Transverse fracture of the clivus

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Three young patients with transverse fracture of the clivus exhibited clinical findings of progressive cranial nerve palsies associated with a Horner's syndrome. Clinical and radiological findings of this syndrome are described.

Key Words: skull fracture, cranial nerve injury, clivus, carotid-cavernous fistula, Horner's syndrome.

Although there are clinical and roentgenographic findings that indicate the presence of a basilar skull fracture, the exact location and extent of the fracture are difficult to define prior to autopsy. In recent years tomography has been helpful in demonstrating these fractures suspected upon clinical examination. Longitudinal fractures of the clivus have been reported in which there was entrapment of the vertebral or basilar artery. These fractures per se were not demonstrated before death. Robinson, et al., and Reynolds describe cases in which sphenoid sinus effusion indicated the presence of a fracture at the base of the skull. However, none of their cases had a fracture through the clivus.

The three patients we are reporting were admitted to the Denver General Hospital with progressive cranial nerve palsies and a Horner's syndrome associated with transverse fracture of the clivus.

Case Reports

Case 1

This 16-year-old girl was involved in an automobile accident while a front-seat passenger. Her only immediate neurological deficit was a right 6th nerve palsy, but over a period of 7 days she developed a right 5th and 7th cranial nerve deficit and a right Horner's syndrome. Throughout this period she was conscious, cooperative, and well-oriented, and there were no signs of increased intracranial pressure. Initial skull films demonstrated an unusual spicule of bone situated behind the dorsum sellae and a fracture at the apex of the right temporal bone (Fig. 1 left). The sphenoid sinus was partially opacified but there was no true air fluid level. Hypocycloidal tomograms of the sellar region and temporal bones demonstrated the fracture at the apex of the petrous portion of the right temporal bone, the previously mentioned spicule of bone, and a transverse fracture through the clivus (Fig. 1 right). Cerebral angiography demonstrated an irregular lumen in the right internal carotid artery in the precavernous and cavernous portions (Fig. 2). The cavernous sinus filled during the venous phase and drained anteriorly by orbital veins. Within 2 days following arteriography the patient developed signs and symptoms of a carotid cavernous fistula, which was
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Fig. 1. Case 1. Left: Lateral skull film demonstrating spicule of bone (arrow). Right: Lateral tomogram of the sella revealing fracture through the clivus (arrows).

This 26-year-old man was involved in an automobile accident. He was initially unconscious for some minutes and arrived at the hospital disoriented and combative. Three days following the injury he developed a left 6th and 7th cranial nerve deficit and a left Horner's syndrome, and on the treated by ligation of the right carotid artery in the neck. This patient recovered with only residual right 5th and 6th cranial nerve palsies. Six months later she was killed in an auto-pedestrian accident. Autopsy revealed a healing fracture of the clivus and right temporal apex plus an atrophic right internal carotid artery and ossification of the right cavernous sinus.

Case 2

Fig. 2. Case 1. Lateral angiogram (subtraction) showing damage to the precavernous and cavernous portion of the right internal carotid artery (arrow).
Fig. 3. Case 2. Lateral skull film demonstrating air at base of the brain and in the frontal area, an air-fluid level in the sphenoid sinus, and an obscure fracture of the clivus (arrow).

following day a right 7th nerve palsy. Skull films demonstrated a sphenoid sinus effusion and fracture of the squamous portion of the left temporal bone. A fracture through the clivus was suspected from the lateral skull films, which showed a lucent line through the clivus and abnormal soft tissue density in the posterior nasal pharynx (Fig. 3). This fracture was confirmed by lateral hypocycloidal tomography. Total cerebral angiography was normal except for extravasation of a small amount of contrast material from an intracavernous branch of the left internal carotid artery. The basilar artery appeared normal but the dural veins of the clivus suggested a few millimeters of posterior displacement. This patient has made a complete recovery.

Case 3

This 9-year-old girl was involved in an auto-pedestrian accident. On arrival at the hospital she was comatose; her musculature was flaccid and areflexic. The right pupil was larger then the left, and both were fixed to light. Initial carotid arteriography showed no mass lesion and skull films demonstrated a probable fracture through the sphenopituitary synchondrosis of the clivus (Fig. 4). Within one day the patient’s level of consciousness had improved to stupor, and she exhibited paralysis of the right 3rd, 4th, 5th, and 6th cranial nerves as well as a right hemiparesis. A bruit became audible over the right eye as well as over the right side of the neck, and carotid arteriography was again performed. Stenotic lesions were demonstrated in many areas within the right internal carotid artery. One week following injury the patient became alert and began to speak. Examination at this time showed paralysis of the soft palate on the right side and bilateral 7th nerve paralysis in addition to the cranial nerve paralyses. Within 2 weeks the 3rd nerve paralysis had improved, and it was possible to demonstrate a right Horner’s syndrome using chemical agents. The patient then gradually returned to normal except for residual right 4th, 6th, and 9th cranial nerve palsies and a left 6th nerve paralysis.

Discussion

These three patients were admitted with progressive cranial nerve palsies; common to all were 6th and 7th nerve palsies and a Horner’s syndrome (Table 1). In the third patient, who had a right 3rd nerve palsy, the Horner’s syndrome did not become apparent until the 3rd nerve palsy began to improve. In all three patients the facial nerve paralysis was delayed in onset and in two cases involved both sides of the face; in all patients the facial weakness cleared completely. All cranial nerve palsies that were delayed in onset proved to have a good prognosis. Two of the patients also had a temporary conductive hearing loss that was bilateral in one case.

All patients had some degree of injury to the internal carotid artery. The only clinical indication of this injury was the Horner’s syndrome, which was thought to be secondary to a lesion of the pericarotid sympathetic nerves. Two patients developed bruits over the eye on the side of the cranial nerve palsies; in one this was felt to be secondary to internal carotid artery stenosis.
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and in the other to a carotid-cavernous fistula. Although the mechanism of delayed cranial nerve palsies is unknown, we speculate that, because of the common denominator of arterial injury, small expanding hematomas could cause compression or ischemia of adjacent cranial nerves. The displacement of the internal carotid artery at the base of the skull in Case 1 supports this hematoa theory. The delayed 7th nerve palsies can probably be explained on the basis of the 7th nerve in its tight bony canal.

These patients all had a particular type of basilar skull fracture, namely, a transverse fracture of the clivus. In Case 3, that of a 9-year-old child, the saphene-occipital synchondrosis was still open and, therefore, a diastatic sutural fracture resulted. This suture should be 3 mm in a 9-year-old child.1

The skull films combined with clinical

TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Cranial Nerves Deficit</th>
<th>Horner's Syndrome</th>
<th>Carotid-Cavernous Fistula</th>
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<tr>
<td>1</td>
<td>16</td>
<td>F</td>
<td>rt 5th, 6th and 7th</td>
<td>rt</td>
<td>rt</td>
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<tr>
<td>2</td>
<td>26</td>
<td>M</td>
<td>lt 6th, bilateral 7th</td>
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</tr>
<tr>
<td>3</td>
<td>9</td>
<td>F</td>
<td>rt 3rd, 4th, 5th,</td>
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signs and symptoms should lead the radiologist or clinician to suspect this fracture and to resolve the question using tomography. Our experience with these cases suggests that a fracture of the clivus should be considered in instances of unexplained sphenoid sinus effusion or soft tissue density. Moreover, unusual spicules of bone posterior to the clivus may result from an avulsion of the petrous apex rather than the more common calcification in the petroclinoid ligament, particularly in younger patients.

The mechanism of injury producing this fracture remains unclear. The clivus has long been recognized as the strongest buttress at the base of the skull. Whether this fracture is due to some unique injury related to high-speed vehicular trauma, or one that has occurred frequently in the past and now is being demonstrated by more sophisticated roentgenographic techniques, are unanswered questions. The fact that these three patients were seen within a 6-month period supports the latter suggestion.

References
1. Irwin GL: Roentgen determination of the time of closure of the sphenoid sinus. Radiology 75:450-453, 1960

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