Documented reversal of global ischemia immediately after removal of an acute subdural hematoma

Report of two cases

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The authors report two cases of severe head injury with acute subdural hematoma, in which cerebral blood flow (CBF) and cerebral blood volume (CBV) measurements were obtained prior to evacuation of the subdural hematoma and again immediately after removal. The first patient, a 21-year-old man with a motor response localizing to pain, had a global CBF of 18.2 ml/100 gm/min and a decreased global CBV of 3.7 ml/100 gm at 2.3 hours after injury. Immediately after removal of the subdural hematoma (8.1 hours after injury), CBF and CBV measurements revealed increased to 35.5 ml/100 gm/min and 5.8 ml/100 gm, respectively. The second patient, a 49-year-old woman with a normal flexor motor response to pain, had preoperative global values of 15.8 ml/100 gm/min for CBF and 2.0 ml/100 gm for CBV at 3 hours after injury. Postoperatively (9.3 hours after injury), the CBF and CBV values increased to 41.6 ml/100 gm/min and 4.0 ml/100 gm, respectively. The first patient, with only borderline ischemia and removal of the subdural hematoma within 3 hours, made a good recovery, while the second patient, with prolonged lower levels of CBF, remained in a persistent vegetative state. The low values of preoperative CBV argue for compression of the microcirculation as the cause of ischemia.

KEY WORDS • acute subdural hematoma • cerebral blood flow • ischemia • cerebral blood volume • xenon-enhanced computerized tomography • reperfusion

A acute subdural hematoma in severe head injury carries a poor prognosis.14 The majority of patients will die or remain disabled, despite a "lucid interval" in over 30% of the cases.1,2,15,22 The causes for this poor outcome are not known, but postmortem studies have shown ischemic brain damage in two-thirds of those who die, as well as in animal models.12,17 However, the mechanisms and timing of onset of putative ischemic damage remain unknown. Previous measurements of cerebral blood flow (CBF) obtained via the noninvasive 133Xe method in patients with intracranial hematomas have mostly been performed postoperatively and have shown relatively increased CBF in the ipsilateral hemisphere.18,20 In contrast, we have previously reported a decrease in flow ipsilateral to the subdural hematoma, measured using the stable xenon-computerized tomography (CT) method prior to evacuation of the clot.1 Nevertheless, the early values and distribution of CBF before and after evacuation of a hematoma remain unclear.

We report our findings using the stable xenon-CT method at three axial levels7 to obtain CBF measurements before and after removal of an acute subdural hematoma in two patients. We also measured cerebral blood volume (CBV) by concurrently performing a dynamic CT study, rapidly scanning the patient after injecting a nondiffusible contrast medium.1 Blood volume is calculated according to the central volume principle:6 CBV = CBF × MTT, where MTT = mean transit time.

Case Reports

Case 1

This 21-year-old man was involved in a motor-vehicle accident and was found unconscious at the scene.

Examination. At admission to the emergency room, the patient’s Glasgow Coma Scale (GCS) score23 was 10 (eyes (E) = 2, verbal response (V) = 3, and motor response (M) = 5), and adequate blood pressure and oxygenation were documented at all times. He had no
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Fig. 1. Case 1. Middle slices of the diagnostic computerized tomography (CT) scans (left) and corresponding xenon-enhanced computerized blood flow maps (right), before (upper) and after (lower) evacuation of a subdural hematoma. The cerebral blood flow (CBF) doubled after evacuation of the clot. In the outlined areas (usually in areas with high or low density on the diagnostic CT scan) flow data were unreliable and therefore omitted from the global CBF calculations.

systemic or limb injuries and his neurological examination was without focal abnormality, localizing to pain bilaterally. Immediate diagnostic CT was performed, followed directly by a stable xenon-CT CBF study. The CT scan, obtained 2.3 hours after the accident, showed an acute right frontotemporoparietal subdural hematoma with subarachnoid hemorrhage and subfalcine herniation with a midline shift of 6 to 7 mm. All basal cisterns were effaced (Fig. 1 upper). The first CBF measurement showed a global reduction of CBF, with a value of 18.2 ml/100 gm/min. The CBF was 17.6 ml/100 gm/min in the hemisphere ipsilateral to the subdural hematoma and 18.8 ml/100 gm/min in the contralateral hemisphere at a mean arterial blood pressure (MABP) of 111 mm Hg and PaCO₂ of 32 mm Hg (Table 1).

Operation and Postoperative Course. Removal of the hematoma was performed without delay via craniotomy. Immediately after surgery (8.1 hours after injury), a second CBF study was performed (Fig. 1 lower). Mean global CBF had increased to 35.5 ml/100 gm/min at an MABP of 79 mm Hg and PaCO₂ of 25 mm Hg. The global CBV was 3.7 ml/100 gm before evacuation of the subdural hematoma and 5.8 ml/100
gm after (Table 1). Because of slackness of the brain after removal of the hematoma, no intracranial pressure monitor was placed. The patient was extubated a few hours after surgery and his GCS score remained at 10 (E = 2, V = 3, M = 5). He started to follow commands on Day 13 and made a full neurological recovery.

Case 2

This 49-year-old woman fell down about 15 steps and was found unresponsive at the scene. After being intubated and ventilated, she was transported to the emergency room.

Examination. The neurological examination showed a GCS score of 4, a positive gag reflex, some spontaneous respiration, and 5-mm nonreactive pupils bilaterally; corneal reflexes were absent. An initial CT scan revealed a right frontotemporoparietal subdural hematoma, causing a 20-mm midline shift, and a small right temporal tip contusion. A congenital arachnoid cyst was present in the left anterior middle fossa. Xenon-CT and dynamic CT studies were performed 3 hours after injury (Fig. 2 upper). The initial CBF measurement showed a globally reduced CBF, with a value of 15.8 ml/100 gm/min, and the preoperative global CBV was 2.0 ml/100 gm. The CBF was 13.9 ml/100 gm/min in the hemisphere ipsilateral to the subdural hematoma and 17.6 ml/100 gm/min in the contralateral hemisphere, whereas the CBV was decreased to 1.3 ml/100 gm ipsilaterally and 2.6 ml/100 gm contralaterally (Table 1). The preoperative MABP and PaCO₂ were 120 mm Hg and 33 mm Hg, respectively.

Operation and Postoperative Course. The patient was taken to the operating room for emergency evacuation of the subdural hematoma. Immediately postoperatively (9.3 hours after injury), the CBF and CBV measurements were repeated (Fig. 2 lower) and showed considerable increases, with global values of 41.6 ml/100 gm/min and 4.0 ml/100 gm, respectively (Table 1). At 3 months, the patient remained in a persistent vegetative state.

TABLE 1
Cerebral hemodynamic parameters in two severely head-injured patients, measured before and after removal of SDH*

<table>
<thead>
<tr>
<th>Hemodynamic Parameter</th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preop</td>
<td>Postop</td>
</tr>
<tr>
<td></td>
<td>Preop</td>
<td>Postop</td>
</tr>
<tr>
<td>CBF (ml/100 gm/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>global</td>
<td>18.2</td>
<td>35.5</td>
</tr>
<tr>
<td>ipsilateral to SDH</td>
<td>17.6</td>
<td>33.5</td>
</tr>
<tr>
<td>contralateral to SDH</td>
<td>18.8</td>
<td>37.4</td>
</tr>
<tr>
<td>CBV (ml/100 gm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>global</td>
<td>3.7</td>
<td>5.8</td>
</tr>
<tr>
<td>ipsilateral to SDH</td>
<td>3.6</td>
<td>6.1</td>
</tr>
<tr>
<td>contralateral to SDH</td>
<td>3.7</td>
<td>5.5</td>
</tr>
<tr>
<td>arterial CO₂ (mm Hg)</td>
<td>32</td>
<td>25</td>
</tr>
<tr>
<td>MABP (mm Hg)</td>
<td>111</td>
<td>79</td>
</tr>
</tbody>
</table>

* SDH = subdural hematoma; CBF = cerebral blood flow; CBV = cerebral blood volume; MABP = mean arterial blood pressure.

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Discussion

Many authors have speculated that the removal of an intracranial hematoma will restore brain perfusion; however, this has been difficult to prove in humans. Our report represents the first documentation of such a mechanism. The causes of early posttraumatic hyperemia following ischemia are not well understood. Lassen argued that the most likely cause was metabolic acidosis due to lactic acid production during the preceding ischemic episode; since the cerebrospinal fluid (CSF) lactate levels peak within 24 hours, the onset of a hyperemic phase could be partially explained by this. Paulson and coworkers referred to this as “dissociated paralysis” (defective autoregulation and preserved CO$_2$ reactivity); however, previous studies have shown hyperemia to persist for up to 8 days after injury in humans, in contrast to animal studies. The impressive increase in CBF in both of our cases immediately following surgery could not be attributed to arterial CO$_2$ levels or blood pressure changes, as PaCO$_2$ values dropped from 32 to 25 mm Hg in Case 1 and from 33 to 30 mm Hg in Case 2, and MABP had fallen from 111 to 79 mm Hg in Case 1 and from 120 to 105 mm Hg in Case 2.

It has not yet been determined whether the poor clinical condition of patients with an acute subdural hematoma is the cause of an early low CBF value or the low CBF is the cause for the poor clinical condition. An argument in favor of a low CBF being responsible for a poor clinical condition is the significant number of patients who “talk and die.” In these patients, we assume that compression of underlying brain tissue leads to increased intracranial pressure and thus to compression of the microcirculation. This could explain the low CBF and hence the poor clinical condition of the patient. The findings of preoperative CBV well below the normal value of 4.3 ml/100 gm in both of our cases and of considerable improvement in the CBV immediately postoperatively also support such a mechanism. It is noteworthy that in both cases ischemia was reversed immediately after surgery. Nevertheless, a considerable difference in outcome between Cases 1 and 2 occurred, which may be related to differences in patient age, amount of midline shift, CBF, and time of admission to the hospital. These factors were in favor of the patient in Case 1, who had a better outcome.

From the two cases described here, the younger patient had the higher preoperative CBF. Thus, it is possible that the cerebral circulation is more easily compromised in older patients; however, it is also possible that the brain of younger patients is more tolerant of similar low levels of CBF. Further studies with more patients need to be carried out to clarify these points.

References

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