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From Binge to Blindness: Acute Central Retinal Artery Occlusion in Two Chronic Alcohol Users

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Abstract

Background: Central retinal artery occlusion (CRAO) is an ophthalmic emergency characterized by sudden, painless monocular vision loss and is considered an ocular analogue of ischemic stroke [1]. Although typically seen in elderly patients with vascular risk factors, its occurrence in young adults is uncommon and often indicates underlying systemic or vascular pathology.

Case Presentation: We describe two young male patients who presented with acute painless monocular vision loss due to CRAO associated with ipsilateral internal carotid artery (ICA) occlusion and concurrent cerebral infarction. Both patients had a preceding febrile illness and a recent alcohol binge. Fundoscopic examination revealed retinal pallor with a cherry-red spot and a relative afferent pupillary defect. Neuroimaging revealed ICA occlusion with acute cerebral infarction. Cardiac evaluation was unremarkable and neither patient had any conventional vascular risk factors. Laboratory findings showed elevated haemoglobin and haematocrit levels, suggestive of haemoconcentration. Infectious and autoimmune workup were negative. Both patients presented beyond the therapeutic window and were managed conservatively [2].

Conclusion: CRAO in young individuals should prompt an urgent evaluation for underlying vascular pathology. ICA occlusion may simultaneously compromise both retinal and cerebral circulation. Early recognition is critical for stroke risk assessment and the prevention of further neurological morbidity.

Keywords: CRAO (Central retinal artery occlusion); ICA (Internal Carotid Artery); Retinal stroke; Monocular vision loss; Young stroke

Abbreviations: CRAO: Central Retinal Artery Occlusion; ICA: Internal Carotid Artery; FAZ: Foveal Avascular Zone

Key Messages

CRAO is a type of retinal stroke that requires urgent systemic evaluation. In young patients, CRAO warrants investigate carotid pathology and hypercoagulable states. ICA occlusion simultaneously affects retinal and cerebral perfusion. Haemo concentration due to dehydration and alcohol binge drinking may increase the thrombotic risk. CRAO should be treated as a neurovascular emergency.

Introduction

Central retinal artery occlusion (CRAO) is an acute ischemic event resulting from the obstruction of the central retinal artery, leading to sudden painless monocular vision loss. It is widely regarded as the ophthalmic equivalent of ischemic stroke and necessitates urgent systemic and neurovascular evaluations.

The central retinal artery arises from the ophthalmic artery which is a branch of the internal carotid artery (ICA). Therefore, ICA pathology can directly impair retinal perfusion. Although CRAO commonly occurs in older individuals with established vascular risk factors, its occurrence in young adults is rare and often signals atypical etiologies such as hypercoagulable states, carotid artery disease, inflammatory vasculopathies, or hematologic abnormalities. We report two cases of young male patients presenting with CRAO associated with ipsilateral ICA occlusion and cerebral infarction, highlighting the importance of early neurovascular assessment.

Case Presentation

Case 1:

A 25-year-old man presented with fever for four days followed by right-sided headache. He developed sudden painless blurring of vision in his right eye which progressed to complete vision loss within seconds. There was a history of continuous alcohol consumption for four days before the onset (Figure 1a, 1b).

Clinical Findings:

Right Eye: only perception of light present

Relative Afferent Pupillary Defect: present

Left Eye: visual acute was 6/6

Left Pupil: normal and reactive

No focal neurological deficits

1Fundus Examination Revealed:

Fundus examination of the right eye revealed clear ocular media with a cup-to-disc ratio of 0.3 and optic disc pallor. The retina appeared pale and oedematous with sclerosed retinal vessels in all four arcades and segmentation (box carring) of blood columns. A characteristic cherry-red spot was observed in the macula. The fundus of the left eye was normal (Figure 2).

OCT Examination:

OCT imaging of right eye demonstrated increased retinal thickness with hyperreflectivity of the inner retinal layers, consistent with acute retinal ischemia. The central macular thickness was approximately 497 µm, indicating macular oedema secondary to a vascular compromise. These findings support the diagnosis of acute central retinal artery occlusion (Figure 3).

Investigations:

Haemoglobin: 17.3 g/dL

Haematocrit: 49.5%

Lipid profile: low HDL

Autoimmune and Infectious Workup: negative

Echocardiography: normal

Neuroimaging:

CT Brain: Axial CT imaging demonstrates hypodense areas in the right frontoparietal region, suggestive of acute cerebral infarction.

CT Cerebral Angiography:

CT cerebral angiography demonstrates near-complete occlusion of the right distal CCA, distal bulb, all segments of the internal carotid artery, reformed flow noted in the right MCA and ACA, right posterior communicating artery through collaterals (Figure 4a, 4b).

Fundus Fluorescein Angiography:

Fluorescein angiography of the right eye demonstrated normal choroidal flush with delayed arterial filling at approximately 20 seconds and delayed venous filling at around 30 seconds. Early hypo fluorescence in the macular region suggested macular ischemia secondary to CRAO. Non filling areas in the capillaries of the macular region with increased Foveal Avascular Zone (FAZ).

Management:

The patient presented beyond the thrombolysis window period and was treated with antiplatelets, statins, ocular hypotensive agents, and hydration.

Case 2:

A 32-year-old man presented with sudden painless vision loss in the right eye following a febrile illness with chills and vomiting. He reported heavy alcohol intake for seven days before the onset (Figure 5).

Clinical Findings:

Right Eye: only perception of light present

Relative Afferent Pupillary Defect: present

Left Eye: visual acute was 6/6

Left Pupil: normal and reactive

Neurological Exam: left-sided ataxic hemiparesis

Fundus examination of the right eye revealed a mildly hyperaemic optic disc with venous tortuosity, diffuse pale oedematous retina, and a cherry-red spot at the macula. The fundus of the left eye was normal.

OCT Examination:

OCT imaging of right eye demonstrated increased retinal thickness with hyperreflectivity of the inner retinal layers, consistent with acute retinal ischemia. The central macular thickness was approximately 497 µm, indicating macular oedema secondary to a vascular compromise (Figure 6).

Investigations:

Haemoglobin: 17.9 g/dL

Haematocrit: 53.7%

Autoimmune And Infectious Profile: negative

Echocardiography: normal

Neuroimaging:

MRI brain: MRI brain demonstrated complete occlusion of the right internal carotid artery from the petrous to supra-clinoid segment. Acute infarctions were noted in the right corona radiata and Posterior limb of internal capsule,

CT Cerebral Angiography:

CT Cerebral angiography revealed complete occlusion of the cervical and intracranial segments of the right ICA. Distal thin reconstituted flow in the clinoid segment and collateral reconstitution of the right MCA and ACA (Figure 7a, 7b, 7c).

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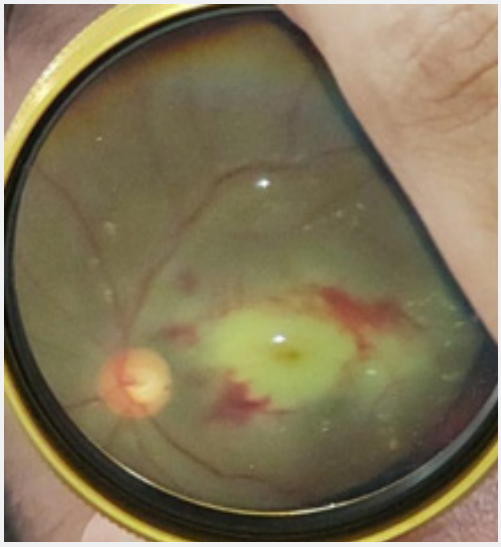

Figure 1a: Right eye fundus.

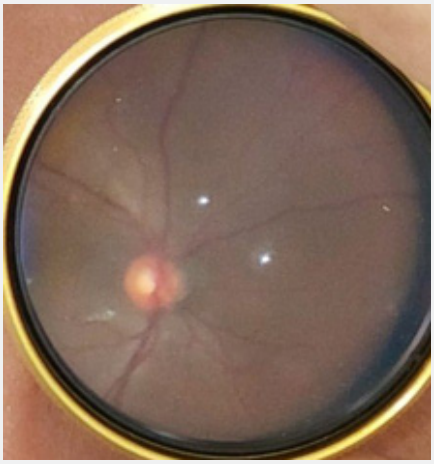

Figure 1b: Left eye fundus.

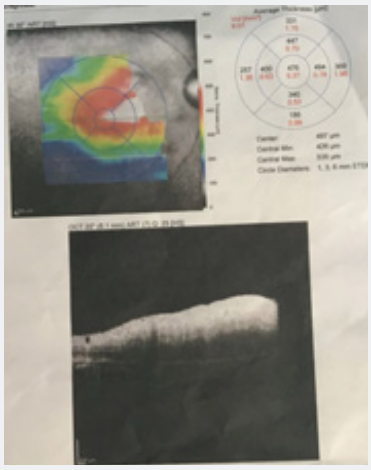

Figure 2: OCT Image of Right eye.

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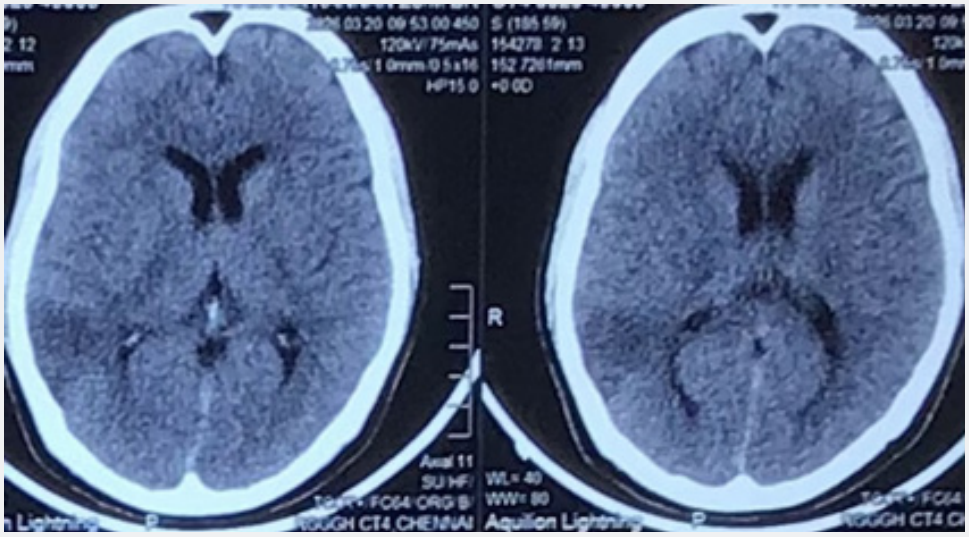

Figure 3: CT Brain.

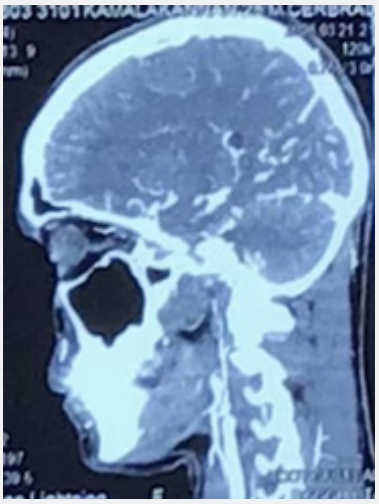

Figure 4a: Right CT cerebral angiography.

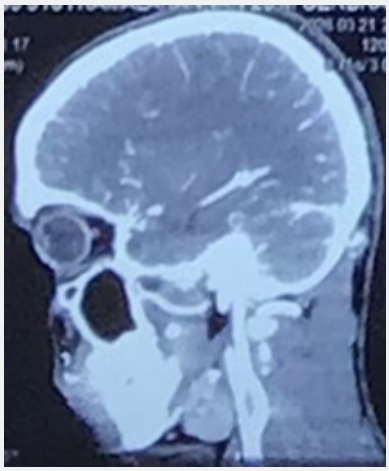

Figure 4b: Left CT cerebral angiography.

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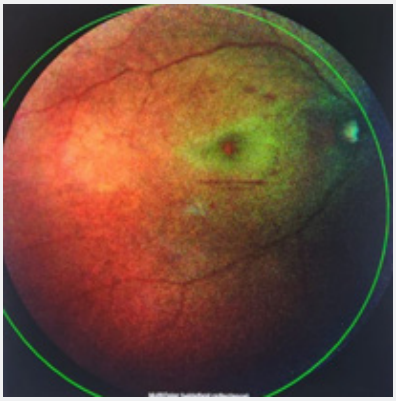

Figure 5: Right eye fundus.

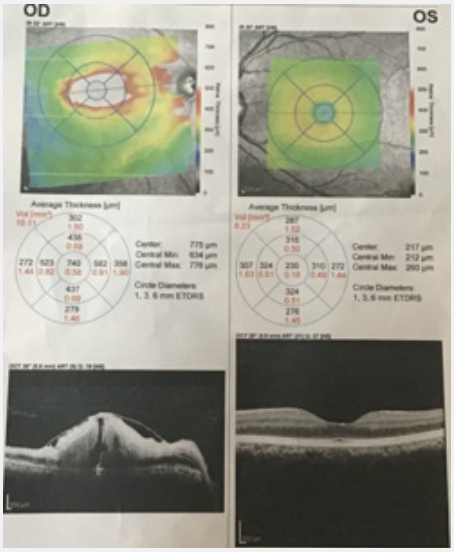

Figure 6: OCT image.

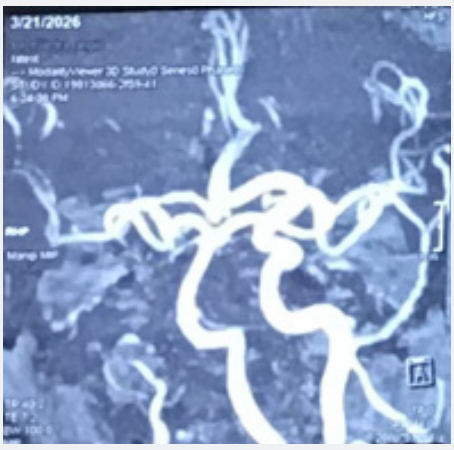

Figure 7a: MR Angiography.

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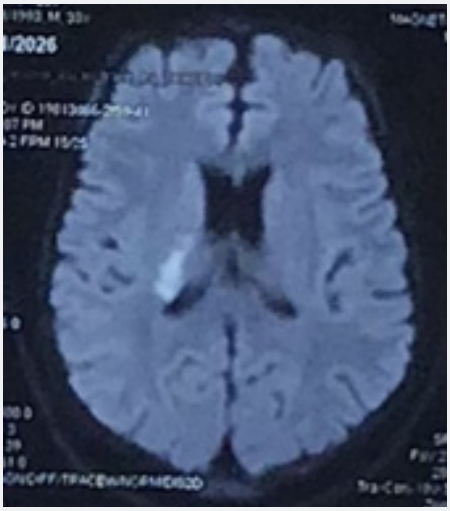

Figure 7b: MRI Brain DWI.

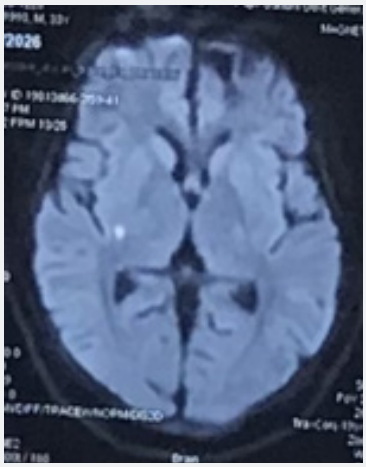

Figure 7c: MRI Brain DWI.

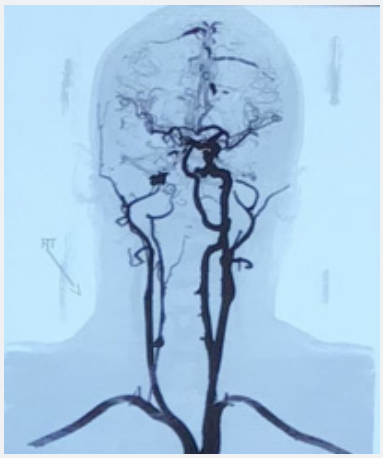

Figure 8: CT Cerebral angiography.

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Management:

Conservative management like Case 1 was initiated.

Discussion

CRAO represents an acute retinal ischemic insult and shares pathophysiological mechanisms with cerebral ischemia [3]. The close anatomical relationship between the ICA and retinal circulation explains the coexistence of retinal and cerebral infarctions in these patients [4].

Both cases highlight ICA occlusion as the underlying etiology, likely contributing via:

- Embolic mechanisms
- Hemodynamic compromise

Notably, both patients lacked conventional vascular risk factors. Elevated haemoglobin and haematocrit levels suggested haemoconcentration, likely secondary to dehydration from febrile illness and alcohol binge. Increased blood viscosity may predispose to thrombosis [2] (Figure 8).

8

Clinical Implications

CRAO in young individuals should prompt evaluation for:

- Carotid artery disease or dissection
- Hypercoagulable states
- Vasculitis
- Hematologic abnormalities
- Cardioembolic sources

CRAO carries a high risk of concurrent or subsequent ischemic stroke, especially in the early period, necessitating urgent stroke workup [4].

Management Considerations

Treatment options for CRAO remain limited. Conventional therapies such as ocular massage and intraocular pressure reduction are often attempted but lack strong evidence. Current emphasis is on:

- Early stroke evaluation [5]
- Secondary prevention strategies [1]

Conclusion

Both patients presented with sudden painless loss of vision beyond the thrombolysis window period and were treated with antiplatelet, statin, ocular hypotensive agents and hydration. There was no vision improvement for both patients, even after one month of the onset of illness. CRAO in young adults is uncommon and often indicative of underlying systemic vascular pathology. The coexistence of ICA occlusion underscores the shared vascular supply of the retina and brain. Early recognition and comprehensive neurovascular evaluation are essential to prevent further neurological complications [2].

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