

Secondary Glaucoma Following Facial Trauma

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Abstract

A healthy, 35-year old, male patient reported with left-sided facial trauma and ipsilateral lower extremity injury following a road traffic accident. Two weeks later, he developed hyperesthesia, pain and loss of vision in the left eye. Ophthalmic evaluation revealed cornea haziness, a dilated and fixed pupil and tonometry revealed an intra-ocular pressure of 60mm Hg. Thus, secondary glaucoma was diagnosed. Without delay anti-edema measures consisting of mannitol, a carbonic anhydrase inhibitor (acetazolamide) and β -adrenergic antagonistic eye-drops (timolol) were instituted.

Results: Within 24 hours, the intra-ocular pressure reduced to 28mm of Hg and his vision gradually improved. The patient thereafter was put on topical anti-glaucoma medication and topical steroids for two weeks following which the IOP returned to normalcy; no inflammatory signs were noted and his medications were gradually tapered.

Conclusion: Health care professionals need to be aware of the possible occurrence of such emergencies in cases of facial trauma, especially those presenting with head injury. Because head injury can similarly present with unilateral head ache, vomiting and photophobia, such symptoms have to be differentiated from those observed in secondary glaucoma, clinically. This consequently would eliminate diagnostic ambiguity. Since glaucoma is a sight-threatening emergency, extreme vigilance for such signs and symptoms is deemed necessary for immediate referral and management.

Keywords: Secondary glaucoma, facial trauma, orbital injuries

Case report

A healthy, 35-year old, male patient reported with left-sided facial trauma and ipsilateral lower extremity injury following a road traffic accident; the patient was conscious and oriented with no history of any other neurological deficit. Local examination revealed multiple, left-sided, facial lacerations with ipsilateral circumorbital edema/ecchymosis and sub-conjunctival hemorrhage. Palpation elicited left infra-orbital tenderness; however, ophthalmic evaluation revealed no impairment in visual acuity, ocular motility and pupillary responses (direct and consensual).

CT of face revealed an undisplaced left

infra-orbital rim fracture and anterior (maxillary) sinus wall comminution for which wound debridement and primary closure was done. MRI of left knee joint revealed an inter-condylar fracture of tibia for which open reduction and internal fixation of the fracture was done. An orbital chart was maintained over the next 72 hours to assess visual acuity and pupillary responses.

After 2 weeks, the patient developed hyperesthesia, pain and sudden loss of vision in the left eye. Immediate ophthalmic evaluation revealed a hazy cornea; the pupil was fixed and dilated; and tonometry revealed an intra-ocular pressure of 60mm Hg. Ophthalmoscopy of the fundus revealed a deep anterior chamber with multiple sphincteral and choroidal tears involving the macula. In view of these findings, a condition of secondary glaucoma was diagnosed. Anti-edema measures consisting of mannitol, a carbonic anhydrase inhibitor

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(acetazolamide) and β -adrenergic antagonistic eye-drops (timolol) were instituted. Within 24 hours, the intra-ocular pressure reduced to 28mm of Hg and his vision gradually improved. The patient thereafter was put on topical anti-glaucoma medication and topical steroids for two weeks following which the IOP returned to normalcy; no inflammatory signs were noted and his medications were tapered.

Discussion

Glaucoma is an ocular emergency that, if neglected, could irreversibly affect vision; types include primary and secondary (open and angle closure) glaucoma.¹ Traumatic glaucoma is a heterogeneous group of entities arising from a variety of pathomechanisms causing increase in the intra-ocular pressure during the early phase or the late phase following injury (blunt/ penetrating).² A 3-4% incidence of glaucoma after ocular contusion has been reported within 6 months and up to 10% within 10 years after trauma.^{3, 4, 5} Glaucoma, following trauma, is significant in patients who have clinically evident pigmentation (more so at the angle), hyphema, lens displacement and angle recession $>180^\circ$ - these constitute early predictors of traumatic glaucoma.⁶ In angle-closure glaucoma, elevated intraocular pressure is one of the most important factors which cause the visual field loss.⁷ However, in our case, the possible causes of the secondary glaucoma (2nd week post-trauma) may be multiple iris (sphincter) tears, causing inflammation, leading to outflow obstruction due to blockage of the trabecular network by inflammatory cells.

Unique to this case is that glaucoma occurred within a short span of time (2 weeks) following trauma. Clinicians and surgeons from allied specialties need to be aware of the possible occurrence of such emergencies in cases of facial trauma, especially those presenting with head injury. Because head injury can similarly present with unilateral head ache,

vomiting and photophobia, such symptoms have to be differentiated from those observed in secondary glaucoma clinically. This would consequently eliminating diagnostic ambiguity.⁸ Since glaucoma is a sight-threatening emergency, extreme vigilance for such signs and symptoms is deemed necessary for immediate referral and management..

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