Epidural hygroma of the posterior fossa

Case report

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A case of posttraumatic epidural hygroma is reported. This child presented with occipital injury. Slowly but progressively he developed bilateral Wallenberg's syndrome and retrocollis. Correct preoperative diagnosis was made with the aid of computerized tomography. The case is discussed with emphasis on the mechanism of formation of an epidural hygroma and its clinical presentation.

KEY WORDS □ epidural hygroma □ epidural hematoma □ Wallenberg's syndrome □ retrocollis □ head injury

Since Payr's original description of posttraumatic acute subdural hygromas, including one of the posterior fossa, there has been an increasing awareness of the syndrome; yet, cerebellar subdural hygroma has only rarely been reported. No previous case of epidural hygroma has been reported in the literature. Recently, we had an opportunity to treat a patient with this entity, who presented with occipital head injury and a bizarre clinical picture. He made a complete recovery following evacuation of the epidural hygroma and an insignificant amount of associated hematoma.

Case Report

This 2-year-old boy was admitted to our unit on August 30, 1981, having sustained head injury 20 minutes earlier. While at play, he fell down a slide approximately 5 feet high and landed on the back of his head and shoulders. He immediately became unconscious and flaccid. Flaccidity soon disappeared, and he started to move.

Examination. On initial examination the child was unconscious, moving limbs spontaneously and warding off painful stimuli. Both pupils were equal, 3 mm in size, and reacted to both direct and consensual light. There was a small subgaleal hematoma over the occiput extending to the right side. He resisted movement of the neck. Pulse rate was 120/min, blood pressure 160/70 mm Hg, respiratory rate 24/min, and rectal temperature 39.5°C. During the next few hours, the child regained consciousness. He kept both eyes open spontaneously, and occasionally followed toys and objects with them. Blink reflex and extraocular movements were normal. He had no dysconjugate eye movement, skew deviation, or nystagmus. There were no other abnormal neurological signs. Within 24 hours, the temperature settled down to 37°C, and the pulse rate was reduced to 100/min. He appeared to make slow but progressive improvement.

At 3 p.m. on September 2, 1981, it was noted that the patient kept his neck extended. There were no localizing signs. Traumatic subarachnoid hemorrhage or aseptic meningitis was suspected, and observation continued. Computerized tomography (CT) could not be performed due to technical difficulties. By the next day, the child showed bilateral lower motor neuron facial paresis, bilateral blunting of the corneal reflexes, regurgitation of food through the nose, choking, bilateral hypalgesia of the limbs and trunk, and marked neck retraction (retrocollis). There were no other neurological changes; in particular there was no decerebration, decortication, deterioration of the level of consciousness, pupillary changes, or changes in cardiac or respiratory rate and rhythm. Now the clinical picture was strongly suggestive of a bilateral Wallenberg's syndrome plus retrocollis.

Hemoglobin was 12.7 gm/dl; serum electrolytes and urea were within normal range. Serum calcium was 2.5 mmol/liter and blood glucose 4.2 mmol/liter. X-ray films of the skull, including Towne's view, did
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FIG. 1. Preoperative computerized tomography scans. Left: Scan showing a linear fracture of the right occipital bone, passing into the foramen magnum. Center: An extradural collection of fluid, with a density ranging from 0 to 4 Hounsfield units, is seen. The average densities of the membrane were in the region of 45 units. Right: A small extradural blood clot, with densities varying from 40 to 65 Hounsfield units, is visualized.

not reveal a fracture line, and films of the cervical spine and chest were also normal.

A CT scan revealed a fracture of the right occipital bone extending into the foramen magnum, and a lesion was seen occupying the posterior cranial fossa (Fig. 1 left). This lesion was clearly extradural, and pushed the cerebellum forward and somewhat to the left, causing a shift of the fourth ventricle (Fig. 1 center). Attenuation values varied from 0 to 4 Hounsfield units in most parts of the lesion (Fig. 1 center) to 40 to 65 units in others (Fig. 1 right). The anterior limit of this lesion was formed by a membrane with an average density of 45 units. Briefly, it appeared to be a large cerebrospinal fluid (CSF) collection, in association with a small blood clot in the epidural space.

Operation. Immediately following CT scanning, a small suboccipital craniectomy was performed through a right paramedial skin incision. On retraction of the muscles, a hairline fracture of the right occipital bone was identified, and CSF started to leak through the fracture. A burr hole was then drilled. As soon as the inner table was penetrated, clear fluid resembling CSF gushed out under very high pressure. A finger was immediately placed in the burr hole to permit slow decompression. Fluid (60 ml) was collected for various laboratory examinations, and at least the same amount escaped or was removed by suction. The burr hole was enlarged, and a blood clot, approximately 30 ml in volume, was found lying in the epidural space. From the midline it extended in the epidural space over the left cerebellar hemisphere. The clot lay mainly below the lateral sinus, while straddling it. The clot was evacuated, and the sinus underneath was found intact. Blood escaped from an emissary vein, which was coagulated. On close inspection, a 3-mm long tear was found in the dura of the right half of the cisterna magna. The tear was roughly midway between the fracture and the blood clot. Through this tear, the CSF leaked out with every pulsation of the brain. The dura was closed with a single silk stitch. Since there was a large epidural dead space, a vacuum drain was inserted and the wound closed in layers.

Postoperative Course. During the first 24 hours, the retracted position of the neck resolved, and the child looked generally well. The Hemovac drain was removed, having accumulated 45 ml of blood-stained fluid. Biochemical analysis of the fluid from the epidural space revealed glucose 3.6 mmol/liter, protein 0.2 gm/liter, and chloride 118 mmol/liter. Thus, the fluid closely resembled CSF. During the first 48 hours there was marked improvement. Pulse rate became steady at 85/min, neck stiffness disappeared, and the child started to play with toys and responded to his parents. Feeding as well as crying became normal.

Five days postoperatively the child began to vomit and showed slight evidence of neck stiffness. Inspection of the wound revealed that the craniectomy site was bulging. By needle aspiration 80 ml of clear fluid was withdrawn, followed by complete amelioration of symptoms within 24 hours. Subsequently, aspiration was required twice more, on the 8th and the 12th postoperative days. Since then, the child has remained perfectly well and is being seen regularly in our outpatient clinic.

Discussion

Meninges are torn in approximately one-half of patients with depressed skull fractures. The meninges
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are deliberately opened in almost every case requiring exploratory burr hole; yet, a collection of CSF under pressure in the epidural space has not been reported previously. Fluid will not collect into the epidural space if the arachnoid mater remains intact. Neither will it collect if the tear in the arachnoid and dura is large enough to permit free flow of CSF in either direction, or if the torn meninges remain firmly in contact with the inner table of the skull. Clearly, the present case failed to comply with the above criteria.

From the information available on our patient, it is possible to reconstruct the mechanics whereby the CSF accumulated under pressure in the epidural space. A small epidural blood clot may have initially stripped the dura off the bone, allowing CSF to leak out through a small tear in the meninges which, somehow, acted as a one-way valve, a mechanism akin to the formation of a subdural hygroma. It is generally accepted that, with rising intracranial pressure, the brain will eventually plug a small tear in the dura. Unfortunately, in this case the dura was torn over the cisterna magna, and consequently large amounts of CSF escaped. The presence of only a hairline fracture and intact pericranium may have contributed by preventing the escape of CSF into the extracranial tissues.

Clinically, the patient presented with slight neck stiffness which later developed into a frank retrocollis. Neck stiffness and torticollis are well known presentations of a space-occupying lesion of the posterior cranial fossa. The mechanism is believed to be due to a reflex action to guard against movement, and is probably related to tonsillar herniation for the former, and due to interference with labyrinthine pathways affecting the relationship between muscle tone and the position of the head for the latter. The mechanism of retrocollis appears to be entirely different. A diffuse epidural space-occupying lesion of the posterior cranial fossa will push the cerebellum and the brain stem forward and upward, producing a reversed tentorial herniation and thus ischemia of the brain stem. Neck retraction thus produced is associated with decerebrate rigidity. More appropriately, Connor's experimental work1 may afford an alternative explanation.

He excised the anterior centralis in dogs, cats, and monkeys, and observed neck retraction limited only to the neck muscles. Other clinical signs could be explained by partial bilateral Wallenberg's syndrome. Etiologically, whether it was due to partial occlusion of both posterior inferior cerebellar arteries or to mechanical compression of the structures supplied by these vessels cannot be ascertained. However, its rapid disappearance following evacuation of the hygroma would favor the latter.

Reaccumulation of epidural hygroma in this patient indicates either that the single stitch employed to close the dura was not enough to achieve a water-tight closure, or that the vacuum drain placed in the epidural space prevented dural coaptation. It is also possible that another tear in the dura at another site was overlooked.

A CT scan proved extremely useful in making a correct preoperative diagnosis. At times, however, it may be difficult to differentiate from CT scans of liquid epidural hematoma and subdural hygroma. Nevertheless, with the demonstration of a thick inner membrane (dura), absence of external membrane, and densities of the accumulated fluid compatible with CSF, confusion should rarely arise.

References


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