diencephalon will form scaffold for axons of optic nerve.



Retina is a thin delicate transparent membrane forming the innermost tunic of eyeball.It extends from optic disc to ora serrata having surface area of 266sq/mm.Four anatomical landmarks of retina are

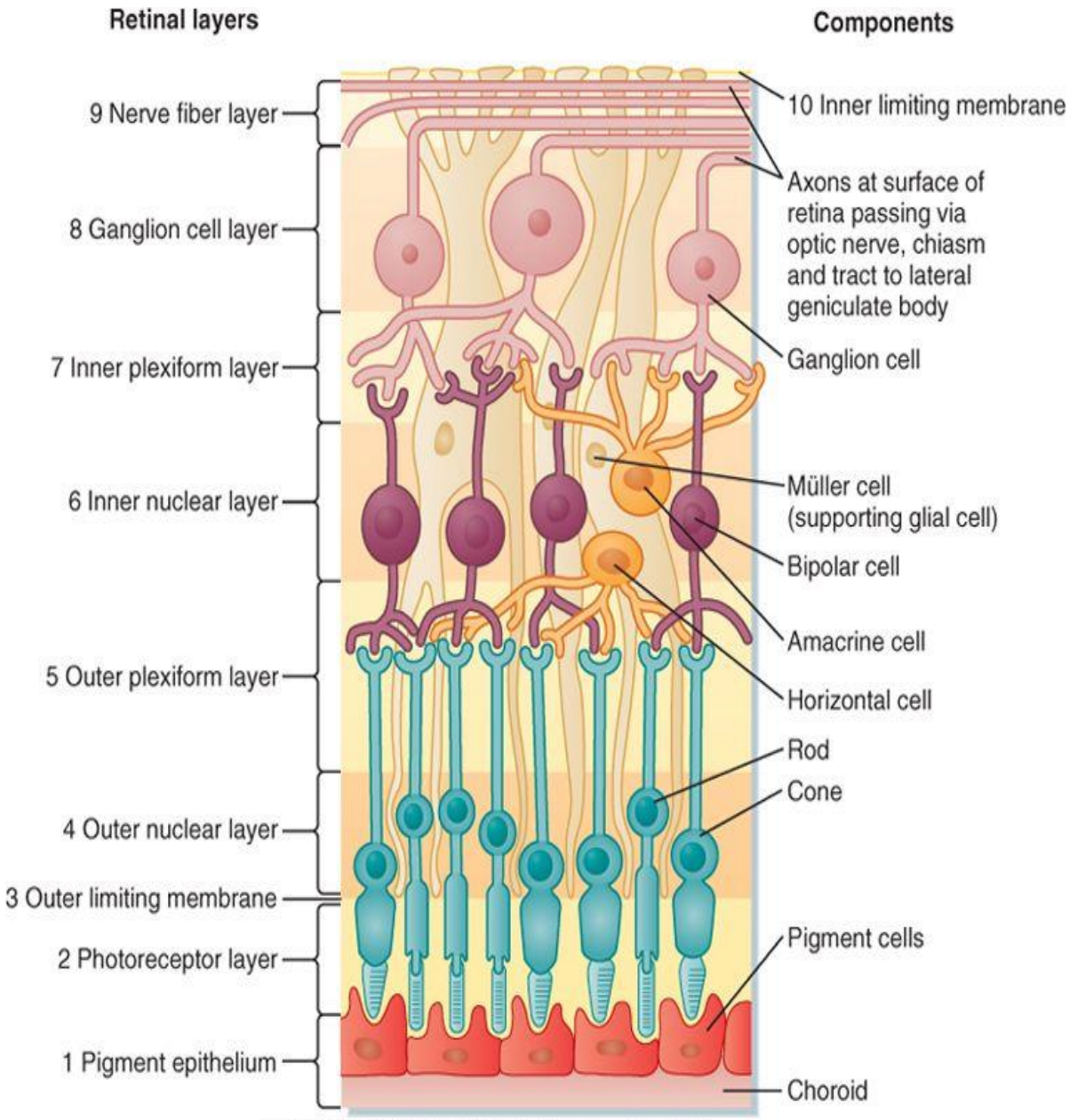
- OPTIC DISC-pale pink,circular area of 1.5mm diameter.All retinal layers terminate here except nerve fibre layer which pass through lamina cribrosa.
- MACULA LUTEA/AREA CENTRALIS-Dark area of 5.5mm diameter situated in posterior pole temporal to optic disc.

  FOVEA CENTRALIS is central depression of 1.5mm diameter in macula. FOVEOLA is the central floor of fovea measuring 0.35mm diameter.It is the most sensitive part of retina.UMBO is tiny depression in centre of foveola.
- PERIPHERAL RETINA-4 regions
  - NEAR PERIPHERY
  - MID PERIPHERY
  - FAR PERIPHERY
  - ORA SERRATA.



MICROSCOPIC STRUCTURE OF RETINA-10 LAYERS:

- RETINAL PIGMENT EPITHELIUM
- LAYER OF RODS AND CONE
- EXTERNAL LIMITING MEMBRANE
- OUTER NUCLEAR LAYER
- OUTER MOLECULAR(PLEXIFORM) LAYER
- INNER NUCLEAR LAYER
- INNER MOLECULAR(PLEXIFORM) LAYER
- GANGLION CELL LAYER
- NERVE FIBRE LAYER
- INTERNAL LIMITING MEMBRANE.



Koeppen & Stanton: Berne and Levy Physiology, 6th Edition.
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CENTRAL RETINAL VEIN

The central retinal vein exit through the lamina cribrosa and courses in the centre of optic nerve.It is surrounded by fibrous tissue.It leaves the optic nerve 10 mm from the eyeball.Each quadrant of retina is drained by minor retinal veins which coalesce to form a main retinal vein. The superotemporal and superonasal main retinal veins form superior papillary vein and inferotemporal & inferonasal main retinal veins form inferior papillary vein.The superior and inferior papillary vein converge to form central retinal vein.

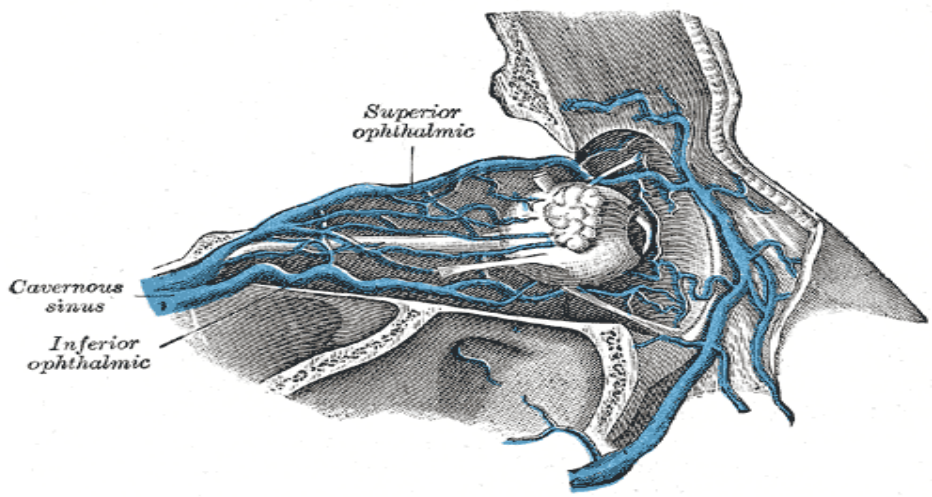
TRIBUTARIES OF RETINAL VEIN:

- From the Retina
- From the Optic nerve head.

It drains into superior or inferior ophthalmic veins and directly into cavernous Sinus.

STRUCTURE OF CENTRAL RETINAL VEIN:

The central retinal vein lies on the basement membrane with the endothelium which is continuous.There is presence of connective tissue adventitia in it. Outside of this is tunica media which is smooth muscle cell.

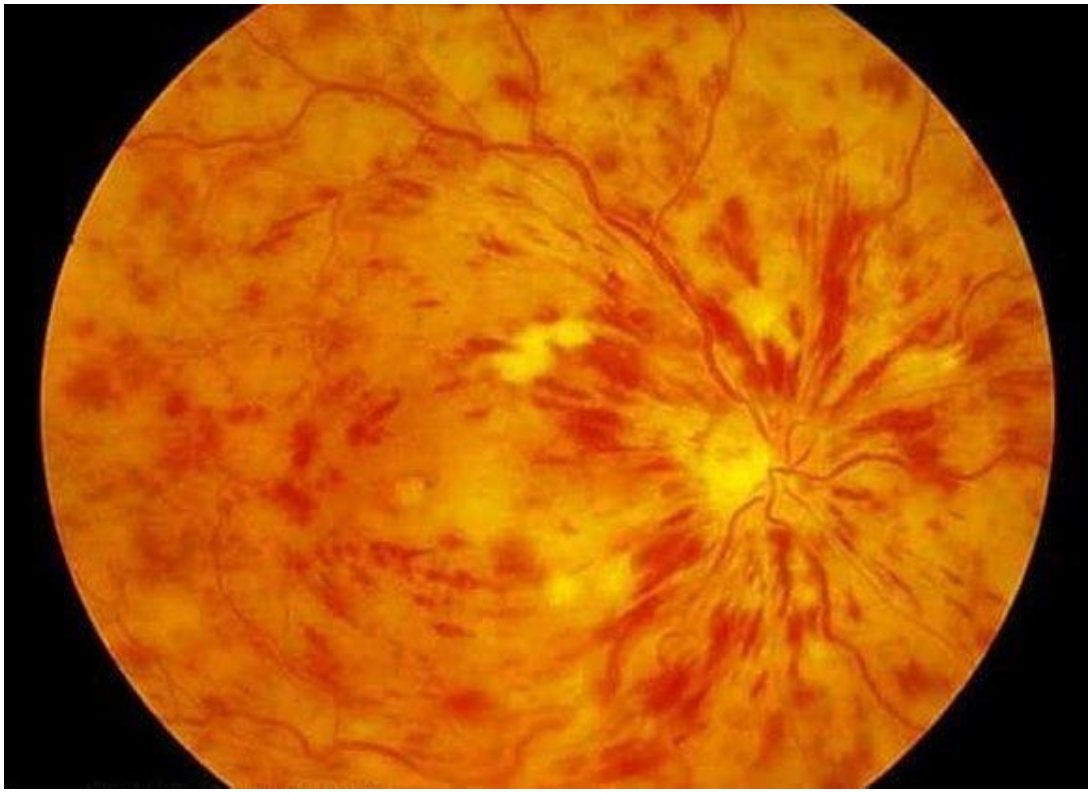


CENTRAL RETINAL VEIN OCCLUSION

Central retinal vein occlusion is the occlusion of retinal vein at or posterior to lamina cribrosa.It is the second common cause of blindness next to diabetic retinopathy.It is associated with multiple riskfactors. It is classified into 2 types

- ISCHEMIC CRVO OR HAEMORRHAGIC RETINOPATHY
- NON-ISCHEMIC CRVO OR VENOUS STASIS RETINOPATHY

Major case of vein occlusion 75-80% are of non-ischemic variety and 20-25% are of ischemic variety.The CVOS study reported a conversion rate from non-ischemic CRVO to ischemic CRVO of 3.3% by 4 months after study and incidence rate 10 times higher by 3 years.

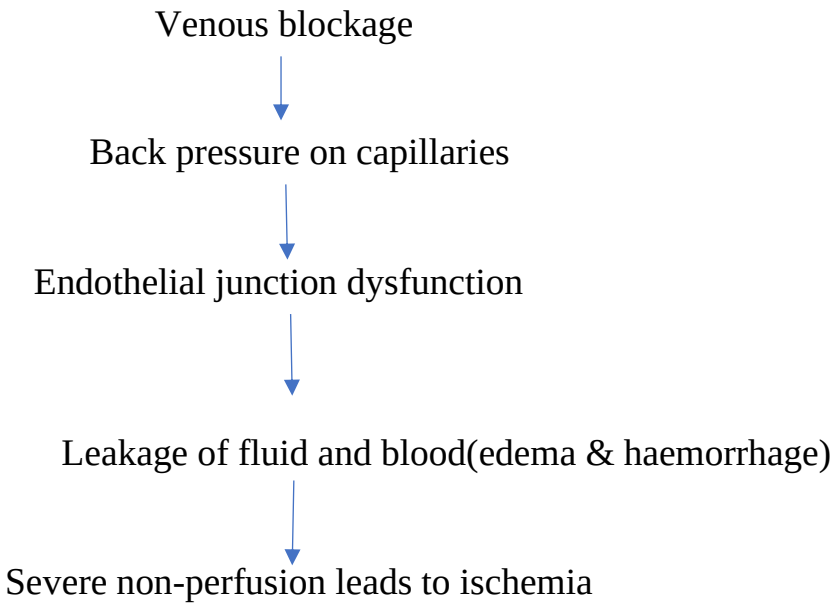


DEMOGRAPHIC FEATURES:

It commonly affect males more than the females and occurs predominantly in person over age of 65 years. Younger individuals who present with clinical picture of CRVO have underlying hypercoagulable or inflammatory etiology. Older people have associated systemic vascular disease such as diabetes and Hypertension. Population based studies report the prevalence of CRVO at 0.1%-0.4%. [(6)] It is usually an unilateral disease but risk of developing vein occlusion in the fellow eye is 1% per year. CRVO demonstrate a significant decrease in vision related quality of life especially in bilateral CRVO.

ETIOPATHOGENESIS:

Common mechanism of vein occlusion is



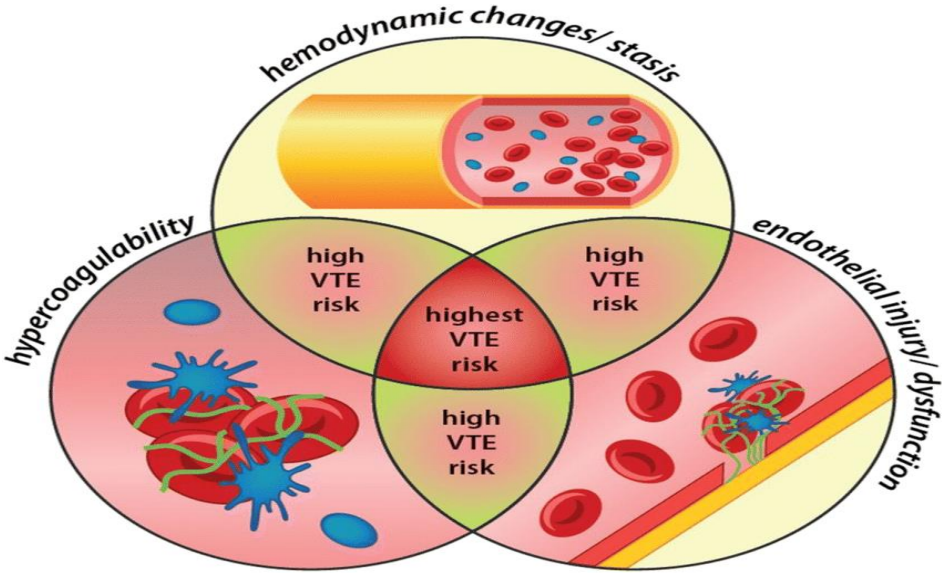
Both central retinal artery and vein shared a common adventitial sheath as they leave the head of optic nerve and passes along a narrow opening in region of lamina cribrosa.Thus the vessels situated in this compartment are tight with limited space to displace.This critical anatomical position leads to formation of thrombus in the vein by some factors such as altered blood flow.

Hemodynamic alterations-VIRCHOW TRIAD plays a role in CRVO

- Loss of vessel wall integrity
- Altered blood flow
- Hypercoagulable state-blood rheology changes

These disturbances lead to thrombus formation and vessel occlusion.

CRVO leads to resistance to venous flow resulting in blood stagnation and ischemia which stimulates the production of Vascular endothelial growth factor(VEGF).This leads to neovascularization and capillary leakage resulting in edema.



Klein & Olwin postulated three mechanism of CRVO- [4]

- Compression of vein by arterosclerotic central retinal artery.
- Occlusion by primary vessel wall disease (degenerative or inflammatory).
- Hemodynamic disturbances.

➢ Causes of External compression of vein are-

- Arteriosclerosis of Central retinal artery
- (Hypertension,Diabetes mellitus,Hyperlipidemia)
- Glaucoma
- Papilledema

- Thyroid eye disease
- Orbital space occupying lesions
- Cavernous sinus thrombosis
- Retrobulbar injection
- Closed head trauma.

Causes of primary vessel wall disease:

- Systemic vascular disease (Diabetes mellitus, Hypertension)
- Systemic vasculitis-TB, AIDS, Syphilis ,SLE
- Localized inflammation-Sarcoidosis, Serpiginous choroiditis.

Hemotological disorders:

- Clotting disorders-Activated protein C and S deficiency ,Lupus anticoagulant deficiency, Anticardiolipin antibodies ,Antithrombin III deficiency, Antiphospholipid antibodies.
- Paraproteinemia-Multiple myeloma, cryoglobulinemia
- Drugs-Oral contraceptives and diuretics
- Blood dyscrasia-Lymphoma, Leukemia, Sickle cell disease, Polycythemia vera.

RISK FACTORS & ASSOCIATED SYSTEMIC & OCULAR CONDITIONS:

The Eye disease case control study found an increased risk of any type of CRVO in persons with Systemic hypertension and Diabetes mellitus. Diabetes mellitus was more prevalent in individuals with non-perfused CRVO than in matched controls. Hyperlipidemia ,arteriosclerosis , Chronic renal failure , smoking have been linked to development of vein occlusions.

ASSOCIATED MEDICAL CONDITIONS:

- **HYPERTENSION**-

  It is the most common cause of CRVO in older patients >55 years accounting for 73% and 25% of younger patients according to Eye disease case control study where odd's ratio was higher for non-perfused venous occlusive disease. A longitudinal analysis of risk factors associated with CRVO showed 92% increased risk of CRVO in individuals with systemic hypertension. [7]

- **DIABETES MELLITUS**-

  One of the systemic vascular disease which causes CRVO in older individuals due to microvascular angiopathy & thromboembolic phenomenon accounting for 13-34%.It is also most common in individuals with non-perfused venous occlusive disease.

- **HYPERLIPIDEMIA**-

  CRVO is also associated with hyperlipidemia in older individuals and it's

incidence is low in previous study of meta-analysis of risk factors of CRVO. Individuals with high cholesterol level are screened for retinal vein occlusion.

- **HYPERCOAGULABLE STATES-**

Hematological abnormalities have been identified in patients with CRVO. There is greater association of hypercoagulable states and inflammatory conditions in individuals <60 yrs of age compared to higher incidence of systemic vascular disease risk factors in older persons.

Lahey and colleagues suggested systemic hypercoagulability of 27% in patients younger than 56 yrs of age [(36)] .Studies have demonstrated increased incidence of coagulation cascade abnormalities such as presence of Factor V Leiden, Lupus anticoagulant, antiphospholipid antibody, hyperhomocysteinemia, Factor XII deficiency , dysfibrinogenemia ,Protein C & S deficiency ,antithrombin III deficiency, prothrombin gene mutations, dysproteinemias, blood dyscrasias etc.

In multivariate analysis of thrombophilic risk factors of CRVO, Hyperhomocysteinemia and elevated PAI-1 levels were independent risk factors for CRVO.[(9)]

Andrew k Vine suggested that hyperhomocysteinemia was present in 5 of 9 individuals with bilateral disease,9 of 30 patients with ischemic occlusion . [(10)]

In Polycythemia and Waldenstrom's macroglobulinemia bilateral CRVO may be seen.

- **CORONARY ARTERY DISEASE**-

  Cardiovascular risk factors for retinal vein occlusion has suggested association of CRVO with coronary heart disease, & carotid artery plaque. Gorden et al has reported in his study that 7 out of 11 patients with vein occlusion showed evidence of Mitral valve prolapse.

- **MIGRAINE**-

  Felix Benninger in his study stated that 9% of patient who had migraine are associated with CRVO. (11)

- **SYSTEMIC VASCULITIS**-

  Inflammatory disease associtaed with occlusive periphlebitis such as Sarcoidosis ,SLE ,Behcet syndrome & Wegener's granulomatosis are also associated with CRVO.

  Steven A Teich & Joseph Sonnabend reported a case of CRVO in AIDS patient which was due to toxic effect of virus on endothelium. (12)

- **MEDICATIONS**-

  Use of Oral contraceptives have been associated with CRVO due to hypercoagulable state. Stowe et al reported the initimal proliferation with occlusion in patients who are on oral contraceptives for 3-4 years.

  Gutman showed that patients who are on diuretics may develop CRVO due to secondary hemoconcentration effect. (13)

ASSOCIATED OCULAR CONDITIONS:

- An increased risk of CRVO is present in eyes with open angle glaucoma (POAG) in elderly people.
- Ocular conditions causing mechanical pressure on the optic nerve head and lamina cribrosa including ,tilted optic nerve head, Optic nerve head drusen, Ischemic optic neuropathy &Pseudotumor cerebri have also been associated with CRVO.
- Thyroid related orbitopathy, mass lesions or head trauma with orbital fracture result in CRVO due to external pressure on optic nerve and globe.

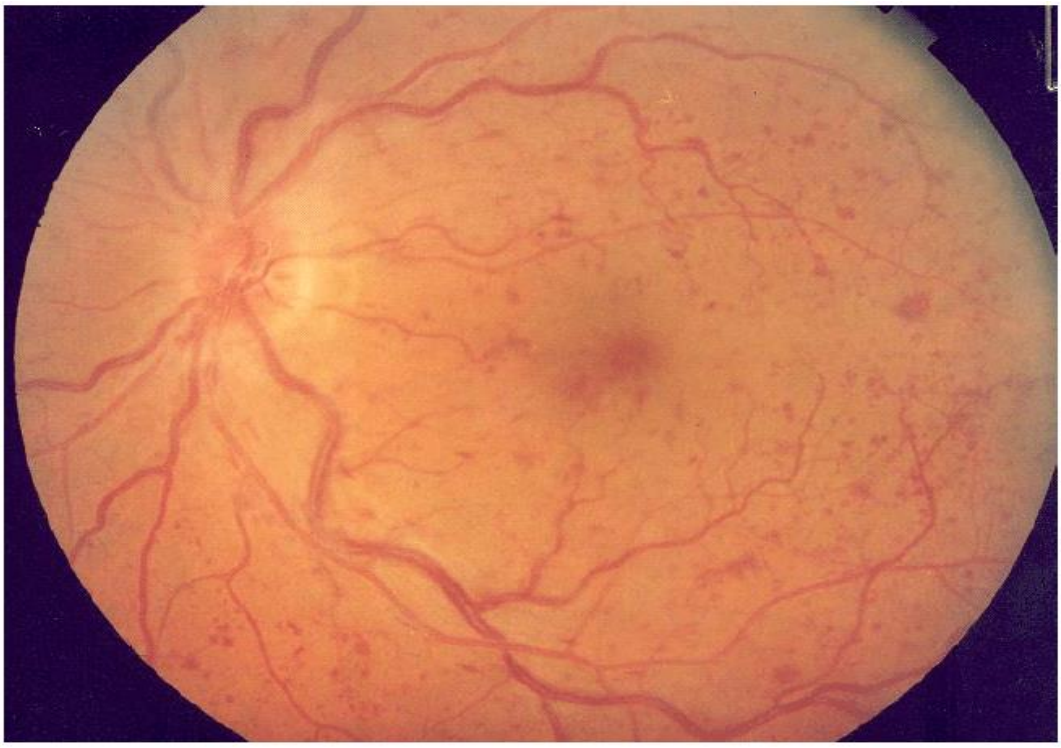
CLINICAL FEATURES SEEN IN ISCHEMIC AND NON-

ISCHEMIC CRVO:

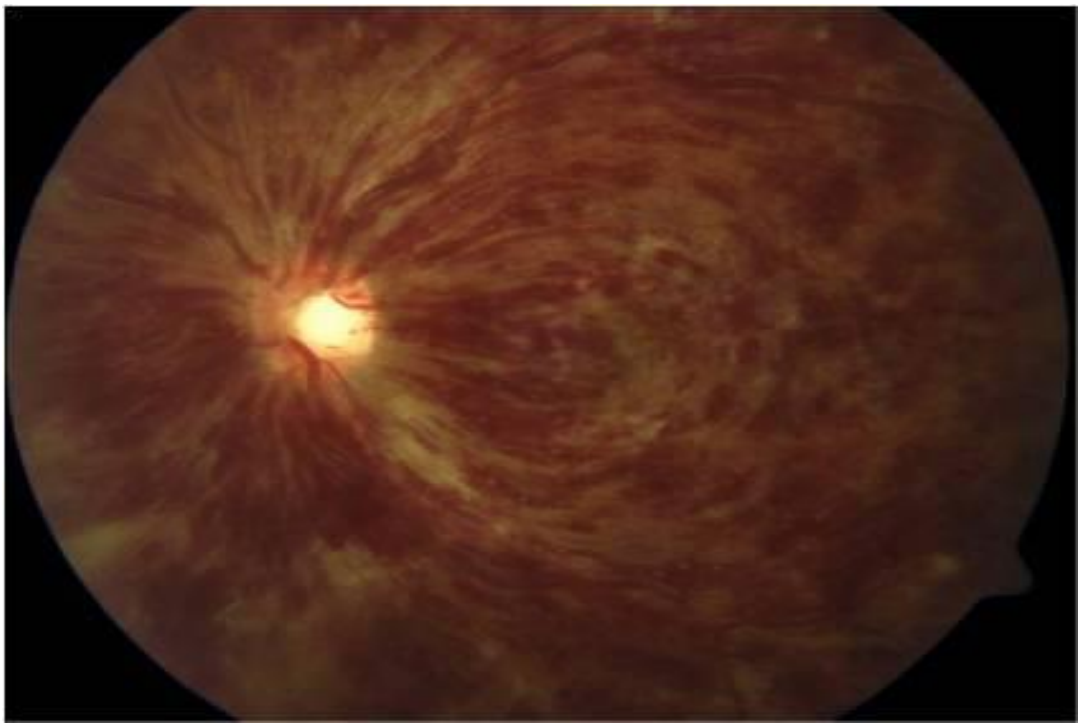
	NON-ISCHEMIC CRVO	ISCHEMIC CRVO
FREQUENCY	75-80%	20-25%
BEST CORRECTED VISUAL ACUITY	>6/60	<6/60
RAPD(RELATIVE AFFERENT PUPILLARY DEFECT	Absent	Present
VISUAL FIELD DEFECT	Rare	Common (using Goldmann perimeter)
FUNDUS APPEARANCE	Mild venous tortuosity & dilatation, less superficial & deep haemorrhage, cotton wool spots, macular edema. Patchy perivenular ischemic retinal whitening (PIRW) at posterior pole is an early sign in young patients.	Disc edema with Severe venous tortuosity & dilatation ,more superficial & deep haemorrhage, cotton wool spots, macular edema-"TOMATO SPLASH APPEARANCE"
FUNDUS FLUORESCEIN ANGIOGRAPHY	Delayed arteriovenous transit time with Less area of capillary non-perfusion.	Delayed arteriovenous transit timw with More than 10 disc area of retinal capillary non-perfusion.
ELECTRORETINOGRAM	Normal	Reduced b-wave amplitude with reduced b/a ratio
PROGNOSIS	Good, less chance of anterior segment neovascularization & neovascular glaucoma (<10% develops	Poor, high chance of anterior segment neovascularization & neovascular glaucoma. (>35% develop NVI/NVA.

	NVI/NVA)	The visual prognosis is worse than central arterial occlusion.

NON-ISCHEMIC CRVO



ISCEMIC CRVO

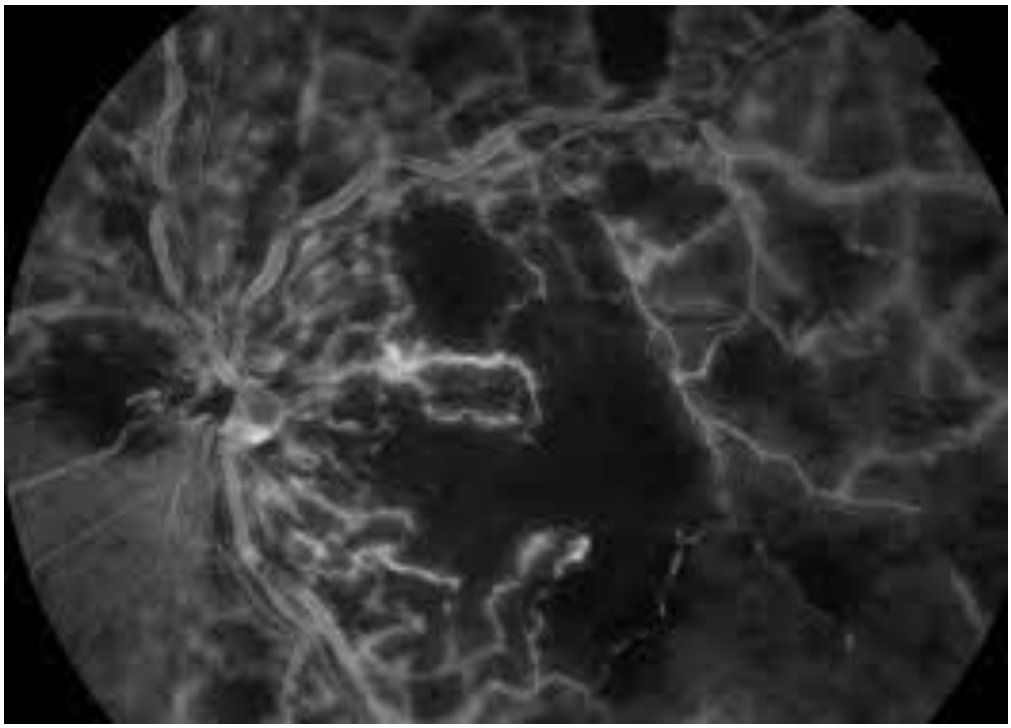


IMPENDING CENTRAL RETINAL VEIN OCCLUSION:

- Occurs in younger patients.
- Prognosis is usually good but a proportion may convert to Ischemic CRVO.
- Symptoms are mild blurring of vision which is worse on waking.
- On fundus examination, there is mild venous dilatation and tortuosity with few scattered dot and blot haemorrhages .
- There may be mild macular edema.
- FAF shows fern like perivenular appearance
- Fluorescein angiography shows impaired retinal circulation

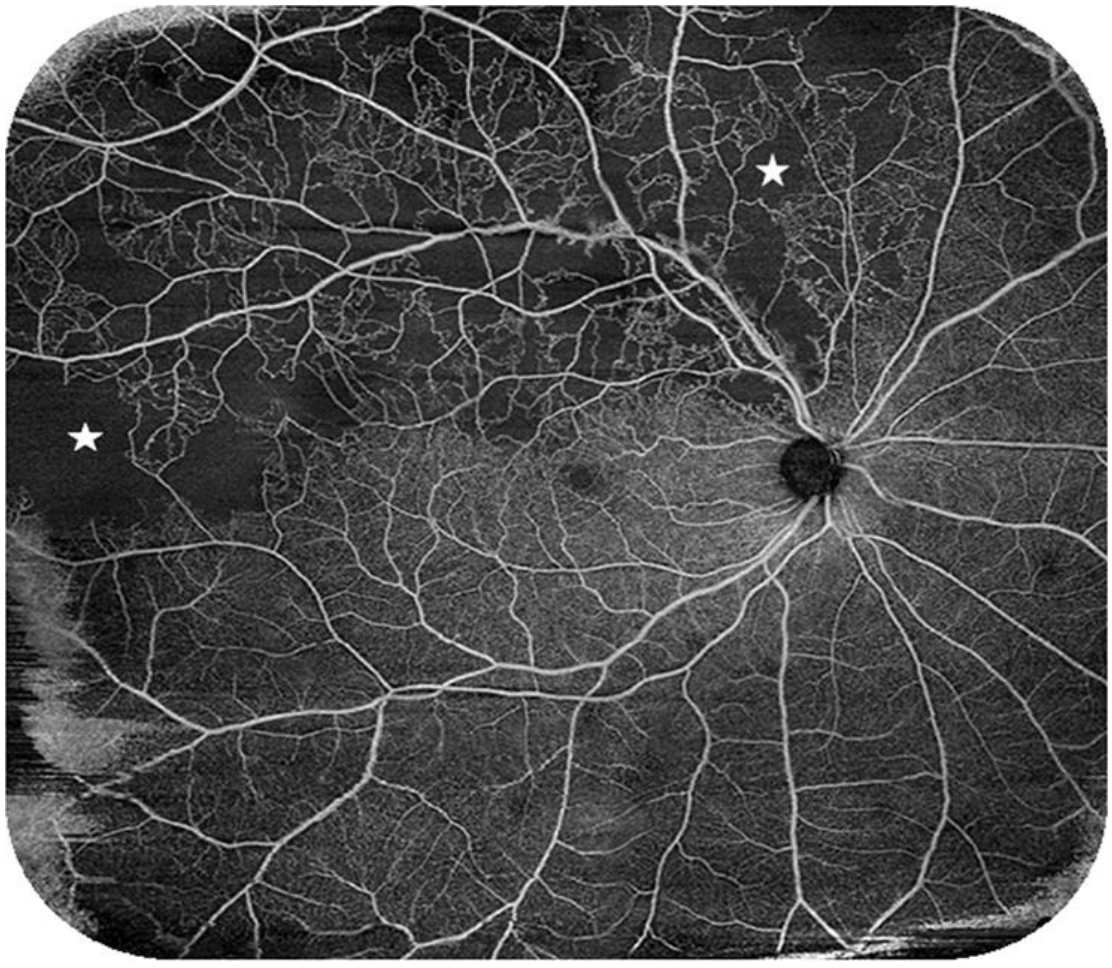
FUNDUS FLUORESCEIN ANGIOGRAPHY OF ISCHEMIC CRVO:

- Prolonged arteriovenous transit time.If it is more than 20 seconds-ISCHEMIA
- Staining of walls of vein -Ischemia.
- Presence of capillary non-perfusion areas may not be seen due to haemorrhages.
- If non-perfusion seen close to fovea-poor prognosis.
- Ischemia causes increase in permeability of vessels results in macular edema with petalloid pattern.
- Decrease in perifoveal blood flow result in enlargement of capillary avascular zone with pruning of vessels in ischemic area.



OCT-ANGIOGRAPHY:

- Abnormalities are seen both in superficial and deep retinal capillary network which is more in deep vessels.
- Mean foveal avascular zone will be larger.
- Inter capillary areas are larger in perifoveal capillaries.



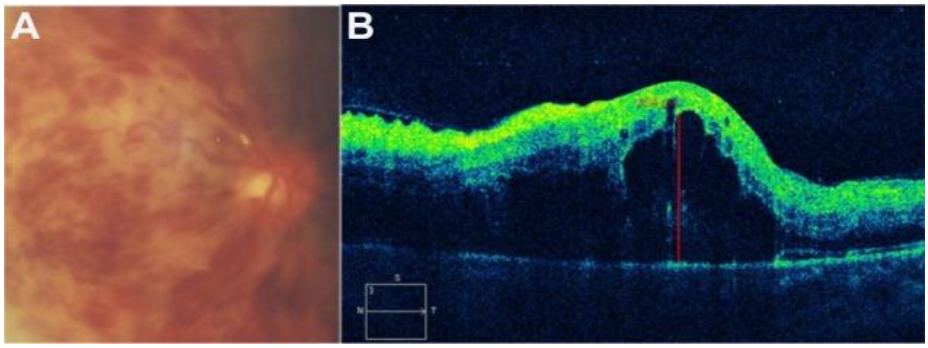
NATURAL COURSE OF CENTRAL RETINAL VEIN OCCLUSION (OCULAR SEQUELAE)

CRVO is a common cause of sight threatening retinal disease. The thrombus formation leads to increased retinal capillary pressure which causes transudation into extracellular space and macular edema.Visual loss from CRVO is most commonly caused by macular edema but may also be caused by macular ischemia , neovascular glaucoma & retinal neovascularization.

The acute features usually resolve within 3-6 months and replaced by exudates , Venous sheathing, collaterals with residual haemorrhages.

- **MACULAR EDEMA**:

- In CRVO, hypoxic retinal tissues releases Vascular endothelial growth factor and inflammatory mediators producing macular edema. Macular edema for chronic period result in epiretinal membrane formation with pigmentary disturbances and macular degenerations.
- Optical Coherence Tomography of Macula is used for quantification of Cystoid macular edema.
- Intravitreal anti-VEGF (Ranibizumab,Bevacizumab) and steroid therapy for refractory cases (IntravitrealTriamcinolone and dexamethasone Implant) are main stay of treatment according to CRUISE and SCORE Study.

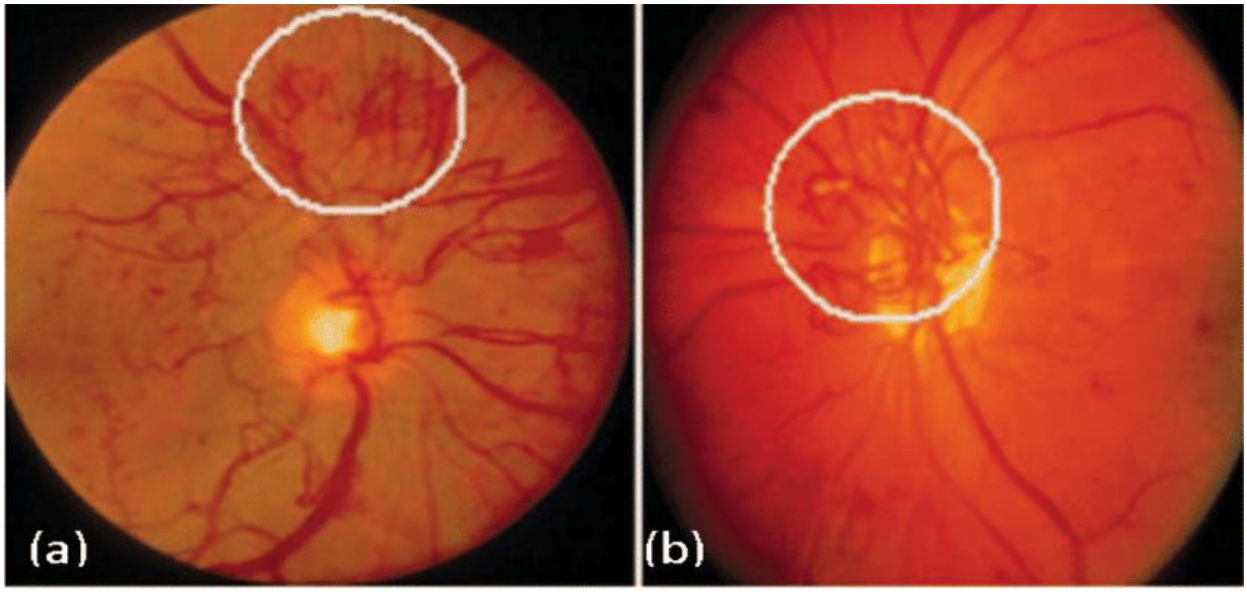


- **NEOVASCULARIZATION:**

- It is one of the dreadful complication of ischemic type of CRVO .It can present as NVE (Neovascularization elsewhere in peripheral retina), NVD (Neovascularization of disc) , NVI (Neovascularization of Iris) or NVA (Neovascularization of angles) resulting in Neovascular glaucoma(NVG).
- The commonest site of ocular neovascularization is anterior segment and less frequently posterior segment and greatest risk for developing anterior segment neovascularization is during the first 7 months after which the risk falls.
- Retinal neovascularization that is within 1 disc diameter of disc is NVD and that is 1 disc diameter away from disc is NVE which occurs as “Raspberry” new vessels in posterior pole which result in vitreous haemorrhage.
- Rubeosis iridis typically develops at the pupillary margin and extends in a skipped pattern to involve anterior chamber angle.
- NVA occurs as fine vessels bridging across scleral spur .

- Neovascular glaucoma which is called “100 days glaucoma” presents with corneal edema, raised intraocular pressure with severe neovascularization of iris and angles & on gonioscopy reveals closed angles. The maximum risk of developing NVG is 45%.
- K Evans et al 1993 studied 73 patients of CRVO with secondary new vessel formation. Ocular neovascularization has occurred in 60% from which 39% developed neovascular glaucoma. Patient with pre-existing primary open angle glaucoma are more likely to develop ocular neovascularization which was less responsive to laser therapy. [14]
- Andrew J Rong et al suggested that risk factors for Neovascular glaucoma development included history of worse visual acuity and RAPD on presentation, history of systemic hypertension . [15] Intravitreal anti-VEGF therapy delayed but did not prevent Neovascular glaucoma.

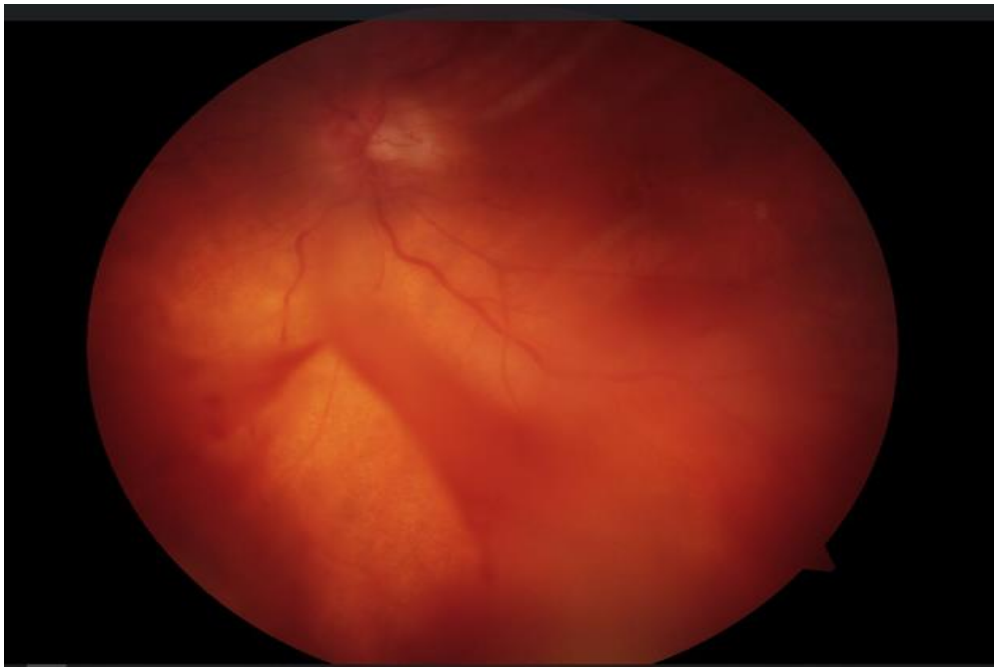
- Hayreh et al suggested that severity of retinal ischemia and followup duration plays a role in incidence of neovascular glaucoma. [17]
- Laser photocoagulation are the mainstay of treatment according to CVOS group



- **VITREOUS HAEMORRHAGE**:

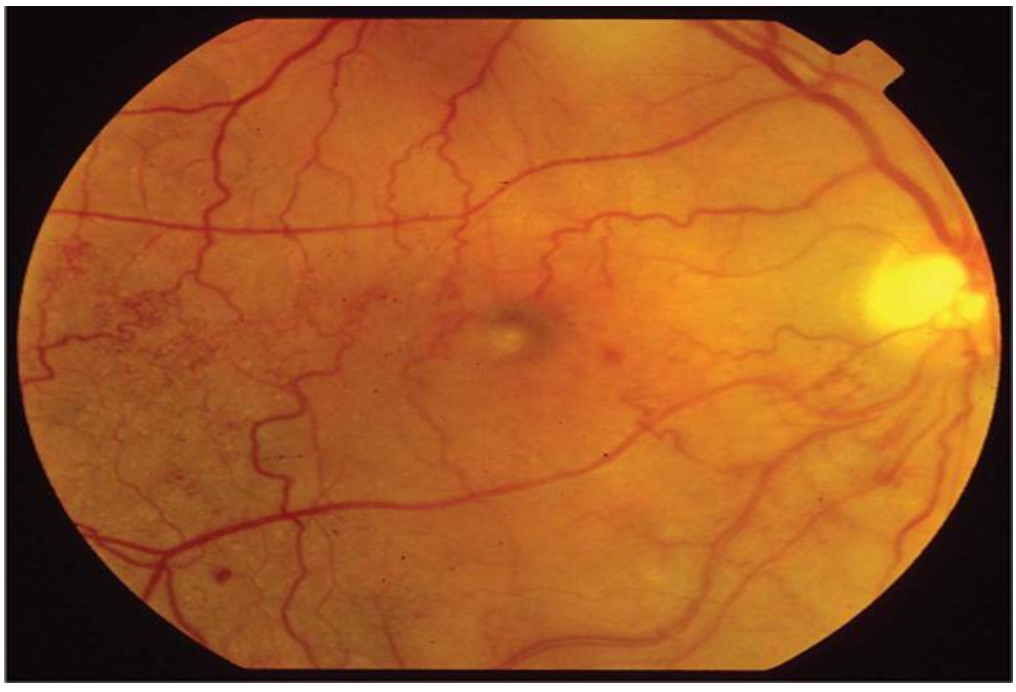
It is may be secondary to retinal & optic disc neovascularization which occurs during late stages of disease or due to rupture of blood through internal limiting membrane in patients with sub-internal limiting membrane haemorrhages.

It can also occurs due to development of posterior vitreous detachment and from intraretinal microvascular abnormalities secondary to CRVO. Incidence of vitreous haemorrhage in CRVO is upto 7%. It result in fibrovascular sequelae of Tractional retinal detachment.



- **COLLATERALS:**

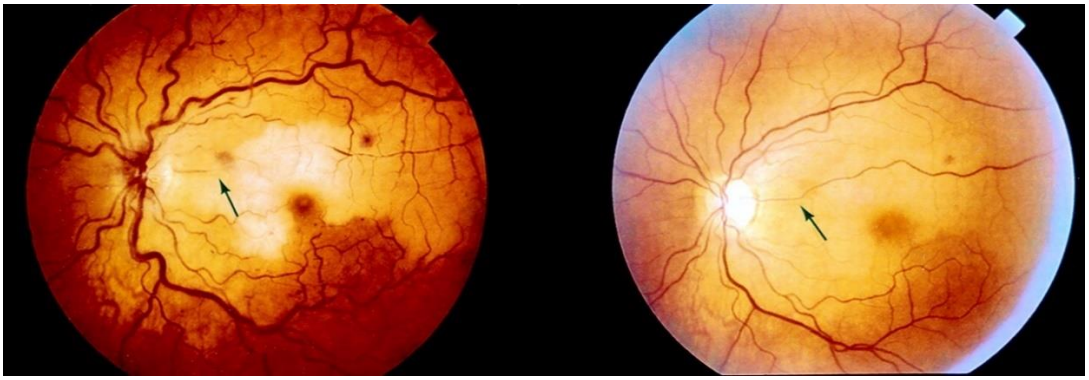
They appears within 4-6 months of onset of CRVO. It is characterized by slightly tortuous veins that develop locally or across the horizontal raphe between superior and inferior vascular arcades and are best detected on Fluorescein angiography.



- **CILIORETINAL ARTERY OCCLUSION:**

Hayreh et al in his study suggested that 38 patients who had CRVO are associated with Cilioretinal artery occlusion. [17]

The pathogenesis of cilioretinal artery occlusion in CRVO is due to transient hemodynamic blockage of cilioretinal artery which is caused by sudden rise in intraluminal pressure of retinal capillary bed due to CRVO above the level of that in cilioretinal artery



TREATMENT

PRIMARY TREATMENT:

- After thorough evaluation of the cause of CRVO ,therapy is directed at treating the primary systemic or ocular pathology as primary treatment or as prophylaxis for prevention of similar disorder in fellow eye.
- Patient with systemic disease should be started on appropriate systemic drugs.
- Patient with raised IOP should be started on antiglaucoma drugs

RHEOLOGICAL THERAPY:

- Hyper or isovolemic hemodilution can be performed which acts by reducing blood viscosity and improving retinal blood flow.
- Contraindications for isovolemic hemodilution are severe cardiorespiratory or renal disease.
- Other rheologically active substances include Pentoxifylline and Troxerutin which act by improving microcirculation in capillaries and venules by inhibiting erythrocyte and platelet aggregation improving erythrocyte deformability.

SECONDARY TREATMENT:

- They are directed at sequelae of CRVO particularly macular edema and neovascularization.

- Macular edema is treated with monthly injection of intravitreal anti-VEGF for 3-6 months
    - ❖ CRUISE STUDY- Intravitreal Ranibizumab 0.5mg in 0.05ml
    - ❖ COPERNICUS & GALLILEO STUDY-Intravitreal Aflibercept 2mg.
- Intravitreal corticosteroid therapy can be given for refractory cases of macular edema.
    - ❖ SCORE STUDY-Intravitreal Triamcinolone 4mg in 0.1ml
    - ❖ GENEVA STUDY-Intravitreal dexamethasone implant including Ozurdex or Retisert.
- Treatment of ocular neovascularization include laser photocoagulation (PRP) which is delivered promptly in patients with NVD/NVE to avoid anterior segment neovascularization.
- Prophylactic PRP is indicated in high risk cases such as
    - ❖ Male gender
    - ❖ Short duration of CRVO.
    - ❖ Cases in whom frequent ophthalmologic followup is not possible.
    - ❖ Extensive retinal nonperfusion & haemorrhages.

ALTERNATIVE TREATMENT:

- Oral Pentoxifylline which is potent vasodilator.
- Local thrombolysis with tissue plasminogen activator either by intravitreal injection or during vitreoretinal surgery by direct injection into the occluded vessels.
- Chorioretinal venous anastomosis
- Pars plana vitrectomy for eyes with non-clearing vitreous haemorrhage from secondary retinal neovascularization.

PART-II

AIMS AND OBJECTIVES

1) To study the most common risk factors and systemic association of CRVO in Non-diabetic individuals.
2) To compare the demographic characteristics such as age , sex between Ischemic and non-ischemic variety of CRVO.
3) To study the natural course and ocular sequelae of CRVO
4) To compare the final ocular outcome in non-ischemic and ischemic CRVO.

MATERIALS AND METHODOLOGY

MATERIALS:

All patients with symptoms & signs of Central retinal vein occlusion attending RIOGOH services who are satisfying inclusion criteria are taken up for study after taking informed and written consent for a period of 12 months.

METHODOLOGY:

- Patient presenting to Vitreoretina services RIOGOH was registered and evaluated.
- Detailed medical history will be taken. Complete general examination with vitals measurement such as BP,PR & systemic examination was

performed to rule out the presence of any systemic disease predisposing to a vein occlusion. Duration of systemic disease and its control will be recorded. In doubtful cases, specialist opinion was obtained.

- A detailed ocular history which included history of any previous ocular disease such as glaucoma, and their treatment will be elicited.In female patient history of oral contraceptive usage was elicited.
- Ocular examination at time of initial presentation of the patient included the following:

  - ❖ Best corrected visual acuity using snellen's chart
  - ❖ Pupillary reaction
  - ❖ Slit lamp biomicroscopy of anterior segment.
  - ❖ IOP measurement with Golmann's applanatation tonometer.
  - ❖ Slit lamp biomicroscopy with 90D lens.
  - ❖ Direct and Indirect ophthalmoscopy.
  - ❖ Fundus Fluorescein Angiography(FFA)(If needed)
  - ❖ Optical coherence tomography of macula(to quantify macular edema)

- Baseline investigation will be carried out immediately and thorough lab workup will be carried as follow,

❖ Haemoglobin

- ❖ Complete blood count
- ❖ Fasting and post prandial blood sugar levels.
- ❖ Lipid profile.
- ❖ Blood Urea,Serum Creatinine.
- ❖ Erythrocyte sedimentation rate(ESR)
- ❖ Serum homocysteine in young patients
- ❖ Coagulation studies
- ❖ Autoantibodies like ANA,ANCA - selected cases.
- ❖ Chest x-ray - selected cases
- ❖ Blood VDRL for Syphilis
- ❖ ELISA for HIV
- ❖ Electrophysiological test:
    - Electrocardiogram
    - Echo cardiogram
- ❖ Carotid doppler for selected patients.

- The appearance of unilateral disc edema with dilatation and tortuosity of major blood vessels of retina and varying amount of retinal haemorrhage were considered to have Central Retinal Vein Occlusion.
- After the initial visit ,patient will be followed up every month for a minimum period of 3 months.
- The following criteria is chosen to classify the patient in ischemic and

non-ischemic :

- ❖ Poor visual acuity <6/60
- ❖ Presence of Relative Afferent Pupillary Defect(RAPD)
- ❖ Fundus showing extensive haemorrhages and cotton wool spots.
- ❖ Fundus Fluorescein Angiography showing capillary non perfusion areas greater than 10 disc diameters.

- INCLUSION CRITERIA:

❖ Confirmed presence of CRVO

❖ Patient of all ages presenting with CRVO.

❖ Non-diabetic individuals with CRVO.

- EXCLUSION CRITERIA:

❖ Presence of Branch Retinal Vein Occlusion(BRVO)

❖ Diabetic Retinopathy or any other retinal vascular disease or with Vitreous haemorrhage.

❖ Previous history of any photocoagulation done for retinal vascular disease.

❖ Uncooperative patients who are not willing to adhere to the followup.

❖ Patient with media opacity whose evaluation of fundus is not possible

like mature cataract will be excluded.

OBSERVATIONS AND RESULTS:

TABLE 1: AGE DISTRIBUTION

Age distribution		
	Frequency	Percent
45 - 50 yrs	6	12.0
51 - 55 yrs	11	22.0
56 - 60 yrs	16	32.0
61 - 65 yrs	15	30.0
66 - 70 yrs	2	4.0
Total	50	100.0

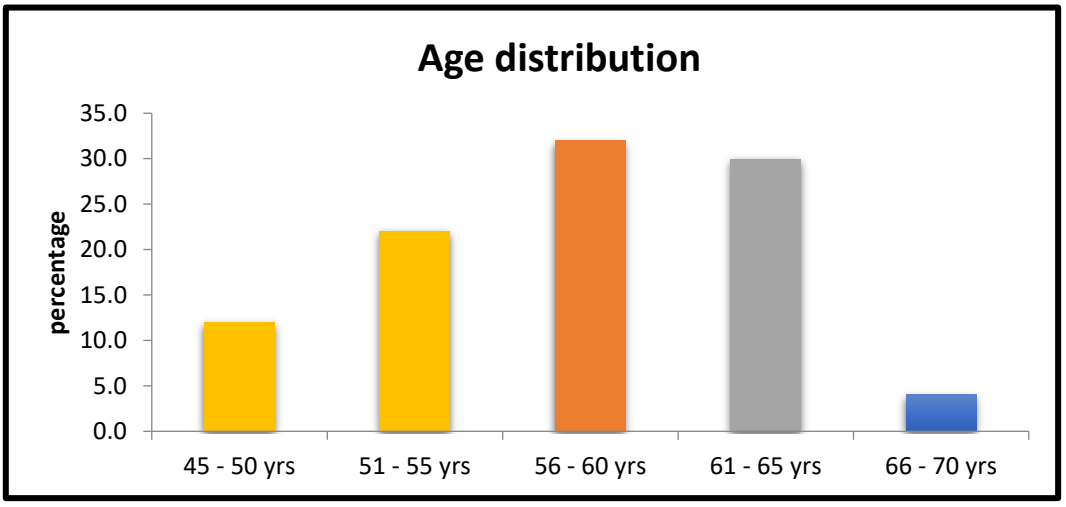


Figure 1

The above table shows Age distribution were 45-50 years is 12.0%, 51-55 years is 22.0%, 56-60 years is 32.0%, 61-65 years is 30.0%, 66-70 years is 4.0%. This study shows that majority of the patients are in age group of 56-65 years (62%)

TABLE 2: GENDER DISTRIBUTION

Gender distribution		
	Frequency	Percent
Female	15	30.0
Male	35	70.0
Total	50	100.0

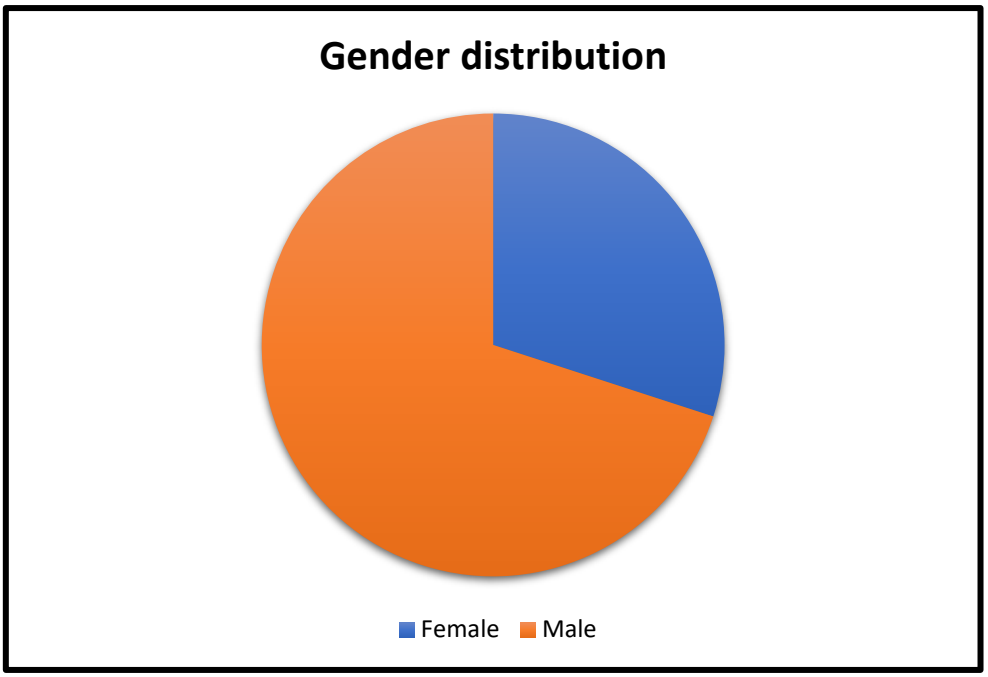
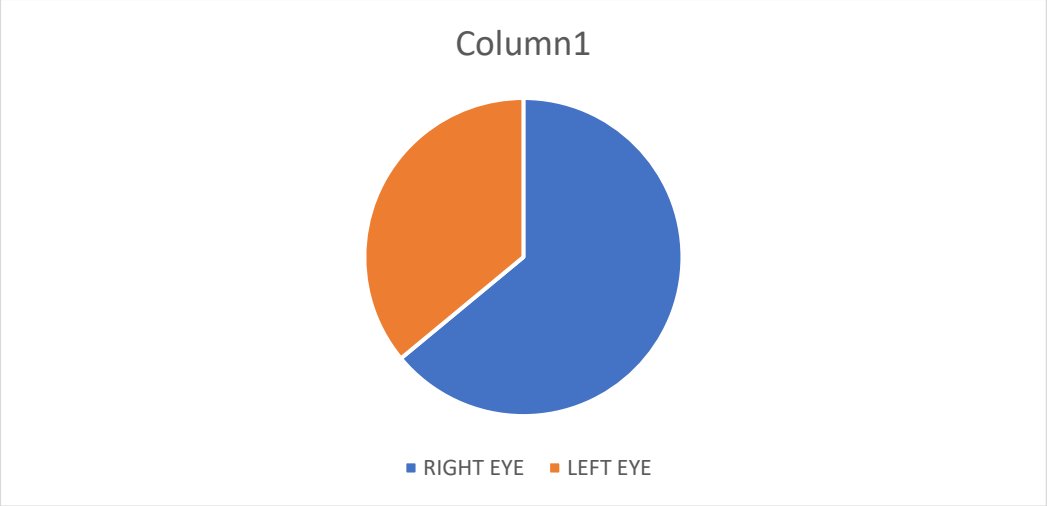


Figure 2

The above table shows Gender distribution were Female is 30.0%, Male is 70.0%.This study group consists of 35 males(70%) and 15 females(30%).

TABLE 3:DISTRIBUTION OF LATERALITY OF CRVO

Laterality of CRVO distribution		
	Frequency	Percent
Right eye	32	64.0
Left eye	18	36.0
Total	50	100.0



In this study all the cases are unilateral with right eye affected in 32 cases(64%)

And left eye affected in 18 cases (36%)

TABLE 4: DISTRIBUTION OF TYPE OF CRVO

Type of CRVO		
	Frequency	Percent
Ischemic	16	32.0
Non-Ischemic	34	68.0
Total	50	100.0

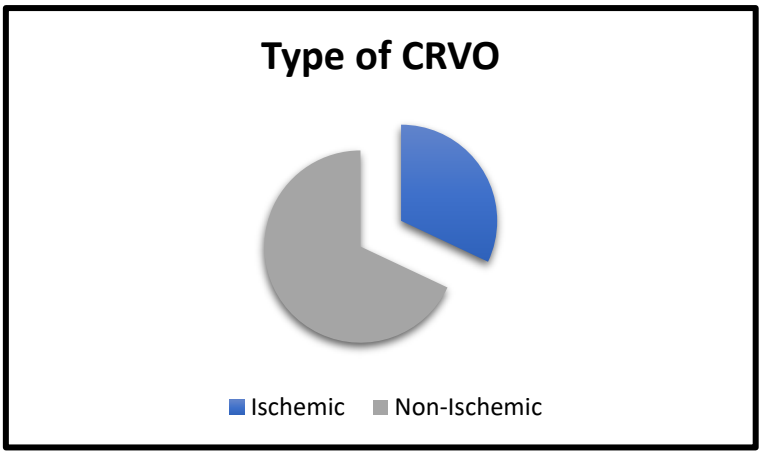


Figure 3

The above table shows Type of CRVO distribution in which out of 50 cases 34 cases were non-Ischemic (68%) and 16 cases were ischemic(32%).

In the end of study out of 34 case of non-ischemic CRVO 5 cases(10%) converted to Ischemic CRVO.

TABLE 5: DISTRIBUTION OF ASSOCIATED SYSTEMIC AND OCULAR CONDITION

Associated Systemic & Ocular condition		
	Frequency	Percent
Chronic Renal Failure	4	8.0
Coronary Artery Disease	1	2.0
Homocysteinemia	2	4.0
Hyperlipidemia	14	28.0
Primary Open Angle Glaucoma	3	6.0
Systemic Hypertension	26	52.0
Total	50	100.0

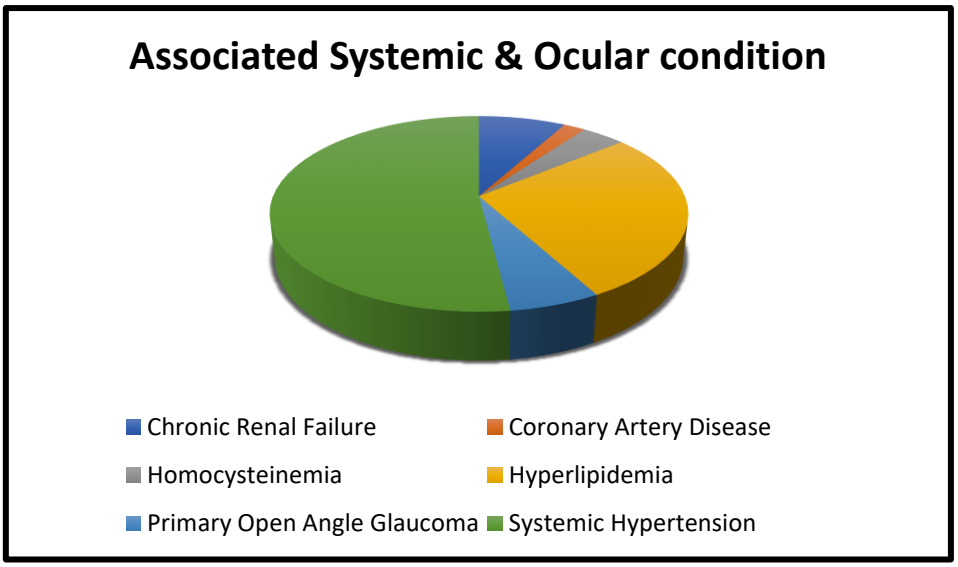


Figure 4

The above table shows distribution of Associated systemic & Ocular condition were Chronic Renal Failure is 8.0%, Coronary Artery Disease is 2.0%, Homocysteinemia is 4.0%, Hyperlipidemia is 28.0% Primary Open Angle Glaucoma is 6.0%, Systemic Hypertension is 52.0%.

Out of 50 patients ,26 patients had associated systemic hypertension which is the largest cause among them. Out of 26 patients of associated systemic hypertension 10 patients were on irregular treatment, 10 patients were on treatment but without adequate dosage, 6 patients were diagnosed to have systemic hypertension after onset of CRVO.

TABLE 6:DISTRIBUTION OF CLINICAL MANIFESTATION

Clinical manifestation		
	Frequency	Percent
Sudden Blurring of Vision	33	66.0
Sudden Blurring of Vision with RAPD	1	2.0
Sudden Painless Loss of Vision	1	2.0
Sudden Painless Loss of Vision with RAPD	15	30.0
Total	50	100.0

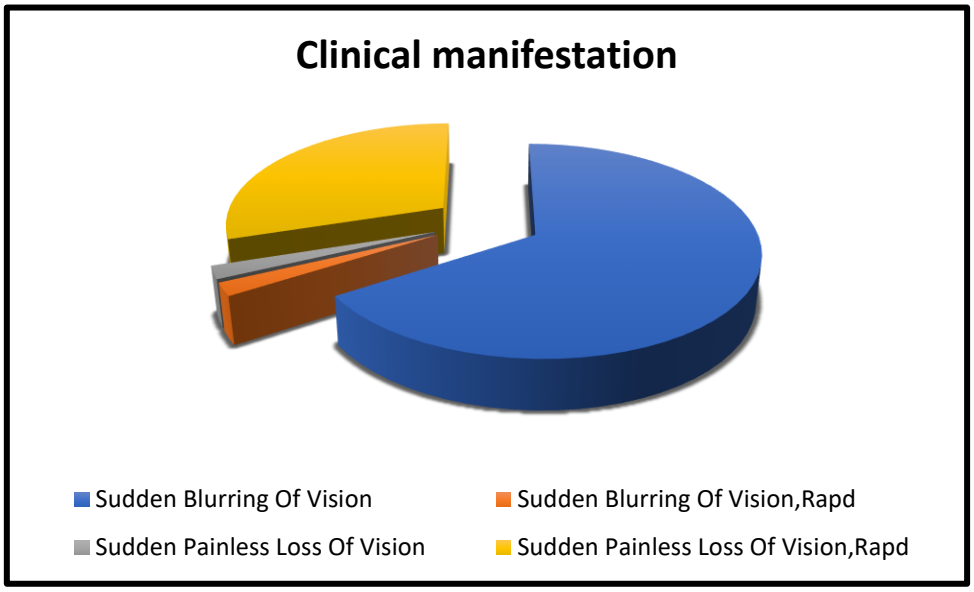


Figure 5

The above table shows Clinical manifestation distribution were Sudden Blurring Of Vision is 66.0%, Sudden Blurring Of Vision with RAPD is 2.0% Sudden Painless Loss Of Vision is 2.0%, Sudden Painless Loss Of Vision with RAPD is 30.0% .

TABLE 7 :DISTRIBUTION OF OCULAR SEQUELAE DURING 1-3 MONTHS FOLLOW-UP

Occular sequelae during 1 - 3 months follow up		
	Frequency	Percent
Collaterals With Sheathing of Vessels	5	10.0
NVI	1	2.0
Converted To Ischemic	4	8.0
Hard Exudates	9	18.0
Macular Edema	19	38.0
Neovascular Glaucoma	7	14.0
NVE	2	4.0
Vitreous Haemorrhage	3	6.0
Total	50	100.0

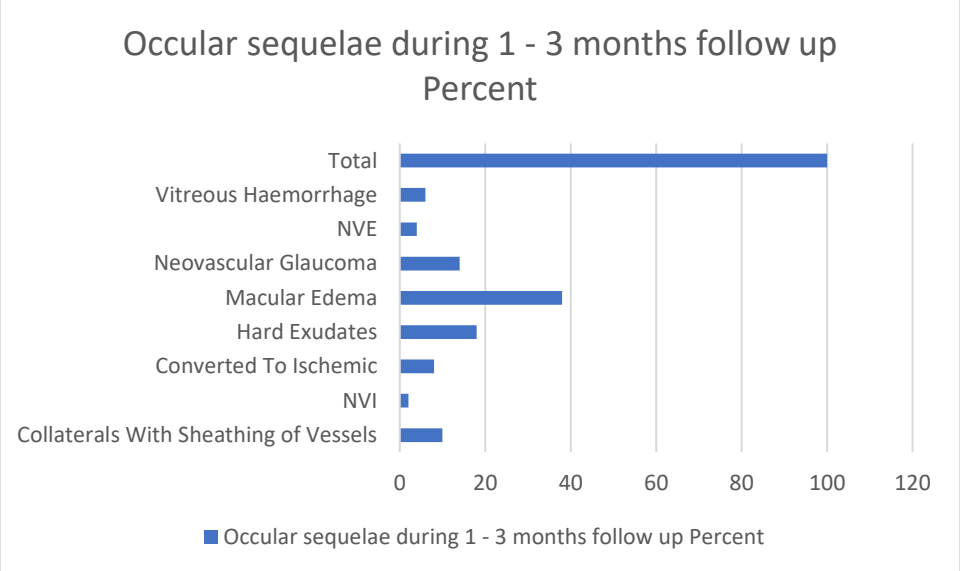


Figure 6

The above table shows Ocular sequelae during 1 - 3 months follow up distribution were Collaterals with Sheathing Of Vessels is 10.0%, Converted To Ischemic is 10.0%, Hard Exudates is 18.0%, Macular Edema is 38.0%,

Neovascular Glaucoma is 14.0%, NVE is 4.0%,Vitreous Haemorrhage is 6.0%.

Table 8: Comparison of Age with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
AGE	45 - 50 yrs	Count	0	6	6
		%	0.0%	17.6%	12.0%
	51 - 55 yrs	Count	1	10	11
		%	6.3%	29.4%	22.0%
	56 - 60 yrs	Count	5	11	16
		%	31.3%	32.4%	32.0%
	61 - 65 yrs	Count	8	7	15
		%	50.0%	20.6%	30.0%
	66 - 70 yrs	Count	2	0	2
		%	12.5%	0.0%	4.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

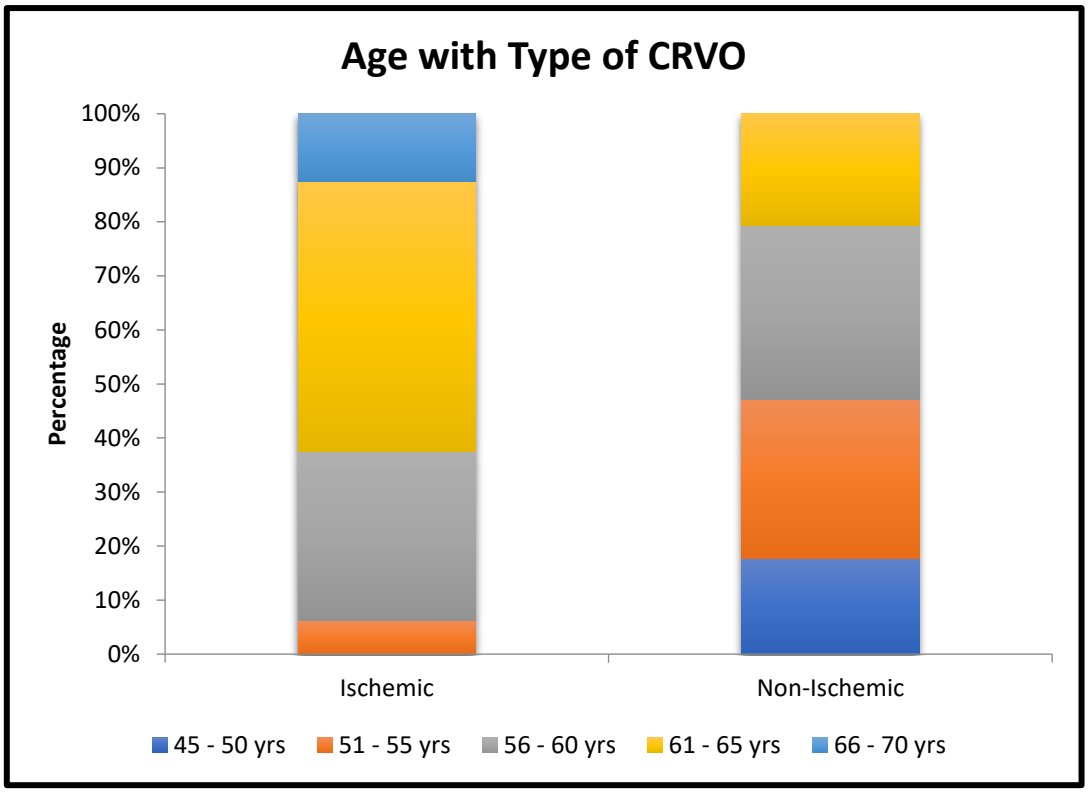


Figure 7

The above table shows comparison of Age with Type of CRVO by Pearson's Chi-Square test were $\chi 2=12.868$, $p=0.012<0.05$ which shows statistical significance with Age and Type of CRVO.

- Majority of non-ischemic CRVO patients are in age group of 50-60 yrs Contributing to 61.8%.
- Majority of ischemic CRVO patients are in age group of 55-65 yrs Contributing to 81.3%

Table 9: Comparison of Gender with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
Gender	Female	Count	6	9	15
		%	37.5%	26.5%	30.0%
	Male	Count	10	25	35
		%	62.5%	73.5%	70.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

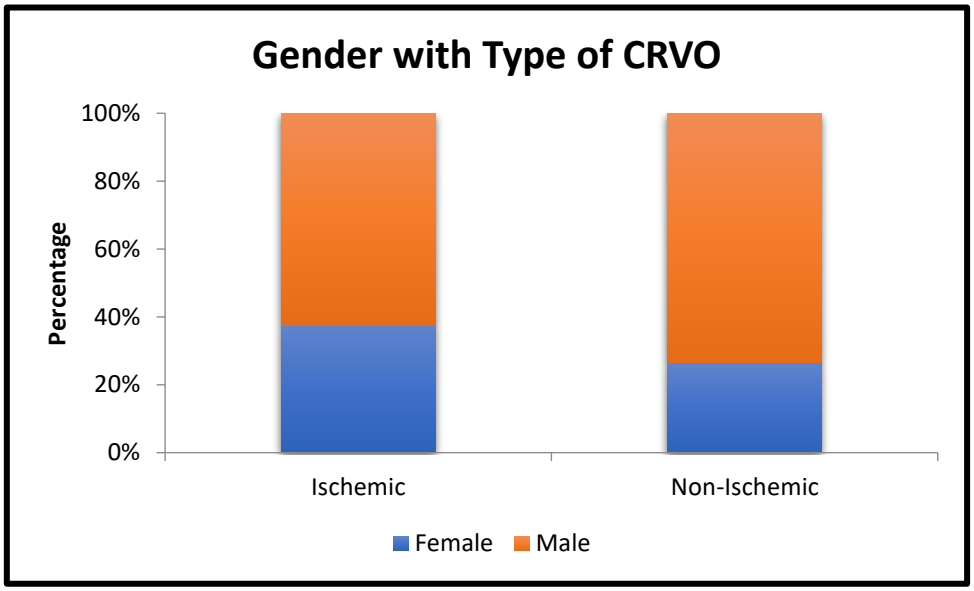


Figure 8

The above table shows comparison of Gender with Type of CRVO by Pearson's Chi-Square test were $\chi 2=0.630$, $p=0.427>0.05$ which shows no statistical significance with Gender and Type of CRVO.

- In our study most of patients are males contributing to 62.5% in ischemic group and 73.5% in non-ischemic group.

- Female patients in our study contributes to 37.5% in Ischemic group and 26.5% in non-ischemic group.

Table 10: Comparison of Leterality with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
Leterality	Left eye	Count	10	8	18
		%	62.5%	23.5%	36.0%
	Right eye	Count	6	26	32
		%	37.5%	76.5%	64.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

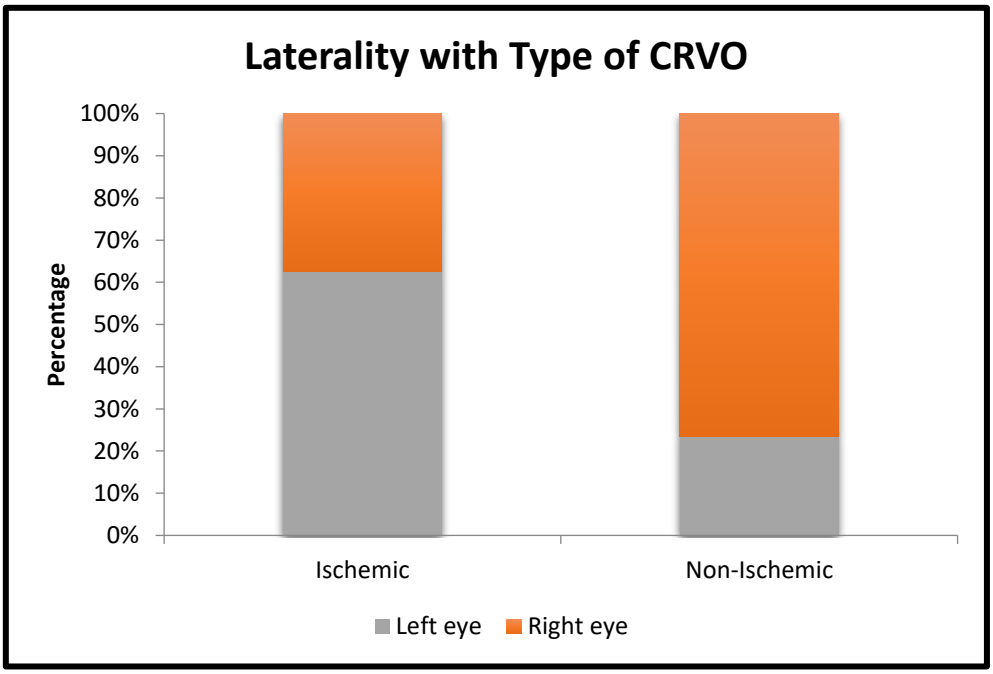


Figure 9

The above table shows comparison of Laterality with Type of CRVO by

Pearson's Chi-Square test were $\chi 2=7.172$, $p=0.007<0.01$ which shows highly statistical significance with Laterality and Type of CRVO.

- In our study Right eye was affected as 76.5% in non-ischemic group and 37.5% in ischemic group.
- Left eye was affected as 23.5% in non-ischemic group and 62.5% in Ischemic group.
- Thus in our study Right eye was more affected in non-ischemic group And Left eye was more affected in ischemic group.

Table 11: Comparison of Associated Systemic & Ocular condition with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
Associated Systemic & Ocular condition	Chronic Renal Failure	Count	2	2	4
		%	12.5%	5.9%	8.0%
	Coronary Artery Disease	Count	0	1	1
		%	0.0%	2.9%	2.0%
	Homocysteinemia	Count	0	2	2
		%	0.0%	5.9%	4.0%
	Hyperlipidemia	Count	3	11	14
		%	18.8%	32.4%	28.0%
	Primary Open Angle Glaucoma	Count	0	3	3
		%	0.0%	8.8%	6.0%
	Systemic Hypertension	Count	11	15	26
		%	68.8%	44.1%	52.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

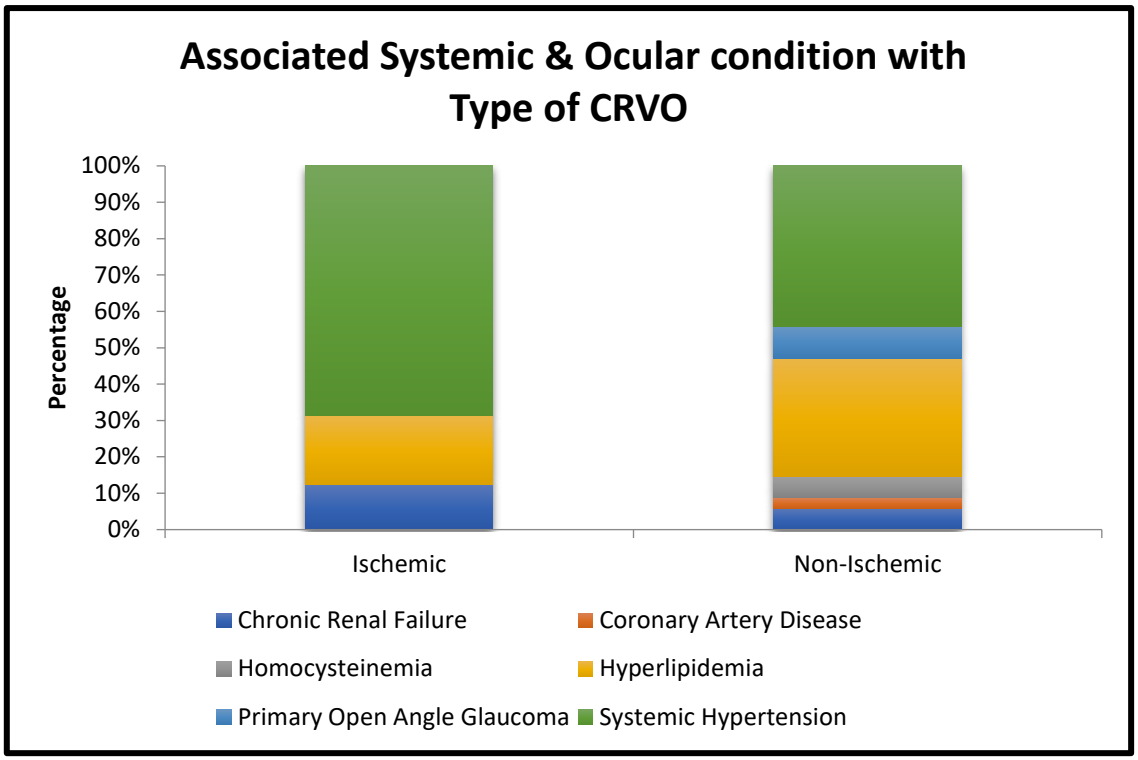


Figure 10

The above table shows comparison of Associated Systemic & Ocular condition with Type of CRVO by Pearson's Chi-Square test were $\chi 2=5.408$, $p=0.368>0.05$ which shows no statistical significance with Associated Systemic & Ocular condition and Type of CRVO.

- In both ischemic and non-ischemic CRVO ,Systemic hypertension was the most common systemic association contributing to 68.8% in ischemic and 44.1% in non-ischemic CRVO.

Table 12: Comparison of Clinical Manifestation with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
Clinical Manifestation	Sudden Blurring of Vision	Count	1	32	33
		%	6.3%	94.1%	66.0%
	Sudden Blurring of Vision,RAPD	Count	0	1	1
		%	0.0%	2.9%	2.0%
	Sudden Painless Loss of Vision	Count	0	1	1
		%	0.0%	2.9%	2.0%
	Sudden Painless Loss of Vision,RAPD	Count	15	0	15
		%	93.8%	0.0%	30.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

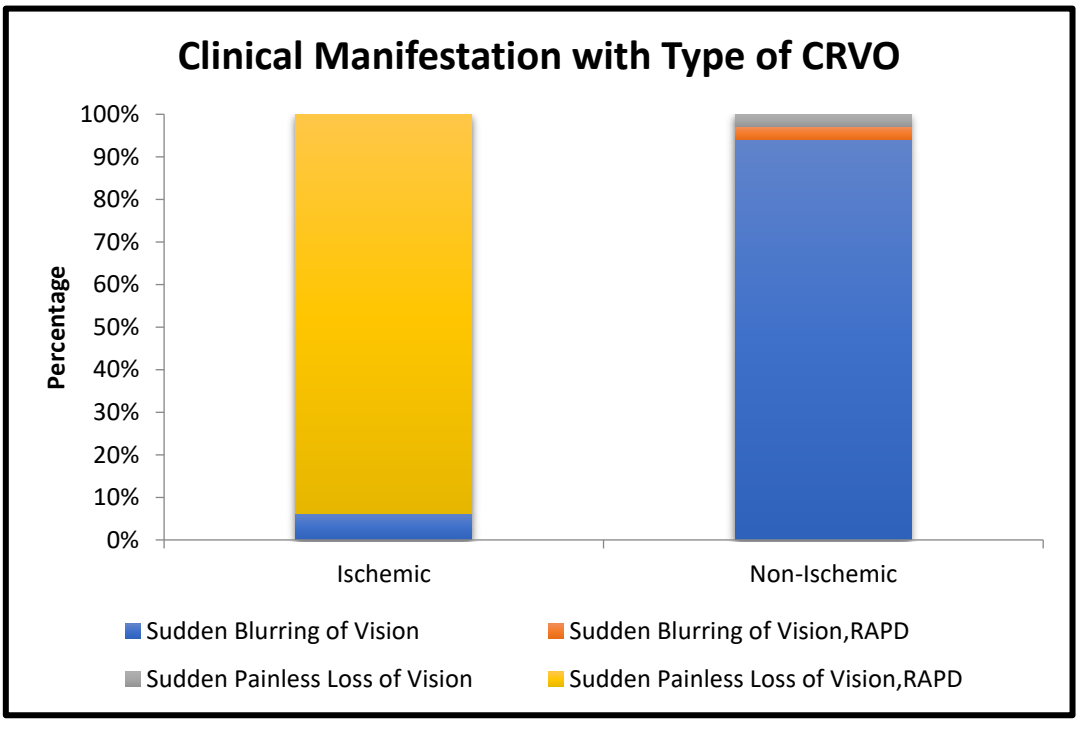


Figure 11

The above table shows comparison of Clinical Manifestation with Type of CRVO by Pearson's Chi-Square test were $\chi 2=45.544$, $p=0.0005<0.01$ which shows highly statistical significance with Clinical Manifestation and Type of CRVO.

- In Ischemic CRVO , there was sudden painless loss of vision with RAPD
- In non-ischemic CRVO, there was sudden blurring of vision only.
- Non-ischemic CRVO converted to ischemic CRVO presented with Sudden blurring of vision and RAPD.

Table 13: Comparison of Ocular squelae during 1-3 months followup with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
Ocular sequelae during 1-3 months followup	Collaterals With Sheathing of Vessels	Count	0	5	5
		%	0.0%	14.7%	10.0%
	Neovascularization of iris	count	1	0	1
		%	6.25%	0.0%	2.0%
	Converted to Ischemic	Count	0	4	4
		%	0.0%	11.8%	8.0%
	Hard Exudates	Count	0	9	9
		%	0.0%	26.5%	18.0%
	Macular Edema	Count	3	16	19
		%	18.8%	47.1%	38.0%
	Neovascular Glaucoma	Count	7	0	6
		%	43.8%	0.0%	14.0%
	NVE	Count	2	0	2
		%	12.5%	0.0%	4.0%
	Vitreous Haemorrhage	Count	3	0	3
		%	18.8%	0.0%	6.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

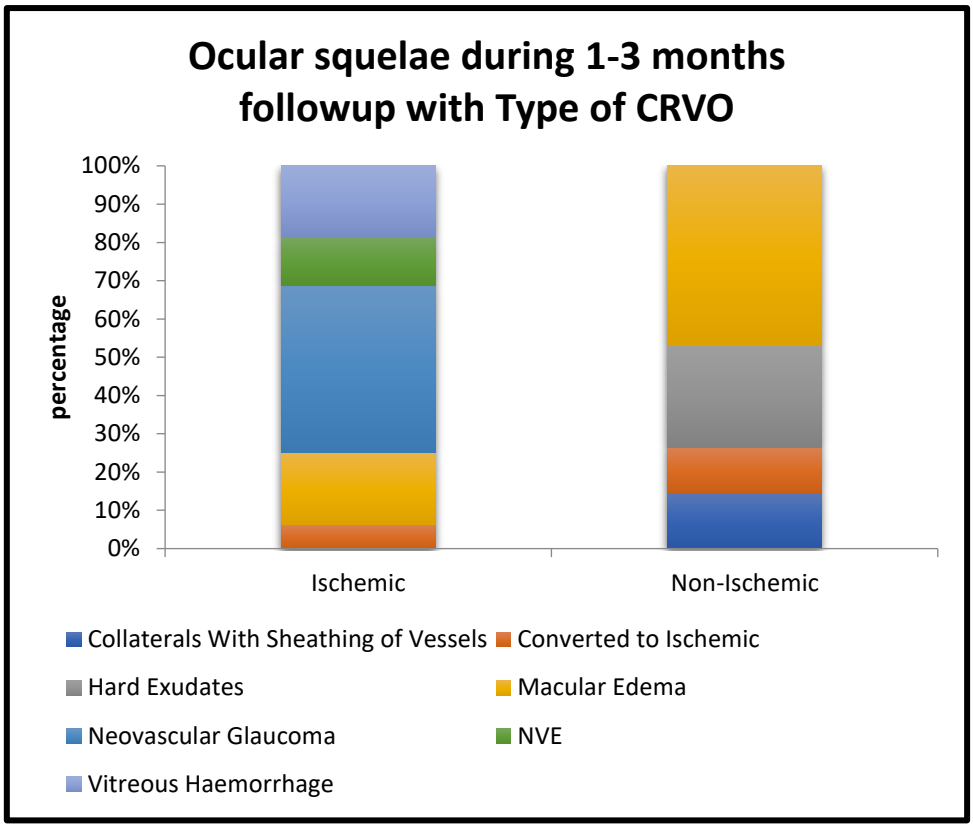


Figure 12

The above table shows comparison of Ocular squelae during 1-3 months followup with Type of CRVO by Pearson's Chi-Square test were $\chi 2=34.714$, $p=0.0005<0.01$ which shows highly statistical significance with Ocular squelae during 1-3 months followup and Type of CRVO.

- Most of Non-ischemic CRVO leads to macular edema (47.1%), Hard exudates (26.5%). Some patient got converted to ischemic Variety(11.8%).Some patients resolved with collateral formation & Sheathing of vessels.
- Most of ischemic CRVO patients lead to Neovascular glaucoma(43.8%).

18.8% of ischemic CRVO patients lead to macular edema and vitreous Haemorrhage.12.5% patients lead to NVE, 6.25% developed NVI.

Table 14: Comparison of Initial BCVA with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
Initial BCVA	6/6 - 6.12	Count	0	1	1
		%	0.0%	2.9%	2.0%
	6/18 - 6/24	Count	0	14	14
		%	0.0%	41.2%	28.0%
	6/36 - 6/60	Count	0	18	18
		%	0.0%	52.9%	36.0%
	< 6/60	Count	0	1	1
		%	0.0%	2.9%	2.0%
	< 2/60	Count	15	0	15
		%	93.8%	0.0%	30.0%
	HM-PL+	Count	1	0	1
		%	6.3%	0.0%	2.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

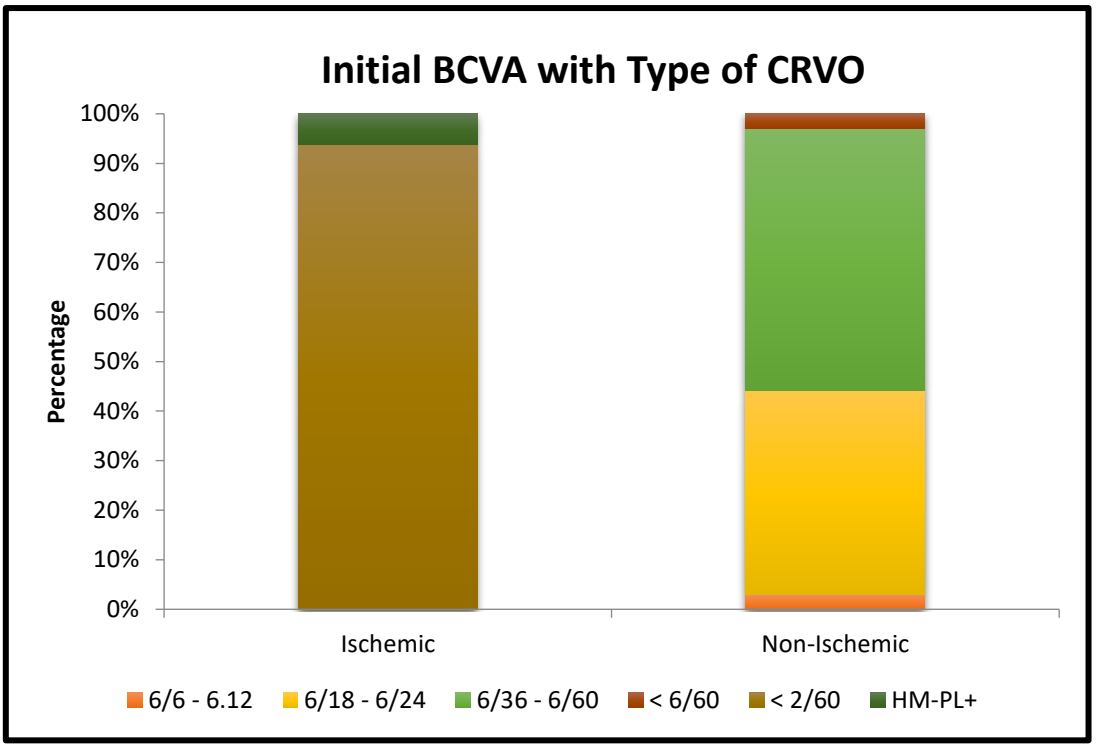


Figure 13

The above table shows comparison of Initial BCVA with Type of CRVO by Pearson's Chi-Square test were $\chi 2=50.000$, $p=0.0005<0.01$ which shows highly statistical significance with initial BCVA and Type of CRVO.

- Most of non-ischemic CRVO patients presented with initial visual acuity of 6/36-6/60.
- Most of ischemic CRVO patients presented with initial visual acuity of Less than 2/60.

Table 15: Comparison of BCVA During followup with Type of CRVO by Pearson's Chi-Square test

			Type of CRVO		Total
			Ischemic	Non-Ischemic	
BCVA During followup	6/6 - 6.12	Count	0	3	3
		%	0.0%	8.8%	6.0%
	6/18 - 6/24	Count	0	9	9
		%	0.0%	26.5%	18.0%
	6/36 - 6/60	Count	0	10	10
		%	0.0%	29.4%	20.0%
	< 6/60	Count	0	12	12
		%	0.0%	35.3%	24.0%
	< 2/60	Count	8	0	8
		%	50.0%	0.0%	16.0%
	HM-PL+	Count	8	0	8
		%	50.0%	0.0%	16.0%
Total		Count	16	34	50
		%	100.0%	100.0%	100.0%

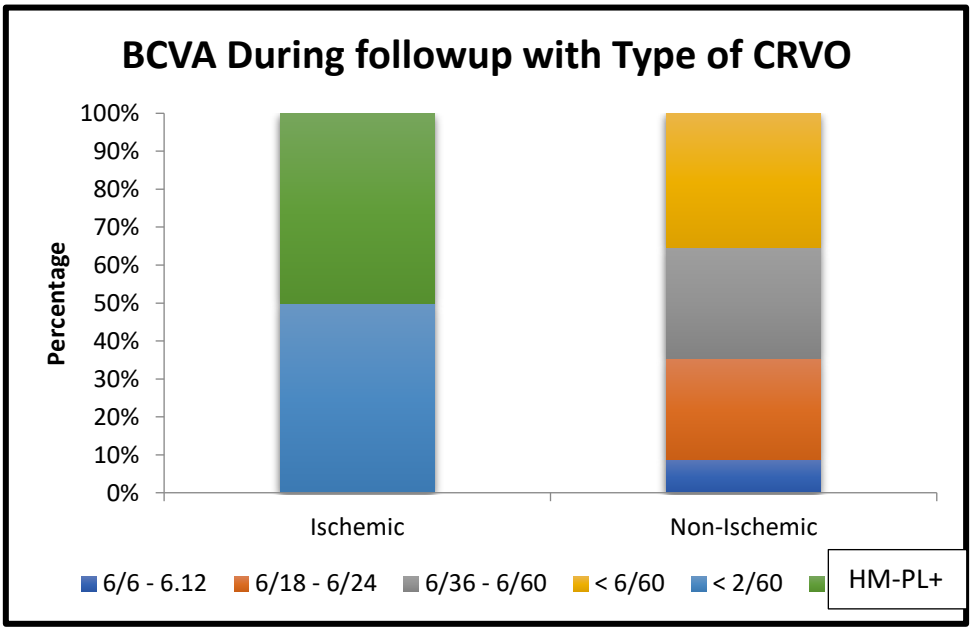


Figure 14

The above table shows comparison of BCVA During followup with Type of

CRVO by Pearson's Chi-Square test were $\chi 2=50.000$, $p=0.0005<0.01$ which shows highly statistical significance with BCVA During followup and Type of CRVO

- Thus ischemic CRVO group showed marked decrease in visual acuity from baseline during followup compared to non-ischemic CRVO.

Table 16: Comparison of Ischemic CRVO with BCVA During Initial visit and followup by Fisher's Exact test

			BCVA During followup		Total
			< 2/60	HM-PL+	
Initial BCVA	< 2/60	Count	8	7	15
		%	50.0%	43.8%	93.8%
	HM-PL+	Count	0	1	1
		%	0.0%	6.3%	6.3%
Total		Count	8	8	16
		%	50.0%	50.0%	100.0%

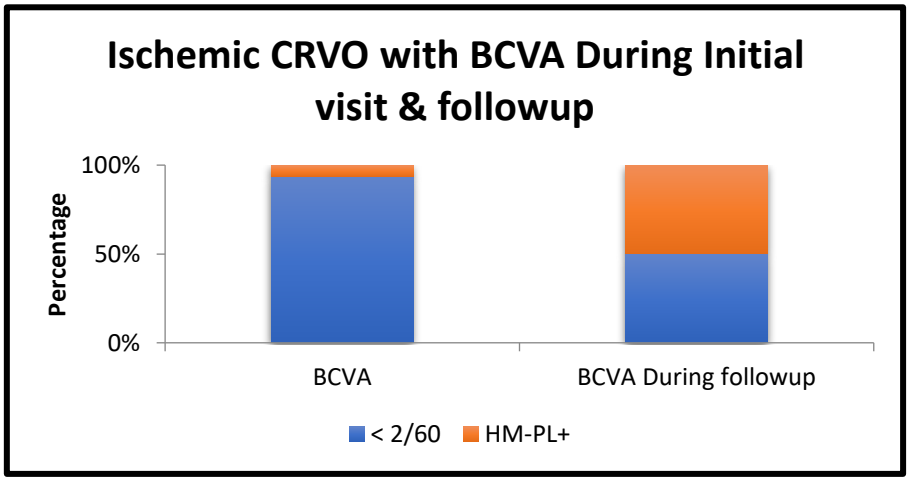


Figure 15

The above table shows comparison of Ischemic CRVO with BCVA During Initial visit and followup by Fisher's Exact test were $\chi 2=1.067$, $p=1.000>0.05$ which shows no statistical significance with Ischemic and BCVA During initial visit & followup..

- In our study in ischemic group majority of patients (93.8%) presented with initial visual acuity of less than 2/60 to CFCF(counting finger close to face) and 6.3% presented with initial visual acuity of HM-PL+.
- At the end of study during the followup 56.3% retained the same visual acuity of less than 2/60 to CFCF, and remaining 43.8% of patients had decreased visual acuity from initial baseline to HM-PL+.

Table 17: Comparison of Non-Ischemic CRVO with BCVA During Initial visit & followup by Pearson's Chi-Square test

			BCVA During followup				Total
			6/6 - 6.12	6/18 - 6/24	6/36 - 6/60	< 6/60	
Initial BCVA	6/6 - 6.12	Count	1	0	0	0	1
		%	2.9%	0.0%	0.0%	0.0%	2.9%
	6/18 - 6/24	Count	2	8	3	1	14
		%	5.9%	23.5%	8.8%	2.9%	41.2%
	6/36 - 6/60	Count	0	1	7	10	18
		%	0.0%	2.9%	20.6%	29.4%	52.9%
	< 6/60	Count	0	0	0	1	1
		%	0.0%	0.0%	0.0%	2.9%	2.9%

Total	Count	3	9	10	12	34
	%	8.8%	26.5%	29.4%	35.3%	100.0%

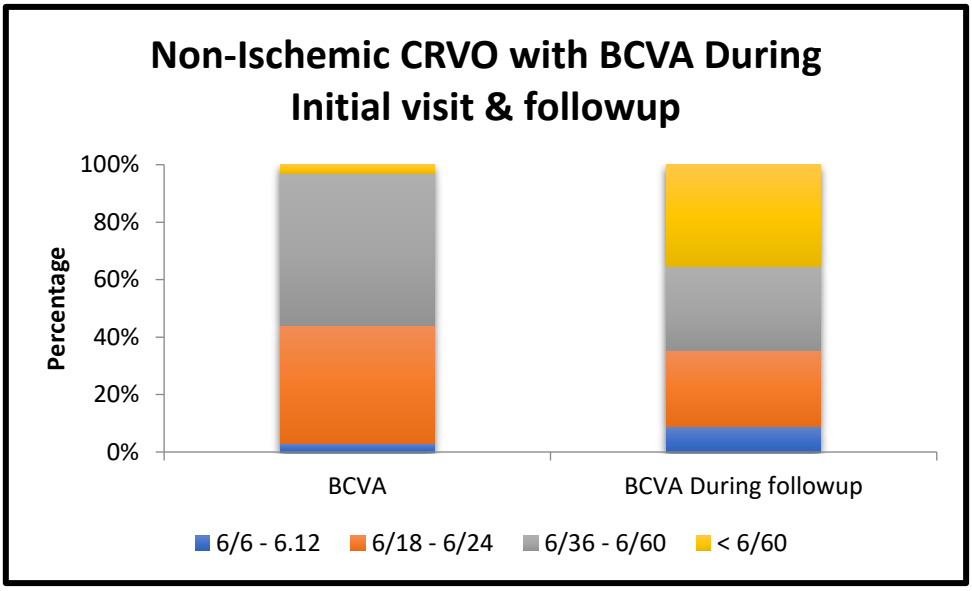


Figure 16

The above table shows comparison of Non-Ischemic CRVO with BCVA During Initial visit and followup by Pearson's Chi-Square test were $\chi 2=28.269$, $p=0.001<0.01$ which shows highly statistical significance with Non-Ischemic CRVO and BCVA During initial visit and followup.

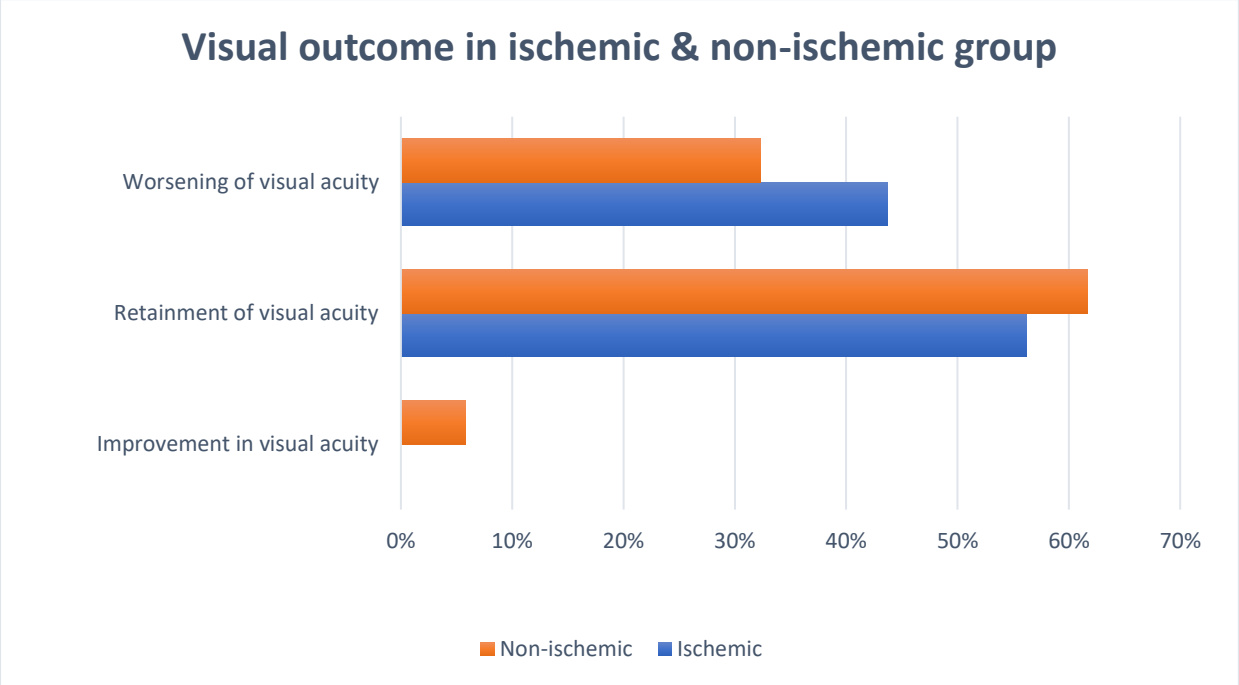
- In non-ischemic type of CRVO, 18 patients (52.9%) presented with initial Visual acuity of 6/36-6/60, 14 patients (41.2%) presented with initial Visual acuity of 6/18-6/24, and 1 patient(2.9%) presented with initial visual acuity of 6/6-6/12 ,and only 1 patient (2.9%) presented with initial visual acuity of less than 6/60.
- At the end of study during followup, 10 patients (29.4%) retained the same visual acuity of 6/36-6/60, 9 patients (26.5%) retained the visual

Acuity of 6/18-6/24, 12 patients (35.3%) showed the visual acuity of

Less than 6/60 and 3 patients (8.8%) showed visual acuity of 6/6-6/12.

Table 18: Comparison of and visual outcome with type of CRVO:

	ISCHEMIC CRVO	NON-ISCHEMIC CRVO
IMPROVEMENT OF VISUAL ACUITY	-	2 (5.9%)
RETAINMENT OF VISUAL ACUITY OR DECREASE IN VISUAL ACUITY BY 1 LINE.	9 (56.3%)	21 (61.8%)
WORSENING OF VISUAL ACUITY BY >2 LINES	7 (43.8%)	11 (32.4%)
	16 (100.0%)	34 (100.0%)



- Thus in our study in ischemic group 56.3% showed retainment of visual

acuity or decrease in visual acuity by 1 line during followup and 43.8% showed worsening of visual acuity by more than 2 lines during followup.

- In non-ischemic group 61.8% showed retainment of visual acuity , 32.4% showed worsening of visual acuity by >2 lines and 5.9% (2 patients) showed improvement in visual acuity by 1-2 lines during followup.
- Thus ischemic group showed more worsening of visual acuity when compared to non-ischemic group.2 patients with non-ischemic CRVO showed improvement in visual acuity by 1-2 lines by resolution of vein occlusion spontaneously followed by collaterals formation with sheathing of vessels.

DISCUSSION

This study is a prospective study of 50 patients presenting with Central Retinal Vein Occlusion attending RIOGOH services, chennai for a period of 12 months.

- **AGE DISTRIBUTION**:

In this study , majority of non-ischemic CRVO patients are in age group of 56-60 yrs contributing to 32.4% and 29.4% are in age group of 51-55 yrs and 20.6% are in age group of 61-65 yrs. Young patients in non-ischemic group in our study contributed to 17.6%.

Majority of ischemic CRVO patients are in age group of 61-65 yrs contributing to 50% and 31.3% are in age group of 56-60 yrs and only 12.5% are in age group of 66-70 yrs which is in concordant with Hayreh et al study.

This showed statistical significance (p=0.012) with age and type of CRVO.

Retinal vein occlusion study conducted by Sohan Singh Hayreh 1994, Suggested that the onset of first episode of retinal vein occlusion occurred between the ages of 14-92 yrs with 51% having first episode at age>65 yrs. On comparison of two groups of CRVO showed significantly higher Proportion of cases with ischemic CRVO with first onset at age> 65 yrs(67%) compared to non-ischemic CRVO(44%).In CRVO patients with age<45 yrs

are more in non-ischemic type(18%) than ischemic type(7%). [18]

Raba Thapa et al in his study of 1860 cases found that mean age of presentation was 69 +/-7 years. [19]

Kyu Sang Eah et al in his study of 263 cases suggested that the age of presentation was between 60-65 years. [20]

- **SEX DISTRIBUTION:**

In this study 70% are males and 30% are females with male/female ratio of 7:3 which is in concordance with Hayreh et al study.

In our study both non- ischemic and ischemic CRVO were common in males Contributing to 73.5% and 62.5% respectively since increased predisposing factors are seen in males .Female patients accounts for 26.5% and 37.5% of non-ischemic and ischemic CRVO .

In this study among females ischemic CRVO are more common 37.5% compared to non-ischemic CRVO of 26.5%.

Hayreh et al in his study found that the male/female ratio in non-ischemic CRVO was 279/221, and in ischemic CRVO it was 100/84. He also found that CRVO in patients older than 65 years were more common in females than males. [18]

Delaram Mirzania et al found that the average age of onset of CRVO was lower in males .Incidence of CRVO was more in males 54% compared to females 45%. [21]

- **LATERALITY OF CRVO:**

In this study right eye involvement was common 64% compared to left eye involvement which was 36%.

In our study also left eye involvement was common in ischemic group 62.5% compared to right eye involvement 37.5% whereas in non-ischemic group right eye involvement was common 76.5% compared to left eye involvement 36% . This showed highly statistical significance (p=0.007) with laterality and type of CRVO. Many recent studies have shown equal incidence.

CRVO is typically unilateral. Hayreh et al in his study concluded that right eye involvement is common in non-ischemic and left eye involvement is common in ischemic CRVO. [16]

Dong Nam Byun et al found that left eye was most commonly involved in CRVO with right eye involvement was common in CRAO. [22]

- **CLASSIFICATION OF CRVO:**

In this study Non-ischemic CRVO type was common 68% (34 cases) compared to Ischemic CRVO 32% (16 cases). 4 patients of non-ischemic CRVO were converted to Ischemic CRVO contributing to 11.8% which is in corcordant with Hayreh et al.

Hayreh et al in his study of 620 consecutive CRVO cases showed that 81%

were non-ischemic type and 19% were ischemic type. Non-ischemic CRVO are mostly asymptomatic and resolves spontaneously with collateral formation hence they are more common. Conversion of non-ischemic CRVO to ischemic type was 9.4% within 6 months & 12.6% within 18 months from onset. In patients older than 65 years showed higher rate of conversion than young patients. History of cerebrovascular disease was common in converted group. [18]

Blair K concluded that non-ischemic CRVO were more common accounting for 70% of cases and ischemic CRVO 30%. [23]

- **ASSOCIATED SYSTEMIC AND OCULAR CONDITIONS:**

In this study the most common risk factor was systemic hypertension contributing to 52%(26 cases),then hyperlipidemia 28%,Primary open angle glaucoma 6%, Chronic renal failure 8%,Homocysteinemia 4% and coronary artery disease 2%.

In both ischemic and non-ischemic group ,systemic hypertension was the common risk factor contributing to 68.8% and 44.1% respectively which is in concordant with the above study.

In ischemic variety next common risk factor were hyperlipidemia 18.8% and chronic renal failure 12.5%.

In non-ischemic type , next common risk factors were hyperlipidemia 32.4%, Primary open angle glaucoma 8.8%, chronic renal failure 5.9%,

Hyperhomocysteinemia 5.9%, coronary artery disease 2.9%.

In this study comparison P value was found to be =0.368 which is not Statistically significant due to limited sample size and lesser duration of Study.

Hayreh et al concluded in his study that ischemic CRVO group had increased prevalence of systemic hypertension (p=0.052) and diabetes mellitus (p=0.018) compared to non-ischemic type. [18]

Eitan Z Rath in his study of 87 patients concluded that the risk factors of CRVO were systemic hypertension (95% confidence interval) , open angle glaucoma (95% confidence interval) whereas diabetes mellitus, coronary artery disease or Stroke were not significant risk factors. [24]

Maxwell S Stem in his study showed the diagnosis of stroke increased the risk of CRVO by 44% and hypercoagulable state was associated with 145% increased CRVO risk. Individuals with end organ damage such as hypertension & diabetes had 92% and 53% increased hazard of CRVO. [7]

Paul R.A.O'Mahoney et al in his study of 2900 cases concluded that both hypertension & hyperlipidemia were significantly associated with any type of CRVO.Percentage attributed to hypertension was 47.9%, diabetes 4.9% & Hyperlipidemia 20.1%. [25]

- **CLINICAL MANIFESTATIONS:**

In this study in ischemic CRVO 93.8% patients presented with sudden Painless loss of vision with RAPD and only 6.3% presented with sudden Blurring of vision without RAPD.

In non-ischemic type 94.1% patient presented with sudden blurring of vision alone whereas 5.8% presented with sudden painless loss of vision with RAPD which got converted to ischemic type which showed highly statistical significance (p=0.0005) with type of CRVO and clinical manifestations.

- **OCULAR SEQUELAE DURING 1-3 MONTHS FOLLOWUP:**

In this study overall , macular edema was the most common sequelae(38%)

In non-ischemic type, macular edema was the most common ocular sequelae(47.1%) followed by hard exudates formation(26.5%), 14.7% resolved with collateral formation and sheathing of vessels.11.8% of non-ischemic CRVO converted to ischemic CRVO.

In ischemic type , Neovascular glaucoma was the most common complication (43.8%) followed by macular edema(18.8%), vitreous haemorrhage(18.8%), NVE(12.5%), NVI(6.3%).

This showed highly statistical significance with ocular sequealae during followup with type of CRVO.

Non-ischemic CRVO resolves spontaneously with time due to collateral formation with no specific treatment.

Hayreh et al in his study concluded that the major complication in non-ischemic CRVO was the chronic macular edema leading to cystoid macular degeneration and permanent central scotoma. About 12% of patient may change to ischemic CRVO within 18 months of onset which is more common in older than younger Individuals. [18]

Occurrence of complications in ischemic CRVO was about 45% which included Ocular neovascularization, vitreous haemorrhage, neovascular glaucoma , optic atrophy , macular degeneration.

Neovascular glaucoma , the dreadful complication of CRVO was seen in about 50% of ischemic cases.Retinal neovascularization developed in about 41.9%, Optic disc neovascularization 29%, iris neovascularization 1.6%, angle Neovascularization 0.5%.Anterior segment neovascularization was more common than the posterior segment neovascularization in his study.

Browning et al in his study of 100 eyes with ischemic CRVO, concluded that 32% developed neovascularization of anterior segment, 12% neovascularization of angle without iris NV. [26]

- **BCVA DURING INITIAL VISIT AND FOLLOWUP:**

In this study , in ischemic CRVO, majority of patients (93.8%) presented with initial visual acuity of <2/60 to CFCF and 6.3% presented with initial visual acuity of HM-PL+.

At the end of study during followup 56.3% showed retainment of visual acuity

or decrease in visual acuity by 1 line from initial visual acuity and 43.8% showed worsening of visual acuity by >2 lines from initial visual acuity.

In non-ischemic CRVO, 18 patients (52.9%) presented with initial visual acuity of 6/36-6/60, 14 patients (41.2%) presented with initial visual acuity of 6/18-6/24, 1 patient(2.9%) presented with visual acuity of 6/6-6/12 and only 1 patient (2.9%) presented with visual acuity of <6/60.

At the end of study during followup, 61.8% showed retainment of visual acuity, 32.4% showed worsening of visual acuity by >2 lines and 5.9%(2 patients) showed improvement in visual acuity by 1-2 lines during followup.

In non-ischemic CRVO, the primary cause of poor visual outcome is due to Macular edema resulting in cystoid macular degeneration, foveal pigmentary degeneration, epiretinal membrane formation whereas in ischemic CRVO , the poor visual outcome is due to retinal ischemia resulting in neovascularization.

In non-ischemic CRVO resolution of haemorrhages and exudates occurs with formation of collaterals and sheathing of vessels resulting in good visual outcome spontaneously without any treatment.

Mc Intosh et al in his study found that visual acuity better than 6/60 was found in 58.4% of non-ischemic type and only 1.7% in ischemic CRVO. Visual acuity of worse than 6/60 was seen in 19.1% of non-ischemic and 93.3% of ischemic CRVO respectively. [27]

Sohan singh Hayreh et al in his study of 667 cases found that the visual acuity in the eyes seen within first 3 months was 20/100 or better in 78% with non-ischemic CRVO and only 1% with ischemic CRVO($p<0.0001$) & visual field defects were mild in 91% & 8%($p<0.0001$). On resolution of macular edema Final visual acuity was 20/100 or better in 83% with non-ischemic CRVO & 12% in ischemic CRVO .Visual field defect were mild in 95% and 18% respectively. [18]

JC Chen et al in his study of 59 eyes with perfused CRVO were followed for 1 year. At the final followup visit the visual acuity had improved by 2 or more lines in 9 eyes(15%), remained the same in 33 eyes(56%) & decreased by 2 or more lines in 17 eyes(29%). [28]

SUMMARY

- Non-ischemic CRVO type was common 68% (34 cases) compared to Ischemic CRVO 32% (16 cases). 4 patients of non-ischemic CRVO were converted to Ischemic CRVO contributing to 11.8% which is in corcordant with Hayreh et al study.

- In this study , majority of non-ischemic CRVO patients are in age group of 56-60 yrs contributing to 32.4% and 29.4% are in age group of 51-55 yrs and 20.6% are in age group of 61-65 yrs. Young patients in non-ischemic group in our study contributed to 17.6%.
- Majority of ischemic CRVO patients are in age group of 61-65 yrs contributing to 50% and 31.3% are in age group of 56-60 yrs and only 12.5% are in age group of 66-70 yrs which is in concordant with Mc Intosh et al study.
- In this study 70% are males and 30% are females with male/femaleratio of 7:3 which is in concordance with Hayreh et al study.
- In this study both non- ischemic and ischemic CRVO were common in Males contributing to 73.5% and 62.5% respectively since increased predisposing factors are seen in males . Female patients accounts for 26.5% and 37.5% of non-ischemic and ischemic CRVO .

- In this study among females ischemic CRVO were more common 37.5% compared to non-ischemic CRVO of 26.5%.
- In this study all patients presented with unilateral involvement . Right eye involvement was common 64% compared to left eye involvement which was 36%.
- In this patients left eye involvement was common in ischemic group 62.5%compared to right eye involvement 37.5% whereas in non-ischemic group right eye involvement was common 76.5% compared to left eye involvement 36%.
- In this study the most common risk factor was systemic hypertension contributing to 52%(26 cases),then hyperlipidemia 28%,Primary open angle glaucoma 6%, Homocysteinemia 4% and coronary artery disease 2%.
- In both ischemic and non-ischemic group ,systemic hypertension was the common risk factor contributing to 68.8% and 44.1% respectively which is in concordant with Hayreh et al study.
- In ischemic variety next common risk factor were hyperlipidemia 18.8%and chronic renal failure 12.5%.
- In non-ischemic type , next common risk factors were hyperlipidemia 32.4%,Primary open angle glaucoma 8.8%, chronic renal failure 5.9%, Hyperhomocysteinemia 5.9%, coronary artery disease 2.9%.

- In this study in ischemic CRVO 93.8% patients presented with sudden Painless loss of vision with RAPD and only 6.3% presented with sudden blurring of vision without RAPD.
- In non-ischemic type 94.1% patient presented with sudden blurring of vision alone whereas 5.8% presented with sudden painless loss of vision with RAPD which got converted to ischemic type .
- In this study overall , macular edema was the most common sequelae(38%) seen in 19 patients.
- In non-ischemic type, macular edema was the most common ocular sequelae(47.1%) followed by hard exudates formation(26.5%), 14.7% resolved with collateral formation and sheathing of vessels.11.8% of non-ischemic CRVO converted to ischemic CRVO.
- In ischemic type , Neovascular glaucoma was the most common complication(43.8%) followed by macular edema(18.8%), vitreous haemorrhage(18.8%), NVE(12.5%), NVI(6.3%).

- In this study , in ischemic CRVO, majority of patients (93.8%) presented with initial visual acuity of <2/60 to CFCF and 6.3% presented with initial visual acuity of HM-PL+.
- At the end of study during followup 56.3% showed retainment of visual acuity or decrease in visual acuity by 1 line from initial visual acuity and

43.8% showed worsening of visual acuity by >2 lines from initial visual acuity similar to study of Jc Chen et al

- In non-ischemic CRVO, 18 patients (52.9%) presented with initial visual acuity of 6/36-6/60, 14 patients (41.2%) presented with initial visual acuity of 6/18-6/24, 1 patient(2.9%) presented with visual acuity of 6/6-6/12 and only 1 patient (2.9%) presented with visual acuity of <6/60.
- At the end of study during followup, 61.8% showed retainment of visual acuity,32.4% showed worsening of visual acuity by >2 lines and 5.9% (2 patients) showed improvement in visual acuity by 1-2 lines during followup.
- In non-ischemic CRVO, the primary cause of poor visual outcome is due to Macular edema resulting in cystoid macular degeneration, foveal pigmentary degeneration, epiretinal membrane formation .
- In ischemic CRVO ,the poor visual outcome is due to retinal ischemia resulting in neovascularization.

CONCLUSION

- Though the systemic hypertension was one of the most important comorbid condition associated with vein occlusion, Hyperlipidemia, Primary open angle glaucoma, Homocysteinemia, Coronary artery disease, Chronic renal failure also play an equally important role in etiology.
- Both ischemic and non-ischemic type of CRVO have systemic hypertension as a common etiological factor. However visual outcome in non-ischemic CRVO is always found to be better than ischemic CRVO. Hence strict control of systemic blood pressure is mandatory.
- In addition, other risk factors should also be ruled out to enhance the visual outcome.
- Thorough workup to rule out all systemic co-morbidities will influence the visual outcome in affected eye and also protect the uninvolved eye.

PART-III

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PROFOMA

NAME DATE

AGE/SEX HOSP.NO

CHIEF COMPLAINTS :with duration

PAST HISTORY:

DURATION YES NO

- H/o Hypertension
- H/o Coronary artery disease
- H/o Chronic renal failure
- H/o Vascular occlusive events(CVA)
- H/o drug intake
- H/o trauma
- H/o Previous intraocular surgery
- H/o Previous PRP
- H/o Migraine
- H/o Oral contraceptive usage(females)
- Chronic illness like HIV,Syphilis,TB.

PERSONAL HISTORY:

DURATION YES NO

- Smoking
- Alcohol

OCULAR EXAMINATION:

	RE	LE
VISION (BCVA)		
IOP		

PUPILLARY REACTION:

ANTERIOR SEGMENT EXAMINATION

- SLIT LAMP EXAMINATION
- GONIOSCOPY

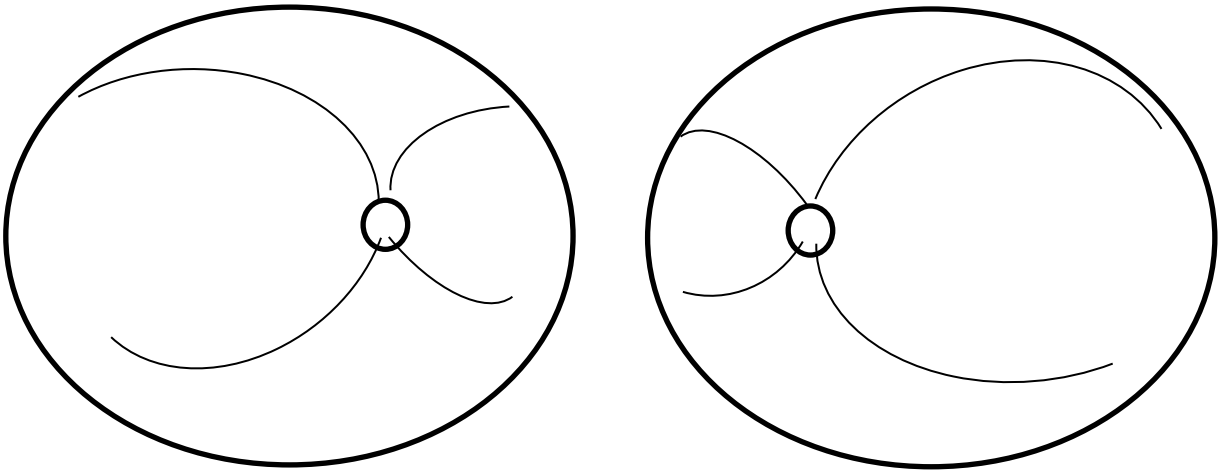
POSTERIOR SEGMENT EXAMINATION:

- SLIT LAMP BIOMICROSCOPY WITH 90D
- INDIRECT OPHTHALMOSCOPY

OPTICAL COHERENCE TOMOGRAPHY:(Macular edema)

FUNDUS FLUORESCEIN ANGIOGRAPHY:

FUNDUS



FINDINGS

RE	LE

DIAGNOSIS:

INVESTIGATIONS:

- Haemoglobin
- Complete blood count
- Fasting and post prandial blood sugar levels.
- Lipid profile.
- Blood Urea,Serum Creatinine.
- Erythrocyte sedimentation rate(ESR)
- Serum homocysteine in young patients
- Coagulation studies
- Autoantibodies like ANA,ANCA-selected cases
- Chest x-ray-selected cases
- Blood VDRL for Syphilis
- ELISA for HIV
- Electrophysiological test:
    - Electrocardiogram
    - Echo cardiogram
    - Carotid doppler for selected patients.

KEY TO MASTER CHART

INITIAL BCVA-Initial best corrected visual acuity

Code 1-6/6-6/12

2- 6/18-6/24

3-6/36-6/60

4-<6/60

5-<2/60

6-HM-PL+

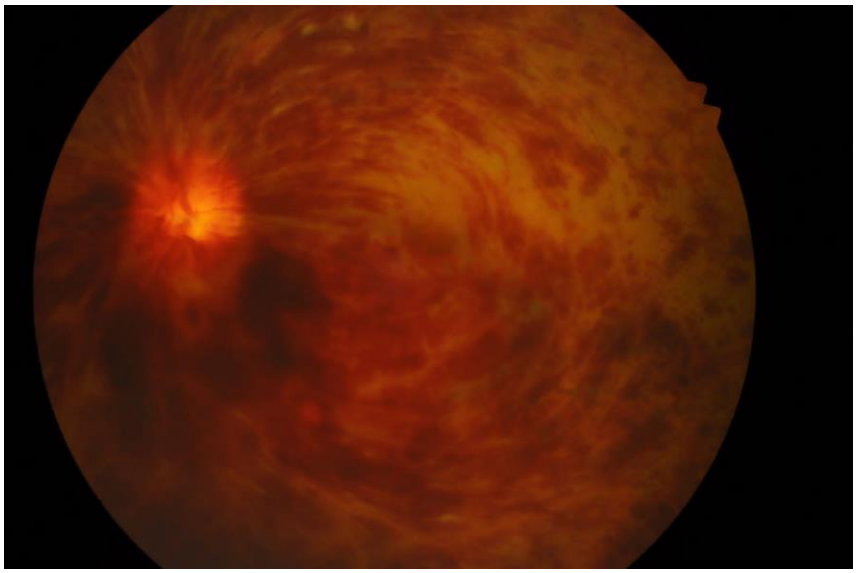
NVE-Neovascularization elsewhere

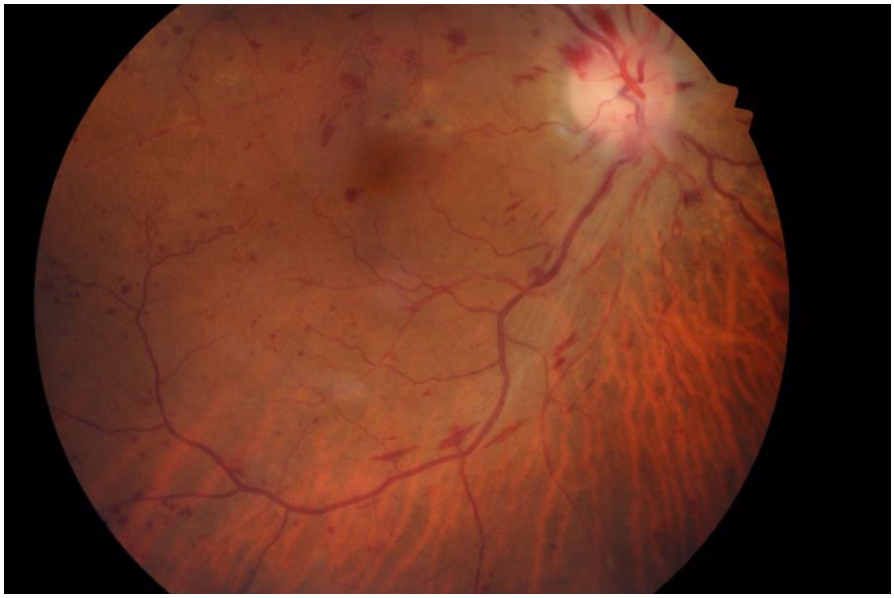
NVI- Neovascularization of iris

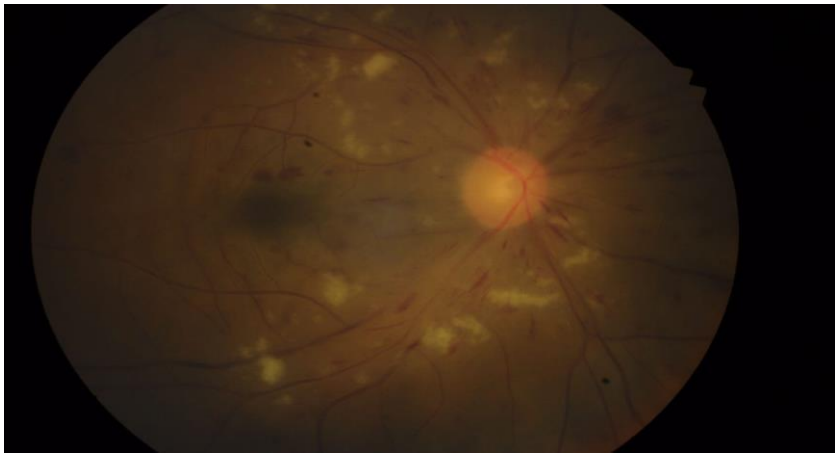
SHTN-Systemic Hypertension.

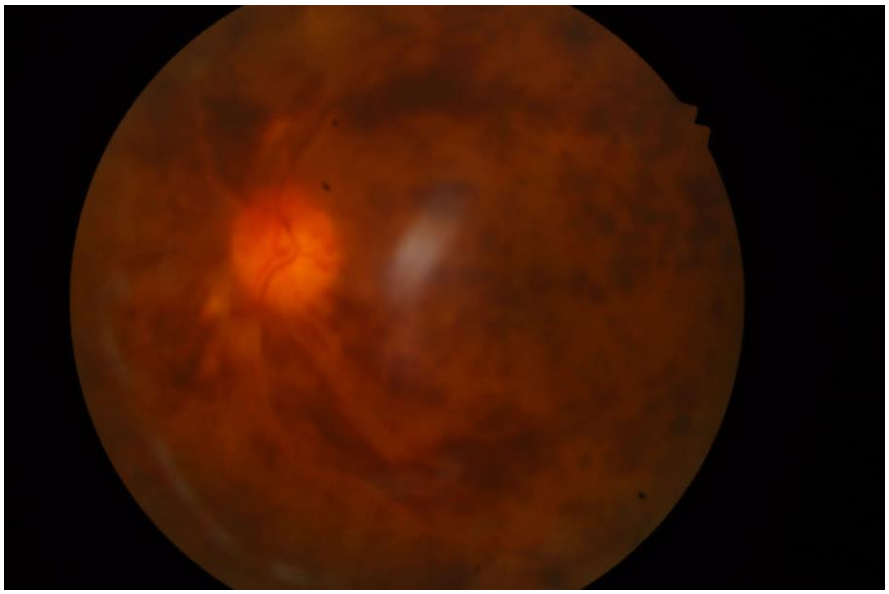
MASTER CHART

S.NO	S.NO	S.NO	S.NO	S.NO	S.NO	S.NO	S.NO	SYSTEMIC DISEASE & ASSOCIATED OCULAR CONDITIONS	CLINICAL MANIFESTATION	OCULAR SEQUELAE DURING 1-3 MONTH FOLLOWUP	BCVA DURING FOLLOWUP
1	KARUNANITHI	57	M	367183	RIGHT EYE	2	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	HARD EXUDATES	2
2	RANGANATHAN	55	M	377188	RIGHT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	MACULAR EDEMA	5
3	DHANASEKAR	50	M	456341	RIGHT EYE	3	NON-ISCHEMIC	PRIMARY OPEN ANGLE GLAUCOMA	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
4	KUPPAMMAL	54	F	482474	LEFT EYE	1	NON-ISCHEMIC	CHRONIC RENAL FAILURE	SUDDEN BLURRING OF VISION	COLLATERALS WITH SHEATHING OF VESSELS	1
5	ZAHIDA	60	F	567065	LEFT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	NEOVASCULAR GLAUCOMA	5
6	KAMALAKANNAN	47	M	512842	RGHT EYE	4	NON-ISCHEMIC	CORONARY ARTERY DISEASE	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
7	JAGANATHAN	52	M	436140	RIGHT EYE	2	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	HARD EXUDATES	1
8	RAMAIAH	62	M	461928	RIGHT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	3
9	VISHWANATHAN	52	M	419643	LEFT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	3
10	KUPPUSAMY	61	M	491452	RIGHT EYE	3	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	CONVERTED TO ISCHEMIC	4
11	SAMUEL	60	M	471293	LEFT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	NEOVSCULAR GLAUCOMA	5
12	KULANTHAIVELU	60	M	478219	RIGHT EYE	2	NON-ISCHEMIC	CHRONIC RENAL FAILURE	SUDDEN BLURRING OF VISION	MACULAR EDEMA	2
13	ANNAMMAL	50	F	428517	RIGHT EYE	2	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	COLLATERALS WITH SHEATHING OF VESSELS	1
14	KANDHASAMY	58	M	431904	RIGHT EYE	5	ISCHEMIC	HYPERLIPIDEMIA	SUDDEN PAINLESS LOSS OF VISION,RAPD	CONVERTED TO ISCHEMIC	5
15	PALANIKUMAR	45	M	417921	RIGHT EYE	3	NON-ISCHEMIC	HOMOCYSTEINEMIA	SUDDEN BLURRING OF VISION	MACULAR EDEMA	3
16	KALAISELVI	50	F	481023	LEFT EYE	2	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	HARD EXUDATES	2
17	ANNAMALAI	56	M	491621	RIGHT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
18	ANNAMARY	62	F	401829	RIGHT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	MACULAR EDEMA	5
19	WILLIAMS	53	M	491623	RIGHT EYE	2	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	HARD EXUDATES	2
20	MANIKANDAN	57	M	418211	RIGHT EYE	3	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	MACULAR EDEMA	3
21	ABDUL KADHAR	60	M	452921	RIGHT EYE	2	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	3
22	KUMUDHA	55	F	421933	LEFT EYE	3	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	HARD EXUDATES	3
23	SENTHILKUMAR	62	M	417420	LEFT EYE	5	ISCHEMIC	CHRONIC RENAL FAILURE	SUDDEN PAINLESS LOSS OF VISION,RAPD	NVE	5
24	KARUPAYEE	70	F	467341	RIGHT EYE	6	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	NEOVASCULAR GLAUCOMA	6
25	RAJESHWARI	65	F	432154	LEFT EYE	2	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	HARD EXUDATES	2
26	SASIKUMAR	63	M	458201	LEFT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	CONVERTED TO ISCHEMIC	4
27	VATCHALA	72	F	417321	RIGHT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	NVE	6
28	KANNIYAPPAN	55	M	461272	RIGHT EYE	2	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
29	VASANTHI	57	F	431642	RIGHT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
30	PANEERSELVAM	64	M	421753	RIGHT EYE	5	ISCHEMIC	CHRONIC RENAL FAILURE	SUDDEN PAINLESS LOSS OF VISION,RAPD	NEOVASCULAR GLAUCOMA	5
31	ANANDHAN	55	M	415238	RIGHT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION,RAPD	CONVERTED TO ISCHEMIC	4
32	JOSEPH	60	M	427319	RIGHT EYE	3	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	HARD EXUDATES	3
33	MANOJKUMAR	48	M	495216	LEFTEYE	2	NON-ISCHEMIC	HOMOCYSTEINEMIA	SUDDEN BLURRING OF VISION	COLLATERALS WITH SHEATHING OF VESSELS	2
34	SANTHANALAKSHMI	60	F	402751	LEFT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION ,RAPD	NEOVASCULAR GLAUCOMA	6
35	VIJAYAKUMAR	63	M	471423	LEFT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	VITREOUS HAEMORRHAGE	6
36	RAMAMOORTHY	62	M	492152	RIGHT EYE	3	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
37	SRINIVASALU	55	M	461324	RIGHT EYE	2	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	HARD EXUDATES	2
38	PERUMAL	60	M	431932	LEFT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	MACULAR EDEMA	6
39	AARAYEE	64	F	451985	LEFT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	NEOVASCULAR GLAUCOMA	5
40	THIRUMALAI	55	M	481932	RIGHT EYE	2	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	3
41	VEERAMANI	58	M	491532	RIGHT EYE	3	NON-ISCHEMIC	PRIMARY OPEN ANGLE GLAUCOMA	SUDDEN PAINLESS LOSS OF VISION	COLLATERALS WITH SHEATHING OF VESSELS	2
42	SHAMUGANATHAN	64	M	483041	LEFT EYE	5	ISCHEMIC	HYPERLIPIDEMIA	SUDDEN PAINLESS LOSS OF VISION,RAPD	VITREOUS HAEMORRHAGE	6
43	ESHWARI	60	F	458120	LEFT EYE	2	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	3
44	VANI	55	F	437521	RIGHT EYE	2	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	COLLATERALS WITH SHEATHING OF VESSELS	2
45	RAMESH	58	M	453961	RIGHT EYE	3	NON-ISCHEMIC	HYPERLIPIDEMIA	SUDDEN BLURRING OF VISION	HARD EXUDATES	3
46	VELMURUGAN	65	M	482051	RIGHT EYE	3	NON-ISCHEMIC	PRIMARY OPEN ANGLE GLAUCOMA	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
47	LAWRENCE	62	M	418420	LEFT EYE	5	ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD	VITREOUS HAEMORRHAGE	6
48	ETHIRAJ	61	M	439173	LEFT EYE	5	ISCHEMIC	HYPERLIPIDEMIA	SUDDEN PAINLESS LOSS OF VISION,RAPD	NEOVASCULAR GLAUCOMA	6
49	KOTEESHWARAN	58	M	461082	RIGHT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	MACULAR EDEMA	4
50	TAMILARASI	65	F	471523	RIGHT EYE	3	NON-ISCHEMIC	SYSTEMIC HYPERTENSION	SUDDEN BLURRING OF VISION	CONVERTED TO ISCHEMIC	4









NON-ISCHEMIC CRVO RESOLVING WITH SHEATHING OF VESSELS