Invited Article

Traumatic optic neuropathy – our experience

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Abstract A great deal of controversy surrounds the physiology and management of traumatic optic neuropathy. Needless to say, it has formed the topic of much debate in the past, especially with regard to its surgical management. With the advances in sinus endoscopic procedures, and their extended applications to the orbit and optic nerve, endoscopic optic nerve decompression offers a very good chance for salvaging vision in patients with traumatic optic neuropathy. However, there is no definite protocol laid down in the world literature for this condition, owing partially to the fact that a majority of such cases are not amenable to surgery within the critical period, due to the coexisting morbidities of head injury. There is also much controversy regarding medical versus surgical management of traumatic optic neuropathy. We present here our experience with this condition, and outline the management protocol followed.

Keywords Traumatic optic neuropathy · Optic nerve decompression

Introduction

Traumatic optic nerve damage after craniofacial injury was first described by Hippocrates [1]. Indirect damage to the optic nerve is the most common form of traumatic optic neuropathy, occurring in about 0.5–5.0% of all cases of closed head trauma [2, 3]. Patients usually suffer craniofacial trauma but occasionally mild orbital or eye injury [4, 5]. Traumatic optic neuropathy can occur following an innocent ipsilateral injury over the superior temporal orbital rim and is characterized by vision loss without external or internal ophthalmic evidences of injury to the eye and its nerve [6]. In many cases, due to the coexistent head injury and its associated comorbidities, the visual status may not be amenable for assessment. In fact, major brain injury occurs in 40–72% of patients with traumatic optic neuropathy, the management of which takes precedence [7]. This may often lead to a delay in diagnosis and subsequent timely treatment for a potentially reversible visual loss. Patients with head injury who are suspected to have a coexistent optic nerve trauma (which may be indicated by contusions around the eye, an afferent papillary defect, with corresponding fundoscopic changes of disc edema and vascular congestion, or an actual complaint of loss of vision in one/both eyes) warrants urgent radiological investigations. While a high resolution CT scan of the paranasal sinuses and orbit would reveal any obvious fracture, hemosinus and coexisting trauma such as fractures of the skull base, it would also serve as a road map for surgery. An MRI would be helpful in delineating the integrity of the optic nerve, and also help in ruling out nerve sheath hematomas. Usually a clinical suspicion coupled with positive radiological findings may provide enough grounds for surgical intervention; nevertheless, there are certain other investigations which may be performed when in doubt.
Pathophysiology

The part of the optic nerve most vulnerable to blunt trauma of the head is the intra-canalicular segment, which by virtue of its bony course is vulnerable to the fractures and compressive-elastic forces of its surrounding bone, which also being unyielding, allows for no space for inflammatory expansion or hemorrhage.

Optic neuropathy following accidental trauma usually results from two distinct mechanisms: a primary injury as a result of a direct contusive force on the optic canal and nerve, or an elastic deformation of the sphenoid sinus with a transfer of force to the intra-canalicular portion of the optic nerve disrupting its axons and blood vessels [7], which may result in compression of the nerve by bony fragments or a sheath hematoma [8], and if untreated may succumb to a secondary ischemia and continued axon loss due to the swelling of the nerve within its sheath and bony canal, compressing its blood supply [7, 9].

Iatrogenic injury of the optic nerve may occur due to a variety of factors which include; anatomic variations of the course of the nerve such as the type III/IV optic nerve or a nerve coursing through a dominant sphenoid sinus of the opposite side (Figs. 1–3), dehiscent bony canal, erosion of the bony canal due to some disease process, excessive hemorrhage impeding visibility during surgery, etc. Most of these problems can be averted by a thorough study of the CT scan prior to surgery and if required, even review during surgery.

Investigations

Certain specific investigations are warranted in a case of traumatic optic neuropathy, and their principles and indications are briefly outlined below:

- **Afferent papillary defect**: an absolute or relative afferent papillary defect indicates that vision is being compromised. It can be tested by shining a bright focused light for a few seconds on the ‘unaffected’ eye, in a dark room, with the patient looking at a distance, and noting the pupillary response, which is normally characterized by miosis. Following this, the light is shone on the ‘affected’ eye, and the pupillary response is compared to its ‘normal’ counterpart. Features sought after are a lack of briskness/sluggish miosis as compared to the normal side, a partial or complete absence of miosis. This usually indicates a defect in the afferent pupillary pathway, and has been termed as a ‘Marcus Gunn’ pupil. It is best interpreted along with fundoscopic and radiologic signs. It may also be elicited subjectively, by asking the patient to compare the difference in the brightness perceived between the unaffected and affected eyes.

- **Fundoscopic examination**: various appearances can manifest in traumatic optic neuropathy namely, disc edema, congestion of vessels, disc pallor, etc. but the most common presentation is usually a normal looking disc, especially in the early stages.

- **Color vision**: loss of color vision, namely red color vision – patients with optic neuropathy often have red