Posttraumatic cerebral hemispheric swelling

Analysis of 55 cases studied with computerized tomography

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The authors have analyzed the clinical course and intracranial pressure (ICP) changes in 55 severely head-injured patients presenting with bulk enlargement of one cerebral hemisphere within a few hours after trauma. These patients represent 10.5% of a series of 520 patients with severe head injury studied with computerized tomography (CT). Cerebral hemispheric swelling has the highest mortality rate and the shortest survival period after trauma in all series of severe head injury. In this series, it was associated with an ipsilateral subdural hematoma of variable size in 47 patients (85%) or with a large epidural hematoma in five patients (9%); in three patients (5.4%) it occurred as an isolated lesion. Evacuation of an associated extracerebral hematoma, which was performed within 4 hours after injury in only 20% of cases, scarcely changed the patients' preoperative neurological status.

The high incidence of arterial hypotension and/or hypoxemia at admission (47% of cases) and the severity of the clinical presentation (82% of patients scored 5 points or less on the Glasgow Coma Scale, 74% had unilateral or bilateral mydriasis, and 80% had an initial ICP above normal) correlated with a very poor final outcome (87% mortality). Only one of the 11 patients with normal initial ICP continued to have normal pressure throughout the course. High-dose thiopental failed to control severe intracranial hypertension in 24 patients (51%) who had a fulminant, malignant course. A transient decrease in ICP elevation was achieved in 15 patients (31.4%) and definitive control in eight patients (17%), among whom were the seven survivors in this series. In the authors' experience, once ICP is controlled, barbiturate administration should not be discontinued until a control CT scan shows complete disappearance of the mass effect.

KEY WORDS: head injury, barbiturate, cerebral edema, brain swelling, computerized tomography

To improve the outcome of severely head-injured patients, factors that adversely influence the clinical course should be identified. The insights provided by computerized tomography (CT) allow the establishment of clinicopathological correlations that delineate the prognostic significance of the major intracranial lesions seen during the immediate posttraumatic period. One of the most threatening posttraumatic lesions is massive swelling (primarily defined in terms of volume) of a single cerebral hemisphere, which is most commonly associated with acute subdural hematoma and has a rapidly devastating course. Bulk enlargement of a cerebral hemisphere occurring within a few hours after injury may be due to increased intravascular blood volume, increased water content, or both. Until new developments in neuroimaging provide a means of determining the intimate mechanism of brain swelling in the clinical routine, one must rely on the correlation between the clinical and CT findings in patients with this type of lesion, and on a comparison of CT scans obtained shortly before death with brain slices obtained at autopsy.

Since autopsies of traffic-accident victims in Spain cannot be performed in the hospital setting, our study is limited to analyzing the clinical, CT, and intracranial pressure (ICP) findings. The clinical material consisted of 55 consecutive severely head-injured patients who developed bulk enlargement of a cerebral hemisphere within a few hours following trauma.
Clinical Material and Methods

This study includes 55 patients selected from a total series of 520 cases of severe head injury. The mechanism of trauma was a traffic accident in 41 patients, a fall in nine, and assault in five. Eighteen patients had relevant associated extracranial injuries. Nearly two-thirds of the patients were brought to our unit directly from the scene of the accident; the others were referred from local hospitals.

All patients were comatose as defined by the Glasgow Coma Scale (GCS) and all underwent conventional x-ray studies and a CT scan immediately after admission. Control CT scans were performed in 36 cases, the first generally within 24 hours after admission and then every 2 to 3 days until normalization of the midline structures or death. Nineteen patients died too soon for follow-up CT scans. The radiological criterion for inclusion in this study was evidence of expansion of one cerebral hemisphere causing marked midline shift (septal and pineal displacement) either alone (three cases) or in association with acute extradural hematoma (52 cases). Some patients had subarachnoid hemorrhage and two had intraventricular hemorrhage. We excluded patients who evidenced focal mass lesions such as brain contusion or intraparenchymal hematoma on the initial CT scan or follow-up CT scans. Also excluded were patients who showed a residual discrete midline shift with visible basal cisterns after surgery for acute extradural hematoma. Cerebral angiography was not performed so the rare possibility of posttraumatic carotid artery occlusion was not ruled out.

All patients with acute extracerebral hematoma, except 12 who showed very thin subdural collections, underwent hematoma evacuation immediately after admission. Four had a second operation several days later consisting of temporal decompressive craniectomy and resection of the temporal lobe tip. Removal of the extra-axial hematoma was performed within 4 hours after trauma in 20% of cases and four to 12 hours after trauma in the remainder. An intraventricular catheter or epidural fiberoptic sensor was implanted in all cases for continuous measurement of ICP immediately after admission or operation. Intracranial hypertension was managed according to a standardized protocol reported elsewhere which includes hyperventilation, administration of osmotic agents, and cerebrospinal fluid (CSF) drainage. When these conventional measures failed to keep ICP below 20 mm Hg, high doses of thiopental (4 to 10 gm/day, average 6 gm/day) were administered as continuous infusions; the doses were titrated according to ICP response. Patients so treated underwent pulmonary artery pressure monitoring. If systemic arterial hypotension occurred, it was managed with dopamine infusion and volume replacement. Thiopental plasma levels ranged from 2 to 18 mg/dl (average 8.5 mg/dl).

TABLE 1
Age distribution in 55 severely head-injured patients with posttraumatic cerebral hemispheric swelling

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10</td>
<td>6</td>
</tr>
<tr>
<td>11-20</td>
<td>11</td>
</tr>
<tr>
<td>21-30</td>
<td>19</td>
</tr>
<tr>
<td>31-40</td>
<td>8</td>
</tr>
<tr>
<td>41-50</td>
<td>4</td>
</tr>
<tr>
<td>51-60</td>
<td>4</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>3</td>
</tr>
</tbody>
</table>
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The final result was graded according to the Glasgow Outcome Scale as: good recovery, moderate disability, severe disability, vegetative state, and dead.

Results

The mortality rate was 87.2% in the 55 patients with cerebral hemispheric swelling (47 of whom had associated subdural hematoma), 41% in the entire series of severely head-injured patients (520 cases), and 62.5% in the total group of patients with acute subdural hematoma (107 cases). The 47 patients with hemispheric swelling and associated subdural hematoma were on average younger (28 vs. 35 years), had a higher incidence of peritraumatic hypotension-hypoxemia (47% vs. 9%), and were more frequently involved in traffic accidents (80% vs. 68% with high-speed trauma) than were other patients with acute subdural hematoma (cases of pure hematoma or hematoma associated with brain contusion).

The age distribution of the 55 patients in this study is shown in Table 1. Ages ranged from 3 months to 69 years, and 80% of the patients were under 40 years. Skull x-ray films showed a linear fracture in 25 patients. The fracture was ipsilateral to the affected cerebral hemisphere in 16 patients (including the five patients with associated epidural hematoma), contralateral in six cases, and bilateral in three cases.

Cerebral hemispheric swelling appeared as an isolated lesion on the admission CT scan in only three patients. In the other 52 patients, it was associated with a thin subdural hematoma (32 cases), a large subdural hematoma (15 cases), or a large epidural hematoma (five cases) (Figs. 1 and 2). In most patients with a laminar subdural collection, there was a striking disproportion between clot thickness and the degree of midline displacement. The involved cerebral hemisphere appeared either isodense (24 cases) or hypodense (31 cases) in comparison to the contralateral hemisphere. The amount of midline displacement seen on the admission CT scan and follow-up CT scans is reflected in Table 2. It can be appreciated that 82.9% of the patients had marked (> 8 mm) midline displacement, which diminished in only 21.8% of the patients after extracerebral hematoma evacuation. Accordingly, pupillary changes and motor reactivity improved in only 14 of these patients. Rapid intraoperative brain reexpansion with herniation through the craniotomy window or burr holes occurred in the majority of the patients operated on for associated extracerebral clot.

Table 3 shows the correlation between neurological status at admission, the occurrence of a lucid interval, peritraumatic hypotension and/or hypoxemia, pupil-

<table>
<thead>
<tr>
<th>Midline Displacement on Initial CT Scan</th>
<th>Cases</th>
<th>Percent</th>
<th>Changes on Control CT Scan</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td></td>
<td>Diminished*</td>
</tr>
<tr>
<td>4-8 mm</td>
<td>10</td>
<td>18.1</td>
<td>2 (2)</td>
</tr>
<tr>
<td>9-12 mm</td>
<td>29</td>
<td>52.7</td>
<td>6 (5)</td>
</tr>
<tr>
<td>&gt; 12 mm</td>
<td>16</td>
<td>29</td>
<td>4</td>
</tr>
<tr>
<td>total</td>
<td>55</td>
<td>100</td>
<td>12 (7)</td>
</tr>
</tbody>
</table>

* Figures in parentheses represent the number of survivors in each group.

Fig. 2. Computerized tomography (CT) scans in a 17-year-old boy who was admitted in a comatose state 15 minutes after injury. A: Admission CT scan performed when the patient was recovering consciousness just before undergoing emergency splenectomy. This scan was considered normal. B: Control CT scan following laparotomy and observation of a maximally dilated right pupil, showing a large epidural hematoma. After hematoma evacuation, right mydriasis was discretely reduced and the intracranial pressure (ICP) ranged between 40 and 50 mm Hg. An immediate follow-up CT scan (not illustrated) showed bulk enlargement of the right cerebral hemisphere with marked midline displacement. Raised ICP was controlled with high-dose thiopental which was not discontinued until midline normalization was appreciated. C: Control CT scan performed 6 days after hematoma evacuation showing decreased but persistent midline shift. D: Control CT scan performed 14 days after injury.
TABLE 3
Correlation between the admission GCS score, clinical findings at admission, and the final outcome*

<table>
<thead>
<tr>
<th>GCS Score</th>
<th>Cases</th>
<th>Lucid Interval</th>
<th>Hypotension/Hypoxemia</th>
<th>Pupillary Changes</th>
<th>MIDLINE SHIFT (mm)</th>
<th>ICP (mm Hg)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-5</td>
<td>45</td>
<td>81.8%</td>
<td>6 (1)</td>
<td>23</td>
<td>5 (1)</td>
<td>25 (2)</td>
<td>15 (1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7 (2)</td>
<td>22 (2)</td>
<td>16</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9 (2)</td>
<td>13 (2)</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 (1)</td>
<td>5 (1)</td>
<td>3 (1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 (1)</td>
<td>1 (1)</td>
<td>1 (2)</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>100%</td>
<td>8 (3)</td>
<td>26</td>
<td>14 (4)</td>
<td>26 (2)</td>
<td>15 (1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11 (3)</td>
<td>28 (4)</td>
<td>16</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11 (3)</td>
<td>18 (3)</td>
<td>26 (1)</td>
</tr>
<tr>
<td></td>
<td>total</td>
<td>55</td>
<td>100%</td>
<td>26</td>
<td>14 (4)</td>
<td>26 (2)</td>
<td>15 (1)</td>
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<td></td>
<td></td>
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<td>11 (3)</td>
<td>28 (4)</td>
<td>16</td>
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<tr>
<td></td>
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<td>18 (3)</td>
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</tbody>
</table>

* Figures in parentheses represent the number of survivors in each subgroup. GCS = Glasgow Coma Scale; ICP = intracranial pressure; GR = good recovery; MD = moderate disability; SD = severe disability; D = dead.

lary changes, midline shift, and the final outcome. Forty-five patients (81.8%) scored 5 points or less on the GCS. Only eight patients (14.5%, including three of the five with associated epidural hematoma) had a short lucid interval, the rest remaining unconscious throughout the course. Arterial hypotension and/or hypoxemia occurred in 26 patients (47.2%), and 74.2% had unilateral or bilateral mydriasis at admission. The incidence of peritraumatic brain ischemia, pupillary changes, marked midline displacement, and high ICP was significantly higher in patients with a GCS score of 3 to 5 than in those with a score of 6 to 8 (p < 0.001). However, mortality was very high in both subgroups (91% vs. 70%), the difference not being significant (χ² = 3.5; p > 0.05). Only five patients (9%) made a functional recovery.

Initial ICP was over normal limits in 44 patients (80%), and 26 patients (47.2%) had ICP over 40 mm Hg. Only one of the 11 patients with normal initial ICP continued to have low ICP throughout the course. Forty-seven patients (85.4%) received high doses of thiopental to control raised ICP. This type of therapy was omitted in eight patients because of impending arterial hypotension (seven cases) or normal ICP (one case). The ICP response to thiopental and the ICP curves throughout the course are reflected in Table 4 and Fig. 3, respectively. The ICP did not respond to thiopental in 24 patients (51%). Most of the nonresponders were treated in the years 1977 to 1982, when a ceiling dosage of 4 to 6 gm/day had been established. Fulminant, uncontrolled intracranial hypertension killed 15 of these patients within 1 to 3 days following admission. Another nine patients showed a more slowly progressive ICP elevation, dying 5 to 8 days after initiation of therapy. Resection of the temporal lobe tip and decompressive craniectomy led to transient ICP normalization in three of these patients, but pressure rose to uncontrollable levels within 1 to 3 days after surgery.

The ICP was initially controlled in 15 patients (31.4%) who died thereafter. In six of these patients, the thiopental dose was tapered following 2 to 4 days of normal ICP while there was a marked midline shift resulting in a fatal pressure rebound. In five other patients, thiopental had to be reduced or discontinued because of side effects also resulting in a refractory ICP rebound. Finally, four more patients had a delayed ICP elevation which could not be controlled in spite of increasing the thiopental dose.

Elevation of ICP was definitely controlled in eight patients (17%), among them the seven survivors in this series. Thiopental withdrawal in these patients was not initiated until complete disappearance of the mass effect and midline normalization were appreciated in the follow-up CT scan (Fig. 2). Temporal decompressive craniectomy apparently contributed to ICP control in one of the survivors.

Table 4 shows the correlation between ICP response to thiopental administration, different clinical variables, and final outcome. Most nonresponders had a very low GCS score (87.5% scored 3 to 5 points), marked pupillary changes (79% had unilateral or bilateral mydriasis), marked midline shift (91% had > 8-mm midline displacement), and a very high initial ICP (> 40 mm Hg in 41% of cases). The clinical condition of patients manifesting a transient response to thiopental was as poor as that of nonresponders: 80% had a GCS score of 3 to 5, 66% had unilateral or bilateral mydriasis, 80% had a GCS score of 3 to 5, and 66% had unilateral or bilateral mydriasis.
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TABLE 4

ICP response to thiopental related to different clinical variables and survival in 55 patients*

<table>
<thead>
<tr>
<th>Response to Thiopental</th>
<th>No. of Cases</th>
<th>GCS Score</th>
<th>Mydriasis</th>
<th>Midline Shift (mm)</th>
<th>Initial ICP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>3-5</td>
<td>6-8</td>
<td>Unilat</td>
<td>Bilat</td>
</tr>
<tr>
<td>none</td>
<td>24</td>
<td>21</td>
<td>3</td>
<td>12</td>
<td>7</td>
</tr>
<tr>
<td>transient</td>
<td>15</td>
<td>12</td>
<td>3</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>sustained</td>
<td>8 (7)</td>
<td>5 (4)</td>
<td>3 (3)</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>untreated</td>
<td>8</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

* Figures in parentheses represent the number of survivors in each subgroup. ICP = intracranial pressure; GCS = Glasgow Coma Scale.

driasis, 80% had a midline shift greater than 8 mm, and in 60% the ICP was over 40 mm Hg. All of these patients died. The patients who showed a definitive ICP response had a better clinical status (62.5% with a GCS score of 3 to 5), less midline shift (62.4% with shift > 8 mm), and lower initial ICP (25% with pressure > 40 mm Hg).

Discussion

In spite of improved diagnostic methods and aggressive therapeutic modalities, the mortality rate of severely head-injured patients is still 20% to 50% in most series. The highest mortality rate observed among patients with acute subdural hematoma (41% to 90%), with overall series mortality being directly proportional to the number of subdural hematoma cases. The lethality of acute subdural hematoma is largely explained by its frequent association with primary (impact damage) and/or secondary brain damage consisting of contusion, laceration, swelling, or edema (the so-called “complicated subdural hematoma”). Perhaps the most severe lesion associated with subdural hematoma is acute swelling of the ipsilateral cerebral hemisphere, which is almost always associated with severe intracranial hypertension and leads to death within hours or a few days after trauma.

Since the radiological definition of this lesion is not yet well established, its true incidence in severely head-injured patients is difficult to estimate. According to the conventional CT definition used in this study, 55 (10%) of our patients with severe head injury had cerebral hemispheric swelling, which was associated with a more or less voluminous ipsilateral subdural hematoma in 85.4% of the 55 cases and with a large epidural hematoma in 9%; in only three patients (5.4%) did this appear as an isolated pathology. Cerebral hemispheric swelling was associated with not only the highest mortality rate in our series of severely head-injured patients, but also the highest incidence of uncontrollable intracranial hypertension and the shortest survival period after injury. After examining the records of our patients, it becomes clear that, independent of the role played by the extraaxial hematoma in the production of cerebral hemispheric swelling, it was the latter lesion that dictated the clinical course. The limited or null effect of extra-axial hematoma evacuation on the clinical status and midline displacement strongly suggests that bulk enlargement of the cerebral hemisphere was predominant in the patients’ course. This was particularly evident in nearly half of the cases in which the amount of extracerebral blood collection was rather insignificant.

Jamieson and Yelland classified 40.5% of the 553 traumatic subdural hematomas in their series as “complicated hematoma.” This type of lesion was most often observed after severe acceleration-deceleration injuries and carried the highest mortality rates in the series (2.5 times greater than simple subdural hematoma and 1.3 times more than hematoma plus brain contusion). According to these authors, “the swelling of the damaged brain added to the sometimes relatively minor accumulation of blood in the subdural space may explain the rapid course of complicated subdural hematoma.” McEvoy et al. found “hemisphere pulping and massive edema” in 11 fatal cases in their series of 125 patients with traumatic epidural hematoma. Heiskanen observed widespread diffuse cerebral contusion of the ipsilateral cerebral hemisphere in seven of the 10 patients who died in his series of epidural hematoma. However, neither these reports nor other studies on extracerebral hematoma performed during the CT era make a distinction between multifocal brain contusion-hematoma and diffuse swelling as a cause of cerebral hemisphere enlargement. Using the CT scan to differentiate these conditions, we excluded from this study patients showing brain contusive foci, hematoma, or focal infarct. Unfortunately, CT scanning does not accurately distinguish whether hemispheric enlargement is due to increased water content or increased cerebral blood volume and cannot differentiate between brain edema and the early stages of infarction.

The physiopathology of posttraumatic brain swelling is a matter of controversy. Taking into account its rapid development after injury, a sudden increase in cerebral blood volume resulting from cerebrovascular dilatation seems a more likely mechanism than acute edema formation. Diffuse swelling of a cerebral hemisphere occurring as early as 20 to 30 minutes after injury has been documented in two pa-
tients in whom the CT density numbers of the involved hemisphere were higher than in the contralateral unaffected hemisphere. The rapid resolution of the swelling effect in these two cases supports the interpretation that hemisphere enlargement was due to vascular engorgement of the type described by Langfitt and his colleagues in the 1960's. However, laboratory work and direct measurement of brain water content in head-injured patients have demonstrated that posttraumatic brain edema may occur much more rapidly than is usually thought. Densitometric and dynamic CT studies seem to indicate that acute edema formation is a common cause of bulk brain enlargement in cases of fatal head injury. Although planimetric analysis was not performed in our patients, it should be noted that in 56% of them the affected cerebral hemisphere appeared hypodense in comparison with the contralateral hemisphere. On the other hand, the prolonged duration of the mass effect and the null effect of hyperventilation on high ICP suggest edema formation as the mechanism of cerebral hemisphere enlargement in our patients.

This type of brain swelling is probably different from the generalized swelling most commonly observed in children and young adults as a self-limited phenomenon causing increased CT numbers and moderately raised ICP, apparently due to transient hyperemia and easily controlled by hyperventilation. These two basic mechanisms of brain expansion (hyperemia and edema) are not mutually exclusive, and vascular engorgement that is severe and persistent may result in widespread brain hypoxia and edema formation. It has been postulated that severe brain trauma may damage the hypothalamic and brain-stem motor centers, triggering vasomotor paralysis with a sudden increase in cerebral blood volume, which in turn would lead to a rise in ICP, compression of cerebral veins, increased cerebrovascular resistance, cerebral blood flow decreased to ischemic levels, and edema formation. Vasodilation might also be a direct response of cerebral vessels to mechanical injury. In patients with associated large extracerebral hematoma, a critical reduction in cerebral blood flow may result from the local pressure exerted by the clot plus the reduction in cerebral perfusion pressure caused by ICP elevation to almost systemic blood pressure. A delay of more than 4 hours between trauma and hematoma evacuation occurred in five of our patients with epidural hematoma and in 80% of those with large subdural hematoma in this series. The fact that we have never observed cerebral hemisphere swelling in patients operated on for acute extracerebral hematoma within 2 hours of injury corroborates the decisive influence of rapid hematoma evacuation.

Finally, peritraumatic hypotension and/or hypoxemia (recorded in 47% of our patients) and posttraumatic cerebrovascular spasm may also contribute to edema formation.

High ICP, usually associated with cerebral hemispheric swelling, is extremely difficult to control. Posttraumatic brain swelling is not amenable to surgical treatment, and large decompressive craniectomy is useless or even detrimental. Temporal lobe tip resection with temporal craniectomy decreased ICP for only a short period of time in our patients. High-dose barbiturates given immediately after diagnosis produce a rapid decrease in ICP followed by overall inhibition of metabolic activity with the consequent reduction in cerebral oxygen substrate requirements and seems the most effective available therapy. High-dose thiopental prevents elevation of blood pressure, which may increase intraluminal pressure in brain areas with altered blood-brain barrier permeability aggravating vasogenic edema. Use of thiopental requires meticulous control of extracerebral parameters since arterial hypotension may intensify interference with cerebral blood flow in ischemic regions. The seven survivors in this study were among the eight responders to thiopental. The majority of patients (60%) showed only a discrete response or none, but a retrospective analysis indicates that some nonresponders received either short doses of thiopental or adequate doses for only a short time. According to our experience, once ICP is controlled, thiopental should not be discontinued until complete disappearance of the mass effect is seen in the control CT scan, an event which may occur as late as 15 days after onset of therapy.

**Conclusions**

Patients with severe head injury who present with acute bulk enlargement of one cerebral hemisphere have a small but real chance for a functional recovery. Rapid diagnosis and removal of acute extracerebral hematoma seem critical for preventing the development of this posttraumatic complication which requires immediate control of raised ICP to avoid a fulminant, malignant course. Laboratory and clinical research to improve our understanding of the multifaceted mechanisms of brain edema and forthcoming developments in neuroimaging should provide a basis for a more rational and specific treatment of this grave posttraumatic lesion.

**References**


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