Abstract

Purpose: To present a rare case of optic nerve avulsion due to gun shot injury.

Case Report: A 30-year-old man with sudden visual loss on the Right eye because of optic nerve avulsion after a gun shot injury. The patient was treated with high dose steroid, but there was no improvement of his RE visual acuity.

Conclusion: Optic nerve avulsion is a rare case with devastating results because there’s still no definite therapy to improve the outcome.

Introduction:

Eyes are part of our head and they can get simultaneously affected in head injuries. From patients with ocular manifestations of head injury, vehicle accident accounted for 70.3 % of all injuries, assault for 19.1 %, fall from height for 5.3 % and others 5 % including fall from train, stone injury on head, gunshot, etc. Traumatic optic neuropathy (TON) is an uncommon cause of visual loss following blunt or penetrating head trauma with a reported incidence of 0.7-2.5% in published case series. TON is rare, but can cause severe, irreversible vision loss. In this paper we would like to describe a case of optic nerve avulsion as a form of severe TON due to gun shot injury.

Case Illustration:

A 30-year-old man presented with sudden visual loss on the Right Eye (RE) after a gun shot on his left frontal bone. On examination of the RE, he had periorbital hematoma and edema, and also conjunctival chemosis. There was no light perception of the RE, and his pupil was dilated and nonreactive to light. There’s also an eye movement resistance. On the examination of the Left Eye, he had periorbital hematoma and edema, visual acuity was 1.0 and other examinations was within normal limit.

B-scan USG of the RE showed a hyperechoic image on the retinal projection in front of the optic nerve similar with T-sign in posterior scleritis. CT-scan showed multiple metal density fractions in the subcortical cortical left frontal lobe and right retrobulbar. On funduscopic examination, there was bleeding in the inferior vitreous originating from the optic nerve papilae. The patient underwent craniotomy, debridement, and corpus alienum extraction by neurosurgeon and plastic surgeon. The patient was treated with high dose steroid (methyl prednisolone 4 x 250 mg iv). After a week of therapy, there was no improvement of his RE visual acuity.

Figure 1. Bilateral Periorbital Edema

Figure 2. Head X-ray shows corpus alienum at right maxillaries region
Discussion:

Traumatic optic neuropathy (TON) is an acute injury to the optic nerve as a result of trauma resulting in loss of vision along with visual field deficits, color perception, and damage to the optic nerve.\(^4\) TON can occur due to direct or indirect injury. Traumatic optic neuropathy is classified as direct when the nerve is injured directly by a projectile, knife, or other object that penetrates the orbit to damage the optic nerve. Indirect optic neuropathy is diagnosed when the injury to the nerve results from the nonpenetrating effects of trauma, including hemorrhage, edema, and concussion. Indirect traumatic optic neuropathy has been the subject of more clinical research than direct injury.\(^5\) Direct injury is characterized by radiologically detectable compression by bony fragments and penetrating foreign bodies, expanding lesions, and fractures of the optic nerve canal itself. Indirect injury does not correlate with any radiologic findings.\(^6\) Both direct injury and indirect injury cause optic nerve ischemia. The mechanism of optic nerve injury can be divided into primary injury and secondary injury. Primary damage occurs as a result of external forces at the moment of trauma, such as rupture of nerve fibres or of capillary vessels. Secondary damage may not be present initially, but may occur later and results from the compromised blood supply to the optic nerve, for example, following oedema or angiospasm.\(^7\) Reperfusion injury and ischemia will lead to peroxidation of fat cell membranes and cause the emergence of free radicals causing tissue damage.\(^4\) A national epidemiological survey of TON in the United Kingdom found a minimum prevalence in the general population of one in 1,000,000. The vast majority of affected patients are young adult males (79-85\%) in their early 30s.\(^2\) Vision loss is typically immediate and often severe (24\%–86\% of patients have no light perception at presentation).\(^8\)
In this case, the fractions of the bullet caused direct injury to the optic nerve resulting in optic nerve avulsion. Optic nerve avulsion (ONA) is a severe TON, where the optic nerve is forcibly disinserted from the retina, choroid and the lamina cribrosa. The diagnosis of ONA is most often made clinically. If the media is clear, the disc is seen to be replaced partially or totally by a hole with variable amounts of surrounding retinal hemorrhages. However, if there is vitreous hemorrhage or pre-retinal bleed which obscures the clinical optic nerve head evaluation, USG and MRI can play an important role in the diagnosis.

Simsek et al. from Taju reported that B-Scan USG can detect ONA clearly, whereas Foster et al reported B-Scan USG evaluation failed to detect ONA in 4 cases. From a case report of optic nerve avulsion from Taju, A B-Scan ultrasound showed alteration in the normal contour of the optic nerve/ globe junction and hypo echogenic area adjacent to the nerve.

The management of TON is controversial, between medical therapy of steroids, surgery, or both, however, the data in the literature to date has not shown any treatment to be superior to observation.

A visual recovery rate of 40-60% has been reported for indirect TON cases managed conservatively. Direct TON is a distinct category that results in severe, irreversible visual loss with little likelihood for recovery, and no intervention is of proven benefit.

References: