Intracerebral hematoma following evacuation of chronic subdural hematomas

Report of two cases

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Two cases of intracerebral hemorrhage occurring after evacuation of bilateral chronic subdural hematomas are reported. Possible pathogenic mechanisms included hemorrhage into previously undetected areas of contusion, damage to cerebral vasculature secondary to rapid perioperative parenchymal shift, and sudden increase in cerebral blood flow combined with focal disruption of autoregulation; of these, the latter mechanism seemed most likely to be responsible for the hematoma formation. The need for clinical awareness of this nearly uniformly devastating complication, as well as prompt use of computerized tomography scanning in assessing the postoperative course, are stressed.

KEY WORDS • subdural hematoma • intracerebral hemorrhage

ALTHOUGH the potential for cure for chronic subdural hematomas (SDH’s) is very high, unexpected neurological deterioration may occasionally complicate the postoperative course. Failure of the brain to reexpand, tension pneumocephalus, and recurrence of the hematoma are well recognized complications which may be responsible for a patient’s poor progress. Intracerebral hemorrhages occurring after the rapid removal of a chronic SDH have been reported recently as a rare but nearly uniformly devastating postoperative occurrence.3

This report documents two additional cases of postoperative intracerebral hemorrhage causing progressive neurological deterioration that occurred after the evacuation of bilateral chronic SDH’s.

Case Reports

Case 1

This 62-year-old man was admitted to our institution 3 weeks after minor head trauma caused by a motor vehicle accident. He had complained of progressive left frontal headaches and became confused with incontinence and weakness on the left side. A computerized tomography (CT) scan showed large, slightly hyperdense, bilateral chronic SDH’s. There was ventricular compression bilaterally, but no shift of the midline structures (Fig. 1 left).

The subdural collections were evacuated through multiple burr holes and by closed-system drainage. The patient’s medical history revealed arterial hypertension, and during surgery he had transient increases in arterial pressure up to 200/100 mm Hg. After an initial improvement, his neurological condition deteriorated and he became lethargic and paretic on the right side. A repeat CT scan revealed an intracerebral hematoma deep in the left hemisphere associated with moderate bifrontal pneumocephalus (Fig. 1 right). The patient was treated conservatively. After a complicated postoperative course he died in a comatose condition.

Case 2

This 73-year-old woman developed headaches, confusion, and a right-sided hemiparesis 3 weeks after receiving minor head trauma. A CT scan showed bilateral chronic SDH’s with moderate shift of the midline structures to the right (Fig. 2 left). The hematomas were evacuated via multiple burr holes and closed-system drainage.

Initially the patient improved, but 6 days after surgery her level of consciousness deteriorated. Angiography revealed a persistent SDH on the left side without any underlying vascular anomaly. A solid clot with thick membranes was evacuated at craniotomy. Postoperatively, the patient did not awake fully from anesthesia.
Intracerebral hematoma after evacuation of chronic SDH's

Recently, Modesti, et al., published a detailed clinical review of intraparenchymal hemorrhages occurring after open decompression of chronic SDH's, reporting a 5% incidence of this often devastating complication among 140 surgically treated cases of chronic extracerebral fluid collections. One more case was added to the literature in 1984 by Richter, et al., who reported a patient with evacuation of a chronic SDH who died 5 days postoperatively following a massive deep intracerebral hemorrhage on the side of the previous hematoma. The two cases described here represent 4% of all patients undergoing surgery for chronic SDH between 1977 and 1984 at our institution. Our patients both had clinical signs of increased intracranial pressure and mild focal neurological deficits. Clotting profiles, including prothrombin time, partial thromboplastin time, and platelet count, failed to reveal any anomaly. In both cases, CT scanning revealed bilateral hypodense subdural collections that appeared to be symmetrical in Case 1 and predominantly on the left side with moderate shift of the midline cerebral structures in Case 2. The first patient had a history of arterial hypertension and was repeatedly hypertensive during the operation. The second patient, however, did not have increased arterial blood pressure at any time during the operation or in the postoperative period. The cause of intracerebral bleeding in our cases is unclear. This occurrence is clearly distinct from the clinical picture of the classic "traumatische Spat-Apoplexie." Bollinger's delayed posttraumatic intracerebral hemorrhage occurs with a different time course and without any causal relationship to previous surgery.

Hemorrhage into undetected areas of contusion has also been listed as a possible pathogenic factor. This seems to be an unlikely mechanism for hemorrhage in our patients, in whom preoperative CT failed to show evidence of cerebral contusion or hematoma at the time of the initial diagnosis. The theory of rapid perioperative parenchymal shift causing direct vascular damage does not fit well with the phenomenon of intracerebral hematoma formation in our patients, who had previous bilateral subdural collections with minimal or no shift of the midline structures. Therefore, although speculative, the pathogenic mechanism that seems most likely to be responsible for these hemorrhages may involve a sudden increase in cerebral blood flow combined with defective vascular autoregulation. Labile hypertension and wide swings in blood pressure during operation, as in our Case 1, may be contributory. Preoperative cerebral blood flow in patients with chronic SDH's is known to be uniformly decreased over the compressed brain.

Surgical decompression allows cerebral blood flow to return to normal values. It seems therefore reasonable to hypothesize that sudden restoration of normal perfusion pressure in areas of faulty cerebral vascular autoregulation due to subcortical swelling underlying surface compression, focal impedance of the venous drainage, or ischemic loss of CO2 reactivity might in
turn lead to the vascular damage that resulted in intraparenchymal hemorrhage.

In light of this, we must stress, along with Modesti, et al., the need for slow decompression of chronic SDH's, possibly with controlled reexpansion by means of lumbar saline instillation, with a gradual emergence from anesthesia and careful control of the blood pressure lability. Clinical awareness of this potential complication and the immediate use of CT scanning if the patient does not awake easily from anesthesia or if new neurological deficits develop are the most important keys to the early diagnosis of this fortunately rare complication.

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References


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