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Traumatic Central Retinal Artery Occlusion Following Minor Facial Trauma: A Case Report

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Case Report

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Abstract

Background

Central retinal artery occlusion is a rare but devastating complication of blunt ocular trauma, particularly in young patients without traditional vascular risk factors. Prompt recognition and treatment are critical, but therapeutic options remain limited and time-sensitive. We report this case to highlight the importance of immediate visual assessment in periocular trauma and to underscore the narrow therapeutic window for potential interventions.

Case presentation

A 28-year-old male presented to the emergency department with acute, painless vision loss in his left eye immediately after his eye struck a faucet during a fall. On examination, visual acuity was no light perception, with a relative afferent pupillary defect. Funduscopy revealed diffuse retinal edema, attenuated retinal arteries, and a cherry-red spot. Fluorescein angiography confirmed delayed arterial filling, and optical coherence tomography demonstrated acute inner retinal ischemia. Immediate conservative management was initiated, including ocular massage, carbogen inhalation, and pharmacologic interventions. At two-week follow-up, visual acuity remained no light perception.

Conclusions

This case demonstrates that central retinal artery occlusion can occur as a severe complication of blunt ocular trauma in young, otherwise healthy individuals. It emphasizes the need for prompt visual assessment in all patients with periocular trauma and early neurovascular consultation, as therapeutic windows for interventions such as intra-arterial thrombolysis are narrow and frequently limited by delayed presentation or resource availability.

Background

Central retinal artery occlusion (CRAO) is an ophthalmological emergency characterized by acute, painless monocular vision loss (1). While typically associated with atherosclerosis in older adults, traumatic CRAO following minor blunt injury is exceptionally rare, particularly in young patients without traditional vascular risk factors(2, 3). Emergency physicians must maintain a high index of suspicion for vascular occlusion in all trauma patients presenting with visual symptoms, as delayed diagnosis results in irreversible visual loss within hours(1, 3). We report a case of traumatic CRAO in a 28-year-old male following seemingly trivial facial trauma, with emphasis on the diagnostic challenges and management limitations in resource-constrained settings.

Case presentation

A 28-year-old male presented to the emergency department with a 5-hour history of decreased vision in his left eye. The vision loss was instantaneous (within seconds) after his left eye struck a faucet during a fall. He reported associated ocular redness and mild periocular pain. His medical history was significant for a traumatic brain injury sustained in a motor vehicle accident seven years prior, resulting in residual dysarthria and gait disturbance. There was no history of hypertension, diabetes, hyperlipidemia, or tobacco use.

On examination, best-corrected visual acuity was no light perception (NLP) in the left eye. Intraocular pressure was 16 mmHg (normal range: 10–21 mmHg). Slit-lamp examination revealed a nasal conjunctival laceration. The left pupil measured 5 mm with a positive relative afferent pupillary defect (RAPD). Funduscopy of the left eye showed diffuse retinal edema, markedly narrowed retinal arteries, and a cherry-red spot in the macula (Figure 1). The right eye was unremarkable.

Fluorescein angiography (FA) demonstrated delayed arterial filling consistent with CRAO (Figure 2). Optical coherence tomography (OCT) revealed significant thickening and hyperreflectivity of the inner retinal layers in the posterior pole (Figure 3). Computed tomography of the orbits and brain showed no evidence of optic canal fracture or intracranial hemorrhage. Carotid duplex ultrasound and transthoracic echocardiography revealed no potential embolic sources. Laboratory investigations, including complete blood count, coagulation profile, and inflammatory markers, were within normal limits.

Immediate conservative measures included ocular massage, inhalation of carbogen gas (95% O_2 + 5% CO_2), sublingual nitroglycerin, oral acetazolamide, retrobulbar injection of anisodamine, intravenous dexamethasone, and daily injections of compound anisodamine near the superficial temporal artery. Interventional thrombolysis was considered but not pursued due to financial constraints and presentation beyond the conventional therapeutic window (6 hours). The conjunctival laceration was surgically repaired under local anesthesia. At the two-week follow-up, visual acuity remained NLP, and no further improvement was observed.

Discussion and Conclusions

This case illustrates several critical practice points for emergency physicians. First, even seemingly minor facial trauma can result in catastrophic vascular occlusion. The patient's injury mechanism—a faucet strike during a fall—would typically be considered low-risk for serious ocular sequelae. The immediate onset of vision loss (within seconds of impact) suggests direct vascular compromise rather than delayed embolic or inflammatory mechanisms, distinguishing traumatic from non-traumatic CRAO(4).

The differential diagnosis of acute vision loss after facial trauma includes traumatic optic neuropathy (TON), retrobulbar hemorrhage, orbital compartment syndrome, and vascular occlusion. While TON typically presents without a cherry-red spot or retinal edema, and orbital compartment syndrome is associated with proptosis and elevated intraocular pressure, the funduscopic and angiographic findings in this case confirmed CRAO. The normal intraocular pressure (16 mmHg) effectively ruled out acute

angle-closure glaucoma or orbital compartment syndrome. Differentiating these entities is critical, as management strategies differ substantially.

The pathogenesis of traumatic CRAO remains multifactorial. Proposed mechanisms include transient extreme elevation of intraocular pressure exceeding retinal perfusion pressure, direct mechanical stretching of retinal vessels causing endothelial disruption, or optic nerve sheath hemorrhage compressing the artery(5). Notably, this patient's history of severe prior traumatic brain injury (TBI) may have altered perineural anatomy, potentially predisposing him to vascular compromise from even minor subsequent injury—a "two-hit" hypothesis. Previous TBI can cause structural changes to the optic nerve sheath, periorbital vasculature, and orbital architecture(6, 7). In this context, even low-energy trauma that would normally be tolerated may precipitate vascular occlusion. While this mechanism remains speculative, it warrants further investigation in future studies.

Despite timely conservative management, no visual recovery was achieved, underscoring the rapid and irreversible nature of retinal ischemia following arterial occlusion. Current guidelines favor intra-arterial thrombolysis within a narrow time window (6 hours); however, real-world barriers including cost, limited access to interventional neuroradiology, and delayed presentation frequently preclude this option (1, 8). This case highlights the need for early consultation with neurovascular intervention teams, even in complex or resource-limited settings.

Finally, this case serves as a critical reminder that basic visual acuity and pupillary assessments should be performed in all patients with periocular trauma, regardless of apparent severity. Emergency physicians must facilitate urgent ophthalmological referral for those with visual symptoms, as therapeutic windows are measured in hours.

Traumatic CRAO is a rare but vision-threatening condition that can affect young patients following minor blunt injury. Maintaining a high index of clinical suspicion, performing rapid visual assessment, and ensuring urgent referral to centers with interventional capabilities are essential components of emergency management.

Declarations

Ethics approval and consent to participate

This retrospective case report was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Shenshan Medical Center, Memorial Hospital of Sun Yat-sen University. Written informed consent was obtained from the patient for participation in this case report.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

PG and **XZ** contributed equally to data collection, manuscript drafting, and literature review. **YL** assisted with clinical data interpretation and critical revision. **XW** (Xiaopei Wei), **ZY**, and **XW** (Xin Wen) participated in patient care and clinical management. **XW** (Xin Wen) conceived the study, supervised the project, and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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References

1. Mac Grory B, Schrag M, Biousse V, Furie KL, Gerhard-Herman M, Lavin PJ, et al. Management of Central Retinal Artery Occlusion: A Scientific Statement From the American Heart Association. Stroke. 2021;52(6):e282-e94.
2. Chen C, Singh G, Madike R, Cugati S. Central retinal artery occlusion: a stroke of the eye. Eye (London, England). 2024;38(12):2319-26.
3. Tiwari V, Bagga SSJ, Prasad R, Mathurkar S. A Review of Current Literature on Central Retinal Artery Occlusion: Its Pathogenesis, Clinical Management, and Treatment. Cureus. 2024;16(3):e55814.
4. Hayreh SS. Ocular vascular occlusive disorders: natural history of visual outcome. Prog Retin Eye Res. 2014;41:1-25.
5. Dalma-Weiszhausz J, Meza-de Regil A, Martínez-Jardón S, Oliver-Fernández K. Retinal vascular occlusion following ocular contusion. Graefes Arch Clin Exp Ophthalmol. 2005;243(5):406-9.
6. Zvorničanin J, Kuhn F, Halilbašić M, Mušanović Z. Central retinal artery occlusion and traumatic optic neuropathy following blunt ocular trauma. Int J Ophthalmol. 2018;11(5):893-7.
7. Noble MJ, Alvarez EV. Combined occlusion of the central retinal artery and central retinal vein following blunt ocular trauma: a case report. Br J Ophthalmol. 1987;71(11):834-6.
8. Lee KE, Tschoe C, Coffman SA, Kittel C, Brown PA, Vu Q, et al. Management of Acute Central Retinal Artery Occlusion, a "Retinal Stroke": An Institutional Series and Literature Review. Journal of stroke

and cerebrovascular diseases : the official journal of National Stroke Association. 2021;30(2):105531.

Figures

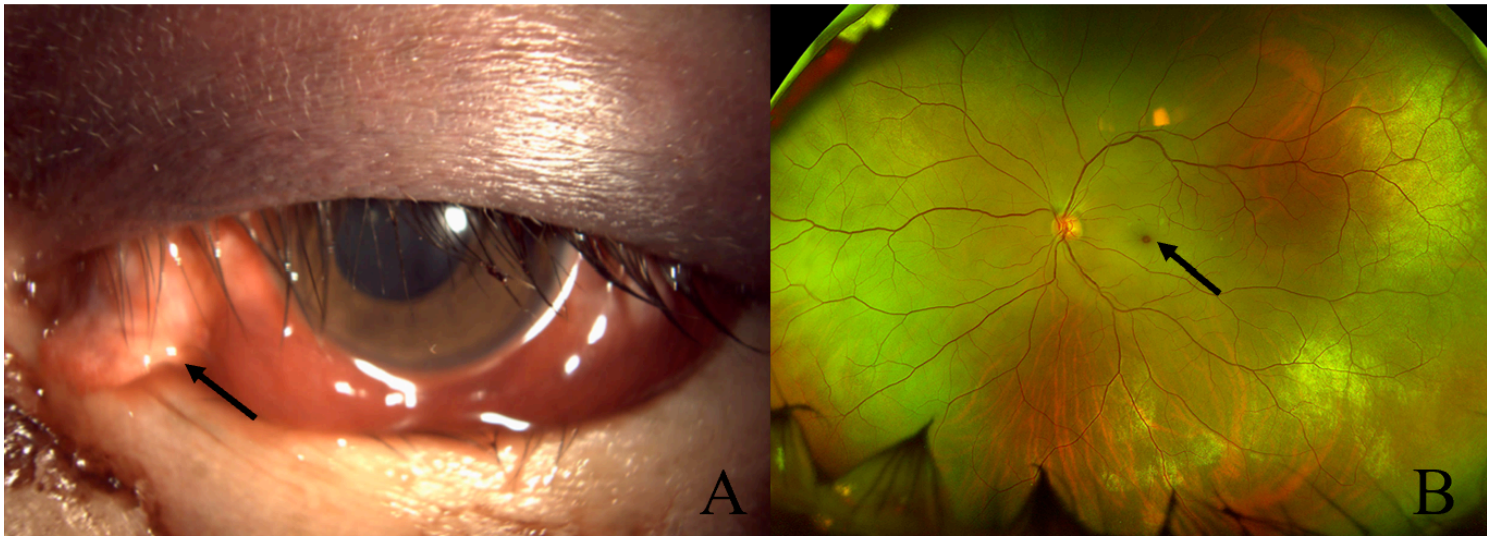


Figure 1

External ocular appearance and fundus findings of the left eye. (A) External photograph showing dilated pupil, nasal conjunctival laceration (arrow), and mild periorbital swelling. (B) Fundus photograph demonstrating diffuse retinal edema, markedly attenuated retinal arteries, and a cherry-red spot in the macula (arrow).



Figure 2

Fluorescein angiography of the left eye (early phase, 30 seconds). Note the delayed arterial filling and hypoperfusion consistent with central retinal artery occlusion.

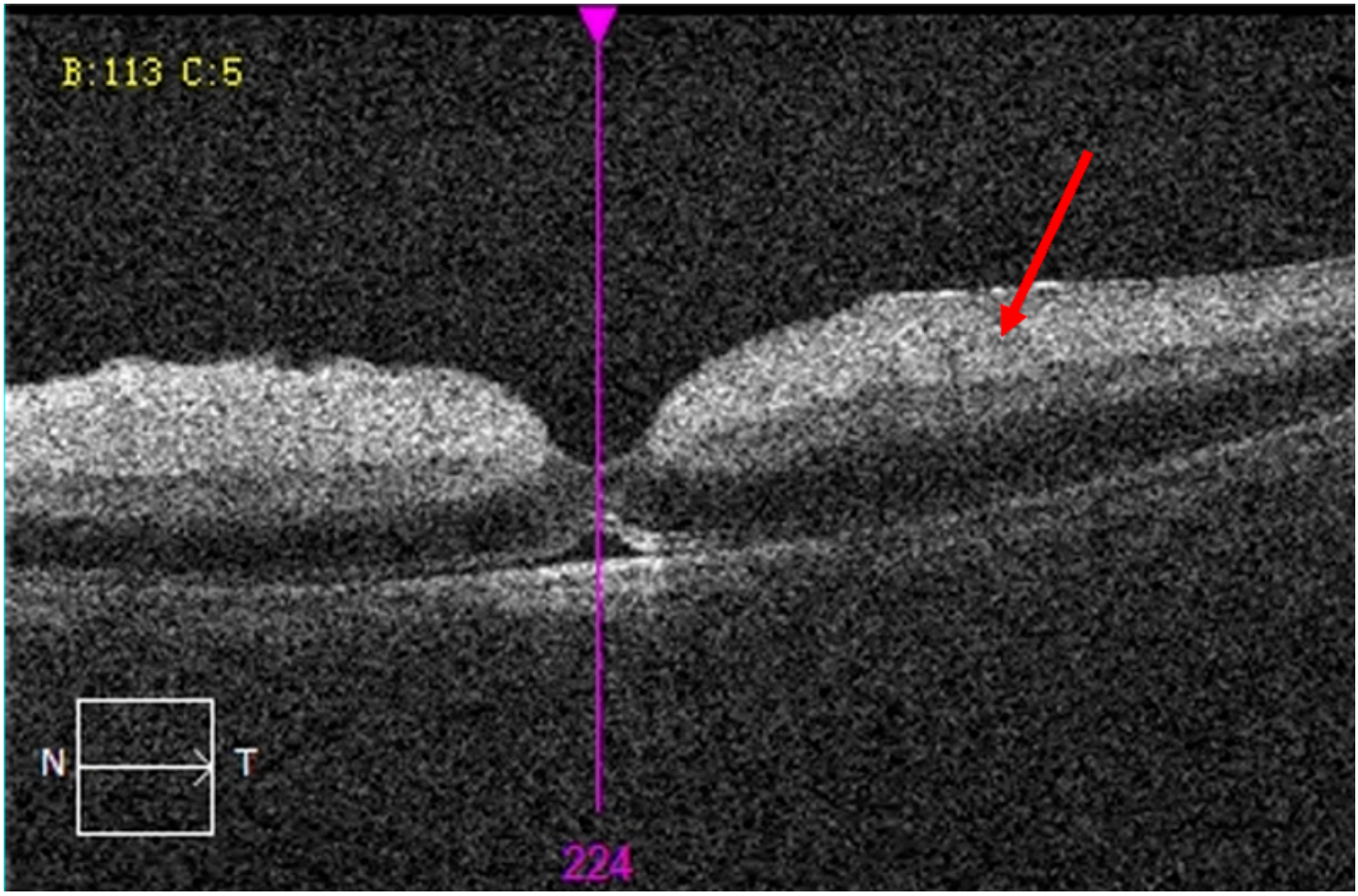


Figure 3

Optical coherence tomography through the macula of the left eye. Reveals marked thickening and hyperreflectivity of the inner retinal layers (arrow) with loss of the normal foveal depression, consistent with acute inner retinal ischemia.