CASE REPORT

Traumatic Chiasmal Syndrome: A Midline Axonal Injury

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ABSTRACT

We report a 26-year-old man who developed cerebrospinal fluid (CSF) rhinorrhoea and complete bitemporal hemianopia after having sustained a blunt frontal region trauma. The chronology of midline frontal trauma and CSF rhinorrhoea, followed days later by transient diabetes insipidus and discovery of a bitemporal visual field loss, makes up a unique syndrome and must be evaluated for. Mechanism of such injuries, their pertinent complications, and review of literature are herein discussed.

Keywords: Bitemporal hemianopia, blunt frontal region trauma, midline axonal injury, traumatic chiasmal syndrome

INTRODUCTION

Since its first reporting in 1960, bitemporal hemianopia is being increasingly recognised and reported as a complication of head injury.¹ The exact pathophysiology of this syndrome is still unclear, although many hypotheses are put forward in literature.

CASE REPORT

A 26-year-old man involved in a motor vehicle accident having sustained a head injury came to us with a Glasgow Coma Scale of E1M5V2; pupils were equal and reactive. Initial computed tomography scan showed a midline frontal bone fracture (Figure 1), with the fracture line running to the basi sphenoid, along with large intraparenchymal haematoma in left frontal region. Patient was also noted to have cerebrospinal fluid (CSF) rhinorrhoea. Patient underwent decompressive craniectomy and intracerebral haematoma evacuation with anterior cranial base repair. Postoperatively he was noted to have a transient period of hypotonic polyuria and hypernatraemia, which improved over 2 weeks. After gradually regaining consciousness over a week, he complained

of diplopia on upgaze and decreased vision in both eyes. Detailed visual evaluation, which was now possible in view of his improved neurological status, revealed visual acuity of 6/6 in the right eye and 6/36 in the left eye. Diplopia on upgaze was attributed to direct injury of the right superior rectus muscle. Humphrey visual field charting revealed the presence of complete bitemporal hemianopia (Figure 2). He was given a trial with parenteral steroids, but he failed to show any improvement in vision. CSF leak had resolved post-anterior cranial base repair. MRI scanning 2 weeks postoperatively revealed presence of central chiasmal contusion with thinning of nasal fibres (Figure 3). On follow-up after 3 months, patient's visual acuity had improved to 6/6 in both eyes with resolution of diplopia. But patient failed to show any improvement in the visual field defect.

The patient has consented to the submission of the case report for publication.

DISCUSSION

Traumatic chiasmal syndrome usually follows severe frontal region trauma accompanied by prolonged loss of consciousness. Affected patients are noted to have

midline basilar skull fractures that traverse the mid-clivus through the sella turcica floor, dorsum sella, and sphenoid sinus.² The visual field defect usually starts immediately after the insult but is diagnosed

late in view of altered sensorium in these patients with severe head injury, although the resultant degree of visual loss may not necessarily reflect the severity of the head injury.

Mechanism

It is postulated that traumatic chiasmal syndrome results from tearing of the optic chiasm on a microscopic, if not macroscopic, level.² We believe that it belongs to the same spectrum as diffuse axonal injury, but more specific where the injury is a result of shearing forces concentrated only on the midline or junctional structures. These forces may cause selective tearing and stretching of the chiasmal crossing fibres and pituitary stalk. The pathogenesis can be explained based on the direction of impact on the head, with maximum force being exerted on midline structures such as the nasal decussating fibres and the pituitary stalk. Another hypothesis being tearing and rupture of fine branches of the internal carotid artery that supplies the medial chiasmal region inducing ischaemia and softening of the crossing fibres. Contusional necrosis of the chiasm caused by compression by herniated gyrus rectus is also postulated.³ These mechanisms should not be considered to be mutually exclusive. Even though iatrogenic causes of such midline chiasmal injury are reported in transphenoidal approaches,⁴ they are unlikely and have never been reported in open anterior cranial base repair. CSF rhinorrhoea in these patients reiterates the presence of

FIGURE 1 Midline frontal bone fracture.

FIGURE 2 Humphrey automated static perimetry shows complete bitemporal hemianopia.

midline skull base fracture caused by blunt trauma with force directed towards central structures.

Diabetes insipidus often reported with such injuries⁵ can be easily explained based on the anatomical proximity of the pituitary stalk to the optic chiasm (Figure 4). Also, its transient nature can also be a consequence of cerebral oedema, which is most marked at 3 to 5 days post injury, functionally impairing the hypothalamic neurosecretory cells that are structurally intact. Such transient diabetes insipidus should resolve once oedema subsides. Presence of dyselectrolytaemia in patients with head injury may point towards presence of chiasmal injury.

Investigation

Clinical evaluation and visual field charting are sufficient to reach a diagnosis in such cases. Magnetic resonance imaging (MRI) usually on careful inspection should demonstrate damage in the midline structures of the base of the brain ranging from traumatic disruption to complete transaction of chiasm.⁴

FIGURE 3 Coronal section at the level of chiasm demonstrating thinning of decussating central fibres on FLAIR sequence and central dot hyperintensity on T2-weighted images representing contusion.

MRI of our patient revealed central contusion and thinning out of nasal fibres at chiasmal level with no detectable pituitary abnormality (Figures 3 and 4).

Complications

The breach in the anterior cranial base was responsible for the CSF rhinorrhoea in our patient. Transient diabetes insipidus is a well-recognised complication in these patients due to proximity of the neurosecretory fibres to the chiasm. Other associated complications are panhypopituitarism, carotid cavernous

FIGURE 4 T1-weighted image demonstrating normal pituitary and diagram showing proximity of neurosecretory fibres to the chiasm.

fistula, cranial nerve palsies, carotid aneurysm, meningitis, pneumatocele, or intrasellar haematoma.³ Post-traumatic bitemporal hemianopia mainly remains as an early warning bell for such critical complications. Our patient showed significant recovery in visual acuity on 3-month follow-up with 6/6 vision in both eyes, but failed to show any improvement in field defects. Most of such patients have a final visual acuity of 6/12 or better in at least one eye.⁵ Damage to chiasmal fibres is usually associated with irreversible visual field defects. None of the reported cases in the literature demonstrate an improvement in visual field deficits in patients with traumatic chiasmal syndrome.

CONCLUSION

Traumatic chiasmal syndrome may belong to a broader spectrum of diffuse axonal injuries caused by midline blunt trauma to forehead. Field defects as a consequence of such injuries are usually irreversible. MRI of perichiasmal region is useful in demonstrating radiographic evidence of such injuries. Keen eye for the clinical sequence of frontal region trauma with

CSF rhinorrhoea, followed by electrolyte disturbances and discovery of bitemporal visual defects, will lead to early identification of these cases.

Declaration of interest. The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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