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Bilateral abducens palsy in closed head injury: A comprehensive review of literature based on a case report

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ABSTRACT

Isolated bilateral sixth nerve palsies are rare, particularly in the setting of trauma. Most post-head injury cases with bilateral abducens palsy involve either basal skull fractures, particularly clival fractures. We present a case of bilateral abducens palsy after closed head injury in a young male who presented to the emergency department and a comprehensive literature review based on our clinical case. A Medline search for bilateral abducens palsy in closed head injury showed 89 results. Articles were excluded if crush head injury, non-traumatic bilateral abducens nerve palsy, associated vascular malformations were reported. After thorough search and filtering of those articles, fifty-one publications were found which reported and discussed about traumatic bilateral abducens palsy with closed head injury. In these 51 articles, a total of 139 cases were recorded. Several theories have been postulated to explain mechanisms of abducens nerve injury in trauma both in immediate and delayed settings. In our case, patient presented with immediate onset of bilateral abducens palsy. On imaging, clival fracture was seen in CT brain, which can be attributed for the nerve injury. Cases with retroclival extradural haematoma had higher chances of multiple cranial nerve injuries. Cases with multiple basal skull fracture involving petrous temporal bone fracture had higher chances of facial nerve injury. Along with bilateral involvement, the poorer outcome for recovery can be related with the severity of the adduction deficit. Our case showed no improvement in bilateral abduction during follow-up at 6 months. Clinical presentation of traumatic bilateral abducens nerve palsy is rare following closed head trauma and is usually associated with other injuries which are incompatible with life. It can be associated with other nerve injuries depending on basal skull fractures.

Key words: Bilateral abducens palsy; Closed head injury; Clival fracture

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INTRODUCTION

Unilateral abducens nerve palsy has been reported to occur in 1% to 2.7% of all head trauma.[1,2] The extended intracranial course of 6th nerve along with its anatomical relationship to the petrous apex, makes it more susceptible to traumatic injury.[3] Isolated bilateral sixth nerve palsies are rare, particularly in the setting of trauma. Most post-head injury cases with bilateral abducens palsy involve either basal skull fractures, particularly clival fractures or other intracranial pathologies with or without cervical spine injury.[1]

We present a case of bilateral abducens palsy after head injury in a young male who presented to the emergency department of our institute and a comprehensive literature review based on our clinical case. To the best of our knowledge, no publication in the literature discusses the total number of cases along with the imaging findings and clinical course of those cases.

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MATERIALS AND METHODS

A Medline search for key words "bilateral abducens palsy" and "closed head injury" showed 89 results. Publications were limited to those published in English. Articles were excluded if crush head injury, non-traumatic bilateral abducens nerve palsy, associated vascular malformations were reported. After thorough search and filtering of those articles, fifty-one publications were found which reported and discussed about traumatic bilateral abducens palsy with closed head injury. In these 51 articles, a total of 139 cases were recorded.

RESULTS

The results are summarized in Table 1. The results include author, year of publication, associated cranial nerve injury, radiological findings, presentation and recovery wherever mentioned by the author in tabular form.

CLINICAL CASE

A 22-year-old male, pillion rider on a two -wheeler was involved in motor vehicle accident when they accidentally skid and fell. He sustained injury to the head. Patient had 2 episodes of vomiting and loss of consciousness post injury for 2 minutes with no history of seizures and nasal/ear bleed post injury. On arrival at the emergency department, he was haemodynamically stable. He was conscious, oriented and moving all four limbs. A secondary survey did not reveal any additional signs of injury or burns.

After the patient was stabilized, a detailed neurological examination revealed bilateral abducens nerve palsies along

Table 1: List of publications

Sl. No.	Author	Year	No. of cases	Associated cranial nerve	Radiology of the cranium	Presentation	Recovery
1	Schneider et al	1971	2	Nil	Normal	Immediate	No
2	Roberts et al	1972	1	Nil	Normal	Delayed	No
3	Keane et al	1976	11	Not known	Not known	Not known	Not known
4	Takagi et al	1976	3	Nil	Normal	Immediate	2 cases recovered
5	Goubran et al	1978	1	Nil	Middle cranial fossa fracture through the sella turcica	Immediate	Yes
6	Limnaios et al	1980	1	Nil	Normal	Immediate	Partial
7	Arias et al	1985	1	Nil	Normal	Immediate	No
8	Dire et al	1987	1	Nil	Not done	Immediate	Not known
9	Ghorayeb et al	1987	3	Not known	B/L temporal bone fracture	Not known	Not known
10	Marconi et al	1994	1	Nil	Basal skull fracture	Immediate	Yes
11	Lazow et al	1995	1	Nil	Normal	Immediate	No
12	Lepore et al	1995	4	One patient U/L 3rd, 4th palsy	Not known	Not known	Not known
13	Celikoz et al	1996	1	Nil	Fronto-naso-ethmoidal fracture	Immediate	No
14	Mutyala et al	1996	13	Not known	Not known	Not known	12% patients
15	Lahbabi et al	1997	1	Nil	Normal	Immediate	No
16	Holmes et al	1998	8	Not known	Not known	Not known	38% patients
17	Mizushima et al	1998	1	Nil	Retro-clival extradural hematoma	Immediate	Yes
18	Holmes et al	2002	22	Not known	Not known	Not known	Not known
19	Lee et al	2002	1	Left 7th palsy	Fracture line extending from one temporal bone crossing to the contralateral temporal bone including clivus.	Immediate	Yes
20	Gaul et al	2002	1	Nil	Normal	Immediate	No
21	Advani et al	2003	1	Nil	Normal	Immediate	No
22	Dobbs et al	2003	1	Nil	Normal	Immediate	No
23	Binder et al	2004	1	B/L 4th palsy	Midline parietal depressed fracture obliterating flow in the subjacent sura-sagittal sinus	Delayed	Yes
24	Dwarakanath et al	2006	1	Nil	Pneumocephalus in the prepontine cistern. Right temporal and sphenoid sinus fracture extending into the petrous apex	Immediate	Yes
25	Calisaneller et al	2006	1	Left 7th, 12th palsy	Retro-clival extradural haemorrhage	Immediate	Yes

(Contd...)

Table 1: *(Continued)*

Sl. No.	Author	Year	No. of cases	Associated cranial nerve	Radiology of the cranium	Presentation	Recovery
26	Durkin et al	2006	29	Not known	Not known	Not known	Not known
27	Ruiz-de-Río et al	2006	1	Left 3rd palsy	Diffuse axonal injury	Immediate	No
28	Ratilal et al	2006	1	B/L 5th, left 12th palsy	Retro-clival extradural haemorrhage	Immediate	No
29	Katsuno et al	2007	1	Nil	Bilateral petrous bones and clivus fracture extending to the posterior clinoid process	Immediate	No
30	Schneck et al	2007	1	Nil	Pre-pontine extra-axial hematoma	Immediate	Yes
31	Kwon et al	2008	1	Right 9th, 12th palsy	Retro-clival extradural haemorrhage	Immediate	Yes
32	Taskin et al	2009	1	Nil	Normal	Immediate	Yes
33	Nayil et al	2010	1	Nil	Extradural hematoma in vertex with parietal bone fracture	Delayed	Yes
34	Pancko et al	2010	1	Left 7th palsy	Skull base fracture extending transversely across the petrous bone and right occipital bone, transverse fracture across the sphenoid sinus with extensive pneumocephalus	Immediate	No
35	Tubbs et al	2010	1	Nil	Retro-clival extradural haemorrhage	Immediate	Yes
36	Palmowski-Wolfe et al	2010	1	Nil	Small bleedings in the brainstem and in the left hemisphere	Delayed	No
37	Czyz et al	2011	1	Nil	Normal	Immediate	No
38	Yilmaz et al	2011	1	Right 7th, 9th, 12th palsy	Retro-clival extradural haemorrhage	Immediate	Yes
39	Lopes et al	2011	1	Nil	Avulsion of 6th nerve	Immediate	No
40	Salunke et al	2012	2	Nil	Case 1. Right convexity chronic Subdural hematoma Case 2. Normal	Delayed Delayed	Yes No
41	Yanamadala et al	2012	1	Nil	Normal	Immediate	Yes
42	Nicot et al	2012	1	Nil	Occipital, sellar and clival fractures with pneumatocephalus in interpeduncular cistern, intraventricular hemorrhage	Immediate	No
43	Selçuk et al	2013	1	U/L 12th palsy	Normal	Immediate	Yes
44	Fam et al	2015	1	Nil	Left temporal fracture with frontal and right corona radiata haemorrhagic contusion	Immediate	No
45	Orajiaka et al	2015	1	Nil	Bilateral temporal bone fractures	Immediate	No
46	Salunke et al	2016	1	B/L 7th palsy	Oblique fracture of the left petrous and right longitudinal petrous fracture extending into the temporal squama with pneumocephalus	Immediate	Yes
47	Paiva et al	2016	1	Right 7th palsy	Retroclival and parenchyma pneumocephalus, right temporal bone fracture	Immediate	No
48	Nguyen et al	2016	1	Nil	Retroclival hematoma in both the subdural and epidural space	Immediate	Yes
49	Ravindran et al	2017	2	Nil	Case1. Normal Case 2. Avulsion of 6th nerve	Delayed Immediate	No No
50	Serio et al	2019	1	Nil	Normal	Immediate	No
51	Sahlu et al	2020	1	Nil	Subacute retroclival subdural hemorrhage with left cerebellar and upper cervical spine extension	Immediate	No

with Grade 4 right facial nerve palsy according to House-Brackman grading. Rest of the neurological examination was normal. His ophthalmological examination revealed his uncorrected vision on bedside in the both eyes was finger counting at >3 meters, colour vision was normal in both eyes. Both anterior and posterior segment were normal with no evidence of disc oedema. Abduction testing revealed inability to abduct past the midline bilaterally compatible with -4 abduction deficits. He complained of diplopia on both left and right horizontal gaze.

The CT (Computed tomography) Brain showed an extensive pneumocepahalus with pneumoventricles with a linear undisplaced fracture noted in squamous part of left temporal bone extending into the petrous part of ipsilateral temporal bone along with linear undisplaced

fractures involving the body of sphenoid bone and linear undisplaced fracture noted in mastoid part of right temporal bone. Linear undisplaced fracture was also noted in the clivus [Figure 1]. Cervical spine CT screening was normal.

Patient was admitted in the ICU and treated with oxygen inhalation, head end elevation, antiepileptic and steroids. The serial CT scans of brain showed reduction in pneumocephalus and pneumoventricle. The MRI brain showed ventriculomegaly with pneumoventricle with no evidence of abducens nerve avulsion [Figure 2]. Patient was discharged and is on follow up. Eye signs have not improved on follow-up at 6 months, but facial palsy has improved to grade 2 according to House-Brackman grading.

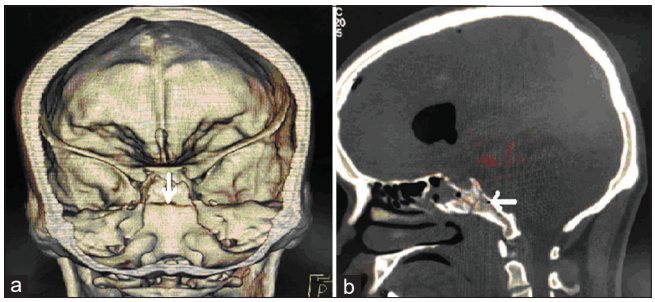

Figure 1: (a & b). Computed Tomography of skull showing linear undisplaced clival fracture

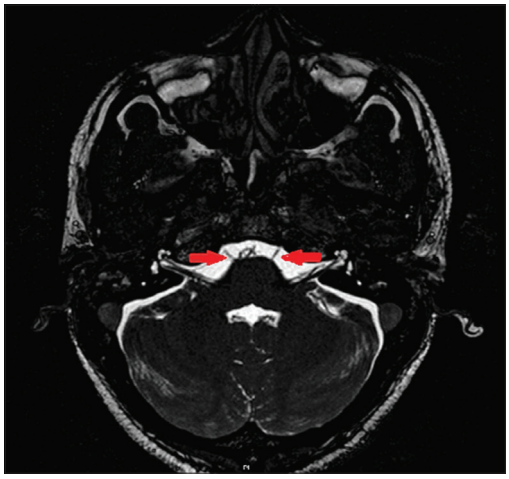

Figure 2: Magnetic resonance imaging showing B/L abducens nerve

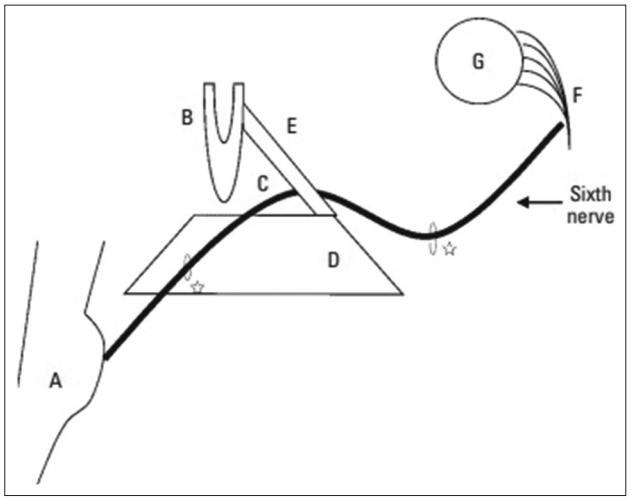

Figure 3: Schematic diagram of the course of the sixth cranial nerve from the pons (A) to the lateral rectus muscle (F). The nerve ascends over the petrous bone (D) and under Gruber's ligament (E) as it passes through Dorello's canal (C). The nerve is tethered by dura before and after the canal (stars). Injury to the peripheral nerve is thought to occur by contusion against the petrous ridge after midfrontal head impact. B, Posterior clinoidal process; G, globe

DISCUSSION

Anatomy of abducens nerve

Abducens nerve vulnerability in traumatic brain injury is due to its tortuous and lengthy intracranial course. Anatomically it is divided into 5 segments: brain stem, subarachnoid space, petrous apex, cavernous sinus and orbit.[4] Arising from the abducens nucleus in the floor of the fourth ventricle deep to the facial colliculus, nerve exits the brainstem at the pontomedullary junction, traversing anteriorly before ascending in the subarachnoid space posterior to the clivus. After approximately 1.5 cm, nerve ascends over the ridge of the petrous bone and later passes under the petroclinoid (Gruber's) ligament.[5] This triangular space demarcated by Gruber's ligament, the posterior clinoid process and the petrous apex is known as Dorello's canal.[6] After passing through the canal, the nerve enters the cavernous sinus and then passes through superior orbital fissure to supply the lateral rectus muscle. Occasionally, the nerve bifurcates as it traverses Dorello's canal with one trunk passing superior to Gruber's ligament and the other passing inferior to it [7] [Figure 3][8]. Bilateral abducens palsy following head injury is a rare but a well-described entity particularly when associated with skull base fractures. Several theories have been postulated to explain mechanisms of abducens nerve injury in trauma.

In 1971, Schneider et al., postulated that upward movement of the brain could result in compression of the abducens nerve against the rigid Gruber's ligament leading to avulsion of nerve.[9] However, a 1976 case series which included 3 cases by Takagi et al., disputed this hypothesis, arguing that the injury of the abducens nerve below Gruber's ligament by the dura and the petrous apex prevented the injury to the abducens nerve by upward cranial displacement. Rather, they hypothesized that the force directed in the mid-frontal bone resulted in stretching of both the abducens nerves with the apex of the petrous bone acting as a fulcrum for compression of the nerves.[10]

Indeed, in the absence of skull fractures, the mechanism proposed by Takagi et al., would appear most appropriate.[4] Following head injury with associated fracture of the skull base involving the clivus, clival epidural haematoma may occur. Epidural haematoma compresses the bilateral abducens nerve resulting in injury.[11] In 1985, Arias et al concluded that the points of entry of the nerve into both the extradural space and the petrous ridge are the most probable locations for the avulsion of abducens nerve.[1] Moreover, post-mortem analysis of ten post-traumatic bilateral abducens palsy cases at autopsy found injury to the nerve to be most evident at the sites of the dural entry point and petrous apex.[12] Keane et al., in 1976 suggested that the incidence of traumatic bilateral abducens palsy is similar to that of unilateral palsy. It has been postulated that the apparent paucity of reported bilateral cases is due to patients not surviving the initial trauma due to severity of injury.[13] A review by Arias et al., in 1985 identified 10 cases of traumatic bilateral abducens palsy, of which 3 had an associated cervical spine fracture without skull fracture.[1] Lazow et al., reported an additional three cases in 1995.[4]

Delayed presentation of post-traumatic bilateral abducens palsy is very rare with only six cases reported in the literature. The mechanism for a delayed presentation of bilateral abducens palsy is unclear. Many mechanisms have been proposed which includes vascular causes like vasospasm and ischemia. It is related to disruption of blood supply from branches of the meningodorsal artery and also tissue edema, similar to the mechanism of delayed onset traumatic 7th nerve palsy.[14,15]

Increase in size of post traumatic retroclival epidural haematoma, causes posterior displacement of brainstem leading to abnormal stretching of the bilateral abducens nerve anywhere along its course. Progressive edema may impinge on the sheath of the nerve, leading to axonal damage of the nerve.[16] Additionally, delayed intracranial haemorrhage has been postulated to be other mechanism, with asymptomatic time of up to 1 month reported in cases of delayed intra cranial haemorrhage.[17] Devin K. Binder et al., had reported a case with depressed skull fracture compressing superior sagittal sinus causing raised Intracranial pressure and bilateral abducens palsy on 4th day. Post operatively after removing the depressed fragments, the nerve palsy improved.[18] Nayil K et al., reported had reported a case with head injury with CT brain suggestive of vertex Extradural haemorrhage with delayed signs of raised intracranial pressure symptoms along with bilateral abducent nerve palsy. Post operatively abducens nerve palsy improved.[19] Elevated intracranial pressure is known to cause bilateral abducens palsy. This may be one of the mechanisms of delayed-onset bilateral abducens paresis following head injury. Thus, there may be other reasons of delayed-onset bilateral abducens nerve palsy apart from direct trauma to the nerve.[14] Ravindran et al postulated that partial avulsion of the abducens nerve, sustained at the time of initial trauma, followed by stretching or compression of the nerve by a combination of the mechanisms could result in delayed complete avulsion of the nerve.[20]

Out of 51 publications which include 139 cases, forty- one cases presented with immediate bilateral palsy, six cases were of delayed presentation while in the rest of the cases the time of presentation was not mentioned. In our case, patient presented with immediate onset of bilateral abducens palsy. On imaging clival fracture was seen in CT brain, which can be attributed for the nerve injury. The most common associated nerves involved along with bilateral abducens nerve injury are facial and hypoglossal nerves. [21-29]

Other nerves involved according to literature review were oculomotor, trochlear, trigeminal nerves.[18,30,31] Cases with retroclival extradural haematoma had higher chances of multiple cranial nerve injuries.[22-24,26] Cases with multiple basal skull fracture involving petrous temporal bone fracture had higher chances of facial nerve injury.[21,30,31] Our case presented with associated right facial nerve involvement which can be attributed to linear un-displaced fracture in mastoid part of right temporal bone.

We cannot comment on the rate of recovery in our review as different publications have chosen different time period for recovery. Few publications mention partial recovery at their follow-up. In our review, twenty-three patients show recovery while 29 patients did not recover irrespective of the follow up period mentioned by the authors. In rest of the cases, the recovery was not mentioned. Holmes et al.,[32] reported spontaneous recovery in 38% of bilateral traumatic abducens palsy at 3 months of follow-up. Mutyala et al.,[33] reported a recovery rate of 12% in the same. However, they did not mention the time period of follow -up. Unilateral 6th nerve palsy has been reported to recover spontaneously in 12 -73%.[32,33]

Our case showed no improvement in bilateral abduction during follow-up at 6 months, however facial nerve palsy improved to grade 2. Along with bilateral involvement, the poorer outcome can be related with the severity of the adduction deficit, with complete deficits having lower rates of spontaneous recovery.[34] Majority of the publications on recovery are limited by the small number of sample size. Cases of bilateral abducens nerve palsy following trauma are probably rare as the force required is usually not compatible with life. For the same reason, traumatic bilateral abducens nerve palsies are frequently accompanied with life-threatening injuries like skull base fracture.[8]

CONCLUSION

To summarize, clinical presentation of traumatic bilateral abducens nerve palsy is rare following closed head trauma and is usually associated with other injuries which are incompatible with life. Due to its long intracranial course, abducens nerve is vulnerable for injury and various mechanisms have been described separately for immediate and delayed presentation. It can be associated with other nerve injuries depending on basal skull fractures. Severity of abduction deficits and bilateral nerve injury are independent risk factors for poor outcome.

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Author's Contribution:
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