

Pathophysiology and diagnostic workup of optic nerve avulsion

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Optic nerve injuries may include infarction, transaction, or avulsion. Compression by perineural hemorrhage or fracture of the optic canal may also occur. Any sites along the anatomic course may be injured, including the intraocular, intracanalicular, or rarely the intracranial chiasmal segment. Optic nerve avulsion is characterized by separation of the optic nerve from the globe at the level of the lamina cribrosa without rupture of the optic nerve sheath, or the adjacent sclera.¹ Several mechanisms of optic nerve avulsion have been postulated, including sudden extreme rotation of the globe, sudden massive rise in the intraocular pressure, or sudden forward displacement of the globe. Moreover, partial and complete avulsions have been described, usually in the setting of blunt orbital trauma. The choice of imaging modality, to diagnose optic nerve injury, is still debatable and shows conflicting opinions among different trauma centers. We report here a challenging case of optic nerve injury, and the benefit of urgent B-scan ultrasonography (US) in the setting of traumatized globe.

A 30-year-old man presented to the emergency department after a motor vehicle accident. On arrival, he stated that he lost vision in his left eye instantly at the time of the impact. Physical examination revealed obvious dystopia with significant periorbital hematoma. Examination revealed that he had no light perception in the left eye. The extraocular movement was normal for the right eye but completely restricted for the left. The left globe was displaced inferiorly by 11 mm and laterally by 6 mm; it was soft on gentle digital pressure with apparent diffuse subconjunctival hemorrhage. The left pupil was dilated and nonreactive to light. The red reflex was absent, and a grade 4+ afferent papillary defect was present. The cornea, anterior chamber, and lens were normal. Fundoscopy view was obscured by dense vitreous hemorrhage. An urgent axial CT scan of the head and orbit was obtained, which confirmed the presence of complex fractures of the midfacial and left orbital walls. No obvious retrobulbar hematoma was apparent, but significant disruption of the orbital apex with possible impingement on the optic nerve was seen. Both intact eye globes were noted on CT scan that is, no ruptured globe; however, there was significant anterior and lateral displacement of the left eye globe with probable injury of both the medial and lateral rectus muscles. Therefore, US was performed to assess the orbit. It showed a posterior ocular wall defect in the region of the optic nerve head with retraction of the

edematous optic nerve into its sheath posterior to the lamina cribrosa, total retinal detachment, and vitreous hemorrhage. The results suggested a diagnosis of acute traumatic optic nerve avulsion (Figure 1). Therefore, he was admitted urgently for surgical decompression of the orbital canal, and open reduction and fixation of the complex facial fractures. No immediate postoperative complications developed. However, postoperatively, neither visual acuity nor the extraocular movements improved during the first week. Unfortunately, at one-month follow-up, he had no light perception.

Optic nerve avulsion remains a devastating complication of orbital trauma and can be challenging to diagnose and manage. It can result from any form of direct or indirect orbital injury. Decreased visual acuity and limitation of extraocular movements are considered the most important emergent signs of serious associated orbital insult and may provide a clue for early diagnosis of optic nerve injury. Posttraumatic injury of the optic nerve is most often associated with an orbital and skull base fracture involving the orbital apex or from serious traction of the nerve that results in optic nerve avulsion.² At the orbital apex, the intracanalicular segment is particularly vulnerable to trauma because of the close attachment of the dural sheath to the periosteum and the bony architecture of the orbit. This will easily transmit force from the forehead and brow directly to the orbital apex. Decreased visual acuity and limitation of extraocular movements is the most important emergency signs to consider. Diagnosis of optic nerve avulsion by ophthalmoscopy is possible if the procedure is carried out in the early stages when the media is clear. However, the associated vitreous hemorrhage, which is almost always present, obscures the view of the fundus in most patients. Orbital imaging studies must be carried out as early as possible to reach

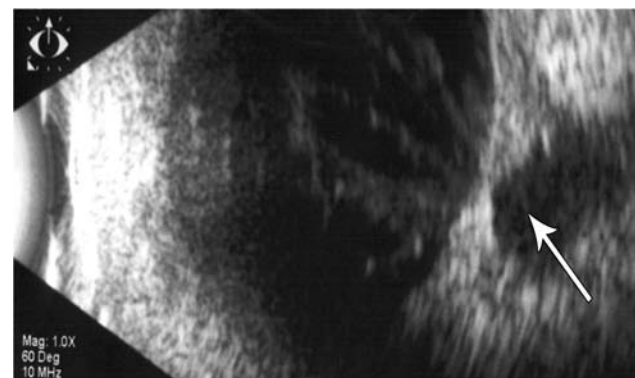


Figure 1 - The B-scan ultrasonography shows vitreous hemorrhage and demonstrates an area of hypolucency at the optic nerve head suggestive of optic nerve avulsion (arrow).

the diagnosis and treat patients who may benefit from urgent surgical intervention. Several modalities have been used, including plain radiographs, CT, MRI, US, and fluorescein angiography. Electrodiagnostic studies, such as visual evoked responses, are used by some centers for trauma patients. The availability of these modalities varies in trauma centers depending on the geographic location of the center and the level of medical care provided. The poor sensitivity of plain radiographs to diagnose orbital fractures makes it unfavorable and no longer used at some hospitals. An MRI has poor resolution for bones, but is satisfactory for defining soft-tissue anatomy particularly at the orbital apex; an MRI is useful for depicting hemorrhage in the sheath of the optic nerve.³ However, the role of MRI in trauma patients is still limited and is not readily available at all centers. Interestingly, MRI failed to detect optic nerve avulsion in some cases.¹ The CT scan has a major role in orbital trauma to assess the severity and extent of orbital injury, particularly in comatose patients with several traumas when physical examination is limited. It is recommended that orbital CT scanning is performed using axial 1-mm and coronal 3-mm slices. Also, 3-dimensional reconstructed CT images of the orbit can be added and are useful adjuncts in planning the surgical repair of complex orbital fractures. Lee et al⁴ reviewed data on 73 patients with orbital trauma and correlated their CT scan images with clinical ophthalmoscopic findings. Most patients with decreased visual acuity or reduced extraocular muscle motility had abnormalities demonstrated by orbital CT scanning. However, in this study, 5 patients (21.7%) with decreased vision had false-negative scans.

Orbital US has not yet been adopted as part of the initial workup for patients with orbital trauma. This is due to its insensitivity for delineation of fractures and lack of soft-tissue differentiation in the presence of an edematous traumatized globe. Moreover, it is operator dependent and may not be available at all times. In the presence of severe facial injury, orbital US is relatively difficult to perform. For the last decade, conflicting

reports on the role of orbital US to diagnose optic nerve avulsion have appeared. In our patient, orbital US clearly demonstrated a posterior defect in the nerve head, which suggested the presence of an avulsed optic nerve head. Our findings are compatible with several case reports with encouraging findings.⁵ These positive orbital US images describe almost the same findings, including attenuation of the optic nerve signal, the presence of hypoechogenic lucency within the optic nerve, and vitreous hemorrhage.

In conclusion, optic nerve avulsion is a true ophthalmic emergency that is difficult to diagnose and challenging to treat. Early diagnosis of this condition will prompt the appropriate medical and surgical interventions for the patient. The use of a specific imaging modality for these patients depends greatly on the treating physician. However, CT scans and orbital US remain the most appropriate initial assessment tools.

Received 28th August 2010. Accepted 11th October 2010.

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