Delayed intracerebral hematoma at the site of a subarachnoid bolt pressure monitor

Case report

HUNT BOBO, M.D., JIMMY D. MILLER, M.D., OWEN B. EVANS, M.D., AND JOHN P. KAPP, M.D., PH.D.

Departments of Neurosurgery and Pediatrics, University of Mississippi Medical Center, Jackson, Mississippi, and Department of Neurosurgery, State University of New York at Buffalo, Buffalo, New York

The authors report development of a delayed intracerebral hematoma following use of a subarachnoid bolt for intracranial pressure monitoring. This complication has not been previously reported.

KEY WORDS • intracranial pressure monitoring • subarachnoid bolt • intracerebral hematoma • coagulopathy

ALTHOUGH the subarachnoid bolt for intracranial pressure (ICP) monitoring has been used successfully for over 10 years, complications continue to occur despite meticulous care. Most of these complications are related to infection or inaccurate pressure measurement. We report the development of a delayed intracerebral hematoma following removal of a subarachnoid bolt. This complication has not been previously reported.

Case Report

This 4-year-old boy with severe chronic pulmonary disease, secondary to an adenovirus pneumonitis at age 15 months, was transferred to the University of Mississippi Medical Center with respiratory failure. In addition to cor pulmonale, he had a known ventriculoseptal defect. On the morning of admission he developed rhinorrhea and cough, and throughout the day his respiratory status declined. Immediately upon arrival, he suffered a cardiopulmonary arrest and was resuscitated.

Examination. The physical examination showed a temperature of 99°F, pulse 163 beats/min, and blood pressure 102/70 mm Hg. He was being mechanically ventilated. He had bilateral ankle clonus, bilateral Babinski signs, an intact oculocephalic reflex, and flexion of the upper extremities in response to deep pain. He developed partial and generalized seizure activity.

Routine laboratory tests were normal except for a white blood cell (WBC) count of 15,000/cu mm. Arterial blood gas measurement revealed: pO₂ 112 mm Hg; pCO₂ 36 mm Hg; and pH 7.50, with a fraction of inspired oxygen (FiO₂) of 100%. Computerized tomography (CT) of the head was normal. Lumbar puncture showed an opening pressure of 260 mm H₂O; cerebrospinal fluid WBC count was 71/cu mm, with 60% segmented neutrophils; protein was 31 mg/100 ml; and glucose was 99 mg/dl. An electroencephalogram revealed periodic lateralizing epileptiform discharges bilaterally in the posterior temporal lobes. Isotope brain scanning showed uptake in both temporal lobes.

Course. The patient was begun on acycloguanosine for presumed herpes encephalitis. He also exhibited intermittent pupillary dilatation which responded to administration of mannitol. A subarachnoid bolt was placed in the right frontal region to monitor ICP. On several occasions the ICP rose to 30 mm Hg and responded to osmotic diuretic treatment. A repeat CT scan 2 days after insertion of the bolt revealed diffuse cerebral edema with no shift or blood (Fig. 1 upper). The bolt was removed on the 6th day after insertion, when the ICP had been less than 20 mm Hg for 24 hours. No bleeding or hematoma was noted when the bolt was removed. On the day after removal of the subarachnoid bolt the patient was found to have clotted blood in the right external auditory canal. Coagulation
studies were obtained with the findings of a prothrombin time of 21.8 seconds with a control level of 10.4 seconds and a partial thromboplastin time of 48.5 seconds with a control level of 30.6 seconds. Fibrin degradation products were greater than 40 mg/dl and platelet count was 349,000/cu mm. The patient was given vitamin K intramuscularly and his clotting studies returned to normal.

Thirteen days later, the patient developed papilledema and his pupils became dilated but slowly reactive. He was hypertensive with a diastolic pressure of 100 mm Hg. The patient could vocalize and was moving all extremities spontaneously. He had marked generalized hypertonicity. Repeat CT scanning showed a hyperdense right frontal mass lesion with some rim enhancement (Fig. 1 lower). Needle aspiration of the lesion removed hematoma and necrotic brain. All cultures, including viral tests, were negative. Follow-up scans documented gradual resolution of the intracerebral hematoma.

The patient’s neurological status improved; however, he remained neurologically impaired with generalized spasticity and minimal responsiveness. He died at home of unknown causes about 1 month after discharge. Permission for autopsy was refused.

Discussion

Delayed intracerebral hemorrhage at the site of a subarachnoid bolt has not been reported previously. The hemorrhage was not present on a CT scan obtained 2 days after the bolt was inserted. Although no bleeding was noted at the time of removal of the bolt, most likely the hematoma developed from traction on fragile adherent cortical vessels during bolt removal in a patient with an unrecognized coagulopathy.

Use of the subarachnoid bolt for monitoring ICP was introduced in 1973 by Vries, et al.6 They reported a subdural hematoma in a patient with liver failure and a coagulopathy who had a subarachnoid bolt in place because of cerebral edema. Narayan, et al.,3 reviewed 207 cases in which ICP was monitored; three of their patients developed intraventricular or intraparenchymal hematoma. They did not specifically mention if any of these three patients had a subarachnoid bolt. In their study, 91% of the patients were monitored with

FIG. 1. Computerized tomography scans 2 days after subarachnoid bolt insertion (upper) and 13 days after removal of the bolt (lower). Slices shown are caudal to the bolt site (left), at the bolt site (center), and rostral to the bolt site (right).
Intracerebral hematoma due to ICP bolt monitor

ventriculostomies and 9% with subarachnoid bolts. They reported that patients with disseminated intravascular coagulation or other coagulopathies and in whom there were catheter placement difficulties were more likely to develop intracranial hemorrhage from ventriculostomy or subarachnoid screw insertion. In 1976, Rosner and Becker reviewed complications arising from ICP monitoring. In their series of 112 patients, four developed subcutaneous fluctuancy at the wound site of the subarachnoid screw. In three, a small hematoma was evacuated and the fourth was treated conservatively. These were not considered to be significant complications and were placed in the "no complication" group.

In 1977, Winn, et al., reviewed 147 patients who had undergone insertion of a subarachnoid bolt. Although they did not report intracerebral hemorrhages, of 24 patients who had postmortem examination, three had evidence of subpial contusion with microscopic evidence of infiltration of the arachnoid with polymorphonuclear leukocytes and red cells.

The subarachnoid bolt remains a useful monitor in the patient with elevated ICP; however, use of this technique may be associated with development of an intraparenchymal hematoma. It is surprising that this complication has not been reported more frequently considering the significant number of patients with cerebral insults and ICP monitors who develop coagulation abnormalities.1,2,5

References


Manuscript received August 8, 1985.

Address reprint requests to: Hunt Bobo, M.D., Department of Neurosurgery, School of Medicine, The University of Mississippi Medical Center, 2500 North State Street, Jackson, Mississippi 39216.