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# CENTRAL RETINAL ARTERY OCCLUSION DUE TO BLUNT OCULAR TRAUMA ASSOCIATED WITH ROAD TRAFFIC ACCIDENT : A RARE CASE REPORT

**Ophthalmology**

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## ABSTRACT

We present a case of central retinal artery occlusion (CRAO) associated with blunt ocular trauma without involvement of optic nerve. A 40 year-old male patient was brought to the emergency department of a tertiary care hospital in the western province of India with a history of head injury due to road traffic accident. External ocular examination of right eye revealed lacerated wound with edema and ecchymosis in upper eyelid, temporal subconjuctival hemorrhage, mid dilated and non reacting pupil on direct light reflex. Fundus examination of right eye with indirect ophthalmoscopy showed disc pallor, severe arteriolar attenuation with absent arterial pulsations on digital pressure, box carring of retinal vein ,cherry red spot at macula and white out retina. A clinical diagnosis of right eye central retinal artery occlusion was made. Patient was immediately treated with ocular massage in right eye and intravenous mannitol 300ml. Repeat fundus examination showed reapperance of arterial pulsation on digital pressure in right eye. To the best of our knowledge, this is a rare case of CRAO without involvement of optic nerve following blunt ocular trauma. We feel that it is important to consider this rare but visually catastrophic condition in the differential diagnosis of acute post-traumatic visual loss.[1]

## KEYWORDS

Central Retinal Artery Occlusion, Blunt Ocular Trauma, Road Traffic Accident, Head Injury

**CASE REPORT:**

A 40 year old male patient was brought to emergency department of tertiary care hospital with history of road traffic accident while going onto 2 wheeler which slipped and hence the patient sustained head injury. Patient had history of loss of consciousness , vomiting, nasal and oral bleed at the time of accident. He did not have a relevant past ocular or medical history and his family and social history were not significant.

On examination patient was conscious and well oriented to time, place and person. On ocular examination of right eye visual acuity was No perception of light, lacerated wound of 3×1 cm with edema and ecchymosis of upper eyelid, sub conjunctival hemorrhage temporally, mid dilated non reacting pupil on direct light reflex. Fundus examination of right eye with indirect ophthalmoscopy showed disc pallor, severe arteriolar attenuation with absent arterial pulsations on digital pressure, box carring of retinal veins, cherry red spot at macula and white out retina.

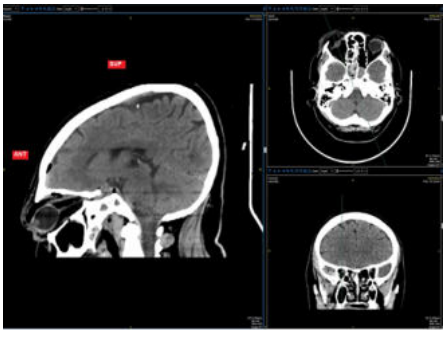
Left eye anterior and posterior segment examination was normal.

Immediate right eye digital ocular massage was given for 10 minutes. Lacerated wound of upper eyelid of right eye was sutured in 2 layers.

Patient was immediately sent for non contrast CT scan examination (fig1).

Non – contrast CT scan of head showed slightly displaced comminuted fracture in the lateral wall of right orbit, extending medially in the posterior part of roof of orbit with linear superior extension in right lateral portion of frontal bone. Linear fracture in right superior orbital rim extending postero medially in posterior wall of right frontal sinus . Mildly displaced fracture in lamina papyracea of right ethmoid air cells. Bilateral pneumo orbits. Periorbital swelling on right side with prominent subcutenous air collection within it.

The CT-SCAN is suggestive of an extra axial hematoma; probably extra dural hematoma in lateral aspect of right antero inferior frontal region. Small extra axial air collection along the calvarium in right frontal region suggestive of pneumocranium.



**(fig1) CT scan showed no discontinuity in the course of intraorbital and intracanalicular part of optic nerve and no direct compression of optic nerve with hematoma or emphysema**

Patient was immediately shifted to intensive care unit for further management. Intravenous mannitol 300ml was given.

On examination of right eye after ocular massage and intravenous mannitol, there was no improvement in visual acuity.

To rule out other causes of CRAO, patient was further investigated for cardiovascular defect by echocardiography (ECG),carotid doppler, bleeding time, clotting time, complete hemogram with platelet count, blood sugar, lipid profile, C-reactive protein, Anti-neutrophil cytoplasmic antibody(ANCA), rheumatoid arthritis (RA) factor, Venereal Disease Research Laboratory (VDRL), and antinuclear antibody (ANA). All these investigations were within normal limits.

On follow up examination of right eye after 24 hours visual acuity was no perception of light ,mid dilated non reacting pupil on direct light reflex . Fundus examination of right eye revealed disc pallor, arterial pulsation present, severe arteriolar attenuation, Box carring of retinal veins, tessalated background, cherry red spot at macula and white out retina.

Patient was vitally stable and was shifted to ward after 6 days. Patient

was called to eye OPD for FFA and OCT examination. Right eye anterior segment examination was same as before.
The right eye fundus findings were same as before on 7th day of examination.(fig 2a)

FFA showed no delay in arteriovenous transit time, normal choroidal filling suggestive of reperfusion of vessels (fig 3).

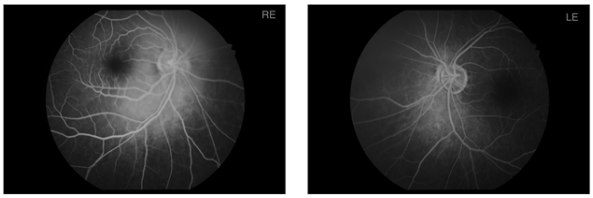



**fig 3**

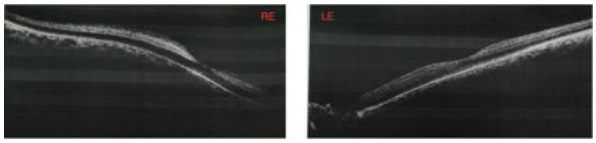



**fig 4**

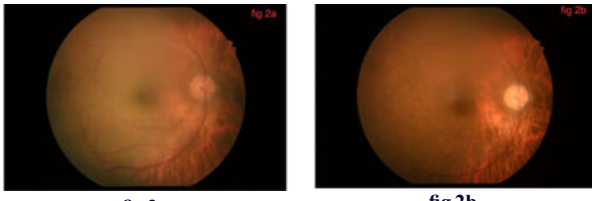



**fig 2a** **fig 2b**

On follow up examination of right eye after one month visual acuity was no perception of light, mid dilated not reacting pupil on direct light reflex. Fundus examination revealed Optic atrophy, minimal arteriolar attenuation, cherry red spot at macula and no retinal edema (fig 2b).

## DISCUSSION:

The incidence of central retinal arterial occlusion is estimated to be around 0.85/100,000 per year.[2] Various causes of arterial occlusion include embolism, thrombosis, atherosclerosis, congenital/acquired thrombophilic states, vasculitis, infectious, iatrogenic, ocular trauma, vasospasm, raised intraocular pressure etc.[2]

CRAO after blunt trauma, though rare, can be caused by compression of the central retinal artery by hematoma [3], by air in case of orbital emphysema [4], or raised intra orbital pressure resulting from swelling of orbital soft tissue. In these conditions, damage to endothelial cells of the artery stimulates platelet aggregation and thrombus formation.[1]

In our case, inspite of orbital wall fractures, there was no injury to optic nerve as per radiological findings. Moreover there were no signs of orbital hematoma or raised intraocular pressure. Hence in our case, the probable mechanism of CRAO could be severe reflex vasospasm initiated as a direct response to concussion injury to the arterial smooth muscle.

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