American Journal of Ophthalmology Case Reports 19 (2020) 100792

Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

journal homepage: www.ajocasereports.com/

Long term follow up after transorbital penetrating injury: A case report

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ARTICLE INFO

Keywords:
Penetrating brain injury
Traumatic brain injury
Central pain syndrome
Autonomic dysfunction

ABSTRACT

Purpose: Traumatic brain injury is the leading cause of mortality and disability among young individuals. Unfortunately, there are few publications concerning long term follow up of patients with these types of injuries. We present a case of trans-orbital penetrating brain injury with an 18 year follow up.

Observations: A 43-year-old, previously healthy, male was accidently impaled on a fencing foil resulting in a penetrating brain injury. Initial symptoms included diplopia, ophthalmoparesis, a non-reactive pupil, decreased visual acuity, decreased sensation across the cheek, dysphagia and dysarthria. CT scan taken on presentation showed a clear tract of the foil traversing the various structures of the brain. One week after the trauma, the patient developed a unique constellation of paroxysmal attacks of autonomic dysfunction consisting of profuse diaphoresis and decreased skin temperature on the left side of the body, as well as dilation of the left pupil. Three months after the accident, the patient suddenly experienced severe constant pain affecting the left side of his body associated with thermal and tactile allodynia. On latest follow up, 18 years after the accident, the patient continues to have chronic pain, allodynia, and lack of temperature sensation throughout the left face, arm, and leg. He has a wide based, hemi-ataxic gait, with the left leg swinging out and around. EMG and nerve conduction studies have found no voluntary activity in the temporalis and masseter muscles resulting in atrophy and fibrosis. An MRI shows linear encephalomalacia along the path of the foil extending to the pons, involving the right spinothalamic tract, and cerebellum.

Conclusions and Importance: Our case illustrates the importance of such a longitudinal follow up. It demonstrates the possible severity of the sequelae from these types of injuries including chronic pain and gait ataxia, as well as EOM and autonomic dysfunction. Due to the potential ongoing needs of such patients, it is important to plan a long-term, team-based approach that centers around physical therapy and improving long term quality of life.

1. Introduction

Traumatic brain injury (TBI) is the leading cause of mortality and disability among young individuals. There are many publications discussing immediate complications of TBI. However, there are fewer publications with long term follow up. We present a case of transorbital penetrating brain injury with a unique constellation of permanent complications including chronic central pain with autonomic dysfunction, extraocular motor (EOM) deficits and ataxic gait disturbances with an 18-year follow-up.

2. Case report

A 43-year-old previously healthy male was accidently impaled on a fencing foil. The foil passed through the right medial orbit without rupturing the globe. The patient experienced immediate weakness and numbness of the left side of his body without loss of consciousness. On admission, he was found to have diplopia and ophthalmoparesis, a non-reactive pupil, and decreased visual acuity (20/200) on the right. He had numbness over the right cheek as well as dysphagia and dysarthria. An initial CT scan showed hemorrhage in the fat around the optic nerve. The tract of the foil extended through the superior orbital fissure along the lateral margin of the cavernous sinus, through the basal cisterns, grazed the posterior midbrain, extended through the lateral pons into the right cerebellar vermis, ending in the medial right cerebellar hemisphere (Fig. 1). An angiogram was negative for cerebrovascular injury. The patient was treated with steroids and prophylactic anticonvulsants and antibiotics. There was no sign of active bleeding, infection, CSF leak or

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https://doi.org/10.1016/j.ajoc.2020.100792
Received 29 January 2020; Received in revised form 1 June 2020; Accepted 20 June 2020
Available online 24 June 2020

retained foreign body so the patient was managed conservatively.

One week after initial presentation the patient continued to have significant restriction of gaze in all directions in the right eye. Visual acuity was improved and visual fields were full bilaterally. There was marked weakness of the right temporalis and masseter muscles and right sided hyperacusis. There was decreased sensation to pinprick and temperature on the left half of the body, but proprioception and vibration were intact bilaterally. He had a severely ataxic gait and poor balance. The patient developed a unique constellation of paroxysmal attacks of autonomic dysfunction consisting of profuse diaphoresis and decreased skin temperature on the left side of the body, as well as dilation of the left pupil. He was discharged from the facility after one month of rehabilitation.

Three months after the accident, the patient suddenly experienced severe constant pain affecting the left side of his body associated with thermal and tactile allodynia. The pain was exacerbated by emotional and physical stressors or by changes in weather or temperature, and was accompanied by the aforementioned autonomic symptoms. The patient was diagnosed with central neuropathic pain affecting left side of the body. Over the following years the pain was partially controlled with different combinations of antiepileptics, antidepressants, muscle relaxants, NSAIDS, and topical analgesics. Pain control was hampered by the side effects of medications and difficulty with accurate dose adjustment to control the fluctuating pain.

On neuro-ophthalmological follow-up three years later, there was continued under-action of abduction, and elevation of the right eye. The patient was able to fuse images in primary gaze but complained of diplopia in eccentric gaze. He had wasting of the right temporal muscle with jaw deviation to the right when he opened his mouth. EMG and nerve conduction studies around the same time found no voluntary activity in the temporalis and masseter muscles indicating severe right trigeminal motor dysfunction. At this time, proposed treatment recommendations for ongoing ocular symptoms included use of Fresnel prisms or use of the Faden procedure to produce the same degree of misalignment in eccentric gaze and prevent double vision. The patient chose to proceed conservatively with the use of patching, prisms and rehabilitation. He continues to follow up with neurology and neuro-ophthalmology.

Eighteen years after initial presentation, on most recent follow up, the patient continues to have anisocoria and significant restriction of gaze in all directions in the right eye. EMG and nerve conduction studies have found no voluntary activity in the temporalis and masseter muscles resulting in atrophy and fibrosis. The patient continues to have balance problems, headaches and chronic pain involving the left half of the body, allodynia, and lack of temperature sensation throughout the face, arm, and leg. He has a wide based, hemi-ataxic gait, with the left leg swinging out and around. An MRI shows linear encephalomalacia along the path of the foil extending to the pons, involving the right spinothalamic tract, and cerebellum (Fig. 2). His last combination of medications included high dose of gabapentin, nortriptyline, clonidine, zolmitriptan, Benadryl, lorazepam, Bystolic, Vitamin B6 and B12. He requires continued physical therapy, stretching and balance exercises.

3. Discussion

We report a unique case of a traumatic brain injury by a fencing foil. In our case, the patient sustained a non-missile transorbital intracranial penetrating brain injury with the entrance point of the foil being at the medial orbit. The horizontal-pyramidal shape of the orbit deflects penetrating objects toward its apex allowing penetration of non-missile projectiles into the skull through the optic canal or superior orbital fissure. Therefore, the sites of possible CNS injury include the frontal lobe, cavernous sinus, temporal lobe, thalamic nuclei, brain stem and basal cistern, of which only the fourth was injured in our case.[1,2]

To our knowledge, this is the first case of transorbital penetrating brain injury resulting in a hemi-body central pain syndrome similar to that described in the literature as central post-stroke pain (CPSP). Classically, this type of pain is described after vascular lesions in the thalamus and spinothalamic tract, however it can be a consequence of lesions located anywhere along the somatosensory pathways. The onset of pain in CPSP is often preceded by a loss of thermal sensation. Like in our patient's presentation, the pain usually develops within several months, but can also develop years after the injury. Pain was reported in the whole hemi-body, similar to our case, in 55% of patients.[3]

Among our patient's permanent disabilities were significant ataxia and balance problems. Neuromotor impairment is a common sequela of severe TBI. Studies have shown that motor function markedly improves between the first and sixth week after TBI, especially with the employment of acute rehabilitation.[4] However, in a long-term, longitudinal study observing neuromotor impairment, more than a third of patients had persistent impairment at 2 years. Ocular convergence seems to significantly reinforce postural stability, even with bilateral loss of vestibular function.[5] Several studies have shown that strabismus worsens postural impairment.[6] In our patient's case, it is unclear how much of his balance problems are related to the direct damage to the cerebellar and pontine injuries, or to the damage to the upper cranial nerves.

4. Conclusions

Many publications on penetrating traumatic brain injuries conclude with the call for an immediate treatment plan by a multidisciplinary team. However, these reports do not mention the importance of long-term follow up. Our case illustrates the importance of such a

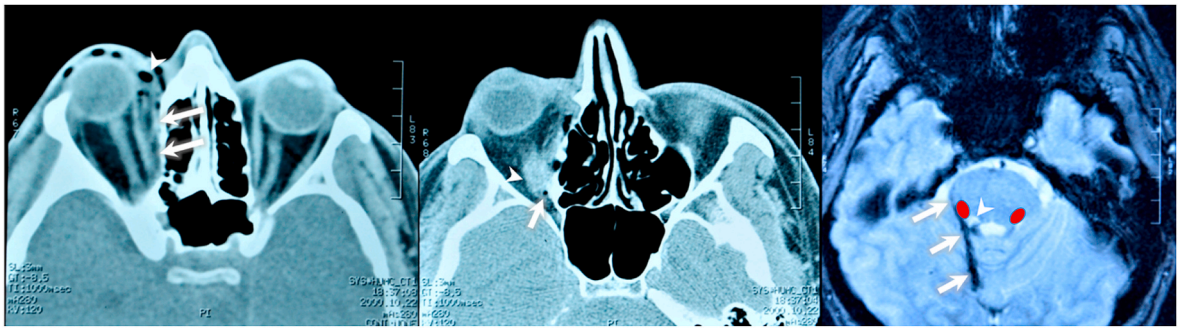

Fig. 1. Orbital and intracranial injury along the course of the foil. (A,B) Contrast enhanced orbits CT shows (A) right preseptal soft tissue thickening, soft tissue emphysema (arrowhead) and small retrobulbar hemorrhage (arrows) in the medial orbit. (B) Punctate soft tissue emphysema (arrow) and small hemorrhage (arrowhead) is just anterior to the inferior orbital fissure along the course of the foil injury. (C) Axial T2 gradient echo shows linear hemorrhage (arrows) and edema (arrowhead) along the course of the foil involving the right lateral basal pons near the spinothalamic tract (ovals) extending into the superior cerebellar hemisphere and lateral vermis.

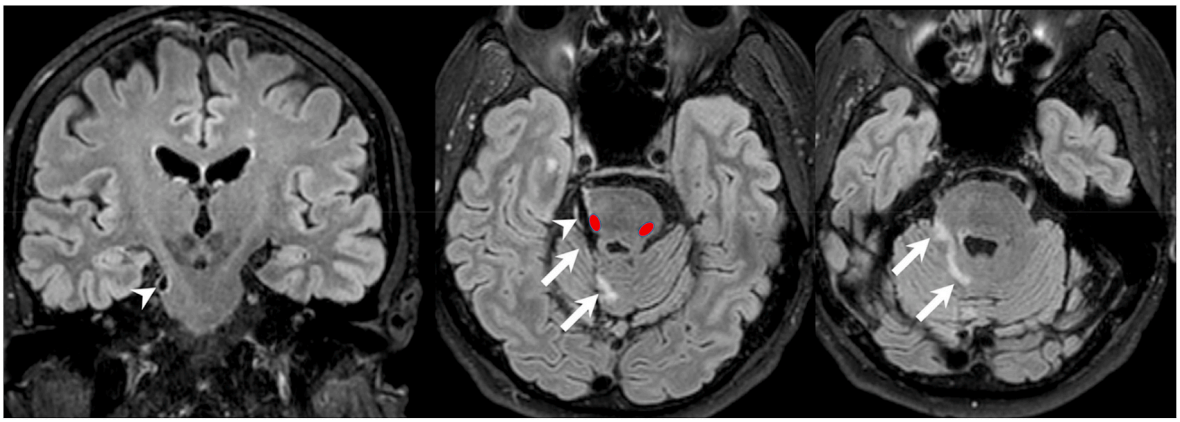

Fig. 2. Eighteen years later, FLAIR images demonstrate gliosis along the course of the foil. (A,B) Encephalomalacia and gliosis (white arrowheads) extends into the right lateral basal pons to involve the spinothalamic tract (ovals), (B,C) the superior cerebellar hemisphere, lateral vermis and superior right brachium pontis (white arrows).

longitudinal follow up. It demonstrates the possible severity of the sequelae from these types of injuries including chronic pain and gait ataxia, as well as EOM and autonomic dysfunction. Due to the potential ongoing needs of such patients, it is important to plan a long-term, team-based approach that centers around physical therapy and improving long term quality of life. Treatment should be individualized to each patient's needs and potentially involve neuro-ophthalmology, physical therapy, neurology and others.

Patient consent

Written patient consent for publication was obtained.

Funding

No funding or grant support.

Declaration of competing interest

The following authors have no financial disclosures:
Caroline Vloka, MD, Alexander Vloka, MD, Tanya Rath, MD, S. Tonya Stefko, MD, All authors attest that they meet the current ICMJE criteria for Authorship.

Acknowledgements

No Acknowledgements.

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