Traumatic central retinal artery occlusion with optic neuropathy: a case report

Sonjoy KD, Situma PW, Kareko CW

Ispahani Islamia Eye Institute and Hospital

Corresponding author: Dr. P. W. Situma. Email: situmawanyama@gmail.com

ABSTRACT

Traumatic optic neuropathy with central retinal artery occlusion is rare. We report a case of an 11 year old male patient who presented with poor vision in the left eye after blunt trauma to the forehead. The visual acuity was light perception and he had relative afferent pupillary defect. Examination and investigations revealed optic neuropathy with central retinal artery occlusion and patent cilioretinal artery. In spite of treatment with oral prednisolone for 2 weeks the visual acuity remained light perception after 3 months of follow up. The right eye was found to be normal. Traumatic central retinal artery occlusion is rare but should be ruled out in patients with poor vision due to trauma to the head.

Key words: Traumatic optic neuropathy, Central retinal artery occlusion, Cilioretinal artery

INTRODUCTION

Central Retinal Artery Occlusion (CRAO) is the occlusion of the central retinal artery with resultant infarction of the retina and vision loss[1]. It's an emergency and the ocular analogue of brain stroke[2]. CRAO occurs mainly among the elderly with evidence of artheromatous emboli[3]. Cases in young patients are often associated with systemic conditions such as hyperhomocysteinemia, antiphospholipid antibody, thrombocytosis, vasculitis, occasionally cardiovascular risks etc[3-5].

Though reported before[6-9], blunt trauma remains a rare cause of CRAO, especially in association with Traumatic Optic Neuropathy (TON). We present a case of an 11 year old male patient with TON and CRAO with cilioretinal artery sparing following blunt trauma to the head.

CASE REPORT

An 11 year old male patient presented to us 5 days after blunt trauma to the forehead with sudden loss of vision in the left eye. There was no associated headache, loss of consciousness, convulsion or drowsiness. There was also no rhinorhoea or otorhoea. He had no complaints about the right eye. He had no known chronic illnesses.

On examination, the right eye was found to be normal with visual acuity of 6/6. In the left eye the visual acuity was light perception (PL), he had a Relative Afferent Papillary Defect (RAPD), and fundoscopy revealed a swollen optic disc and a pale edematous retina in the posterior pole with a cherry red spot at the macula. There was also an area adjacent to the optic disc, which maintained the normal retina colour, though this did not extend to the fovea. Fundus Fluorescent Angiogram (FFA) showed delayed arterial filling and an enlarged foveal avascular zone. The Optical Coherent Tomogram (OCT) showed hyper-reflective and swollen inner layers of the retina with shadowing of the outer layers and cystic space within the fovea.

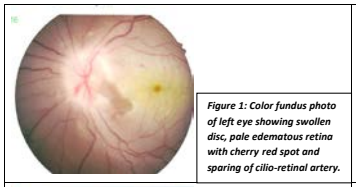

Figure 1: Color fundus photo of left eye showing swollen disc, pale edematous retina with cherry red spot and sparing of cilio-retinal artery.

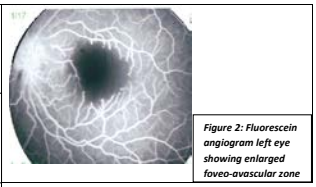

Figure 2: Fluorescein angiogram left eye showing enlarged foveo-avascular zone

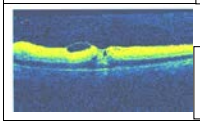

Figure 3: OCT left eye showing thickened hypereflective inner retinal layers with shadowing of the

Systemic examination showed normal blood counts, normal blood sugar, normal kidney function tests and normal total cholesterol levels at 187mg/dl (reference: <200mg/dl). However, he had elevated serum Low Density Lipoprotein (LDL) at 148mg/dl (reference: <130mg/dl) and Low High Density Lipoprotein (HDL) of 19mg/dl (reference:>35mg/dl). The blood pressure was normal at 100/60mmHg, Electro-Cardiogram (ECG) revealed sinus tachycardia and the echocardiogram was normal. CT scan of the brain and orbit revealed tissues swelling of the canalicula portion of the optic nerve but no fractures.

The patient was diagnosed with TON and CRAO with cilioretinal artery sparing. He was subsequently put on oral prednisolone 20mg once per day in the morning for 2 weeks and then tapered off over a period of one week. On subsequent follow up three months later the visual acuity was still PL, the optic disc showed signs of atrophy and the retina remained opalescent.

DISCUSSION

CRAO occurs mostly in the elderly patients with cardiovascular risk factors leading to artheromatous

emboli. In children the condition is rare and when it occurs an underlying systemic cause is often present[3]. Blunt trauma is a very rare cause of CRAO with TON. Our patient had suffered blunt trauma to the forehead and investigations also revealed a deranged lipid profile with high LDL levels and low HDL.

The mechanism of CRAO in blunt trauma is not fully understood. Some of the mechanisms hypothesized include direct compression of the optic nerve, the central retinal artery and vascular supply to the optic nerve[10]. This could be due to edema of orbital tissues, haematomas or direct impingement by bony spicules. Compression forces transmitted to the orbital apex cause a compartment syndrome whereby compression leads to a vicious cycle of swelling and ischemia.

The role of endothelial disruption due to sudden stretching of the vessels with subsequent clot formation has also been ascribed[9]. This is augmented by vaso-spastic response which is part of the normal clotting cascade[7]. The end result is ischemia to the retina and optic nerve with resultant profound loss of vision.

Few reports have been published of TON with CRAO. Cumurcu[10] wrote of a 10 year old child with blunt trauma to the left eye with subsequent total loss of vision in that eye. He was diagnosed with TON and CRAO and received bolus high dose steroids for 72 hours followed by oral steroids without improvement in visual acuity.

Vianna[6] reported a case of traumatic parafoveal arteriolar obstruction and TON in a 32 year old male who also suffered from systemic lupus erythematosus. The patient had suffered blunt trauma to the right eye and developed poor vision within hours. In that patient too, the visual acuity remained poor (HM) despite treatment with oral prednisolone 60mg per day. Karna *et al*[9] also reported a case which had poor outcomes in visual acuity.

In spite of being treated with oral steroids the visual acuity remained PL in our patient. There's generally no consensus on the management of optic neuropathy due to trauma[11] and the few case reports on CRAO with TON point to disappointing outcomes irrespective of the treatment regimen employed[6,10]. The international optic nerve trauma study[12] concluded that neither corticosteroids nor optic canal surgery should be considered the standard of care for patients with traumatic optic neuropathy. It is therefore clinically reasonable to decide to treat or not treat on an individual patient basis.

As far as we know, ours is the first case with TON and CRAO in which the cilioretinal artery remained patent. However the vision was still poor, likely because the area supplied by the cilioretinal artery in this patient did not include the foveal area and also because of the optic neuropathy.

In conclusion, though rare, CRAO can occur in blunt trauma and should be watched out for by clinicians as they manage patients with trauma. Even so, a search for underlying systemic conditions must be carried out to rule out other covert risk factors.

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