Pars Plana Vitrectomy for Central Retinal Artery Occlusion Without Emboli

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ABSTRACT

Purpose: The results of patients who had 23-G pars plana vitrectomy (PPV) unresponsive to medical therapy for central retinal artery occlusion without visible embolization are presented.

Materials and Methods: We operated on eight patients who were treated for acute central retinal artery occlusion without visible emboli in our clinic. We started emergency medical treatment following diagnosis which was unsuccessfull. Afterwards, the patient had PPV in six cases and PPV- trabeculectomy in two cases. During the operation we applied intraoperative hypotonia for ten minutes and we tried to restore retinal circulation with fluid turbulance on optic nerve head. While visual acuity remained at the basal level in five patients (hand movement positive), slight increase was observed in three patients (preop P (-), 20 cmFC, 2 mFC, ; postop 30 cmFC, 30 cmFC, 0.1 respectively). **Discussion and Conclusion:** Primary pars plana vitrectomy and surgical hypotonia may be an early treatment option for central retinal artery occlusion without visible emboli, which should be evaluated in large scale studies.

Keyword: Pars plana vitrectomy, central retinal artery oclussion, retina blood flow.

INTRODUCTION

Central retinal artery is the first branch of ophthalmic artery and nourishes the inner retinal layers. Therefore embolisation and occlusion of retinal artery is seen infrequently. Other causative factor may be arteriosclerosis and risk factors for cardiovascular and cerebrovascular aterosclerosis also apply for central retinal artery occlusion (CRAO)[1].

Diseases causing vasoobliteration in retinal artery occlusions; atherosclerosis, hypertension, periarteritis (polyarteritis nodosa, systemic lupus erythematosus), hematological disorders (protein S and C deficiencies), mitral valve prolapse, retinal migraine and carotid artery disease should not be forgotten[2, 3].

The most important symptom of acute CRAO is the sudden, painless loss of vision[4]. In 10% of patients, amaurosis fugax precedes vision loss. On examination, visual acuity is typically at the level of hand movements or light perception. If the cilioretinal artery supplies the fovea, normal central visual acuity can be found. The visual field shows the intact temporal islet of peripheral vision. There is absolute afferent pupillary defect on the affected side. Ischemic whitening occurs in the posterior pole due to decreased blood flow. Intermittent blood flow in arteries and veins and "cherry red spot" appearance in the macula is typical. There may be splinter retinal hemorrhages on the disc. After about four weeks, the retinal whitening disappears and the optic nerve becomes pale. It causes neovascular glaucoma in 18% of cases[5, 6].

CRAO incidence is approximately 1-2/100.000 of patients in ophthalmic examination; retinal artery branch occlusion incidence is 1/10.000[7-9]. CRAO results in acute, serious, permanent visual acuity loss and final visual results depend on the time period of CRA occlusion, presence of cilioretinal artery supporting macular region and type of embolisation[1, 10]. Current acute emergency medical treatment for CRAO includes ocular massage, reduction of intraocular pressure by medication, anterior chamber paracenthesis and vasodilatotory agents which can rarely reach satisfactory visual results [1]. In theory, acure retinal

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ischemia should be treated just like acute myocardial or cerebral ischemia and circulation should be restored in hours[1, 11-13]. Treatment should be started emergently following CRAO without delay best before the first 6-12 hours[1].

In our case series the patients who had diagnosis of CRAO had emergency medical treatment and hospitalisation first and those who did not benefit were offered 23G PPV surgery and PPV- trabeculectomy combination in two cases, results of which are presented in this manuscript.

MATERIALS AND METHODS

Eight patients with diagnosis of acute CRAO who were hospitalised and treated in XXXXX Hospital Department of Opthalmology in 2017- 2020 were retrospectively evaluated. The study adhered to the tenets of Helsinki and ethical approval was obtained. These patients did not have a visible emboli and all had preoperative fundus fluorescein angiography to evaluate perfusion status. In patients with poor perfusion, medical emergency treatment was started immediately and patients had consultations for cardiology, neurology and internal medicine. Patients had acetazolamid tb 3x1, potassium eff tb 1x1, mannitol 20% 1gm/kg IV, nasal oxygen, digital massage. Patients who were unresponsive to initial emergency medical treatment were offered 3 port 23 G PPV surgery under retrobulber anestesia as a last chance. Triamsinolon acetonide was used to seperate or check for seperation of posterior hyaloid face. Infusion was stopped for ten minutes in all patients to obtain intraoperative hypotonia under observation of fundus with a chandelier illumination, fluid turbulence at the optic nerve head level was created by the help of flute cannula backflushing and activating vitrectomy cutter in front of optic nerve. In all cases retinal artery calibers were increasing under hypotonia and were narrowing under normal surgical intraocular pressures. Partial fluid air exchange was performed, one drop of triamcinolone acetonide was injected and transconjunctival sclerotomy incisions intentionally were not sutured at the end of the operations intending for lower IOP values in postoperative period. In two cases trabeculectomy was combined with PPV to maintain a longer term hypotonia. Rectangular scleral flap with removable suture for IOP control was preferred. In all patients postoperative hypotonia was maintained with additional antiglaucoma medical therapy, if needed, to keep IOP between 10-15mmHg.

CASES 1-2-3

Three patients with CRAO (case 1, 2, 3) were examined one day following sudden visual acuity loss. These three patiens had P (+), P(+) ve HM visual acuity on admission. Medical history revealed an eye trauma and cataract surgery with a sutured IOL implantation in case 2 . We did not observe any emboli pre and peroperatively. These patients did not have systemic comorbid diseases, only one case with a history of ocular trauma. The patients had PPV surgery with intraoperative hypotony and fluid turbulence, on day 3, 3 and 2 after medical treatment. On the first and second postoperative month, visual acuity did not improve which was hand motions in all cases (Figure 1a-b-c).

CASE 4

Case 4 was admitted one hour after sudden visual acuity loss. Patient medical history revealed a previous myocardial infarction two years ago, therefore the patient was on anticoagulation with acetylsalicylic acid. On

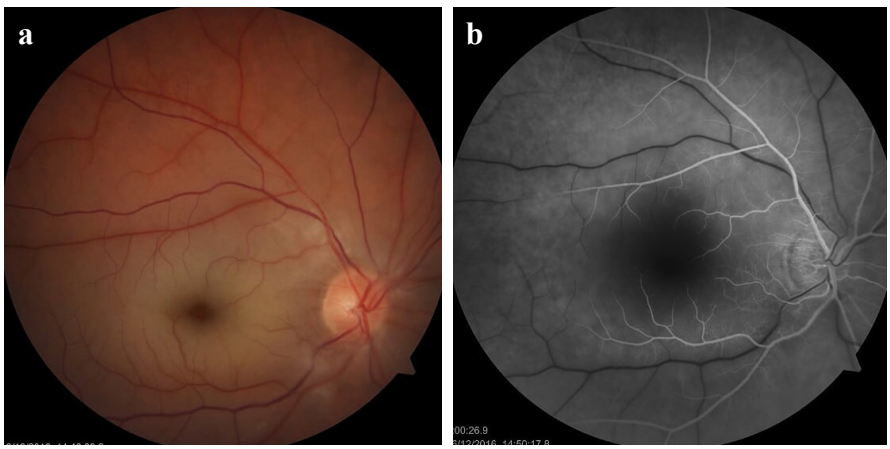


Figure 1a-b: *Color Figure and FFA of case 1 OD on the first day. Cherry red spot is seen and retinal artery perfusion is delayed (26. Sec).*

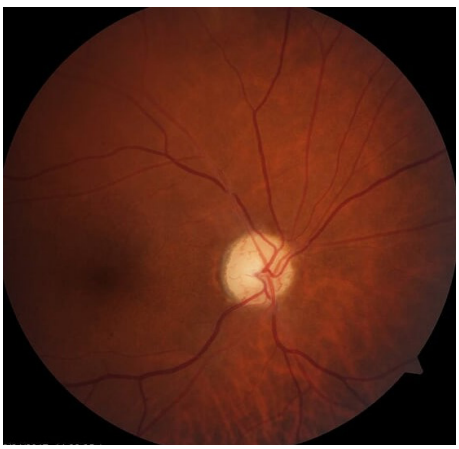


Figure 1c: *Case 1 In 4 month control there is a pale optic nerve and narrow arteries on color Figure.*

ophthalmic examination, visual acuity was 2m FC right eye and 20/20 in OS. Intraocular pressure was 16 mmHg OD, 17 mmHg OS, righ eye had relative afferent pupillary defect. Anterior segment was normal on biomicroscopy. Fundus examination showed a pale retina and cherry red spot/Japanise flag sign in OD. OS fundus apperance was normal. The patient was hospitalised, had emergency medical treatment together with cardiology consultation without benefit. On the second day PPV surgery was performed with predefined technique. On first month control visit visual acuity was 0.05; on second month 0.1 (Figure 2a-b-c).

CASE 5

The patient applied to the hospital with sudden visual loss in OD with two days delay, visual acuity was perception negative which was checked many times. Medical history showed hipertension and chronic obstructive pulmonery disease. and ophthalmic history of ischemic optic neuropathy OU with inferior hemialtitudinal visual field defects two years ago. IOP was 19mmHg. FFA examination revealed enlargement of FAZ and perfusion defect OD, left eye had macular edema in superior temporal area with vascular tortuisity and linear haemorrhages. Diagnosis was CRAO OD, and RVBO OS (Figure 3a-b-c-d-e-f). AntiVEGF injection was started in the left eye.

Following emergency medical treatment the patient had PPV surgery on 4th day. On control examinations on 2nd month visual acuity was 30cmFC OD, 0.2 OS. Fundus examination showed atrophic retina and optic disc pallor OD, ghost vessels superotemporally OS. OCT also showed thin retina (161 micron) and ERM, macular edema OS.

CASE 6

The patient was examined 14 days after visual loss in our clinic. In medical history, there was bilateral cataract operation two years ago. Visual acuity was P(+) OD, 0.7 OS, biomicroscopy showed bilateral pseudophakia with PC IOL. In right eye pupillary light reflexes were weak IOP was 15 mmHg, 14 mmHg OS. In fundus examination right eye optic disc was pale, macula was pale and Japanise flag apperance. In OCT right eye had subretinal fluid with exudative retinal detachment, left eye was normal. FFA showed delayed filling in OD with a localised area of perfusion due to cilioretinal artery in right eye, left eye was normal (Figure 4a-b). Following medical therapy the patient had PPV surgery on 15th day. The patient did not have an increase in visual acuity in short term and later was lost to follow-up.

CASE 7-8

The two cases with CRAO was admitted to the hospital on the first day after visual loss. Both had diabetes mellitus as comorbidity. In ophthalmic examination visual acuity in the affected right eyes were P(+) and 20 cmFC consequtively, IOP values were 17 mmHg, 11 mmHg. These patients were both operated on the second day after emergency medical treatment with PPV combined with trabeculectomy. Long term hypotony thus could be achieved. Postoperative first month visual acuit was Hand motions and 30 cmFC with IOP of 6 mmHg and 11 mmHg. Medical treatment with brimonidine eyedops was added.

The patient characteristics of the study group of SRAO cases who underwent PPV had are shown in table 1.

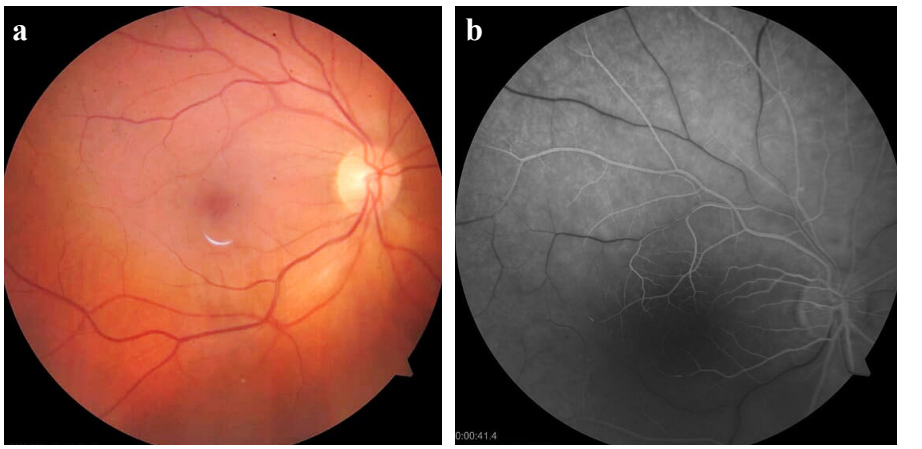


Figure 2a-b: *Color Figure and FFA of case 4 with CRAO OD. Delayed arterial filling on 44 sec is evident.*

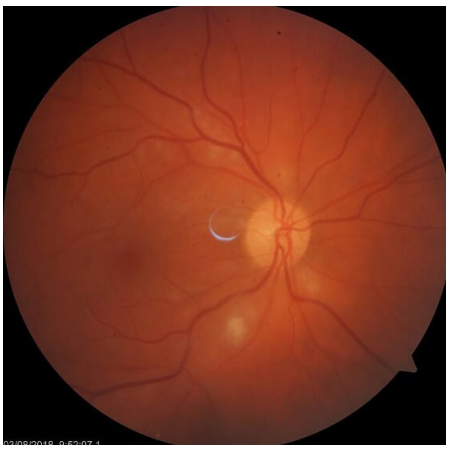
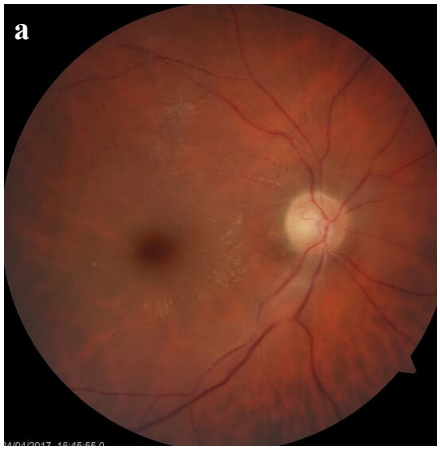


Figure 2c: *Case 4 Color Figure of OD on first month control. There is a slight optic disc pallor.*



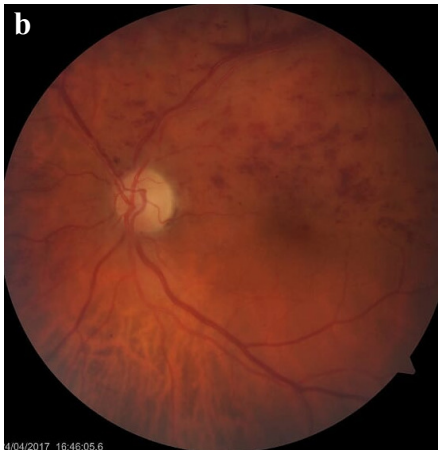
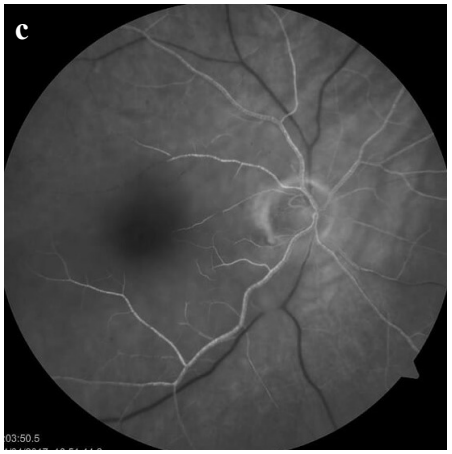


Figure 3a-b: *Case 5 Color Figure of OD with signs of CRAO and OS with superotemporal BRVO. Optic discs are pale.*



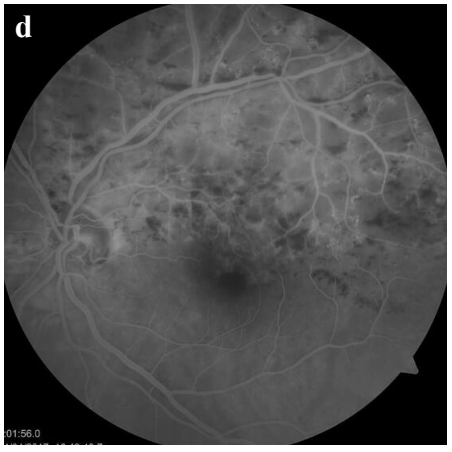
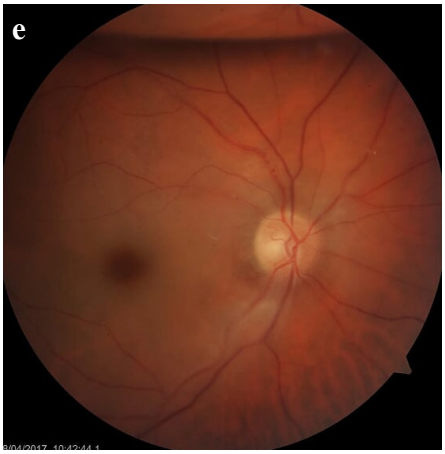


Figure 3c-d: *Case 5 FFA Figures Show delayed filling in OD and superotemporal vascular leakage masking with haemorhages in OS.*



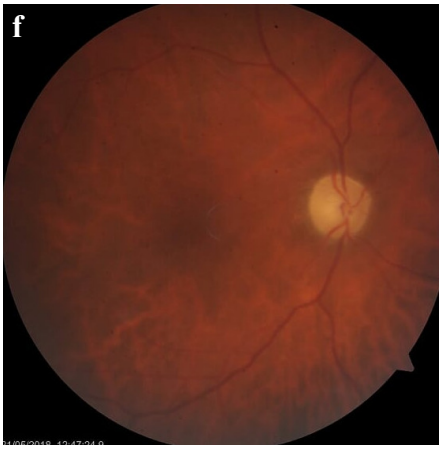
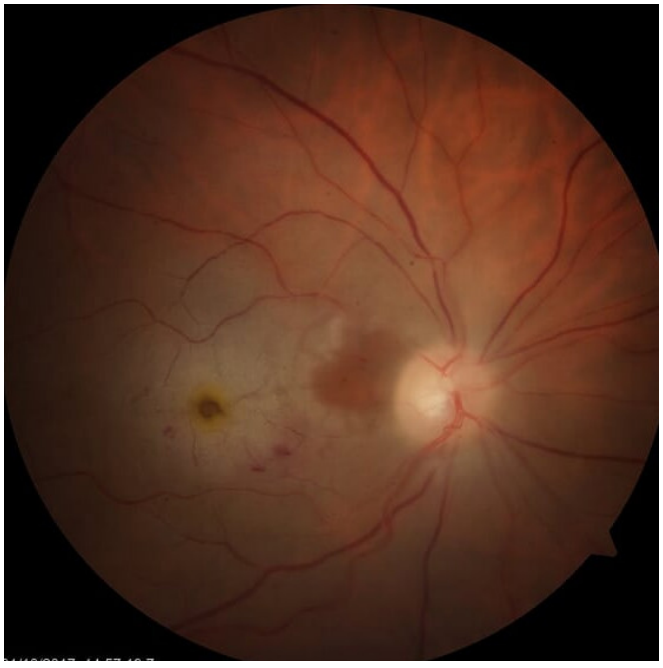


Figure 3e-f: *Case 5 Color Figure of OD postoperative first day with macular edema, partial air in vitreus cavity, and 2nd month with optic mild disc pallor and atrophy.*



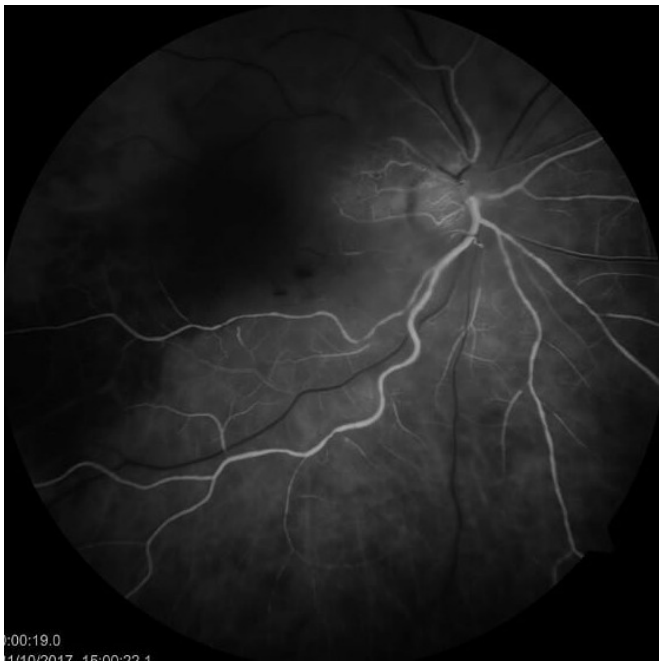


Figure 4a-b: *Case 6 CRAO with a minor cilioretinal artery protection.*

Table 1: *Patient characteristics of the study group of SRAO cases who had PPV surgery*

	Patient age, gender, laterality	Comorbid diseases	Operation Timing (days)	Preop Visual acuity	Postop Visual Acuity	PPV Timing (days)	Preop IOPmmHg	Postop 1. Week IOP mmHg
CASE 1	56/M/Right	nil	1	P+	HM	3	12	7
CASE 2	60/M/Left	Trauma	1	P+	HM	2	10	7
CASE 3	54/M/Right	nil	1	EH	HM	2	20	7
CASE 4	47/M/Right	COPD	1hour	2MPS	0.1	2	16	9
CASE 5	67/M/Right	HT, COPD	2	P-	30 CmFCS	4	19	11
CASE 6	80/M/ Right	nil	14	P+	HM	15	15	10
CASE 7	39/F/ Right	DM	1	P+	HM	2 PPV Trab	17	6
CASE 8	67/M/ Left	DM	1	20 CmFC	30 CmFC	2 PPV Trab	11	11

DISCUSSION

The aims of the treatment strategy for CRAO are; to increase retinal oxygenation and restore retinal blood flow to prevent retinal damage. Retinal oxygenation can be increased by inhalation of carbogen (95% oxygen, 5% carbon dioxide)[14]. The increase in retinal artery blood flow is tried to be achieved by lowering the intraocular pressure. This can be achieved with ocular massage, paracentesis and ocular antihypertensive drugs. Anticoagulants, fibrinolytic agents, intra-arterial tissue plasminogen activator injection with ophthalmic artery catheterization, antioxidant drugs were used and their effects were discussed[15, 16]. High-dose corticosteroids should be used urgently in CRAO associated with temporal arteritis[17].

There is a limited chance for good visual acuity results for patients with CRAO with classical emergency medical therapy, therefore alternative treatment methods are searched. Pars plana vitrectomy creates changes in ocular oxygenation and associated fluid turbulence can reduce vascular resistance and increase retinal blood flow. Vitrectomy can also increase retinal oxygenation which is reported in the literature[18, 19]. Emboli (if present) can move distally or break down to smaller pieces by fluid turbulence[20, 21]. Wen et al studied on rabbit eyes and showed that PPV surgery and aspiration in front of optic nerve with 350-400mmHg for 5 seconds could increase retinal blood flow. After the embolic event, fibrin and platelets activate in 24 hours, so emboli may better be dislodged at that time period before they attach tightly[21]. Intervention in the first hours after CRAO by PPV (Primary PPV treatment) can help to improve visual acuity in CRAO in light of these information.

Only one patient in our group could be treated medically in a very ideal time period (case 4 in one hour), all the other cases presented were treated after 24 hours or more after visual loss. All the patients had cardiac, neurologic and internal medicine consultations and were given emergency medical treatment on admission to the hospital. The earliest time to operate by PPV was 2 days, therefore we think the visual prognosis can be limited with this time loss. Case 4 had the most favorable prognosis who improved from finger counting to 0.1 after PPV. Case 5 was interesting as visual acuity was tested as perception negative multiple times before operation and the benefit of operation was discussed with the patient and operation was performed on patient's demand. Postoperatively visual acuity was 30cm FC which was expressed as 'going from dark to sunlight' by the patient. This particular case was very extraordinary, demonstrating possible benefit of PPV. Cases 1, 2, 3, 6, 7, 8 started with a low visual acuity, which did not change much following the operation

If a cilioretinal artery protecting the macular circulation exists, visual prognosis may be far better. In our cases there was not a case with a total macular protection but in one case (case 6) a limited area of healthy retina and cilioretinal circulation did not help in improvement. Postoperatively IOP was kept below 15mmHg, preop mean IOP was 15.0 ± 3.7 mmHg and postop mean IOP was 8.5 ± 2.0 mmHg on the first week controls. We think that low IOP might help in restoration of retinal circulation, so combination with a trabeculectomy might be feasable.

One of the factors in defining the effectivity of treatment in CRAO may be the charactheristics of retinal emboli. Cho et al reported fibrin-platelet emboli as the most common form and more prone to be dislodged. On the other hand, cholesterol and calcific emboli could not be reperfused easily [22]. In our patient grop, although we did not observe a visible emboli, we hypothetize that as treatment and digital

massage failed, they are more likely to be calcific deep settled emboli. We thought we couldn't see if the embolism was just behind or just behind the Lamina cribrosa.

CRAO is frequently the result of aterosclerotic changes and is considered as end organ damage which can be seen as a comorbid disease for myocardial nfarction and cerebral stroke. [23, 24]

Xiao et al reported in a retrospective study that cerebral stroke can increase the incidence of CRAO and carotid artery aterosclerotic plaques may be significant risk factor[25]. Hayreh et al found that myocardial infarction incidence before and after one year of CRAO was 21%[26]. In our case series two (case 4, 5) had hypertension and obstructive pulmonary disease history. Case 4 had previos myocardial infarction while case 5 died because of myocardial infarction in the first year follow up.

There is a time limit for the irreversible retinal damage to occur in CRAO[27]. This time is about 105 minutes for primates in experimental studies, however considering partial occlusion this important emergency intervention time period can be accepted as 12 to 33 hours [16, 28]. The primary target for treatment can be restoring circulation in this time frame. In our cases only one patient (case 4) could be treated in this critical period who relatively had a better visual acuity result. Even a patient with perception negative visual acuity had an increase to 30cm FC (case 5) which suggests that treatment may be offered to all cases. Even minor improvements may change patient life comfort.

In a study on the effectivity of vitrectomy surgery on CRAO, improvements occured in cases with a duration of 4 days or less, on the other hand after day 6 retinal circulation may restore but visual acuity did not increase. In the study of Lu et al., retinal circulation was observed after direct retinal artery massage in four cases. Although retinal circulation was not observed during the operation in the other four cases[29].

Contrary to the common impression, spontaneous recovery may occur in both visual acuity and visual fields in the first few days after the onset of CRAO, depending on the type of CRAO. So the recovery of some of the patients in the study group may not solely be attributed to surgical intervention[10].

In our study, minimal improvement in visual acuity was observed, except for one patient. This may be due to the NA-CRAO type of our cases, the lack of improvement in visual acuity despite the necessary medical and surgical intervention. At the same time, it may be that it was intervened as late as 48 hours. The visual improvement as a part of the natural history of CRAO limits to understand the beneficial effects of treatments. These may be limitations of our study.

CRAO is an emergency situation and interventions should be promptly done. However all treatment options do not offer a satisfactory visual results yet. Early vitrectomy surgery is a possible solution that can change the natural course, but surgery together with emergency medical treatment should be initiated probabily in hours following the occlusion, just as primary angioplasty in myocardial infaction by cardiologists. Late onset interventions can be of limited benefit which is also reflected in results of our patient group. Further controlled large scale studies by early PPV intervention to CRAO may highlight the best options and time for the treatment of CRAO.

Declaration of conflicting interests

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REFERENCES

1. Dattilo M, Biousse V, Newman NJ. Update on the Management of Central Retinal Artery Occlusion. Neurol Clin 2017; 35: 83-100.
2. Brown GC.Retinal arterial obstructive disease.İn:Schachat AP MR, Patz A, eds.Medical retina, Vol2ofRyan SJ, ed.Retina.St Louis; CV Mosby; 1989:73:1361-77.
3. Golub BM, Sibony PA, Coller BS. Protein S deficiency associated with central retinal artery occlusion. Arch Ophthalmol 1990; 108: 918.
4. Sanborn GE MLAodoteİTWJE, eds.Clinical Ophthalmology, vol 3. Philadelphia:Lippincott;1993;14:1-29.
5. Duker JS, Sivalingam A, Brown GC, et al. A prospective study of acute central retinal artery obstruction. The incidence of secondary ocular neovascularization. Arch Ophthalmol 1991; 109: 339-42.
6. Hayreh SS, Podhajsky P. Ocular neovascularization with retinal vascular occlusion. II. Occurrence in central and branch retinal artery occlusion. Arch Ophthalmol 1982; 100: 1585-96.
7. Leavitt JA, Larson TA, Hodge DO, et al. The incidence of central retinal artery occlusion in Olmsted County, Minnesota. Am J Ophthalmol 2011; 152: 820-3.e2.
8. Park SJ, Choi NK, Seo KH, et al. Nationwide incidence of clinically diagnosed central retinal artery occlusion in Korea, 2008 to 2011. Ophthalmology 2014; 121: 1933-8.

9. Rumelt S, Dorenboim Y, Rehany U. Aggressive systematic treatment for central retinal artery occlusion. Am J Ophthalmol 1999; 128: 733-8.

10. Hayreh SS. Ocular vascular occlusive disorders: natural history of visual outcome. Prog Retin Eye Res 2014; 41: 1-25.

11. Hayreh SS, Jonas JB. Optic disk and retinal nerve fiber layer damage after transient central retinal artery occlusion: an experimental study in rhesus monkeys. Am J Ophthalmol 2000; 129: 786-95.

12. Hayreh SS, Zimmerman MB, Kimura A, et al. Central retinal artery occlusion. Retinal survival time. Exp Eye Res 2004; 78: 723-36.

13. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: visual outcome. Am J Ophthalmol 2005; 140: 376-91.

14. Atebara NH, Brown GC, Cater J. Efficacy of anterior chamber paracentesis and Carbogen in treating acute nonarteritic central retinal artery occlusion. Ophthalmology 1995; 102: 2029-34; discussion 34-5.

15. Fernández FJ, Guelbenzu S, Barrena C, et al. [Selective ophthalmic artery fibrinolysis in acute central retinal artery occlusion]. Arch Soc Esp Oftalmol 2002; 77: 81-6.

16. Richard G, Lerche RC, Knospe V, Zeumer H. Treatment of retinal arterial occlusion with local fibrinolysis using recombinant tissue plasminogen activator. Ophthalmology 1999; 106: 768-73.

17. Matzkin DC, Slamovits TL, Sachs R, et al. Visual recovery in two patients after intravenous methylprednisolone treatment of central retinal artery occlusion secondary to giant-cell arteritis. Ophthalmology 1992; 99: 68-71.

18. Sín M, Sínová I, Chrapek O, et al. The effect of pars plan vitrectomy on oxygen saturation in retinal vessels--a pilot study. Acta Ophthalmol 2014; 92: 328-31.

19. Lim LS, Tan L, Perera S. Retinal vessel oxygen saturation increases after vitrectomy. Invest Ophthalmol Vis Sci 2014; 55: 3851-6.

20. Nadal J, Ding Wu A, Canut M. Vitrectomy With Intrasurgical Control of Ocular Hypotony as a Treatment for Central Retina Artery Occlusion. Retina 2015; 35: 1704-5.

21. Wen X, Yuan M, Li C, et al. Effect of vitrectomy with intrasurgical regulation of intraocular pressure in a rabbit model of central retinal artery occlusion. Exp Eye Res 2019; 189: 107779.

22. Cho KH, Ahn SJ, Cho JH, et al. The Characteristics of Retinal Emboli and its Association With Vascular Reperfusion in Retinal Artery Occlusion. Invest Ophthalmol Vis Sci 2016; 57: 4589-98.

23. Varma DD, Cugati S, Lee AW, et al. A review of central retinal artery occlusion: clinical presentation and management. Eye (Lond) 2013; 27: 688-97.

24. Cugati S, Varma DD, Chen CS, et al. Treatment options for central retinal artery occlusion. Curr Treat Options Neurol 2013; 15: 63-77.

25. Xiao YY, Wei WB, Wang YX, et al. Correlation of the history of stroke and the retinal artery occlusion: a nested case-control study. Int J Ophthalmol 2020; 13: 431-7.

26. Hayreh SS, Zimmerman MB. Ocular arterial occlusive disorders and carotid artery disease.Ophthalmol Retina 2017; 1: 12-8.

27. Tobalem S, Schutz JS, Chronopoulos A. Central retinal artery occlusion - rethinking retinal survival time. BMC Ophthalmol 2018; 18: 101.

28. 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: 818-25.

29. Lu N, Wang NL, Wang GL, et al. Vitreous surgery with direct central retinal artery massage for central retinal artery occlusion. Eye (Lond) 2009; 23: 867-72.