Epidural hematoma of the clivus

Case report

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This 8-year-old boy presented with a rare case of epidural hematoma of the clivus and atlantoaxial dislocation caused by a hyperflexion injury sustained in a traffic accident. Magnetic resonance (MR) imaging demonstrated an epidural hematoma in the posterior fossa that compressed the pons and medulla. On admission, the patient was confused and had bilateral abducens palsy. He was treated conservatively, and 6 months after admission, the epidural hematoma on the clivus had disappeared on MR imaging and the bilateral abducens palsy was cured. Only two such cases have been reported in the literature. In this report, the authors discuss the mechanism of hematoma formation in this region of the brain.

KEY WORDS • clivus • epidural hematoma • hyperflexion injury

EPIDURAL hematomas in the posterior fossa are rare and patients surviving with these lesions are even more uncommon. These hematomas are often sustained during hyperextension or hyperflexion injuries. Lesions in the posterior fossa, including the clivus, cannot always be diagnosed easily on computerized tomography (CT) scans because of artifacts caused by the cranial bones. In our experience, thin-slice CT scanning has been required for diagnosis in these cases. Magnetic resonance (MR) imaging is the best choice for verifying this disease because it facilitates understanding of the relationship between the hematoma, midbrain, and vascular system.

Case Report

History and Examination. This 8-year-old boy was hit by a small truck while walking across a street on July 4, 1996. The truck’s left side door hit the boy’s right temporal bone and he fell to the ground. Thirty minutes after the accident, he was brought to the emergency room of our hospital. On admission, he showed signs of confusion accompanied by nausea and vomiting. A slight paresis of both upper extremities was noted. His consciousness level was assessed as 6 (E1V1M4) on the Glasgow Coma Scale (GCS). Physical examination revealed right-sided Battle’s sign at the posterior auricular lesion, bilateral abducens palsy, and no anisocoria. His blood pressure was 124/78 mm Hg. Brain CT scanning demonstrated subarachnoid blood in the cerebellopontine cistern, but no epidural hematoma and no contusion (Fig. 1). Magnetic resonance studies, which were obtained at the same time as the CT scans, revealed that the mass lesion was located mainly in the left clivus and compressed the brainstem posteriorly. The mass was isointense on T1- and hyperintense on T2-weighted images (Fig. 2). Skull x-ray films showed no fracture, and no abnormalities were noted in the chest and abdomen. The results of the patient’s blood workup were normal except for a finding of leukocytosis. He was initially treated conservatively for a traumatic subarachnoid hemorrhage. One day after admission, repeated CT scanning revealed intraventricular hemorrhage in the fourth ventricle and disappearance of subarachnoid hemorrhage in the cerebellopontine cistern. However, we could not discern the epidural hematoma in the posterior fossa on CT scans because of cranial bone artifacts.

Clinical Course. Two days after admission, the patient’s GCS score had improved to 8 (EVTM). Computerized tomography scanning showed a slight left-to-right shift in the fourth ventricle and a slightly high density mass in the left cerebellopontine angle cistern. However, we were not able to make a diagnosis based on these findings because of motion and bone artifacts. Four days after admission, a lumbar puncture was performed and the fluid was found to be bloody, with a pressure reading of 130 mm Hg. Electroencephalography revealed high-amplitude and generalized slow waves (delta waves). Repeated CT scanning
first demonstrated an epidural hematoma extending from the ventral pons to the lower medulla on July 9 (5 days after admission); the epidural hematoma was shown to be increased in size (4 × 2 × 3 cm) and the fourth ventricle had disappeared (Fig. 3). However, there was no sign of hydrocephalus and the patient’s respiration was not disturbed. As his consciousness level gradually improved, nasal feeding tubes were placed.

Additional Imaging Studies. On July 12, the patient was transported to the university hospital to undergo digital subtraction angiography. Vertebral angiograms revealed a dorsal shift from the vertebral artery (VA) to the basilar artery (BA), especially on the left angiogram. There was no dissecting aneurysm or pseudoaneurysm (Fig. 4). Magnetic resonance images obtained on the same day demonstrated an epidural hematoma on the clivus displaying a low isointense signal on T₁- and an isointense signal on T₂-weighted images. The pontomedullary junction was shown to be especially compressed by the hematoma on the sagittal section of the MR image (Fig. 5). In the x-ray study of the neck, the tip of the odontoid process was dislocated posteriorly and the atlantoodontoid distance was 4 mm within the normal limit, but the distance between the posterior atlantal arch and the odontoid was widened (Fig. 6). Because his neurological signs did not worsen and he wore a cervical collar for atlantoaxial dislocation, we continued to manage this patient conservatively.

Outcome. Repeated MR imaging performed on August 14 revealed that the epidural hematoma had disappeared. The patient’s neurological symptoms began to improve during this period and only left abducens palsy remained when he returned to our hospital on August 19. Twenty-four weeks after admission, his left abducens palsy had vanished; MR images obtained at that time showed no epidural hematoma or contusion (Fig. 7), and MR angiography revealed that the dorsal shift from VA to BA had improved.

Discussion
Epidural hematoma in the posterior fossa was first reported as a disease entity by Coleman and Thomson in 1941. Many case reports in which this entity is described have been published since the advent of CT scanning tech-
According to Hooper, Jamieson and Yelland, McKissock, et al., and Cordobés, et al., posterior fossa epidural hematomas constitute 11%, 7%, 4%, and 1.2%, respectively, of all epidural hematomas. Often, head injury from a blow to the occipital region causes an epidural hematoma of the posterior fossa and there is an 85% incidence of occipital bone fractures. The bleeding may arise from the transverse sinus, transverse confluence, and occipital sinus, followed by a tear in the venous system when the components of the occipital bone fracture compress the venous sinus. Bleeding sometimes may occur without fractures when the injury transiently deforms the occipital bone. Epidural hematomas can occur at any location supra- or infratentorially, including the clivus. Most cases are caused by coup injuries. In our literature review, however, the traumatic mechanism consisted of hyperflexion injuries in two cases and hyperextension in one (Table 1). The mechanism for the formation of epidural hematomas of the clivus remains controversial.

Occipital bone originates from two different components: membranous and cartilaginous. Bone constituting the posterior fossa originates from the latter, and the transverse sinus region is thought to be the border between the two components. This possible mechanism may result in a sinus injury caused by traumatic detachment of the dura mater from the occipital bone when the injury transiently deforms this bone. Furthermore, the dura mater is more easily displaced from the skull in children.

Anatomically, the odontoid process is maintained in relation to the anterior arch of the atlas by ligamentous and membranous tissues. The tectorial membrane is the upward continuation of the posterior longitudinal ligament. This membrane passes superiorly over the cruciform ligament of the atlas and continues anteriorly, covering the inferior aspect of the clivus. Anterior or posterior dislocation of the odontoid process with rupture of the transverse ligament of the atlas would strip this membrane from the inferior aspect of the clivus; in young patients particularly, this dissection can extend to the level of the sella turcica. This would result in venous bleeding and subsequent compression of the brainstem from an extradural hematoma on the clivus.

In Case 2 (Table 1), the patient exhibited diastasis of the sphenoccipital synchondrosis, which was wider than usual for her age. The possible cause of an epidural hematoma on the clivus includes bleeding from the injured dura caused by fracture of the clivus or the skull base surrounding it. In our case, the cause of an epidural hematoma on the clivus may include these mechanisms. According to the literature, two cases similar to ours have been reported. All injuries were sustained in motor vehicle accidents and all those injured were children (Table 1). Each patient’s GCS score on admission was...
between 3 and 7 points. However, their prognoses were relatively good unless they had suffered severe damage to other organs in addition to the clivus. Conservative therapy was chosen in two cases and yielded good results. Pareses of both upper extremities, which might have been caused by central spinal cord syndrome, were noted in two patients (Cases 2 and 3). In Case 2, myelography and MR imaging were performed, good passage of the contrast medium was observed, and there were no signs of cord compression. Magnetic resonance imaging revealed a high-intensity area located between the lower cervical and the upper thoracic vertebrae. Unfortunately we did not perform cervical MR imaging, but we did not observe central cord syndrome from C1–5 on the brain MR image. It is important to remember that head and neck injuries can cause epidural hematomas on the clivus. Magnetic resonance imaging provides a better diagnostic modality than CT scanning because it is not affected by bone artifacts.

References


Fig. 7. Sagittal MR image obtained 6 months after the onset of symptoms demonstrating that the epidural hematoma had disappeared.

TABLE 1

<table>
<thead>
<tr>
<th>Authors</th>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Cause</th>
<th>GCS</th>
<th>Complications</th>
<th>GOS</th>
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<td>Orrison, et al.</td>
<td>1</td>
<td>8</td>
<td>M</td>
<td>MVA (hyperflexion)</td>
<td>3</td>
<td>liver damage, acute death</td>
<td>EDH (parietal)</td>
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<td>Kurosu, et al.</td>
<td>2</td>
<td>11</td>
<td>F</td>
<td>MVA (hyperextension)</td>
<td>7</td>
<td>leg fracture, central cord syndrome (lower cervical)</td>
<td>GR</td>
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<tr>
<td>Mizushima, et al. (present study)</td>
<td>3</td>
<td>7</td>
<td>M</td>
<td>MVA (hyperflexion)</td>
<td>6</td>
<td>none</td>
<td>GR</td>
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