Delayed-onset bilateral abducens paresis after head trauma

Pravin Salunke, Amey Savardekar, Sukumar Sura

Bilateral sixth nerve paresis following closed head injury, though rare, is a known entity. However, delayed-onset post-traumatic bilateral abducens paresis is extremely rare. We present two cases. The first patient had onset of bilateral abducens paresis 2 weeks after closed head injury and the second patient after 3 days. The cause in the former was detected to be chronic subdural hematoma and in the latter is speculated to be edema/ischemia due to injury to soft tissue structures housing these nerves. The delayed onset of bilateral abducens paresis following head injury may vary according to the cause. There may be another mechanism of injury apart from direct trauma. Though rare, it needs to be evaluated and may have a treatable cause like elevated intracranial pressure.

Key words: Bilateral abducens nerve, delayed onset paresis, head injury

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The incidence of unilateral abducens palsy following head injury is as high as 1 to 2.7%.[1] However, bilaterality is rare. Post-traumatic bilateral sixth nerve palsies are usually associated with additional intracranial, skull, and cervical spine injuries.[1] Cases of bilateral sixth nerve palsy without any associated injuries and good Glasgow coma scale (GCS), though rare, have been reported in the past.[2] However, delayed-onset bilateral sixth nerve palsies without significant associated injuries are extremely rare.[3]

We present two cases of delayed-onset bilateral sixth nerve palsy in young patients following head injury with GCS 15. Traumatic bilateral sixth nerve palsies with respect to their time of onset, mechanism of injury, and prognosis have been reviewed.

Department of Neurosurgery, PGIMER, Chandigarh, India

Correspondence to: Dr. Pravin Salunke, Department of Neurosurgery, PGIMER, Sector 12, Chandigarh – 160 012, India. E-mail: drpravin_salunke@yahoo.co.uk

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Case Reports

Case 1

A 20-year-old man skidded off the bike and had transient loss of consciousness. Computed Tomograph (CT) showed thin right acute subdural hematoma at presentation. He was managed conservatively as he had no deficits and GCS was 15. Six weeks later, he came with severe headache and bilateral sixth nerve palsy [Fig. 1]. CT showed right chronic subdural hematoma with mass effect [Fig. 2]. The fundus examination revealed papilledema. Patient was operated using two burr holes and drainage of subdural hematoma. The patient recovered completely.

Case 2

A 30-year-old man sustained head trauma in a road traffic accident. He had transient loss of consciousness. The GCS at presentation was 15 without any focal deficits. The CT at presentation was normal and showed no basal fractures or subarachnoid bleed [Figs. 3 and 4]. Three days later, he presented with bilateral 6th nerve paresis (Right > Left). The fundus examination showed normal optic discs. Repeat CT head showed the same findings. The right-sided paresis did not improve even after three months after injury despite giving steroids.

Discussion

Gaze deficits after head trauma are not uncommon and deficits in the lateral gaze are frequently encountered. The abducens nerve, mostly due to its long intracranial course, is liable to be affected through myriad mechanisms: Injury to the central nervous system, the peripheral nerve, or the motor unit. Physical examination and appropriate imaging help identify the cause. Isolated lateral rectus palsy with no other cranial nerve signs, no pyramidal tract signs, and no signs of muscular entrapment is thought to result from an injury to the peripheral nerve along its course from the brain stem to the lateral rectus.[1]

Anatomically, abducens nerve consists of intracisternal, intracavernous, and intraorbital parts. After exiting the

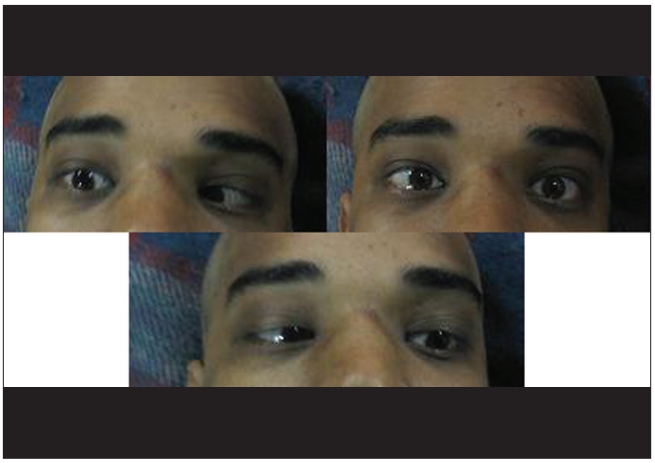

Figure 1: (Case 1) Eye movements in either lateral gaze demonstrating bilateral abducens paresis

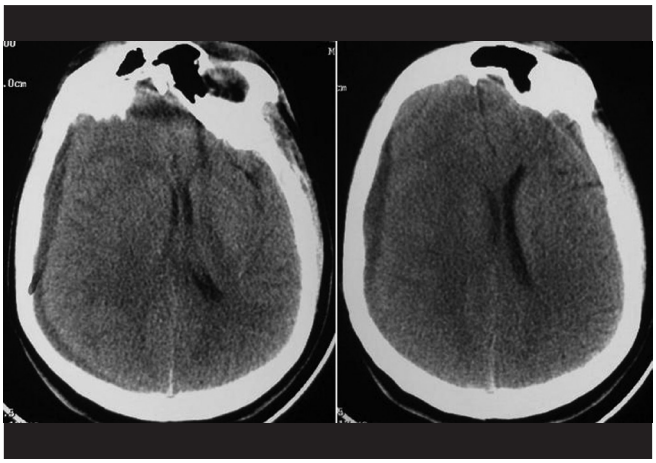

Figure 2: (Case 1) CT scan showing right frontoparietal chronic subdural hematoma

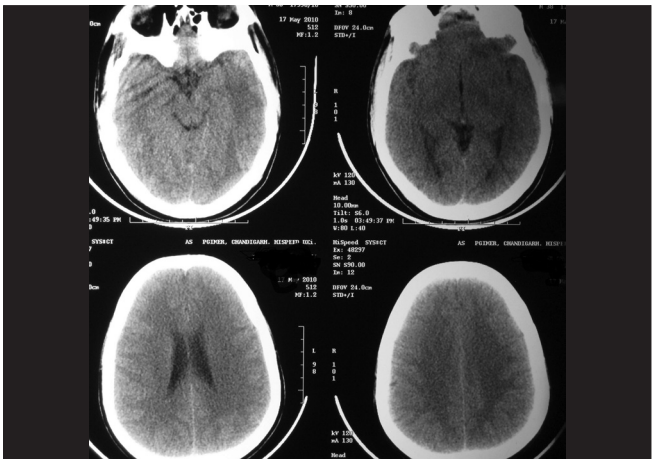

Figure 3: (Case 2) CT scan showing no hematoma

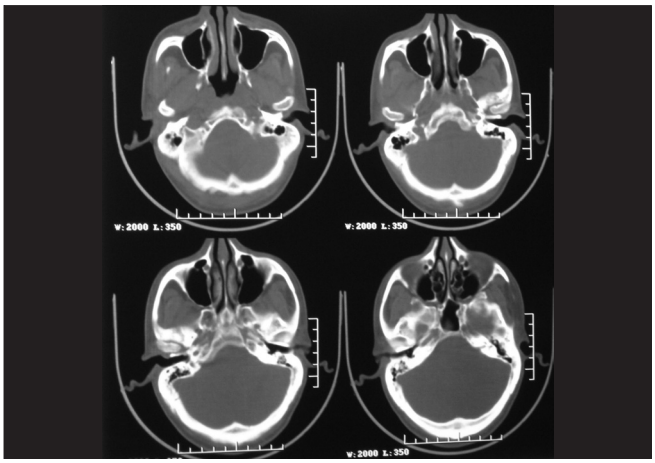

Figure 4: (Case 2) CT scan bone windows showing no fracture of the skull base

pontomedullary sulcus, it courses through the prepontine cistern, then joins the cavernous sinus in the petroclival region, and innervates the lateral rectus muscle in the orbit. The abducens nerve has three angulations in the petroclival region. After its exit from the pontomedullary junction, the nerve ascends vertically over the ridge of the petrous bone. Later, it changes direction approximately 120 degrees forward and passes below the petrosphenoid ligament (Gruber's) and then into Dorello's canal and then passes through the cavernous sinus to enter the superior orbital fissure.[2] These angulations are vulnerable to injury.

The post-traumatic abducens palsy can be variable in its onset, duration, and recovery. Acute onset refers to the immediate post-traumatic palsy which may be due to: (a) head injury causes downward displacement of the sixth nerve in Dorello's canal with contusion of the nerve against the petrous ridge; (b) bony injury of the skull base; (c) static loading force causes avulsion of the petrous bone from the foramen lacerum to the outer side of the bone, which stretches and directly injures the abducens nerve.[4] It is greatly speculated that both the abducens nerves are stretched by the linear accelerated force in mid-sagittal plane at the time of impact. The apex of the petrous pyramid acts as the fulcrum, so that the nerves are compressed, contused, and stretched at this point.[4]

Delayed onset of abducens nerve palsy may be due to vasospasm or nerve ischemia due to injury to branches of meningodorsal artery.[3] The other cause could be due to edema of surrounding tissues, a mechanism akin to delayed traumatic facial palsy.[5]

The abducens nerve injuries have been documented to take place in the petroclival region. Histopathology revealed edema and perineural hemorrhages predominantly at the locations of dural entry point and petrous apex.[3]

Elevated intracranial pressure is known to cause abducens paresis. This may be one of the mechanisms of delayed-onset bilateral abducens paresis following head injury, as was evident in our first patient. Thus, there may be other mechanisms of delayed-onset abducens nerve involvement apart from direct trauma.

Spontaneous recovery rate in unilateral 6th nerve palsy is 12 to 73% in six months. The median time to recovery is 90 days. The predictors of non-recovery may include complete palsy and bilateral palsy.[1] Contrary to the existing reports, our patient showed recovery.

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