

Complete ophthalmoplegia associated with clival fracture following trauma: Case report and clinico-anatomical correlation

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Abstract: Fractures of clivus occur following high energy trauma and are rare. There are a few reports of associated vascular and brainstem injury in literature. We report a patient with transverse clivus fracture and complete ophthalmoplegia and bilateral multiple cranial nerve palsies. A young boy was admitted in altered sensorium following RTA. Initial imaging revealed diffuse pneumocephalus, subarachnoid hemorrhage and diffuse cerebral edema. He was treated conservatively. Clinical review after improvement in sensorium showed that he had visual loss in left eye, bilateral ptosis, complete ophthalmoplegia in horizontal as well as vertical gaze, bilateral trigeminal paresis, right facial weakness and dysarthria. There was right CSF otorrhea. A spiral CT scan revealed transverse clivus fracture extending into bilateral middle cranial base. The fracture line was involving facial canal and mastoid on the right side. An MRI brain demonstrated periaqueductal infarcts and multiple cortical infarcts. He was managed conservatively. In the presence of clinical suspicion of skull base injuries, a spiral CT scan and MRI brain and MR angiography are essential for accurate assessment of bony and neurological involvement. The diagnosis of clivus fracture should prompt the clinician to look for associated brainstem or basilar artery injury.

Keywords: Clivus fracture, post-traumatic ophthalmoplegia, post-traumatic cranial nerve palsy, midbrain injury, post-traumatic cortical infarcts.

INTRODUCTION

Skull base fractures occur following high-energy trauma and are often under-diagnosed. Clivus fractures are rare following trauma and have no specific clinical features to raise a suspicion. An association of basilar artery injury¹ and bilateral internuclear ophthalmoplegia² with clival fractures have been reported. We report a case of complete ophthalmoplegia and multiple cranial nerve palsies associated with transverse clival fracture following trauma.

CASE REPORT

An 18 year-old-boy was admitted in the hospital after sustaining injury to the head in an RTA. He fell down from the top of a van, which was reversing, and his head was crushed between the rear of the van and a wall. On

examination in casualty three hours following trauma, the Glasgow coma score was E2M5V2. Pupils were 5 mm dilated and were not reacting to the light, and there was no focal motor deficit. There was bleeding from both the ears. Other systemic examination was normal. CT scan done at three hours following trauma revealed diffuse pneumocephalus with extension into lateral ventricles. There was subarachnoid hemorrhage in the right cerebellopontine (CP) angle cistern, and moderate diffuse cerebral edema. Bone windows revealed linear fracture of the left anterior cranial fossa base and the roof of right orbit (Fig 1).

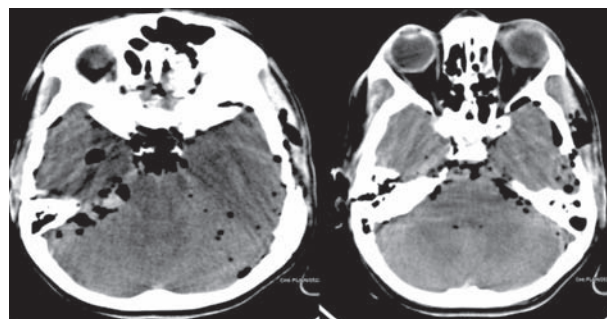


Fig 1: CT scan of brain 3 hrs following trauma shows diffuse pneumocephalus and subarachnoid hemorrhage in the right CP angle cistern.

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He was managed conservatively with anti-edema measures, antiepileptics and antibiotics. His sensorium improved over the next 24 hours and he started obeying commands. A review of his clinical condition revealed that vision in his left eye was poor with finger counting at 4 feet. Visual fields could not be assessed accurately. He had bilateral ptosis and pupillary dilatation. There was complete bilateral ophthalmoplegia, with both horizontal and vertical gaze palsy in all directions. Doll's eye movements and convergence were absent. Other cranial nerve examination revealed bilateral absent corneal reflex, bilateral facial sensory loss involving ophthalmic and maxillary distribution, and weakness of left masseter and temporalis with deviation of the jaw to the left on mouth opening. He also had right lower motor neuron facial weakness. Hearing could not be accurately assessed. He had a hoarse dysarthric voice. The uvula was central and the palatal movements were normal. There was a suggestion of left hypoglossal paresis with deviation of tongue to left on protrusion and mild weakness of tongue muscles on the left. There was no limb weakness. The deep tendon reflexes were normal and the plantars were flexor. He also had right CSF otorrhoea.

In view of the extensive cranial nerve involvement, a spiral CT with reconstruction was performed to look for skull base involvement. An MRI of the brain was done to look for brainstem involvement in view of bilateral complete ophthalmoplegia. The spiral CT of the brain revealed a linear hyperdensity in the ventral midbrain, subarachnoid hemorrhage in bilateral cerebellopontine (CP) cisterns and prepontine cisterns. There were diffuse pneumocephalus in the frontal region, sylvian fissure, bilateral frontal and temporal horns with evidence of pockets of air within the parenchyma of the right temporal, insular and bilateral frontal regions. There were also wedge shaped well-defined hypodensities involving the left parietal and right parieto-occipital regions. Bone windows revealed a vertical fracture oriented in coronal direction involving the right squamous and petrous temporal bone extending to mastoid at the level of the middle ear. Clivus showed a transverse fracture starting at the junction of right petrous apex, running along its entire width, disconnecting it from the sphenoid bone through the sphenoid occipital synchondrosis, extending to the left side involving the greater wing of sphenoid and causing displaced horizontal fracture of the left squamous temporal bone. The lesser wing of sphenoid on the left also showed a horizontal fracture

involving the anterior clinoid and planum sphenoidale. There was also soft tissue edema and air in bilateral retropharyngeal space (Fig 2).

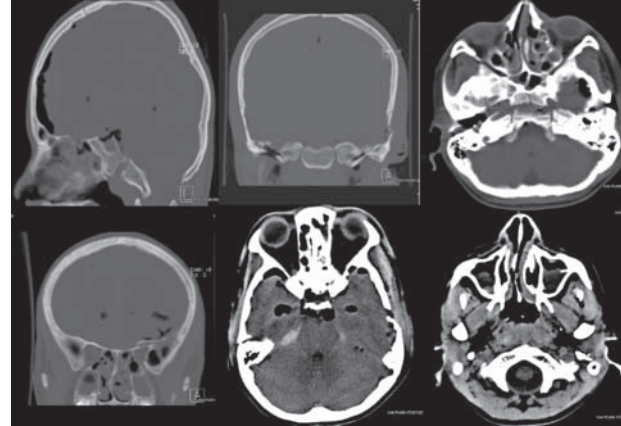


Fig 2: A spiral CT scan 24 hrs following trauma shows a transverse clivus fracture and extension into both petrous temporal bone. A linear fracture involving lesser wing of sphenoid was also seen. Parenchymal windows reveal air in para- and retropharyngeal space. Pneumocephalus shows resolution.

MRI of the brain revealed infarcts in the periaqueductal grey matter of the midbrain, multiple areas of wedge shaped cortical infarcts in the right frontal, bilateral parietal and right occipital regions. Pneumocephalus showed significant resolution (Fig 3).

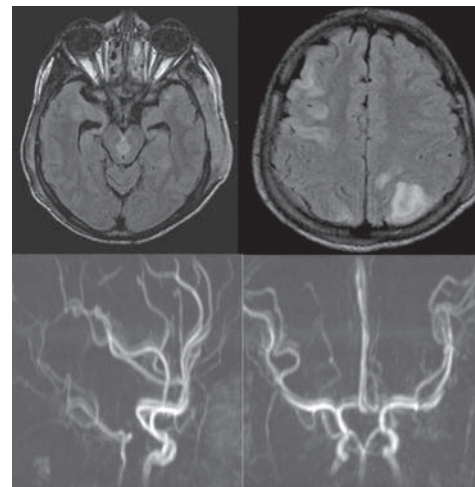


Fig 3: MRI brain (FLAIR) images reveal hyperintense signal changes in the periaqueductal grey matter and right frontal and left parietal regions. MR angiography did not reveal any abnormality.

Patient was managed conservatively. CSF otorrhoea subsided, and at the time of discharge after one week, his eye movements showed some improvement. He was

tolerating semi solid foods administered under supervision. His vision and ptosis remained static. He was advised regular follow up and assessment.

DISCUSSION

The clivus, defined by the basioccipital bone and body of the sphenoid bone, extending from the tuberculum sellae to the foramen magnum, is located deep at the anatomical center of the skull base. The fractures of clivus have been underdiagnosed and underreported. These fractures often resulting following high energy trauma, have been classified into transverse, longitudinal and oblique based on the radiological findings³. Transverse fracture of clivus occurs following lateral crush injury of the head, as in our patient, when the direction of the force runs along the speno-occipital synchondrosis⁴. Interestingly, our patient sustained a low velocity lateral crushing injury which has resulted in multiple cranial nerve palsies with preserved sensorium. Longitudinal fractures are caused by various mechanisms, including forces transmitted along the vertebral column axially.

Spiral CT imaging with bone windows and reconstruction accurately defines the type and extent of the fracture which can be missed in routine axial CT imaging. In the present case, the transverse clival fracture was not identified in the initial CT scan. In the presence of clinical features suggesting brain stem involvement due to mechanical or vascular cause, MRI of the brain and MR angiography (MRA) would be helpful. MRI diagnosis of retroclival epidural hematoma secondary to clival fracture^{5,6} and basilar artery injury¹ have been reported in the literature. In our patient, MRA did not reveal any abnormality of the basilar artery.

In the present case, the various neurological deficits can be explained by the radiological findings. The right-sided facial palsy and CSF otorrhoea occurred as a result of the right petrous fracture involving the facial canal and the mastoid. The extension of the transverse clival fracture along the petrous apex region explains the left trigeminal nerve involvement, probably at the Meckel's cave. The left sided fracture has also extended to the region of the optic canal, probably causing optic nerve injury, which resulted in visual deterioration in the left eye. The cause of dysarthria in our patient was probably because of pharyngeal edema due to local injury. Khan et al⁷ reported a case of post traumatic transverse clival fracture resulting in unilateral CN III and VII palsy and bilateral abducens palsy. Sharma et al⁸ reported a case of

basilar fracture resulting in ipsilateral palsies of cranial nerves IX to XII.

The complete ophthalmoplegia in the patient is probably the result of the midbrain injury around the periaqueductal grey matter, which is evident from the MRI, affecting both the vertical and horizontal gaze fibres. A similar picture can be theoretically caused by the involvement of the CN III, IV, and VI bilaterally at the cavernous sinus region. But there were no signs of cavernous sinus involvement in this patient, either clinically or radiologically. Therefore, we feel that the complete ophthalmoplegia occurred as a result of brainstem trauma. Oddly, this has not obtunded his sensorium significantly. The radiological features of the periaqueductal grey matter involvement probably suggest ischemic injury due to the shearing force on the perforators of basilar artery supplying the midbrain. Bonilha et al² had suggested probable involvement of medial longitudinal fasciculus resulting in bilateral internuclear ophthalmoplegia.

MRI brain also demonstrated multiple cortical infarcts in this patient which are probably of embolic etiology. The exact cause of embolism involving multiple vascular territories was not clear. It is possible that diffuse pneumocephalus in the brain has resulted in multiple air emboli of the brain resulting in cortical infarcts. The other possibility of bilateral internal carotid artery dissection at the level of cavernous sinus due to the fracture resulting in multiple distal embolism can be considered. But the MR angiography did not reveal any abnormality in the vessel caliber.

CONCLUSIONS

This report highlights the rare occurrence of post-traumatic complete ophthalmoplegia in association with transverse clival fracture. In the presence of clinical suspicion of skull base injuries, a spiral CT scan and MRI brain and MR angiography are essential for accurate assessment of bony and neurological involvement. The diagnosis of clivus fracture should prompt the clinician to look for associated brainstem or basilar artery injury.

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