Case report

Traumatic central retinal vein occlusion following a hawk attack to the posterior cranium

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ABSTRACT

Purpose: We describe a patient who developed a central retinal vein occlusion following a diving hawk attack to her posterior cranium.

Observations: A 50-year-old female presented with decreased visual acuity and color perception in the left eye following a diving hawk attack to her right-posterior parietal cranium. Medical history was significant for well-controlled type 2 diabetes, hyperlipidemia, and obesity. Fundoscopy of the left eye revealed pre, intra and subretinal hemorrhages in all 4 quadrants consistent with the “blood and thunder” appearance. Additional findings included optic disk edema, venous dilation and tortuosity. Optical Coherence Tomography of the left eye demonstrated pronounced macular edema with intra and subretinal fluid accumulation. Pattern electroretinography of the left eye was flattened.

Conclusions and Importance: There are few documented cases of central retinal vein occlusion following blunt trauma to the cranium in the literature. To the best of our knowledge, this is the first case of central retinal vein occlusion associated with posterior cranial trauma. Also unique to the case is the mechanism of injury whereby the patient was attacked by a hawk at a high-diving velocity.

1. Introduction

Central retinal vein occlusion (CRVO) is the second most common retinal vascular disorder after diabetic retinopathy. Currently, there is no consensus as to the exact pathogenesis of this condition. The most commonly held belief is that Virchow’s triad - endothelial dysfunction, hypercoagulability, and abnormal blood flow - contributes to thrombus formation in the central retinal vein thus leading to occlusion. Histopathologic studies conducted by Green et al. supports the theory of thrombus formation as the precipitating event for CRVO development. In this study, a fresh or recanalized thrombus was noted in 29 eyes from 28 patients with CRVO. Thrombus formation typically occurs at or anterior to the lamina cribosa. This observation is explained by the narrowing of the central retinal vein at the level of the lamina cribrosa, thus promoting turbulent blood flow and thrombus formation.

We present a patient who developed a central retinal vein occlusion in her left eye following a high-velocity diving attack from a hawk to her right-posterior parietal cranium. The patient was lost to follow-up prior to anti-VEGF therapy. While her long-term visual outcome is unknown, her prognosis is poor.

2. Case presentation

A 50-year-old Caucasian female was walking through a Central Florida forest preserve when a hawk flew into her right-posterior parietal cranium at a high diving velocity. Upon impact, the patient experienced a brief loss of consciousness. The event was observed by another individual who accompanied the patient. The patient presented to the local emergency department complaining of mild pain at the impact site and decreased visual acuity and color perception in the left eye. The wounds appeared superficial and insignificant and were treated appropriately. Head and neck MRI and CT imaging were unremarkable.

The patient was seen within 12 hours post-injury by an ophthalmologist (MJC). Her BCVA was 20/20 OD and 20/HM OS. All exam findings on the right eye were within normal limits. Slit lamp exam of the left eye and vitreous was unremarkable. Fundoscopy of the left eye revealed pre, intra and subretinal hemorrhages in all 4 quadrants consistent with the “blood and thunder” appearance. Additional findings included optic disk edema, venous dilation and tortuosity. These findings were consistent with a diagnosis of central retinal vein occlusion.

Eight days after the injury the patient presented for outpatient follow
up. Visual function in the left eye had not improved. Pupils were equal, round, and reactive to light. A relative afferent pupillary defect was not observed. Funduscopic examination and IVFA studies of the left eye revealed similar findings from the initial presentation (Figs. 1 and 2). Importantly, IVFA demonstrated delayed flow through the venous system thus providing further evidence in favor of CRVO. Vitreous hemorrhage was now present and precluded an adequate view of the retina to assess for ischemia. Optical Coherence Tomography of the left eye demonstrated pronounced macular edema with intra and subretinal fluid accumulation. Macular thickness was increased at 441 μm. Pattern electroretinography of the left eye was flattened (Fig. 3).

The patient was to be re-evaluated 8 weeks status post injury for possible anti-VEGF therapy. However, the patient had to reschedule this appointment to 12 weeks status post injury. At this visit, the vitreous hemorrhages had resolved, but the pronounced macular edema with intra and subretinal fluid accumulation remained unchanged. The patient’s visual acuity had not improved (20/HM OS). Intravitreal Eylea (Aflibercept) was attempted but was unsuccessful due to patient cooperativity. The injection was rescheduled for the following week under anesthesia but the patient was lost to follow up.

The patient’s ocular history was positive for primary open angle glaucoma suspicion. Prior to the trauma, her BCVA was 20/20 bilaterally. Past medical history was significant for well-controlled type 2 diabetes, hyperlipidemia, and obesity. Social history was significant for tobacco and alcohol use. The family history was non-contributory.

3. Discussion

Three previous studies have documented the occurrence of CRVO following cranial trauma. Differences, however, are noted between this case and those described previously. Kline et al. described a patient who experienced a blow to the right anterior cranium with a crowbar without a loss of consciousness. The patient subsequently developed an ipsilateral CRVO 10 days later. Venograms revealed persistent luminal narrowing in the superior ophthalmic vein bilaterally and the authors attributed this to a congenital abnormality, due to the bilateral nature of the variant. Given the decreased diameter of the vein, they concluded that flow in upstream regions was likely turbulent. They speculated that the blow to the head was the inciting event which further perturbed hemodynamics in the region, ultimately leading to thrombosis. Hope-Robertson et al. and Cozzoli documented similar cases, with CRVO development occurring over four to seven days, but failed to provide venograms or evidence of central retinal vein or ophthalmic vein anatomic variants.

Interestingly, the CRVO in our patient differs in two ways from previous reports. First, the visual acuity deterioration in the left eye occurred on the scale of minutes to hours rather than days. Second, our patient experienced a right sided, posterior blow to the parietal region of the skull with a contralateral CRVO.

The cause of CRVO in this patient was multifactorial, with the blow to the right posterior cranium serving as a precipitating event for thrombosis via disruption of normal hemodynamics. The impact likely

Fig. 1. Fundus photo of the left eye eight days after the inciting trauma.

Fig. 2. Fluorescein angiography of the left eye eight days after the inciting trauma.

Fig. 3. Pattern electroretinography of the left eye eight days after the inciting trauma.
caused a sudden jolt in head motion which resulted in a coup-contrecoup injury to the brain, as evidenced by the immediate loss of consciousness. The coup-contrecoup injury provides a plausible mechanism for the contralateral location of the CRVO with respect to the site of trauma. Sudden rapid displacement of the brain and orbital contents within the rigid confines of the cranium and orbit respectively may have led to various compressive and shearing forces acting on the intracranial and orbital contents (including the central retinal vein) resulting in excessive traction on myriad nerves and vessels traversing these structures. This might have precipitated endothelial damage and turbulent blood flow, promoting thrombosis with subsequent occlusion of the vein. While the conditions described above can serve as a nidus for thrombosis in the setting of a traumatic brain injury, this phenomenon is exceedingly rare. Therefore, additional risk factors must have been present which predisposed this patient to CRVO.

The patient had several significant vascular risk factors that predisposed her to thrombosis and increased her risk of CRVO: obesity, hyperlipidemia, type 2 diabetes mellitus, and chronic tobacco use. The patient’s ocular history was significant for primary open angle glaucoma. The patient harbored an anatomic variant as an orbital CT angiogram and/or orbital venogram would have provided a more complete picture of the patient’s vascular anatomy. However, these imaging studies were not ordered as the identification of a vascular anomaly is primarily of academic interest and would not benefit the patient.

4. Conclusion

There are few documented cases of CRVO following blunt trauma to the cranium in the literature. To the best of our knowledge, this is the first case of CRVO with posterior cranial trauma. Also unique to the case is the mechanism of injury whereby the patient was attacked by a hawk at a high-diving velocity. Given that the overwhelming majority of individuals who suffer a traumatic brain injury do not develop a CRVO, we speculate that the presence of numerous vascular risk factors in our patient predisposed her to thrombosis and subsequent CRVO under the tumultuous conditions of the TBI. The presence of a congenital venous anomaly would have further altered the venous hemodynamics thus creating conditions even more favorable for thrombosis. While the patient did not receive a thorough workup for congenital venous anomalies, we surmise that the patient has an increased likelihood of harboring such a variant with respect to the general population.

Patient consent

The patient consented to publication of the case in writing. This report does not contain any personal information that could lead to the identification of the patient.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Intellectual property

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

Declaration of competing interest

No conflict of interest exists.

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