VISUAL OUTCOME FOLLOWING PARACENTESIS IN CENTRAL RETINAL ARTERY OCCLUSION
Tanushree V1, H. T. Venkate Gowda2, Savita Patil3, Vijaylaxmi Patil4, Tejashree T. Chavan5, B. Agni6

HOW TO CITE THIS ARTICLE:
Tanushree V, H. T. Venkate Gowda, Savita Patil, Vijaylaxmi Patil, Tejashree T. Chavan, B. Agni. "Visual Outcome following Paracentesis in Central Retinal Artery Occlusion". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 66, December 01; Page: 14330-14335, DOI: 10.14260/jemds/2014/3922

ABSTRACT: AIM OF THE STUDY: To investigate whether paracentesis improves visual acuity after central retinal artery occlusion (CRAO), depending on the time between first symptoms of CRAO and the implementation of paracentesis. STUDY DESIGN: Prospective study. MATERIALS AND METHODS: Patients with Central retinal artery occlusion attending the outpatient and in-patient department, Department of Ophthalmology, K. R. Hospital, Mysore, fulfilling the inclusion criteria framed were included under the study, between the periods from September 2013 to September 2014 (1 year). Informed and written consent was taken from all the patients. After detailed history, all necessary ocular, systemic examination and investigations was done. Patients received the conservative treatment after the diagnosis was found along with paracentesis. The primary endpoints were the change in visual acuity 3 days after the paracentesis and the time between first symptoms and initiation of therapy. RESULTS: 4(40%) out of 10 patients who presented within 8 hours onset of CRAO, showed a gain in visual acuity following anterior chamber paracentesis from hand movements positive to counting finger at 2 metres in temporal visual field. CONCLUSION: Paracentesis by reducing intraocular pressure improves retinal arterial perfusion pressure, by vasodilatation of central retinal artery leads to dislodgement of the thrombus or emboli from a major retinal branch to the periphery restoring some amount of vision in a particular sectoral field. KEYWORDS: Central retinal artery occlusion, Paracentesis, Visual outcome.

INTRODUCTION: Central retinal artery occlusion (CRAO) is caused by closure of the central retinal artery by a thrombus or emboli. Atherosclerosis related thrombosis at the level of the lamina cribrosa is by far the most common underlying cause of central retinal artery occlusion (CRAO), accounting for about 80% of cases. Embolism is another key cause of retinal arterial compromise, including transient ischaemia. The origin of emboli is most commonly caused by an atheromatous plaque at the carotid bifurcation, and less often the aortic arch and elsewhere. Since the ophthalmic artery is the first branch of the internal carotid artery, embolic material from the heart and carotid arteries has a fairly direct route to the eye. Clinically, the patient notices a sudden and painless unilateral loss of vision. It’s an ophthalmic emergency. Even if there is only a short closure of the central retinal artery, CRAO leads to permanent ischaemic damage of the retina.

Various therapeutic modalities in the treatment of CRAO described in the medical literature include systemic anticoagulation, systemic venous thrombolysis, catheter-guided intraarterial fibrinolysis, ocular massage and reduction of intraocular pressure. Despite the numerous therapeutic approaches there is no effective therapy to restore function of the retina to a satisfying extent. Reduction of intraocular pressure to improve retinal blood flow is frequently discussed as a possible treatment option. Approaches towards lowering of intraocular pressure includes administration of systemic and local medications, ocular massage and paracentesis.
Paracentesis leads to a rapid reduction of intraocular pressure, and therefore is believed to promote improved retinal perfusion, especially in the first few hours after onset of CRAO.\(^7,9\) Paracentesis also leads to vascular dilatation, which shifts the thrombus or embolus from the major vascular branch to smaller branch at periphery improving some amount of vision in a particular sector, provided the procedure is done within few hours of onset of CRAO. In the present study we investigated whether paracentesis improves visual acuity after CRAO depending on the time between first symptoms of CRAO and the implementation of paracentesis.

**MATERIALS AND METHODS:**

**SOURCE OF DATA:** Patients with Central retinal artery occlusion attending the outpatient and inpatient department, Department of Ophthalmology, K. R. Hospital, Mysore, fulfilling the inclusion criteria framed were included under the study, between the period from September 2013 to September 2014 (1 year).

**STUDY DESIGN:** Prospective study.

**SAMPLE SIZE:** 10 patients.

**INCLUSION CRITERIA:** Non-arteritic Central retinal artery occlusion with thromboembolic origin.

**EXCLUSION CRITERIA:** Arteritic, transient ischaemia and with a Central retinal artery occlusion preserved cilioretinal vessel.

**METHOD OF STUDY:** Informed and written consent was taken from all the patients. After detailed history, all necessary ocular examination and investigations such as visual acuity testing by Snellen's chart, Slit-lamp biomicroscopic examination, Intraocular pressure recording by Icare and posterior segment examination by Direct/Indirect opthalmoscopy and 90D examination was done. Systemic examination most importantly cardiac evaluation and carotid palpation was done. Systemic investigations such as complete haemogram, Random blood sugar, blood pressure measurement, carotid doppler and lipid profile was done.

Patients received the conservative treatment after the diagnosis was found along with paracentesis. All CRAO patients were posted for paracentesis. 1% pilocarpine was instilled 1 hour before the procedure. Miosis will prevent damage to the lens while paracentesis procedure. Under sterile aseptic precautions and under topical anaesthesia, parts painted and draped with betadiene. Anterior chamber paracentesis was done with tuberculin syringe at 9'0 clock position with bevel edge up. Brief puncture of the cornea done to drain a few drops of aqueous fluid. Successful execution was verified with an Icare intraocular pressure measurement that had to be under 5 mmHg.

After the procedure antibiotic drops was instilled. Pad and bandage was applied. Best corrected visual acuity (BCVA) was measured at the beginning of treatment and after 3 days. In addition, we recorded current medication, demographic data, the onset of initial CRAO symptoms, and the time of paracentesis. Furthermore, an analysis of cardiovascular risk factors was performed and the test results were documented. In particular, blood pressure was measured routinely four times during the first 24 hours. Finally, any complications associated with paracentesis were
documented. The primary endpoints were the change in visual acuity 3 days after the beginning of treatment and the time between first symptoms and initiation of therapy.

**RESULTS:** Ten patients with non arteritic type of central retinal artery occlusion attending the outpatient and in-patient department, Department of Ophthalmology, K. R. Hospital, Mysore, fulfilling the inclusion criteria framed were included under the study, between the period from September 2013 to September 2014 (1 year). After detailed history, all necessary ocular and systemic examination and investigations was done. Patients received the conservative treatment after the diagnosis was found along with paracentesis. Demographic data, systemic risk factors, time of presentation, presenting best corrected visual acuity and best corrected visual acuity after paracentesis was documented.

In the present study about 4 (40%) patients were in the age group of 50-60yrs and 6(60%) patients in the age group of 61-70yrs (Table 1).All were male patients (Table 2). Patients with non arteritic type of CRAO underwent systemic examination and systemic investigations like complete haemogram, Random blood sugar, blood pressure measurement, lipid profile and carotid Doppler. In our study as shown in table 3, 3 (30%) were diabetic, 2 (20%) were known hypertensive and 5 (50%) patients showed hyperlipidemia. Those patients with significant abnormality on carotid Doppler were referred to cardiologist for further management. Patients presented with sudden painless loss of vision. About 2 (20%) presented within 2 hrs of onset of CRAO, 2 (20%) patients presented within 8 hrs of onset of CRAO. Rest all 6 patients presented within 2-4 days after the onset of CRAO (table 4). Table 5 shows presenting visual acuity. 6 (60%) patients had hand movements positive and 4 (40%) patients had perception of light only. Table 6 shows best corrected visual acuity following paracentesis. Those patients who presented within 8 hrs of onset of CRAO showed significant improvement to counting finger of 2 m in the temporal quadrant. No improvement in vision was seen in patients who presented after 2-4 days of onset of CRAO.

**DISCUSSION:** Retina has a very limited tolerance for ischaemia, which was determined to be 105 minutes in animal experiments. If ischaemia persists longer than this period, inevitable permanent retinal damage occur. Since the extent of retinal damage is a function of the duration of retinal ischaemia, it is important that a counter measure against CRAO be effective quickly and available at any time. Paracentesis leads to a reduction of intraocular pressure, and therefore it promotes to improve retinal perfusion, especially in the first few hours after onset of CRAO.

Advantages of paracentesis are that the implementation of procedure is quick and involves low costs and few resources. On the other hand, paracentesis poses a considerable risk of infection and lens injury.

The present study reveals significant benefit of paracentesis to visual acuity after CRAO done within 8 hours of onset. Paracentesis lowers the intraocular pressure leading to relative rise in the retinal perfusion pressure, possibly resulting in improved retinal blood flow. Implementing paracentesis early and draining ocular fluid offers a quick and effective means to lower the intraocular pressure. In previous studies a relative gain in visual acuity due to paracentesis could not be proven. Experiments showed that a decrease in intraocular pressure measured by applanation tonometry from 15 mmHg to 5 mmHg resulted in a relative increase of the perfusion pressure of only 15% and an increase of arterial blood flow of only 20%.
Schumacher et al\textsuperscript{3} showed a relationship between the eventual visual acuity and the time from central retinal arterial occlusion to the initiation of treatment. The study concluded that a delay in treatment within the first 20 hours after CRAO resulted in an irreversible loss of visual acuity of 0.2 lines per hour delay. Augsburg et al\textsuperscript{6} found that only 35\% of patients with persistent CRAO showed a significant improvement of visual acuity of at least three lines. Moreover, only 8\% of CRAO patients showed an improvement of visual acuity of more than 0.1.\textsuperscript{13,14} In 92\% of patients without therapy, the visual acuity after CRAO is counting fingers or worse.\textsuperscript{15} Hayreh and colleagues\textsuperscript{15} demonstrated that any possible increase of visual acuity after CRAO usually occurs within the first week after the occlusion. It was shown, that a clinically significant increase in visual acuity after weeks is rare.\textsuperscript{16}

With no effective treatment for CRAO available it is important to identify the systemic causes and focus on preventive measures. Cardiovascular mortality rate of affected patients was also shown to increase when risk factors for CRAO were ignored.\textsuperscript{17} Therefore, diagnosis and adequate treatment of cardiovascular risk factors is of utmost prime importance. In 64–82\% of patients with CRAO at least one previously undetected cardiovascular risk factor was diagnosed in the wake of a thromboembolic risk check-up.\textsuperscript{18} In a study done by Achim Fieß, Ömer Cal, Stephan Kehrein, Sven Halstenberg, Inez Frisch, Ulrich Helmut Steinhorst showed that there was no significant difference in the outcome between patients with (BCVA 1.9 ± 0.31) and without paracentesis (BCVA 1.75 ± 0.32) (p = 0.9), nor among the groups with paracentesis (p = 0.8).\textsuperscript{19}

CONCLUSION: Anterior chamber paracentesis by reducing intraocular pressure, improves retinal arterial perfusion pressure, by vasodilatation of central retinal artery leads to dislodgement of the thrombus or emboli from a major retinal branch to the periphery restoring some amount of vision in a particular sectoral field. The primary goal of treatment in patients with CRAO must be a comprehensive investigation of a patient’s individual systemic risk factors for CRAO and their subsequent treatment. This should also contribute to reduce mortality after CRAO and prevent the occurrence of CRAO in the unaffected eye.

REFERENCES:
1. Chen CS, Lee AW. Management of acute central retinal artery occlusion. Nat Clin Pract Neurol 2008; 4 (7): 376–83.
2. Rumelt S, Dorenboim Y, Rehany U. Aggressive systematic treatment for central retinal artery occlusion. Am J Ophthalmol 1999; 128 (6): 733–8.
3. Schumacher M, Schmidt D, Jurklies B, Gall C, Wanke I, Schmoor C, et al. Central retinal artery occlusion: local intra-arterial fibrinolysis versus conservative treatment, a multicenter randomized trial. Ophthalmology 2010; 117 (7): 1367–75.
4. Hattenbach LO, Kuhli-Hattenbach C, Scharrer I, Baatz H. Intravenous thrombolysis with low-dose recombinant tissue plasminogen activator in central retinal artery occlusion. Am J Ophthalmol 2008; 146 (5): 700–6.
5. Kattah JC, Wang DZ, Reddy C. Intravenous recombinant tissue-type plasminogen activator thrombolysis in treatment of central retinal artery occlusion. Arch Ophthalmol 2002; 120 (9): 1234–6.
6. Augsburger JJ, Magargal LE. Visual prognosis following treatment of acute central retinal artery obstruction. Br J Ophthalmol 1980; 64 (12): 913–7.
7. Ffytche TJ. A rationalization of treatment of central retinal artery occlusion. Trans Ophthalmol Soc U K 1974; 94 (2): 468–79.
8. Rassam SM, Patel V, Kohner EM. The effect of acetazolamide on the retinal circulation. Eye (Lond) 1993; 7 (5): 697–702.
9. Atebara NH, Brown GC, Cater J. Efficacy of anterior chamber paracentesis and Carbogen in treating acute nonarteritic central retinal artery occlusion. Ophthalmology 1995; 102 (12): 2029–34.
10. Hayreh SS, Kolder HE, Weingeist TA. Central retinal artery occlusion and retinal tolerance time. Ophthalmology 1980; 87(1):75–8.
11. Hayreh SS, Weingeist TA. Experimental occlusion of the central artery of the retina. IV: Retinal tolerance time to acute ischaemia. Br J Ophthalmol 1980; 64 (11): 818–825.
12. Hayreh SS, Weingeist TA. Experimental occlusion of the central artery of the retina. I. Ophthalmoscopic and fluorescein fundus angiographic studies. Br J Ophthalmol 1980; 64 (12): 896–912.
13. Mueller AJ, Neubauer AS, Schaller U, Kampik A. Evaluation of minimally invasive therapies and rationale for a prospective randomized trial to evaluate selective intra-arterial lysis for clinically complete central retinal artery occlusion. Arch Ophthalmol 2003; 121 (10): 1377–81.
14. Neubauer AS, Mueller AJ, Schriever S, Gruterich M, Ulbig M, Kampik A. Minimally invasive therapy for clinically complete central retinal artery occlusion–results and meta-analysis of literature. Klin Monbl Augenheilkd 2000; 217 (1): 30–6.
15. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: visual outcome. Am J Ophthalmol 2005; 140 (3): 376–91.
16. Ahn SJ, Kim JM, Hong JH, Woo SJ, Ahn J, Park KH et al. Efficacy and safety of intra-arterial thrombolysis in central retinal artery occlusion. Invest Ophthalmol Vis Sci 2013; 54 (12): 7746–55.
17. Wang JJ, Cugati S, Knudtson MD, Rochtchina E, Klein R, Klein BE et al. Retinal arteriolar emboli and long-term mortality: pooled data analysis from two older populations. Stroke 2006; 37 (7): 1833–6.
18. Rudkin AK, Lee AW, Chen CS. Vascular risk factors for central retinal artery occlusion. Eye (Lond) 2010; 24(4): 678–81.
19. Achim Fieß, Ömer Cal, Stephan Kehrein, Sven Halstenberg, Inez Frisch, Ulrich Helmut et al. Anterior chamber paracentesis after central retinal artery occlusion: a tenable therapy?. BMC Ophthalmology 2014; 14: 28.

| RANGE (YRS) | NUMBER | PERCENTAGE (%) |
|------------|--------|----------------|
| 50-60      | 4      | 40             |
| 61-70      | 6      | 60             |

**TABLE 1: AGE DISTRIBUTION**

| GENDER | NUMBER | PERCENTAGE (%) |
|--------|--------|----------------|
| MALE   | 10     | 100            |
| FEMALE | 0      | 0              |

**TABLE 2: SEX DISTRIBUTION**


### Original Article

#### Table 3: Systemic Risk Factors

| Risk Factors      | Number | Percentage (%) |
|-------------------|--------|----------------|
| Diabetes          | 3      | 30             |
| Hypertension      | 2      | 20             |
| Hyperlipidemia    | 5      | 50             |

#### Table 4: Time of Presentation After Onset of CRAO

| Time (in Hrs) | Number | Percentage (%) |
|---------------|--------|----------------|
| < 8           | 4      | 40             |
| > 8           | 6      | 60             |

#### Table 5: Presenting Visual Acuity

| Vision                     | Number | Percentage (%) |
|----------------------------|--------|----------------|
| Hand Movements +           | 6      | 60             |
| Perception of Light +      | 4      | 40             |

#### Table 6: Best Corrected Visual Acuity After Paracentesis

| Vision                     | Number | Percentage (%) |
|----------------------------|--------|----------------|
| Counting Finger at 2m      | 4      | 40             |
| Hand Movements +           | 2      | 20             |
| Perception of Light +      | 4      | 40             |

### Authors:
1. Tanushree V.
2. H. T. Venkate Gowda
3. Savita Patil
4. Vijaylaxmi Patil
5. Tejashree T. Chavan
6. B. Agni

### Particulars of Contributors:
1. Senior Resident, Department of Ophthalmology, Mysore Medical College and Research Institute, Mysore.
2. Professor and HOD, Department of Ophthalmology, Mysore Medical College and Research Institute, Mysore.
3. Junior Resident, Department of Ophthalmology, Mysore Medical College and Research Institute, Mysore.
4. Junior Resident, Department of Ophthalmology, Mysore Medical College and Research Institute, Mysore.
5. Junior Resident, Department of Ophthalmology, Mysore Medical College and Research Institute, Mysore.
6. Junior Resident, Department of Ophthalmology, Mysore Medical College and Research Institute, Mysore.

### Name Address Email ID of the Corresponding Author:
Dr. H. T. Venkate Gowda, #1128, 1st Cross, Paduvana Road, T. K. Layout, Kuvempunagar, Mysore-570023. Email: drvrmc@gmail.com

Date of Submission: 15/11/2014.
Date of Peer Review: 17/11/2014.
Date of Acceptance: 26/11/2014.
Date of Publishing: 29/11/2014.