Primary Brainstem Injury: Benign Course and Improved Survival

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Summary

Primary brainstem injury following head injury is a rare event. The victims often have features of supratentorial injury, and a primary isolated injury to the brainstem occurring due to shearing stresses or to injury from the tentorial edge is extremely rare. In the presence of supratentorial injury, these patients may have altered sensorium. Isolated brainstem injury may manifest itself as internuclear ophthalmoplegia, anisocoria, rigidity and cerebellar tremor. Such injuries are now being diagnosed more often due to improved imaging techniques. We treated nine such cases who had sustained primary brainstem injury in road traffic accidents, all but one of whom were subsequently independent. Primary brainstem injuries need not be associated with poor prognosis and mortality and may run a benign course with good quality of survival.

Keywords: Brainstem; facial myokymia; head injury; internuclear ophthalmoplegia.

Introduction

In head injury, the brainstem is usually injured secondarily, i.e., due to an expanding mass lesion producing brainstem distortion and Duret’s haemorrhages. Primary brainstem injury is an extremely rare occurrence, and when such an injury occurs, it is often accompanied by diffuse supratentorial lesions. Primary brainstem injury in isolation is indeed a rare event, and the rarity of the lesion can be appreciated from a statement made by Adams et al. [2] after analysis of autopsies in patients dying of head injuries, “in a personal series of 600 brains of patients dying as a result of head injury, we have never identified a primary traumatic lesion in the brainstem in isolation”. Recently there have been reports of cases of primary brainstem injury surviving although with devastating neurological deficits [4, 20]. We present nine cases of primary brainstem injury managed over the last seven years, all but one of whom survived with minimal or no neurological sequelae. Relevant literature is briefly reviewed.

Clinical Material

Case No 1

A 24-year-old-army officer hit his head on the ground after a fall from a motorcycle. He was transiently dazed and was able to get up and walk. He was admitted to hospital 30 minutes later for observation, when a neurological examination did not reveal any focal abnormality. Glasgow coma score (GCS) on admission was 15/15. Twelve hours later, he complained of diplopia and neurological examination revealed internuclear ophthalmoplegia. There was no anisocoria or motor deficit. CT brain showed a midbrain haematoma. There was no evidence of supratentorial injury, cerebral oedema or obliteration of cisterns. He was managed conservatively and INO resolved after about six weeks and there was no residual deficit.

Case No 2

A 32-year-old-male struck his head against the body of the vehicle he was travelling in as the vehicle skidded off the road. There was no loss of consciousness. About six hours later, he was admitted with “inability to see clearly”. There was no external injury and Glasgow Coma Score was 15/15. He had INO and there was no other neurological abnormality. CT was done about 24 hours after the injury, which showed a midbrain haematoma. There was no supratentorial injury, or effacement of cisterns. He was managed conservatively and INO resolved after 4 weeks. There was no residual deficit.

Case No 3

A 24-year-old-male was admitted with history of having hit his head against the interior of the bus he was travelling in when it met with an accident. There was no loss of consciousness. He however complained of diplopia. Clinical examination showed scalp contusion over the left frontal region. INO was detected on neurological examination. There was no other neurological deficit. MRI brain could be done eight days after injury, which showed an iso-intense lesion in the midbrain on T1weighted images (T1WI), which was well circumscribed and hyperintense on T2weighted images (T2WI) (Fig. 1). Basal cisterns were normal and there was no evidence of supratentorial injury. He was managed conservatively and showed gradual resolution of INO over 4 weeks. There was no residual neurological deficit.
Case No 4

A 28-year-old soldier was admitted in a state of altered consciousness after having sustained a head injury when the vehicle he was travelling in fell into a ditch. There was no external injury. Neurological examination revealed GCS 9/15 (E2V2M5). Pupils were equal and there was hypertonia in the limbs, brisk tendon jerks with extensor plantar responses. He showed gradual improvement in the level of consciousness with cerebral decongestants. About 36 hours later when he was cooperative enough, he was seen to have INO. CT brain done 48 hours after injury showed diffuse cerebral oedema and a small left sided thalamic haematoma. There was a haematoma in the pons. He continued to have hypertonia of all four limbs with inability to move without support. There was no appreciable recovery and hypertonia persisted in the limbs even after six months of injury. INO too did not resolve. He remained dependent and had to be mobilised in a wheelchair (GCS outcome score 3).

Case No 5

A 30-year-old male was admitted after he had fallen from a speeding two wheeler and struck his head on a hard surface. There was transient loss of consciousness followed by rapid recovery. Neurological examination revealed GCS 15/15 and no focal deficit. Two hours after the injury, anisocoria was noticed without any deterioration in GCS. Left pupil was larger than the right. Reaction to light and convergence was preserved. CT brain done 24 hours after the injury showed midbrain contusion and obliteration of adjacent cistern, haemorrhage and a deep seated periventricular haematoma. There was no other supratentorial lesion. He was managed conservatively and when discharged 4 days later, was asymptomatic with resolution of anisocoria. There was no neurological disability.

Case No 6 & 7

These were almost identical. Patients were admitted with history of head injury in road traffic accidents. There was no loss of consciousness. GCS was 15/15 on admission. INO was noticed 24 hours after admission. CT brain done 48 hours after admission showed a pontine lesion with obliteration of cistern and haemorrhage. There was no evidence of supratentorial injury or cerebral oedema. They were managed conservatively and INO resolved after 6 weeks and 12 weeks respectively. There was no residual neurological deficit.

Case No 8

A 35-year-old male was admitted with history of having sustained a head injury in a fall. There was transient loss of consciousness followed by rapid recovery. On admission, GCS was 15/15 and there was no neurological deficit. He was discharged after observation for 36 hours. He was referred again after seven days with persistent facial movements of the right side. Clinically, the movements were undulating and involved the right side of the face and the face. Voluntary facial movements were present and there was no focal neurological deficit. MRI brain could be done after two months of injury, which showed iso-intense lesion in the pons on the right side on T1WI, which was hyperintense on T2WI. A diagnosis of focal gliosis affecting the brainstem was made and the facial myokymia was treated with two sittings of Inj Botulinum toxin given into the facial muscle. He has remained free from facial myokymia and there has been no fresh neurological deficit over a follow up period of three years.

Case No 9

A 5-year-old male child was admitted 24 hours after having sustained a head injury due to a fall from a height of about 12 feet. There was truncal ataxia and dysmetria and intention tremor involving the left upper and lower limbs. There was no disturbances of ocular movements, anisocoria or facial asymmetry. CT brain (Fig. 2) showed left pontine haematoma with effacement of adjoining cistern. There was no supratentorial injury or cerebral oedema. The child was managed conservatively and discharged after few days. When reviewed two months later, he still had mild cerebellar signs although there was no truncal ataxia.