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Anterior Chiasmal Optic Nerve Avulsion

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Summary: In a case of traumatic avulsion of the optic nerve at the anterior chiasm, MR imaging provided highly specific images of the injury site, including the absence of the optic nerve within the optic canal and the point of transection at the anterior portion of the chiasm. This was confirmed clinically and histopathologically. MR imaging should be considered in cases of suspected chiasmal injury.

Index terms: Eyes, injuries; Eyes, magnetic resonance; Nerves, optic (II); Optic chiasm

There is a small but intriguing body of literature on traumatic optic nerve avulsions resulting from traumatic enucleations. Injuries usually are the result of severe facial trauma, martial arts maneuvers, or autoenucleations in psychiatric patients. Psychiatric patients often blame an irresistible compulsion and quote Matthew’s admonishment from the Bible, “If thy right eye offend thee, pluck it out.” According to one study (1), 38% of these patients enucleate both eyes.

We report a case with radiologic, clinical, and histopathologic correlation in which the optic nerve was traumatically torn from the chiasm, with involvement of all ipsilateral optic nerve fibers as well as fibers of Wilbrand’s knee. Magnetic resonance (MR) imaging proved to be highly specific in revealing the location of the chiasmal injury.

Case Report

A 25-year-old man with a history of temporal lobe epilepsy, treatment for squamous cell carcinoma of the nasopharynx, traumatic injuries, and alcohol and drug use was assaulted. The assailant placed the patient in a headlock and then plunged his fingers into the patient’s left lateral orbit and pulled out the globe. The patient lost consciousness and was brought by ambulance to the emergency department. On ophthalmologic examination, the right eye was uninvolved. However, the left globe was luxated, lying anterior to the eyelids, with obvious disruption of the superior rectus and lateral rectus muscles. The left pupil was dilated and nonreactive. Funduscopic examination showed evidence of an occlusion of the ophthalmic artery. Head and orbital computed tomography (CT) showed an intact left globe, avulsion of the superior and lateral rectus muscles, marked proptosis, and a severed optic nerve with its free end lying within the orbit (Fig 1A). Interestingly, there were no orbital fractures.

Twelve hours later, when the patient was considered medically and neurologically stable, he was brought to the operating room for exploration and replacement of the globe within the orbit. On examination, the superior and lateral recti were completely severed, but the inferior and medial recti were intact. The optic nerve was located and found to be completely avulsed, with a 46-mm segment attached to the globe (Fig 1B). The distal portion of the torn optic nerve was free of overlying dura. Because this length indicated optic canal and probable chiasmal involvement, a piece of the terminal segment of the optic nerve was removed and sent for histopathologic examination. A primary enucleation was considered, but deferred, since the patient had not been conscious to discuss this alternative. The globe was reposited within the orbit. A megadose of intravenous corticosteroids was initiated to treat any potential traumatic chiasmal or contralateral optic nerve edema.

After surgery, the primary concern on examination was the visual acuity and peripheral field of the contralateral right eye. In an attempt to determine better the distal extent of the optic nerve injury, MR imaging was performed at 1.5 T. Serial images showed that the injury extended to the level of the anterior chiasm with no evidence of thalamic, pituitary, or carotid artery injury (Fig 1C–H).

The radiologically established location of the optic nerve transection was confirmed both clinically and histopathologically. Light microscopic analysis of the optic nerve specimen showed fibers cut axially as well as fascicles that were oriented more longitudinally, suggesting that the site of avulsion was within Wilbrand’s knee (Fig 1I). Clinical confirmation was obtained by performing a Goldmann visual field examination of the right eye. The
supertemporal wedge defect corresponded exactly to the inferonasal nerve fibers that form Wilbrand’s knee (Fig 1J and K). Fortunately, central vision in the right eye was 20/20. The visual field defect remained stable on serial examinations. Nine months after injury, the right optic nerve exhibited sectoral pallor from retrograde ganglion cell degeneration.

Discussion

Traumatic enucleations and, specifically, autotenucleations have been described in almost legendary fashion throughout history. Krauss and colleagues (1) thoroughly reviewed this topic, including historical accounts of autotenucleation of Oedipus, St Lucia of Syracuse, and St Triduan of Scotland. Studies in the 1960s, which relied on ophthalmoscopy, surgical exploration, and autopsy examination, were clearly limited (2). The advent of high-resolution CT and MR imaging has the potential to significantly advance the study of this type of injury.

The optic nerve can be divided into four segments: intraocular (1 mm in length), intraorbital (20 to 30 mm), intracanalicular (4 to 9 mm),
and intracranial (10 to 16 mm) (3). The intraorbital section is S-shaped; this redundancy permits unrestricted ocular rotation and limited axial displacement. The intracanalicular portion is immobilized by the dura, which is fused to the periosteum of the optic canal. The intracranial optic nerve begins at the intracranial optic foramen and ends at the chiasm. In the anterior portion of the chiasm, a bundle of nerve fibers known as Wilbrand’s knee, which subserves the inferonasal retina of the contralateral eye, juts anterior into the distal portion of the optic nerve. The total optic nerve length is approximately 45 to 50 mm.

There are three locations at which the optic nerve is susceptible to being avulsed: the optic disk, the orbital apex, and the optic chiasm. In a review of 18 cases of autoenucleation, Krauss et al (1) reported that division of the optic nerve at the orbital apex occurred in the majority of such
cases (55%). Eleven percent of the optic nerve avulsions occurred at or near the optic disk. Disruption of the optic nerve at the anterior chiasm occurred in 33% of autoenucleation cases. Other investigators (4) also reported a high rate of anterior chiasmal avulsion. The location of injury found in studies is heavily influenced by the patient population and the mechanism of injury (eg, battlefield trauma versus autoenucleations). Considering all cases of traumatic enucleation, not limited to autoenucleation, avulsion of the optic nerve at the orbital apex is the most common, most likely due to tethering of the nerve at the attachment site of the dura to the canalicular periosteum, and often occurs with basilar skull fractures (3). Optic disk avulsions are generally associated with rotational injuries with extreme abduction (5). Optic nerve laceration at the chiasm is usually the result of abrupt optic nerve traction, as was shown under experimental conditions (4, 6).

In this case, the entire optic nerve was torn from the chiasm, presumably from strong tractional forces. Gross inspection of the nerve revealed that it was stripped of dura over its distal portion, corresponding to the canalicular segment (Fig 1B). Coronal MR imaging clearly showed the remaining cuff of dura present within the optic canal (Fig 1C–E). The adhesiveness of the dura to the nerve and to the periosteum of the optic canal must partially determine whether abrupt tension on the nerve will result in avulsion at the orbital apex or the chiasm.

Investigators have offered several mechanisms for nerve avulsion, including extension of bone fractures, direct penetration of a foreign body such as bone, shearing forces, anterior luxation of the globe, forced globe rotation, and a sudden explosive rise in intraocular pressure, hemiating the optic disk through the scleral foramen (2, 7, 8). The mechanism in this case appears to be direct axial traction on the optic nerve from the assailant’s fingers, resulting in an avulsing shearing force at the anterior chiasm.

The potential complications that may accompany monocular visual loss with traumatic enucleations include chiasmal injury, endocrine abnormalities from pituitary damage (for chiasmal avulsions), leakage of cerebrospinal fluid, meningitis, thalamic injury, carotid-cavernous fistula, and subarachnoid hemorrhage (9, 10). Endocrine abnormalities can range from diabetess insipidus to panhypopituitarism. In this case, only chiasmal injury developed and the patient had little functional impairment from the visual field deficit. There have been sporadic reports of similar injuries with chiasmal injury and associated temporal field loss in the contralateral eye (1, 2, 9, 11).

Treatment options in direct optic nerve trauma are limited (12, 13). In cases of compressive optic neuropathy from orbital hemorrhage, urgent soft-tissue decompression by lateral canthotomy and cantholysis is indicated. Subdural optic nerve sheath hematoma is managed by optic nerve sheath fenestration. Indirect traumatic optic neuropathy may be treated either with megadose systemic corticosteroids or optic canal decompression (14). Although there is no effective treatment of chiasmal injury, its identification is important for early management of potential injuries to nearby intracranial structures.

In older studies there was doubt as to whether chiasmal field losses were the result of a single lesion. However, present advances in imaging permit confidence in this conclusion with accurate localization of the avulsion. MR imaging provides particularly good definition in the region of the chiasm (15, 16), and is the imaging technique of choice for evaluating soft-tissue abnormalities once the possibility of a metallic foreign body has been ruled out. As illustrated in this case, MR imaging provides specific information about the location and extent of chiasmal injury and the integrity of surrounding anatomic structures.

References