ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 7, Issue, 03, pp.13418-13422, March, 2015

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

CASE STUDY

CENTRAL RETINAL VEIN OCCLUSION, A CLINICAL STUDY

***Dr. K. V. V. Satyanarayana and Dr. K. Vijayasekhar**

Govt. Regional Eye Hospital Visakhapatnam

ARTICLE INFO

Article History:
Received 08th December, 2014
Received in revised form
26th January, 2015
Accepted 23rd February, 2015
Published online 17th March, 2015

Key words:

CRVO,
RAPD,
Macular edema.

ABSTRACT

A prospective study of Central Retinal vein Occlusion conducted at Govt. Regional Eye Hospital Visakhapatnam.

Materials and Methods: 26 cases documented with in a period of one year, all the cases are examined, investigated, treated and followed up for a period of one and half years.

Results: Incidence of ischemic CRVO is more as the age advances. Visual prognosis is good in non ischemic CRVO. Ischemic variant of CRVO not only ended with bad visual prognosis, but also serious complications.

Conclusions: The incidence of ischemic and nonischemic CRVO is almost same. Proper follow up is mandatory to diagnose conversion of non ischemic to ischemic verity and to identify the impending compilations.

INTRODUCTION

Central retinal vein occlusion (CRVO) is a common retinal vascular disorder. Clinically, CRVO presents with variable visual loss, with a clinical picture of retinal haemorrhages, in the posterior pole and giving the "blood and thunder appearance." 1.CRVO can be divided into 2 clinical types, ischemic and nonischemic. A number of ocular and systemic conditions are the aetiology for CRVO. Several clinical futures are taken into account for classifying CRVO, including vision at presentation, presence of relative afferent pupillary defect,(RAPD), extent of retinal haemorrhages, cotton-wool spots, assessment of retinal perfusion by fluorescein angiography, and electro retinographic changes. It is important to differentiate non ischemic and ischemic in prognostic point . Proper follow up required for good visual out come and to notice impending ocular complications 2.

MATERIALS AND METHODS

26 cases (20 males and 6 females) are included in the study, the age groups are between 31 years to 71 years. Risk and aetiological factors are evaluated. Complete ophthalmic examination and systemic, ocular investigations Fundus Florescence angiography carried out. Macular complications are treated with grid laser. All the cases followed up to one and half years.

**Corresponding author: Dr. K. V. V. Satyanarayana*
Govt. Regional Eye Hospital Visakhapatnam

RESULTS

Out of 26 cases male pts are 20, and female pts are 6 in number. The age group varied between 31 years to71 years (4 cases among 30s, 5 cases among 40s, 5 cases among 50s, 6 cases among 60s, 2 cases among 70years of age), Left eye is involved more than Right eye. (OS:OD:16 eyes:10 eyes). Visual status at the time of presentation pl –ve in 2 eyes., hand movements in 6 eyes; 6//60 in 12 eyes, 6//36 –in 1eye, 6/18 –in 3 eyes: 6//9 -2eyes Bilateral CRVO in one case.

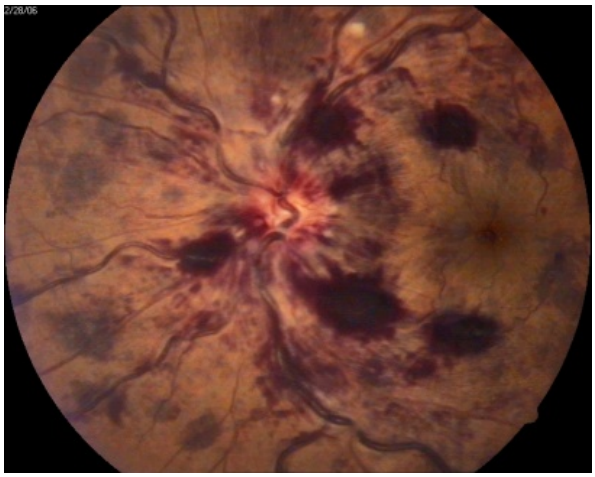


Fig.1. CRVO OS with Macular edema

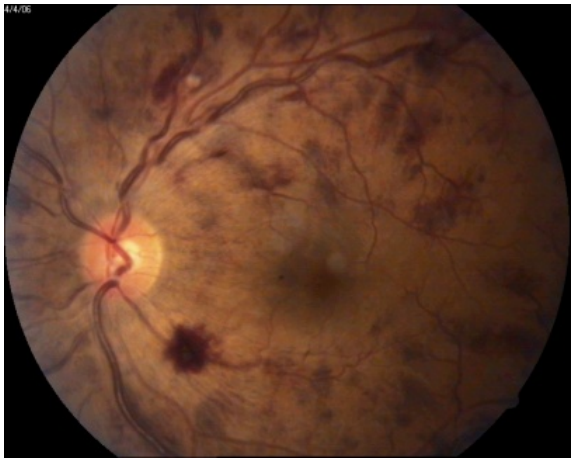


Fig.2. Same case after 2 months resolving CRVO with ersistance of Macular edema

Hemi retinal vein occlusion a variant of CRVO. Normal development of CRV bifurcates in to superior and inferior branches on the optic nerve head, but 20% of cases CRV bifurcates with in the optic nerve. Occlusion any one of the branch may results in a Hemi retinal vein occlusion.

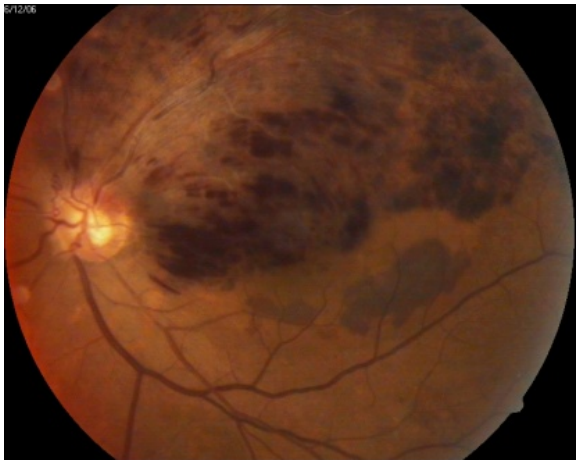


Fig. 3. HRVO OS – venous engorgement Marked superficial haemorrhages, choroidal haemorrhage along the inferior temporal vein 4.5.

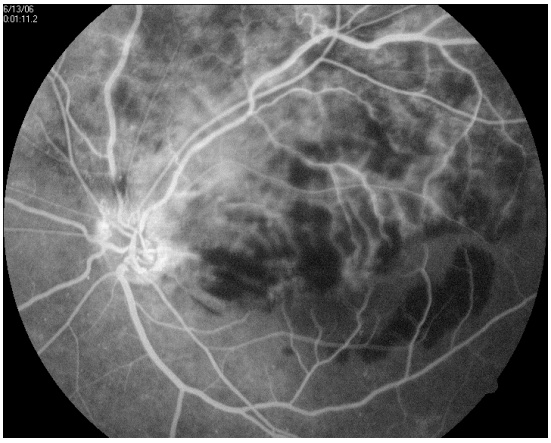


Fig. 4. FFA Delayed arteriovenous transit time-Hypo fluorescence in the areas of superficial haemorrhages, choroidal haemorrhage

Non ischemic CRVO 17 cases and Ischemic CRVO 9 cases: 8 cases of CRVO has the history of Diabetes mellitus, 2 cases has Systemic hypertension and 2 cases are associated with POAG. It is well established that the association of CRVO with POAG.10. Iti Visual prognosis is Good 13 and poor in 13 cases. 6

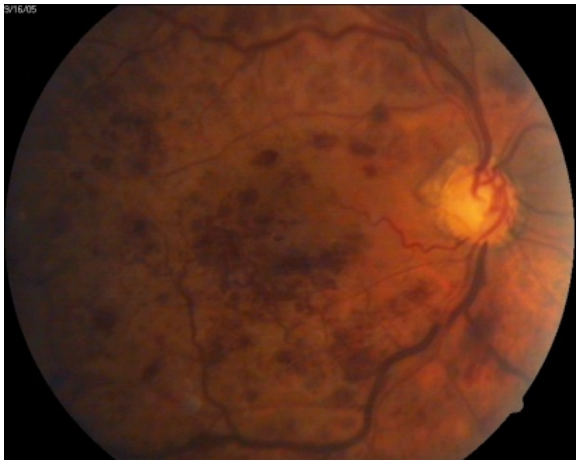
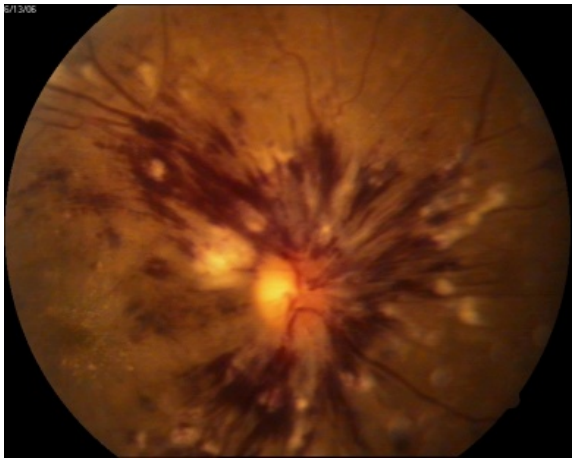


Fig. 5. CRVO with POAG. Note the CD ratio 0.9:1



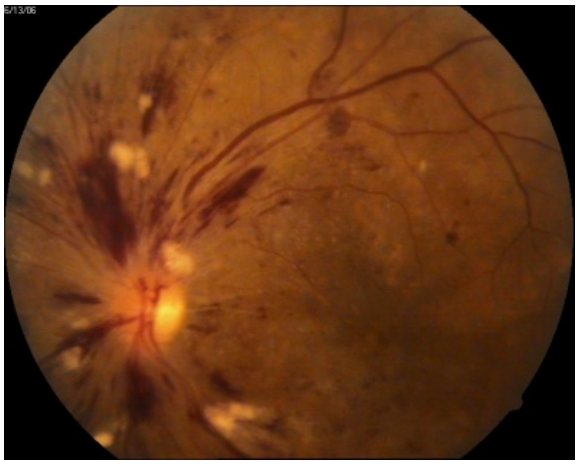
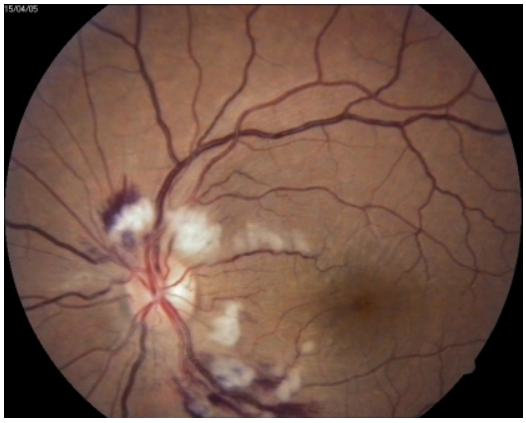
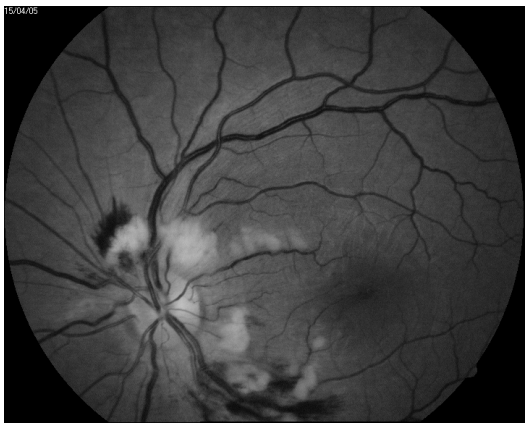


Fig. 6. Bilateral CRVO

Head injury predisposed to CRVO. 2 cases of CRVO presented along with cilio retinal artery occlusion.





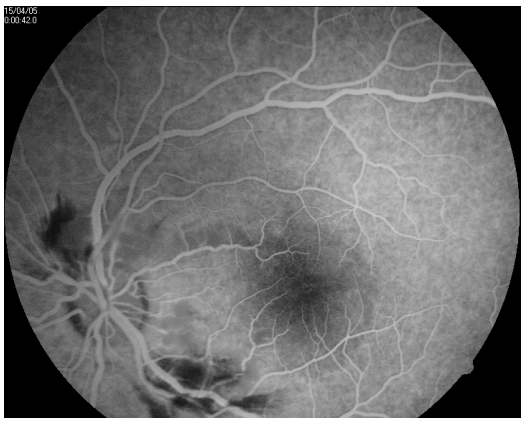


Fig. 7. CRVO OS following head injury, with gross diminution of vision, RAPD: FFA : delayed aretriovenous transit time. Macular edema

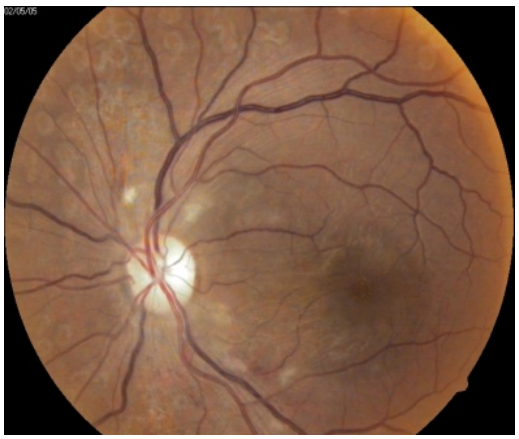


Fig. 8. After three months followup CRVO resolved with persistent macular edema developed optic atrophy

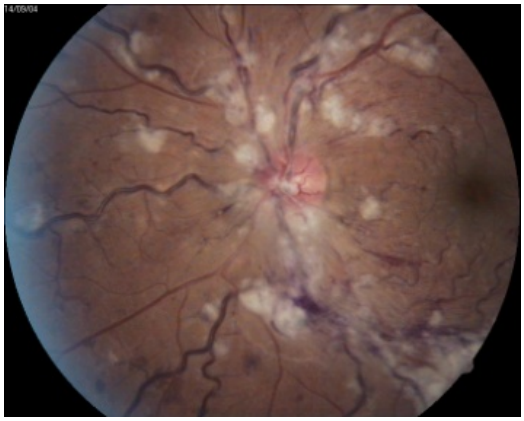
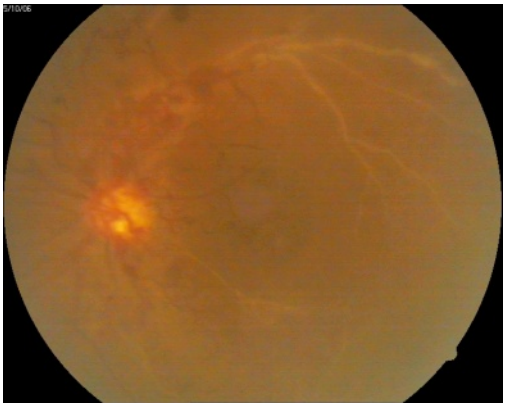


Fig. 9. CRVO OS, Number of soft exudates indicates the severity of ischemia



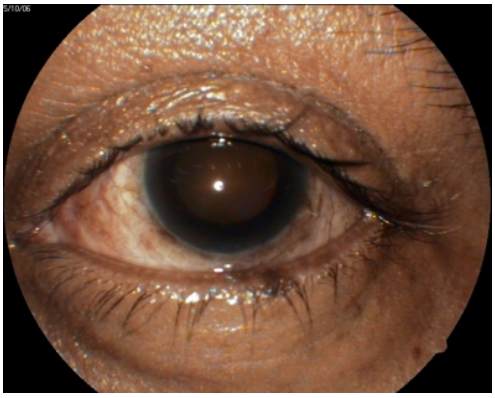


Fig. 10. Ischemic CRVO wih resolving Vit. Haemorrhage Fig: After four months the same eye developed rubiosis and neovascular glaucoma

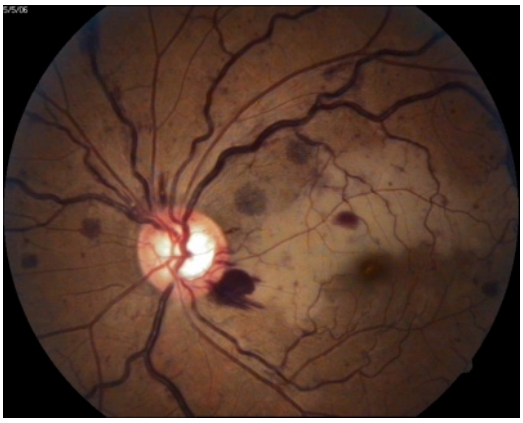


Fig. 11. CRVO OS with cilioretinal artery block

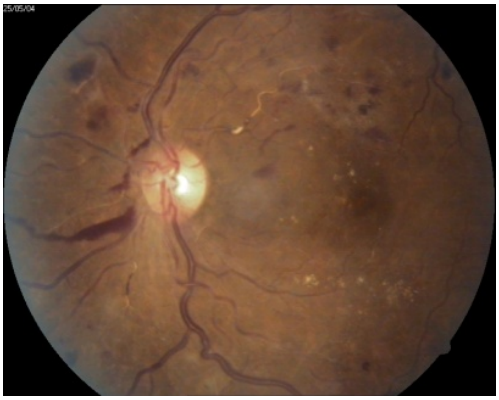


Fig. 12. CRVO OS With Hypertensive retinoparthy

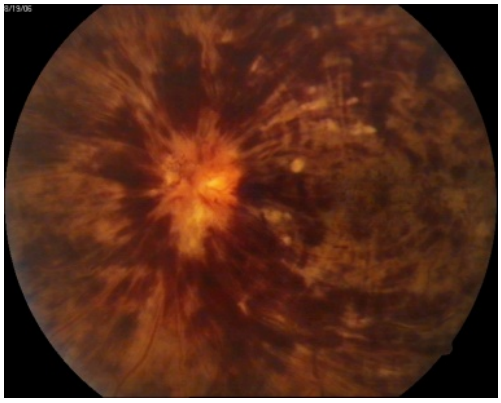


Fig: 13:CRVO OS with disc edema "Blood thunder appearance" severity of haemorrhages indicates ischemic CRVO

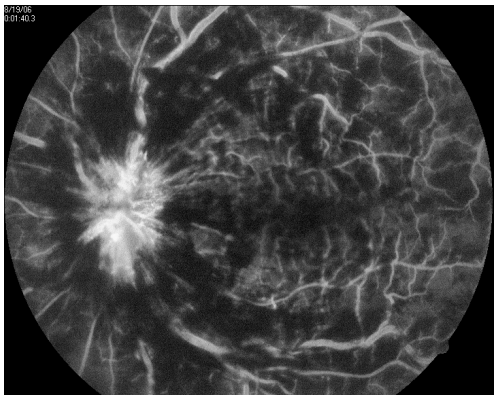


Fig. 14. FFA note hyper fluoresence of the disc indicates disc edema, general fundus at this stage

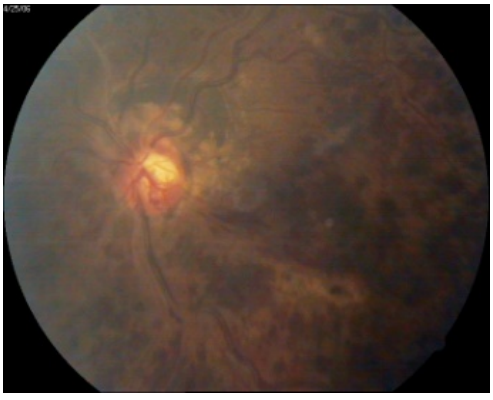
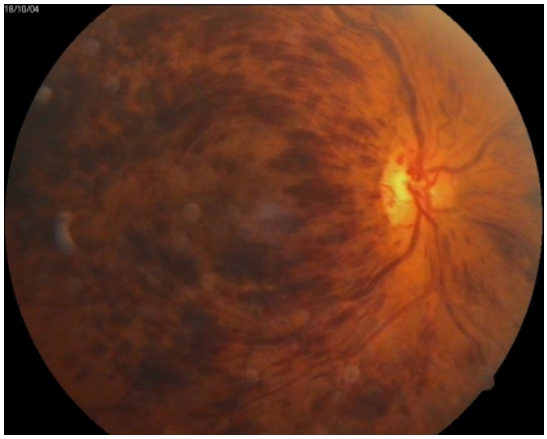
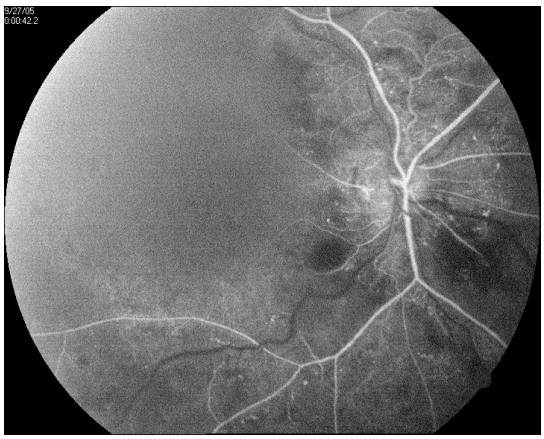
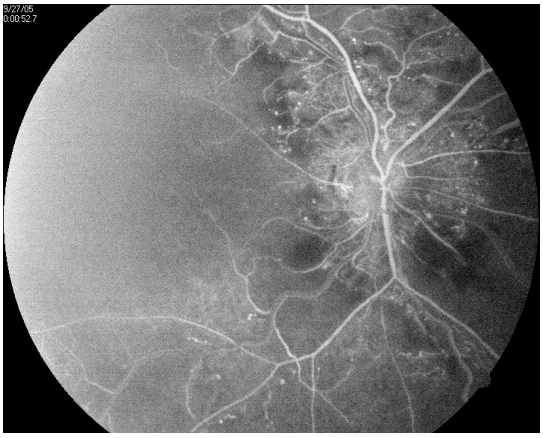
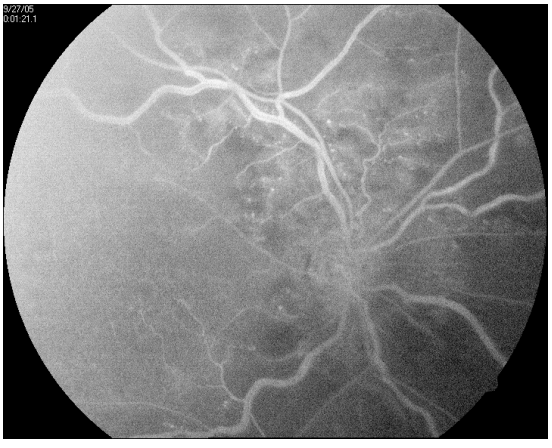


Fig. 15. Ischemic CRVO OS, note neovascularisation of disc









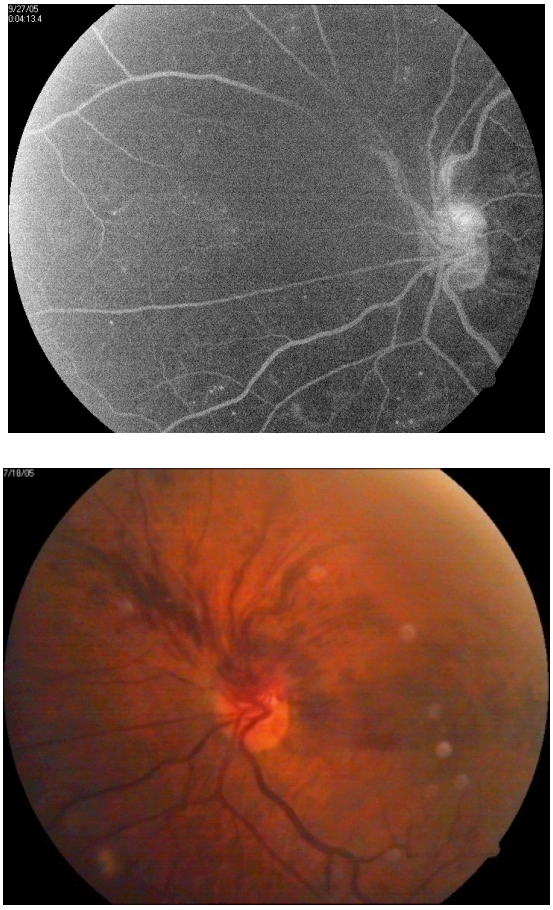


Fig. 16. CRVO OD : HRVO OS in same syst. hypertensive person

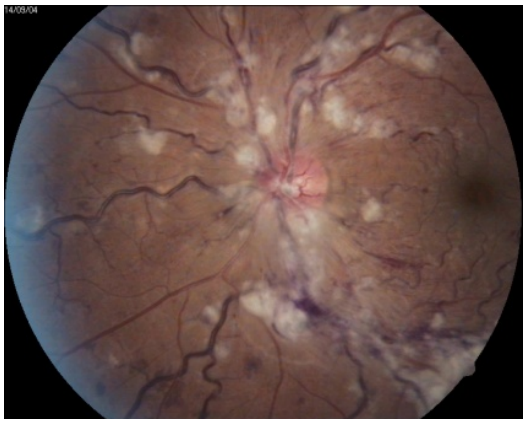


Fig. 17. OS Venous stasis retinopathy with persisted macular edema developed macular hole

During the follow up eye developed macular hole.

DISCUSSION

There are 2 types of CRVO: Non ischemic and ischemic. CRVO arises from thrombosis of the central retinal vein in the vicinity of the lamina cribosa. 7. May be due to Throttle mechanism in the central retinal vein in the region of lamina cribrosa. 8. Compression-from surrounding structures and hemodynamic changes induced changes in the vein, turbulence, endothelial cell damage, and eventual thrombosis.

More the distance of thrombus to the lamina cribrosa less the risk of ischemia; an occlusion distal to the lamina may provide more venous collateral channels and hence improved perfusion and chance of non ischemic CRVO 1. Visual loss can occur due to haemorrhage at macular area and complications macular edema, ischemia. Visual prognosis is good in non ischemic CRVO. Retinal ischemia can be assessed by fluorescein angiography when ever it is possible. The risk of conversion from non ischemic to ischemic predicted by Relative afferent papillary defect, the number of non perfusion areas are more than ten disc dioptres reflects at anterior segment rubiosis and leads to secondary glaucoma known as "Ninty day glaucoma". 2. Recurrent CRVO should be think of if non resolving CRVO fundus clinical picture persisted for more than 6 months. Periodical fundus photographs and FFA will help to differentiate this condition. Non resolving vitreous haemorrhage for more than 6 months should be suspected for recurrent haemorrhage. Macular edema treated with macular grid laser in selective cases.

Conclusions

The incidence of ischemic CRVO is more as the age advances, macular edema resolved in 40% of cases with macular grid laser, long standing macular edema not responded to treatment and developed in to macular hole. CRVO due to head injury landed in optic atrophy after three months . Bilateral CRVO confined to ischemic variant. In case of CRVO with non resolving vitreous haemorrhage probably due to recurrent CRVO with repeated vitreous haemorrhage. CRVO in a single eye may be a combination of two hemiretinal vein occlusion. One may be ischemic the other may be non ischemic. Visual prognosis is good in non ischemic CRVO. Proper follow up is mandatory to diagnose conversion of non ischemic to ischemic verity and to identify the impending compilations.

REFERENCES

Central Retinal Vein Occlusion Study Group. Natural history and clinical management of central retinal vein occlusion. *Archives of Ophthalmology* 1997, 115: 486-491.

Central venous occlusion study (COVS) *Arch Ophthal.,* 1993. Aug 111(8) 1087-95

Chopdar A. Dual trunk central retinal vein incidence in clinical practice. *Arch. Ophthal.,* 1984.102.85-7

Fry. Variations in course of CRA. CRV in optic nerve. Arch. Ophthal. 1930.4.180-87.

Harreyh. central retinal vein occlusion pathogenesis and terminology. *Arch. Ophthal.,* 1996. 114.545-54

Hayreh SS. 1994. Retinal vein occlusion. *Indian J Ophthalmol.,* 42:109-32

Klein R. 2008. Moss SE the 15 year cumulative incidence of retinal vein occlusion. *Arch. Ophthal*., 126 . 513-18.

Verhoeff, FH. 1913. The effect of chronic glaucoma on central retinal vessels. *Arch Ophthal.,* 42.145-152.

Williamson A. 2007. Throttle mechanism in the central retinal vein in the region of lamina cribrosa. *Br. Journal of Ophthalmology,* 91.1190.93.

****