Case Reports

Acute Epidural Hemorrhage Complicating Resection of Acoustic Neurinoma*

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The following case history is reported to draw attention to an uncommonly recorded complication experienced in surgery of tumor of the cerebellopontine angle and to consider the relationship of this complication to the general problem of acute intracranial hemorrhage secondary to ventricular decompression in the hydrocephalic patient.

Case Report

PF 131 09 04, D.S., a 37-year-old Negro female, was admitted to the Los Angeles County General Hospital on April 2, 1962 with a complaint of occipital headaches of increasing severity over a period of 4 months. She had noticed diminished hearing and tinnitus on the right for several months. In March 1962 she was hospitalized in another facility because of the headaches. A lumbar puncture yielded spinal fluid containing 190 mg. per cent of protein. Carotid angiography revealed changes suggesting ventricular enlargement. She was advised to have further diagnostic studies and for this reason was referred to the Los Angeles County General Hospital.

Past History. At age 17 a diagnosis of sickle-cell anemia was made, subsequently requiring numerous hospital admissions for transfusion. Because of the problems related to sickle-cell disease she had a subtotal gastric resection in 1952 and a dilatation and curettage in February 1962, at which time her hemoglobin was 3 gm. per cent.

Examination. Blood pressure was 140/90. There was a Grade II apical systolic murmur.

Neurological findings were normal fundi, intact corneal reflexes and sensory perception on the face and no disturbance of the facial nerve. There was a suggestion of diminished hearing on the right, which was not substantiated by audiometric studies. Caloric studies revealed a hypoactive right labyrinth. Motor and sensory functions were intact. Cerebellar examination revealed no abnormalities.

On lumbar puncture the opening pressure was 140; the cerebrospinal fluid was faintly xanthochromic and content of protein was 900. A hemogram recorded sickle cells, probably of SS type.

Roentgenograms of the skull showed apparent enlargement of the right internal auditory meatus.

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Pneumoencephalography revealed a right pontine-angle tumor with ventricular dilatation (Fig. 1).

Course. During the preoperative period, the patient’s hemoglobin fell to 8.0 gm. per cent. She was transfused with 1000 cc. of whole blood in preparation for operation.

Operation. On April 25, 1962, with the patient in the sitting position, a right suboccipital craniectomy was performed. The dura mater was extremely tense overlying the cerebellum. Therefore, ventriculostomy via a right posterior parietal burr hole was performed with cautious removal of 30 cc. of ventricular fluid. As the tension in the suboccipital region lessened, the dura mater was opened; and a tumor of the cerebellopontine angle was exposed by retraction, facilitated by resection of the lateral one-third of the cerebellum. Intracapsular decompression of the tumor was performed, and the capsule of the tumor was resected in piecemeal fashion. As the resection neared completion and the capsule was being teased from the midbrain, sudden onset of hemorrhagic engorgement and swelling of the cerebellum occurred. The blood pressure rose from 150/90 to 180/120. However, the pulse remained stable at 80. The ventricular drain was opened, revealing no apparent increase in pressure. The swelling was to such an extent that it required further cerebellar resection. Ninety grams of urea were administered intravenously. The medial remnants of the tumor were removed, and the dura mater and wounds were closed. Considerable difficulty was encountered during this period by inability to obtain hemostasis. At the time of closure, hemostasis in the bed of the tumor was reasonably secure. Extensive loss of blood occurred, requiring transfusion of 4000 cc. of whole blood. A tracheostomy was performed at the end of the procedure.

Postoperative Course. The patient was unresponsive to pain and had bilateral dilated, unresponsive pupils. The temperature was maintained at 30–32°C. Twenty-four hours after operation there were feeble responses to pain, and she moved the right upper extremity spontaneously. The ventricular drain was opened and 15 cc. of ventricular fluid were removed. The subocciput was not bulging. At 36 hours after operation, the blood pressure fell; and she became apneic. Vasopressors and assisted respiration were instituted. She expired 48 hours postoperatively.

Postmortem examination revealed a massive acute right parieto-occipital extradural hematoma. This measured 10×12×2.5 cm. and was centered under the burr hole of the ventriculostomy (Fig. 2). There was minimal subdural hematoma at the site of the posterior fossa exploration. The tumor was grossly and microscopically resected totally. A mild degree of ventricular
dilation was found with deflection of the midline structures to the left. The convolutions underlying the extradural hematoma were markedly flattened, and there was herniation of the uncus and hippocampal gyrus into the right tentorial notch. Dissection of the anterior inferior cerebellar artery revealed it to be intact throughout its course and distribution.

**Discussion**

The unexpected nature of the underlying pathological process leading to this patient’s demise caused much consternation among those responsible for her care. Theoretically, this was a surgically remediable condition had its nature been suspected but, unfortunately, this diagnosis was not entertained.

The literature dealing with the diagnosis and treatment of acoustic neurinoma is extensive, and the surgeon is reminded constantly of the hazards of excessive retraction and manipulation of the cerebellum and brain stem. Much emphasis has been placed on the role of impairment of the circulation of the anterior inferior cerebellar artery. The precipitous sequence of events described in the operative report was ascribed incorrectly to compromise of the pontine circulation by interruption of the flow of the anterior inferior cerebellar artery. This decision was made based upon knowledge of a study reported by Atkinson\(^2\) and previous personal experience with 2 similar cases.

The clinical and pathological study cited\(^2\) described a series of 7 cases in which death occurred following complete or nearly complete removal of tumors of the acoustic nerve, and partial infarction was demonstrated at necropsy. The operative and postoperative events described suggest that operative occlusion or injury of the anterior inferior cerebellar artery may be associated with profound autonomic disturbance indistinguishable from severe supratentorial compression. It is of particular interest and irony that the clinical events described in Case 5 of that article are almost an exact duplication of our own experience, except that on that occasion the surgeon’s incorrect impression was that of a severe hemorrhage in the supratentorial space.

However, a search of the English literature has failed to yield a record of the complication reported here in the surgical treatment of acoustic neurinoma. The problem of acute intracranial hemorrhage complicating procedures of ventricular decompression has received attention in recent years. Peet\(^7\) mentioned a case of acute epidural hemorrhage during ventriculography in a hydrocephalic child. Schorstein\(^8\) recorded 3 cases of acute venous subdural or epidural hemorrhage complicating ventricular drainage. These patients ranged in age from 10 to 24 years and in each instance had severe obstructive hydrocephalus. Anderson\(^3\) described 3 cases of acute subdural hemorrhage following ventricular decompression in hydrocephalic infants. In 2 of these children subdural hematomas developed following unilateral resection of choroid plexus. The third child had not had cranial surgery, but bilateral subdural hematomas developed following a spino-ureteral shunt. Davidoff and Feiring\(^2\) reported 3 cases of chronic subdural effusion complicating ventricular decompression in hydrocephalic children. They found the subdural effusions to be particularly refractory to treatment and to require subdural cisterna-magna shunts. Youmans and Schneider\(^9\) described the case histories of 2 adult hydrocephalics with posttraumatic subdural and epi-
dural hematomas. These were giant accumulations that had been insidious in their manifestation. They also reported the case of a 16-year-old hydrocephalic with papilledema and symptoms of elevated intracranial pressure from aqueductal stenosis. Death occurred following ventricular drainage from a 920-cc. right posterior epidural hematoma which arose from a coronal burr hole. Haft et al. reported 2 similar cases of acute postoperative epidural hemorrhage in 15-year-old patients with obstructive hydrocephalus and concluded that they were the product of excessive ventricular decompression in young individuals with loosely attached frontal dura mater. Del Vivo and Armenise reported 5 cases of acute epidural hemorrhage complicating ventricular decompression in hydrocephalic individuals. These patients ranged from 6 to 35 years of age. They could establish no relationship between age of patient, etiology of obstructive process and duration of the hydrocephalus to the occurrence of this complication. Schorstein considered the pathogenesis of the venous hemorrhage to be related directly to the diminished intracranial pressure and reduced volume of ventricular fluid associated with ventricular drainage. Under these circumstances, he postulated that the brain tended to sag in the calvarium and apply traction to the dura mater via the bridging veins, thus encouraging the dura mater to contract (by its elasticity) from the bone and tear the epidural veins. He also postulated engorgement of the intracranial veins as a compensatory mechanism to reduction of brain and ventricular volume and enhancing bleeding from the torn vessels. He also considered this series of events to be more likely in the younger person with less densely attached dura mater.

In the review of this problem, the one factor common to all cases has been the presence of moderately advanced hydrocephalus. Relative to the case reported herein, a moderate degree of hydrocephalus was present. The epidural hemorrhage was almost certainly secondary to ventricular decompression at the time of operation. The mechanisms influencing this surgical catastrophe can be only conjectured. Cerebral hemorrhage as a manifestation of sickle-cell phenomenon has been reported but is not a common complication of this disease. The epidural hemorrhage in our patient was centered at the site of the burr hole placed for ventriculostomy. However, in the cases reviewed from the literature, 9 hematomas were adjacent to a surgical wound and 10 were remote and probably unrelated to a cranial exposure. Therefore, this complication is not necessarily a result of inadequate hemostasis in the surgical wound. Of the 17 reported cases, 9 resulted in fatality and 4 patients of the remainder suffered severe and permanent disability. Those patients that survived did so because of prompt recognition of the nature of the complication and immediate surgical intervention. It is the purpose of this report to aid those performing operation on tumors of the cerebellopontine angle in the recognition of this potential complication, particularly in the patient in whom some degree of obstructive hydrocephalus has developed concurrently. Unfortunately, the clinical events associated with this complication are not specific but comprise alterations in blood pressure, pulse, respiration, pupillary function and conscious state, similar to those that occur with compromise in the function of the brain stem.

Summary
A fatal case of massive extradural hematoma complicating surgical removal of an acoustic neurinoma is described.
The difficulty of making a clinical distinction between this complication and the universally recognized syndrome of acute pontine ischemia is illustrated and discussed.
The relationship of this complication to the general problem of acute extracerebral hemorrhage occurring secondarily to surgical ventricular decompression in obstructive hydrocephalus is considered.

References
2. Atkinson, W. J. The anterior inferior cerebellar artery. Its variations, pontine distribution, and
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