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Ocular Neovascularisation in Central Retinal Artery Occlusion (CRAO)

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Abstract

Presentation
We report the case of a man in his 70s presented with 60 minutes of painless monocular vision loss.

Diagnosis
Following ophthalmological examination, a diagnosis of a right eye central retinal artery occlusion was made. This was confirmed via multimodal imaging consisting of optical coherence tomography (OCT) and fundal fluorescein angiography (FFA).

Treatment
Acute intraocular pressure lowering measures were undertaken without resolution of the obstructing embolus at the bifurcation of the ophthalmic artery. Approximately 6 weeks following the event, this patient developed neovascular glaucoma that required multiple therapies to restore the eye to a comfortable state.

Discussion/Conclusion
CRAO is a uncommon but devastating cause of monocular vision loss. This case highlights that treatment options in the acute setting are controversial with none proven to be beneficial to final visual acuity outcomes. Furthermore, treating physicians should be aware that CRAO may lead to neovascular glaucoma and a resultant painful blind eye.

Introduction

Central retinal artery occlusion (CRAO) is an ocular emergency caused by obstruction of the central retinal artery leading to inner retinal ischaemia and devasting monocular vision loss. The treatment in the acute setting is hotly debated with avoidance of ocular complications a key priority.

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Case Report

A 73-year-old male who presented to the eye-emergency-department with 60-minutes of painless loss of vision from his right eye. He had no associated jaw claudication, scalp tenderness, temporal headache, or focal neurology. His medical history included hyperlipidaemia and hypertension. His best-corrected visual acuity (BCVA) in his left eye was 6/5 while his right eye BCVA was 'Hand movements'. His right eye had a positive relative-afferent pupillary defect and reduced colour vision on Ishihara plates (0/17). Anterior segment examination was unremarkable with an intraocular pressure (IOP) of 17 mmHg in both eyes. Right eye funduscopy showed diffuse intraretinal oedema with a "cherry-red" macula, attenuated retinal arteries, and an embolism at the bifurcation of the ophthalmic artery at the optic nerve head. Optical coherence tomography (OCT) showed hyperreflectivity of the inner retinal layers in the affected eye. Fundus fluorescein angiography (FFA) showed normal choroidal filling but delayed filing of the retinal arteries confirming a diagnosis of central retinal artery occlusion (CRAO). Acute treatment with ocular massage and IOP-lowering medications was commenced followed by urgent referral to the inpatient-stroke service. Investigations revealed >70% internal carotid stenosis on Doppler ultrasound, normal neuroimaging, and normal inflammatory markers.

6 weeks later, the patient noted a red and painful right eye with IOP elevation and iris neovascularisation noted on examination. This neovascular glaucoma required intravitreal bevacizumab injections, panretinal photocoagulation laser, and cyclodiode laser to return the blind eye to a comfortable state.

Discussion

CRAO has an estimated incidence of 1 to in 100,000[1] with a male gender predilection and mean age of onset of 60-65 years. Patients' typically report painless, monocular total vision loss with 74% of affected eyes presenting with a visual acuity of counting fingers or worse [2]. However, in approximately 15-30% of the population the fovea is supplied by the cilioretinal artery (a branch of the short ciliary artery) and these individuals will have preserved central visual acuity[2].

Aetiologies for CRAO are similar to those for cerebral stroke with ipsilateral carotid artery atherosclerosis being the most common cause in up to 70% of cases[3]. Other non-arteritic causes include cardiogenic embolism, hypertension, hypercholesterolaemia and vascular pathologies[3]. Therefore, cardiovascular risk factors optimization is recommended to prevent secondary ischaemic events. CRAO may be associated with giant cell arteritis in approximately 5% of cases and should be ruled out via C-reactive protein (CRP) or erythrocyte sedimentation

rate (ESR) testing [2]. Iatrogenic causes of CRAO include cerebral angiography, carotid stenting or endarterectomy, and coiling of intracranial aneurysms[3].

Experimental studies performed have shown that the retina can only survive for 90 to 100 minutes after occlusion of the central retinal artery and therefore, spontaneous recovery in vision is rare[4]. Partial recovery may be possible if ischaemia is reversed within 240 minutes but occlusions lasting over this time will likely lead to permanent damage[5]. Acute treatment theoretically aims to resolve the arterial occlusion to restore retinal blood flow and may include anterior chamber paracentesis, ocular massage, and IOP-lowering medications. However, these treatment methods have not been shown to improve in clinical outcomes compared to observation[6] and may actually lead to worse outcomes[7].

Importantly, the EAGLE study, a randomised-control-trial comparing intra-arterial thrombolysis by tissue plasminogen activator (tPA) to placebo in CRAO treated within 20 hour of symptom onset noted the group that received thrombolysis had no significant difference in BCVA after 30 days and a significant increased risk of symptomatic intracranial haemorrhage[8]. Recently, a meta-analysis of observation studies suggests intravenous tPA given within 4.5 hours may provide a mild visual benefit but the risk of intracranial or choroidal haemorrhage remains[7].

Overall the visual prognosis for CRAO is poor with the priority of treatment focusing on cardiovascular risk factor optimization [9]. As seen in our patient, ocular neovascularization is a serious complication of CRAO that has been reported in approximately 15% of eyes with CRAO– an important consideration for the treating ophthalmologist[10].

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Figures

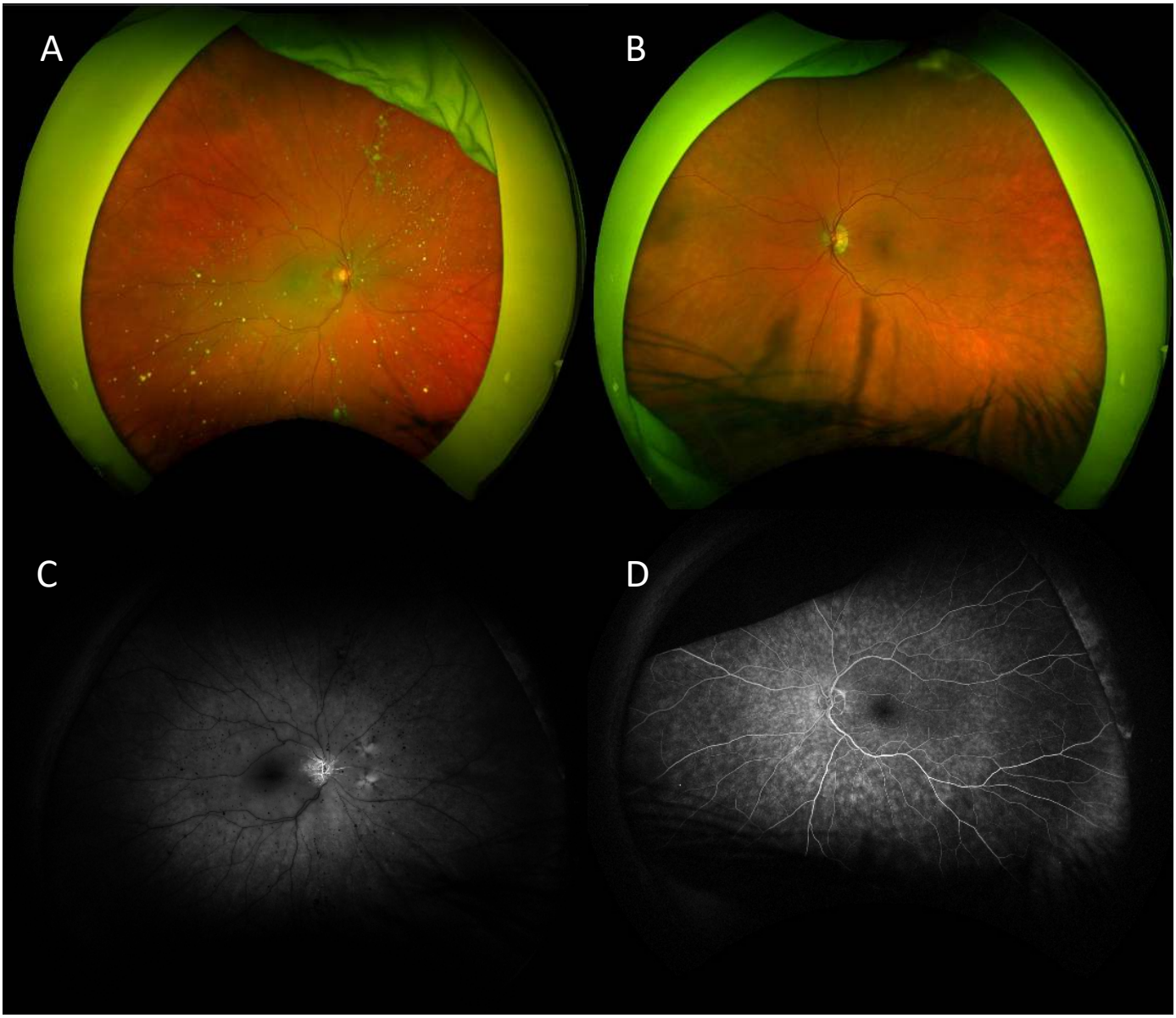

Figure 1: (A & B) Widefield fundus photograph (Optos 'California', Optos plc, Scotland) of the right and left eye respectively showing right eye asteroid hyalosis, retinal oedema, attenuated retinal arteries, and a subtle cherry-red macula. Left eye unremarkable. (C & D) Widefield late phase fluorescein angiogram (FFA) of the right and left eye respectively showing right eye delayed filling of retinal arteries (30s).

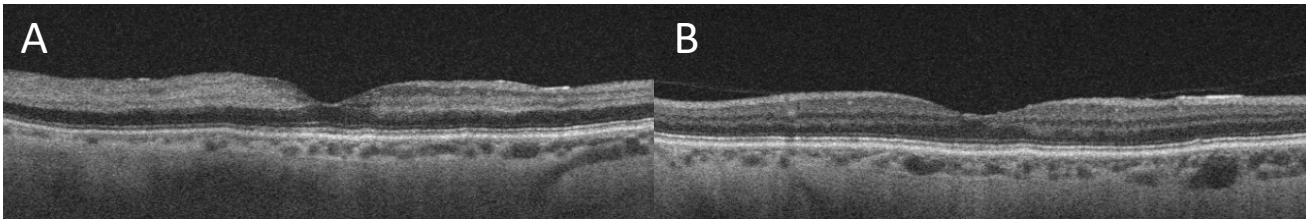

Figure 2: (A & B) Macular optical coherence tomography (OCT, Cirrus 5000, Carl Zeiss Meditec, Dublin, USA) of the right and left eye respectively showing hyperreflectivity of the inner retinal layers in the right eye and a normal left eye.

Declarations of Conflicts of Interest:
None declared.

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