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Delayed bilateral lateral gaze deficit following trivial head trauma in a pediatric patient

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Abstract: *Traumatic bilateral gaze deficits are uncommon in adolescent population. They are typically associated with severe head injuries. We describe a case an unusual case of delayed presentation of complete bilateral Abducens nerve palsy in 12 years old boy following trivial head trauma. This case highlights the approach to diagnosis and possible treatment options for bilateral Abducens nerve palsy.*

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I. Introduction

Gaze disturbances are relatively uncommon after head injury. Traumatic injuries to the brain stem, peripheral cranial nerves, or the orbits have the potential to cause a lateral gaze deficit. Isolated loss of lateral gaze, results from injury to Abducens nerve along its course from brain stem to the lateral rectus. It is most vulnerable to injury due to its long extracerebral intracranial course. Bilateral involvement of Abducens nerve is uncommon in head injury and extremely rare occurrence in trivial trauma [1]. These injuries are typically associated with intracranial, skull, and cervical spine injuries.

Although, the incidence of bilateral involvement accounts for 10% of Abducens nerve paresis, but author's believe that actual incidence may be high as some of the patients remain undiagnosed due to severe head trauma and associated co-morbidities [2]. Due to scarcity of literature, the mechanism for bilateral 6th cranial nerve involvement remains controversial. However; trauma still remains the most accepted theory worldwide. Here we describe an unusual case of 12 years old boy with trivial head injury presented to us with bilateral gaze deficit diagnosed clinically.

II. Case Report

12 years old male presented to our outpatient clinic following a road traffic accident while riding on the back seat of motorbike 2 days back. He presented with sudden onset of diplopia associated with mild headache since morning. Symptoms started 2 days after the injury, although he was apparently alright after the episode of trauma. Immediately following trauma he had a history of transient loss of consciousness for 10 minutes while transferring to the hospital. He was taken to a nearby tertiary care hospital, where primary treatment was provided and was admitted for observation. Medical records from previous treatment showed GCS of 14 points with abrasions over right thigh and foot at the time of arrival in emergency. There was no history of vomiting, seizures, or bleeding from ears. Initial neurological examination was non-focal with no associated systemic abnormality. Computed tomography head was unremarkable and so patient was discharged after 24 hours of observation. Records showed that patient was given bolus dose of parental methylprednisolone and was discharged on oral steroids.

On third day of injury, patient reported a sudden onset of double vision with mild headache after waking up in the morning. He presented to our clinic with persistent diplopia and headache.Mild convergent bilateral squint can be seen in primary gaze (Fig. 1). Ophthalmologic examination revealed, painless restriction to abduct either eye beyond midline, indicating complete bilateral Abducens nerve paresis (Fig. 2). All other extra-ocular movements were intact and normal. Pupils were symmetric and reactive to light and consensually without any impairment of visual acuity. Examination for other cranial nerves was normal and fundoscopic examination was insignificant. He scored full marks in mini mental state examination (MMSE) test. MRI of brain with orbits was done, which also showed normal results.

After arriving at the diagnosis, patient and the guardians were counseled about the natural history of the disease and were asked to follow up at regular intervals. We gradually tapered the dose of oral steroids and stopped the drug after 3 weeks. At 1 month after the injury, bilateral gazed deficit persisted with no signs of improvement, however; diplopia was improved slightly. Patient was lost to follow up after that and revisited us

at 11 months following injury with surprisingly good ocular mobility and alignment in both eyes, although some residual restriction of abduction persist in left eye compared to right.

III. Discussion

Earlier results from a metacentric study showed that neoplastic and traumatic injuries, being the most common cause in children, while vascular and idiopathic are responsible for majority of cases in adult population [3]. However; traumatic injuries to the brain stem, cranial nerves or the orbit have been advocated as the most accepted theory for dysfunction of the ocular mobility [4]. Bilateral Abducens nerve palsies may occur as a result of direct injury to both nerves independently or indirectly from increased intracranial pressure. Dural puncture, whiplash injury, hangman's fracture and halo traction has also been proposed as causative factors for bilateral paresis. Mechanism of injury is usually contusion or stretching of the nerve, due to its long intracranial course and vertical displacement of brain is supposed to be the causative factor for such lesion [5]. An association with other cranial nerve palsies, mostly hypoglossal nerve is seen due to close proximity of nucleus of sixth, seventh and eight nerves.

Clinical presentation is mainly persistent diplopia with associated headache which is seen acutely or may have a delayed or late presentation as seen in our case. Examination shows misalignment of visual axis with restricted abduction of the eyes beyond midline on asking the patient to look on either side. Associated cranial nerve involvement and subtle head injury signs should be carefully looked for. Although, thorough clinical examination confirms the diagnosis, but imaging modalities should be added to rule out other pathologies. While there are well recognized findings on magnetic resonance imaging for other causes of Abducens nerve palsy, abnormal radiological findings in the setting of trauma are rare [6]. Sixth cranial nerve palsy may be a false localizing sign of raised intracranial pressure which needs to thoroughly investigated using various imaging modalities, lumbar puncture and cerebrospinal fluid analysis.

In the setting of a trivial head injury, the most likely mechanism of injury is stretching of the nerve by acceleration in the mid-sagittal plane at the time of impact. However; there seems to be more reports of this injury without fracture or haematoma than with, perhaps because those with associations are deemed less interesting [7]. Cases of bilateral Abducens palsy and unilateral palsy have also been reported after lumbar puncture and following water soluble myelography [8,9].Lateral rectus paralyses usually occur due to direct damage of the sixth cranial nerve, encephalon nuclei or less frequently, diffuse axonal damage. Patients with diffuse axonal injuries usually present with low Glasgow Coma Score, multifocal neurological damage and certainly carry a poor prognosis. Diplopia from Abducens nerve palsy should be differentiated from those caused by mechanical entrapment of orbital contents within a fractured orbit. Children are less likely to sustain traumatic bilateral sixth nerve palsy than adults; however, they have associations with tumor or vascular anomalies.

Due to the lack of enough evidence in the literature, no proper treatment guideline has been established. The aim of management in paediatric cases with isolated sixth nerve palsy should be to identify and treat the predisposing factor and relieve the symptoms. Development of Abducens nerve palsy following minimal head trauma should raise the suspicion of a compressive lesion, and warrants consultation with neurologist to rule out skull base tumor. Overall spontaneous recovery rate of traumatic Abducens nerve palsy is high, however; a complete or bilateral paresis have a poor prognosis [10,11]. Treatment of bilateral post-traumatic palsy should always start with conservative approach using oral steroids, despite low recovery rate initially in 4 months. Minimally invasive technique using subtenon injection of botulinum toxins showed appreciable results compared to the conservative treatment [12]. Stabismus surgery for non-resolving chronic Abducens nerve palsy is an option, but should only be considered when all the other available conservative treatment modalities have been tried with assurance. The maximum time that may be allowed for an isolated sixth nerve palsy to improve is debatable. However; literature suggest to wait for minimum of 6 months before embarking on any surgery, as by this time recovery is usually seen in the ocular mobility.

IV. Conclusion

Bilateral isolated sixth nerve palsy is an extremely rare occurrence in minor head injury in adolescent and very few cases have been reported in literature. Complete or partial recovery during the natural course of the disease, over a period of 6 months is usually expected, which is an important consideration when counseling patients with such injuries. Presence of bilateral Abducens nerve palsy indicates a severe trauma to the head and neck, and patients with bilateral palsy show a tendency to have a low GCS. However, the patient in the present case exhibited a high GCS with no definite head and neck injuries confirmed by imaging.

Conflict of interest Authors report no conflict of interest, financial or otherwise, concerning the material or methods used in this study or the findings specified in this paper.

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Ethical standard This study was approved by ethical committee and was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

V. Figures And Legends

Figure 1: Clinical photograph showing mild convergent bilateral squint in primary gaze.
Figure 2: Picture showing bilateral complete lateral rectus palsy on asking the patient to look on either side.

Figure 1

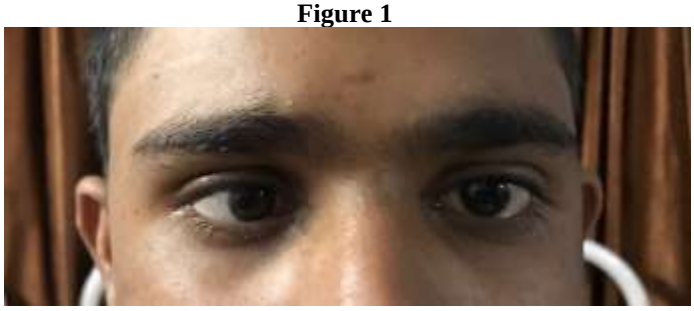
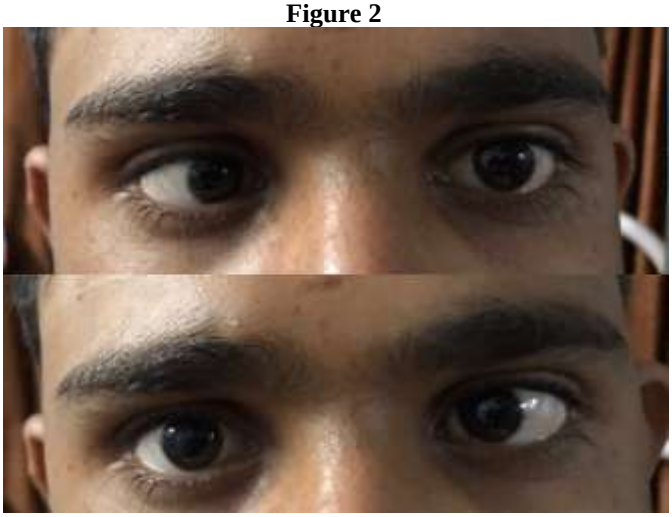


Figure 2



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