Subdural Hematoma of the Posterior Fossa

Review of the Literature with Addition of Three Cases

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In comparison to the large number of head injuries that are seen in the hospital and at autopsy, traumatic lesions of surgical consequence to the posterior fossa are relatively rare and, of those, subdural hematomas over cerebellar hemispheres probably represents one of the rarest forms of hemorrhagic space-occupying lesion. The rarity of this lesion coupled with its confusing clinical presentation justifies a review of the subject and the reporting of 3 new cases.

In the neurosurgical unit of the Boston City Hospital over a 15-year period (1948–1962) 1589 patients were hospitalized because of extensive head injuries. Subdural hematomas over the cerebral hemispheres were verified in 532 of these patients. Subdural collection of blood in the posterior fossa was found in 3 cases only (0.57 per cent). In 1960, McKissock et al.,19 reported on 389 cases of subdural hematoma, of which 2 were in the posterior fossa. Ciarla,8 reviewing the results of 2215 autopsies, found pachymeningitis hemorrhagica interna above the tentorium in 163 cases and only twice in the posterior fossa. Vance,35 on the basis of 507 autopsies on patients in whom death was attributed to head trauma, stated that "subdural hemorrhages below the tentorium were caused by cerebellar lacerations and were rather rare. Most of them were insignificant." In the statistics of Fisher et al.,11 of 135,000 hospitalized patients only 300 were admitted because of trauma to the occipital region and in 4 of them subdural hematoma of the posterior fossa was present. Webster et al.,34 in an analysis of 300 cases of penetrating cranial wounds during World War II, found cerebellar wounds in only 10.

Barnett and Meirowsky2 in their study of Korean War casualties found wounds of the cerebellum in 3.8 per cent of patients. Probably the first successful operative case of subdural hematoma in the posterior fossa was described by Neisser and Pollack22 in 1904. The patient, with a history of headache, vomiting and unsteadiness for a few weeks, was admitted to hospital in coma. In addition to the signs indicating involvement of the posterior fossa, left facial weakness of the upper-motor neuron type was present. After negative exploration over the right motor area, the posterior fossa was trephined twice and 180 gm. of clotted and semiliquid blood were removed. The operation was followed by a complete recovery. The same authors mentioned 2 other patients manifesting cerebellar signs and a chronic course; in them trephination of the posterior fossa revealed blood in the subdural space, the amount and character of which were not clarified. In 1913 Ciarla5 presented a single case of traumatic pachymeningitis hemorrhagica in the posterior fossa, and in the following years other authors1,6,7,10,12,14,19,21–22 added more cases, bringing the total number reported to 31.

Report of Cases

Case 1. E.D., a 47-year-old white male, was admitted on June 29, 1954 and died on June 30, 1954. Shortly before admission the patient was struck on the forehead with a baseball bat, and on falling to the ground he struck his occiput on a curbstone. He did not lose consciousness and there was no headache or vomiting.

Examination. On admission he was fully conscious, alert and talking, but slightly confused. A small laceration of the occipital scalp was recognized. The neurological findings at this time were negative except for a slightly larger left pupil. Pulse rate was 120, blood pressure 120/90, and respiratory rate 20. Roentgenograms showed no

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evidence of fracture of the skull. The laceration of his scalp was sutured and the patient was admitted to one of the general surgical wards in good condition. Half an hour later he suddenly became comatose and unresponsive. Neurological examination at that time revealed faccic extremites and absence of muscular reflexes. Plantar responses were absent. Pupils reacted to light, the left much more than the right. Pulse rate was 80, blood pressure 130/90 and respiratory rate 6–8/min. Lumbar puncture revealed pink fluid under a pressure of 350 mm. of water.

The clinical impression was that of acute epidural or subdural hematoma over the left cerebral hemisphere. He was transferred to the neurosurgical unit.

Operation. Burr holes were made in the parietal, temporal and frontal areas. The only abnormality found was marked brain edema. The posterior fossa was then explored, revealing a discolored dura mater under great pressure. When the dura mater was incised a copious amount of solid blood clot extruded itself, and immediately the patient’s labored and irregular respirations became less stertorous and soon assumed a regular rhythm. The clot was estimated at over 30 cc. and when almost completely removed the presumed source of bleeding, a small artery over the upper part of the right cerebellar hemisphere, was uncovered. The bleeding was controlled with coagulation.

Course. The following day the patient was deeply comatose. The left pupil was slightly larger than the right. Both pupils, however, reacted to light. Pulse rate was 110, blood pressure 150/60, and respiratory rate 18. About 20 hours after operation the patient suddenly stopped breathing and despite intubation and artificial respiration, he expired. Postmortem examination disclosed a swollen brain, weighing 1580 gm., with recurrent blood clot in the posterior fossa, herniation of tonsils, and a flattened hemorrhagic pons. A linear fracture of the skull on the right side extended from the occiput into the foramen magnum.

Comment. The patient sustained a severe injury to the occipital area, which however did not produce unconsciousness. On admission, neurological findings were entirely negative apart from slight anisocoria. Films of the skull failed to reveal the fracture which was later found on autopsy. After a short lucid interval, the patient suddenly became drowsy and lapsed into deep coma rapidly. At this stage there were no lateralizing signs, apart from marked anisocoria. Signs of cerebellar dysfunction were not noticed, presumably escaping detection because of the unconsciousness. The decision to open the posterior fossa was made after several explorations through supratentorial burr holes failed to reveal a lesion.

Case 2. D.E., an 84-year-old white male, was admitted on Feb. 3, 1960 and died on Feb. 5, 1960. Shortly before admission he was struck on the head with a club which rendered him unconscious for half an hour.

Examination. On admission the patient was lucid and alert, complaining only of some headache. He did not vomit. A few small lacerations of the scalp were over the forehead and a larger one in the occipital area. Neurological findings at that time were negative except for an extensor plantar response on the right side. Roentgenogram of the skull disclosed a linear fracture in the right parietal area, and the patient was admitted to the general surgical service with a diagnosis of cerebral contusion.

Course. About 6 hours later, while sitting up in bed, he started to vomit and suddenly became unresponsive and apneic. He was intubated and respirations were maintained by positive pressure. Neurological examination revealed a deeply comatose patient not responding to painful stimuli, flaccid in all extremities and totally areflexic. Pupils were equal, very small and did not react to light.

The patient was transferred to the neurosurgical unit with the diagnosis of brain edema, brain-stem compression and possible expanding lesion of the posterior fossa.

Operation. After negative exploration through burr holes over the line of fracture of the skull, both lateral ventricles were tapped through the burr holes in the parietal area with the recovery of grossly bloody fluid under pressure of 300 mm. of water. Approximately 80 cc. of fluid were drained from each of the ventricles. The posterior fossa was explored. The dura mater over both cerebellar hemispheres was bluish and strongly bulging. After incision of the dura mater about 40 cc. of fresh subdural clot extruded itself. A small area of laceration with a cortical hematoma was found over the right cerebellar hemisphere. Cerebellar tonsils were found herniated through the foramen magnum and were necessarily removed. The cerebellum started to pulsate again. The subdural clot was found to extend down to the level of C2 and to compress the spinal cord severely. It was removed by suction. The wound was closed in the usual manner.

Course. During the operation the patient’s condition deteriorated. Blood pressure had to be maintained with large amounts of Levophed and no spontaneous respirations were recorded. Four hours after operation the patient expired.