Case Reports

An Arterial Posterior Fossa Extradural Hematoma
Demonstrated by Vertebral Angiography

Case Report

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Extradural hematomas of the posterior fossa have been considered a comparatively rare lesion. Reigh and O'Connell found 80 cases in the literature between 1901 and 1962. The fact that slightly over one third of the cases were reported after 1955 indicates an increasing awareness and ability on the part of clinicians to diagnose and treat this condition.

In the typical case, there is a history of trauma, usually to the occiput, and an occipital fracture is often, but not always, found on plain x-rays of the skull. The development of the hematoma may be acute (within 24 hours after the injury), subacute or chronic (weeks or months later). In the acute variety, the signs and symptoms of medullary compression develop rapidly within hours of the injury. The more slowly accumulating hematomas often have the signs and symptoms ordinarily associated with a slowly expanding posterior fossa lesion.

Most posterior fossa extradural clots develop as the result of leakage of blood from a tear in one or several of the large venous sinuses. In reviewing the literature one gets the impression that these hematomas are exclusively venous in origin. Beller and Peyser state: "The condition usually develops more slowly than epidural hematomas in the middle fossa since the bleeding is always venous." Campbell, et al., noted, however, that in one of their four cases, as well as in three other cases reported elsewhere, the hematoma did not extend to any of the venous sinuses. They therefore surmised that the bleeding does not always come from a torn dural sinus. In a patient described in an addendum to their paper, brisk bleeding from small arteries in the exposed dura was found to be the source of the hematoma.

Recently we have treated a patient with a posterior fossa epidural hematoma that was diagnosed preoperatively by vertebral angiography. The arterial origin of the hematoma was established clearly by its angiographic appearance as well as by the findings at operation.

Case Report

History. L.R., M.N.I. 65-117. The patient was a 61-year-old white man who was brought to the Emergency Department of the Royal Victoria Hospital on the evening of January 17, 1965, by the police. He was intoxicated, vomiting, and complaining bitterly of occipital headache. He had been drinking at a bar when he complained of dizziness and fell backward striking the back of his head on the floor. There was loss of consciousness for a few moments, and thereafter he seemed to be stuporous and intoxicated. He was admitted to the Montreal Neurological Hospital for observation.

Examination. The patient was restless and crying with periods of somnolence and irritation. At times he was able to obey simple commands but he did not talk sensibly. There was an occipital scalp hematoma. His pupils were equal and reactive to light, the cranial nerves were normal, there was normal power in all extremities, and the deep tendon reflexes were normally active and symmetrical. The plantar responses were flexor. In the first few hours after admission there was intermittent vomiting. X-rays of the skull showed a linear fracture of the occiput starting near the medial end of the left lambdoid suture and extending down to the
floor of the posterior fossa on the right side of the foramen magnum. The fracture extended across the region of the torcular Herophili. The calcified pineal gland was not displaced in any direction.

**Hospital Course.** On the morning of January 18, the patient was still drowsy but responded. There was ecchymosis about the left eye. His blood pressure and pulse had remained stable, but intermittent vomiting continued. On the morning of January 19, he seemed drowsier and showed no verbal response except moaning. That same afternoon his condition deteriorated and he was no longer responsive. His blood pressure had risen to 170/80, pulse 70. The pupils, although normal in size, did not react to light. There was withdrawal of all limbs to painful stimulation and a response to tickle in the nose by movement of the head and all limbs. There was no papilledema or retinal hemorrhage. Respiration was Cheyne-Stokes in type and shallow. The deep tendon reflexes were increased compared to their state on admission, and in the lower limbs there was spasticity with bilateral ankle clonus and extensor plantar responses. The patient was having frequent hiccups.

An expanding lesion in the posterior fossa was suspected. Angiography via the femoral route was attempted, but it was impossible to pass a catheter into the desired position from either femoral artery due to marked tortuosity of the vessels. After this, a catheter was passed into the right axillary artery and an injection of 28 cc 75% Hypaque was given using a pressure injector. Serial stereoscopic frontal and right lateral views of the skull were made. There was good visualization of the right vertebral and carotid arteries and their intracranial branches. The right posterior-inferior cerebellar artery was displaced to the left and forward (Fig. 1 A, B). There was displacement of the inferior cerebellar superficial arteries upward as well as away from the inner table of the floor of the posterior fossa on the right side. These branches were displaced up to 2.5 cm above their corresponding arteries on the left side.

A meningeal branch of the right vertebral artery was visualized and shown to be displaced away from the internal occipital crest of the skull. It appeared torn in several places and was leaking opacified blood into the epidural space (Fig. 2). Extravasated opacified blood was also seen to collect in the epidural space in the region of the torcular Herophili, suggesting anterior displacement of that structure (Fig. 3). There was some upward displacement of the right superior cerebellar artery as compared with the left (Fig. 3). There was also good filling of the anterior and middle cerebral arteries on the right side. The position of the callosal and Sylvian arteries indicated the presence of some moderate degree of ventricular dilatation (Fig. 1 B). It was felt that this examination showed a rather thick but localized epidural hematoma lying along the posterior and inferior aspects of the posterior fossa on the right side.

**Operation.** After angiography the patient was postured in the prone cerebellar position. The occipital squama was exposed on both sides revealing the fracture line on the right. There was bluish discoloration of bone in this region. A burr hole was placed over the left occipital squama and no epidural hematoma encountered. Dural tension was slightly elevated. As soon as a burr hole had been made in the right occipital squama, a reddish-black jelly-like clot began to extrude through the bony opening. The burr hole was enlarged into a right suboccipital craniectomy and a large amount of clot extruded. The clot was removed with suction, and it was estimated that the dura was pushed in about 3 cm from the inner table of the skull. The clot occupied the entire right side of the posterior fossa from the transverse sinus superiorly to the foramen magnum inferiorly. It extended medially to the midline and laterally to the mastoid air cells. When the clot was removed, bleeding from a tear in a small dural artery was noted in the upper medial corner of the craniectomy near where the occipital sinus joined the torcular Herophili and transverse sinus. This was controlled with coagulation. After the clot was removed the dura came back against the bone, and tension appeared normal. The dura was opened and slight contusion of the cerebellar cortex was noted. There was no subdural fluid or collection of blood.

**Postoperative Course.** The patient began to improve immediately after operation. The next morning his pupils reacted sluggishly to light, the spasticity in both legs and the