Delayed intracerebral haematomas in moderate to severe head injuries in young adults

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Key words: Head injury; Delayed haematoma; Intracranial pressure; Cerebral perfusion pressure

The incidence of delayed intracerebral haematoma formation in moderate to severely head-injured young adults who do not have signs of cerebral contusions on the initial post-trauma head CT scan has been studied. Thirty patients were reviewed and in 6 (20%) a delayed haematoma was diagnosed on a later CT scan, when signs of cerebral contusions were absent initially. Recurrence of intracranial hypertension within 3 to 4 h proved to be a significant indication for the development of these haematomas, despite the initial but inconsistent good response to osmotic diuretics. The average Glasgow outcome score was 3, including two mortalities. Cerebral perfusion pressure directly correlated with the outcome.

Intracranial haematoma is the most common cause of preventable death after head injury (1). Intracranial hypertension is closely associated with increased morbidity and mortality after head injury (2). Early detection and urgent evacuation of a haematoma with continuous monitoring and control of intracranial pressure would be expected to reduce the mortality from severe head injury (3).

Delayed intracerebral haematomas in head-injury patients can occur without indication on preliminary CT scanning. Such haematomas may collect over a span of several hours during which the associated intracranial hypertension will respond initially to medical treatment. This may lead to misinterpretation of the actual cause of the intracranial hypertension and will delay the diagnosis and the appropriate treatment.

Methods and materials

Case notes of moderately to severely head-injured patients admitted to the Neurosurgery Department at St Bartholomew’s Hospital, London, between June 1988 and June 1989 were reviewed. Severity of the head injury was assessed according to the initial Glasgow coma score on admission (4). Patients who scored 8 or less were included. Thirty patients were reviewed: 18 males and 12 females with a mean age of 35.7 and 27.3 years, respectively. Their average scores on the Glasgow coma scale were 6.0 and 6.83. All had head CT scans and the relevant radiographic and blood investigations and were managed according to a defined protocol: pharmacological sedation, paralysis and mechanical hyperventilation; continuous monitoring of intracranial (ICP)*, central venous, and arterial pressures; regular administration of phenytoin, H2-receptor antagonist, and mannitol 20% if the initial ICP was more than 20 mmHg. Fluids and electrolytes were balanced, blood loss was replaced. Inotropes, where indicated, were titrated against blood pressure to maintain a cerebral perfusion pressure above 70 mmHg. Propofol infusion was administered in cases of resistant intracranial hypertension (5,6).

Head CT scans were repeated at 24 h after injury or sooner in cases of persistent intracranial hypertension. Delayed intracerebral haematoma was diagnosed if the second post-trauma CT scan was positive, provided no signs of haematoma or contusions were present on initial scanning.

The cerebral perfusion pressure (CPP) was calculated as the gradient of the mean arterial and intracranial pressures, which were monitored continuously.

The Glasgow Outcome Score (GOS): a continuum scale of 1 to 5, where excellent recovery scores 5, and

* Codman ICP Subarachnoid Bolt # 81 8194
death scores 1, was used to assess the outcome of these patients (7).

Statistical analysis

Results are expressed as means ± standard error of the mean (s.e.m.). Pearson's correlation coefficient for linear regression \( r \), was calculated for the relation between the cerebral perfusion pressure (CPP), and the Glasgow outcome score (GOS).

Student's \( t \) test was applied to obtain the \( P \) value.

Case reports

Case 1
A 16-year-old boy was involved in a road traffic accident as a passenger in a car and sustained craniocerebral and chest injuries. Initial GCS score was 7.

Head CT scan, 3 h after injury showed diffuse cerebral swelling (Fig. 1). A repeat CT scan, 13 h later, showed a large butterfly posterior callosal haematoma (Fig. 2).

Case 2
An 18-year-old girl, hit by a train in a suicide attempt sustained multiple injuries: craniocerebral, chest with right pneumothorax, pelvis, and bilateral femoral fractures. Initial GCS score was 7.

Head CT scan, obtained 4 h after injury, revealed diffuse cerebral swelling (Fig. 3). A large intracerebral left frontotemporal haematoma was shown on subsequent scanning 22 h later (Fig. 4).

Case 3
A 30-year-old man, hit on the forehead by a swinging heavy object at work, sustained a craniocerebral injury. Initial GCS score was 9. At 4 h after injury, a head CT
scan showed diffuse cerebral swelling. A large right frontal intracerebral haematoma was demonstrated on CT scan 17 h later.

Case 4
A 23-year-old woman, hit by a speeding car, sustained craniocerebral, chest and contusional injuries of the lung. Initial GCS score was 5.

At 3 h after the accident, a head CT scan showed ethmoid and sphenoid basal fractures with diffuse cerebral swelling. A repeat CT scan, 9 h later, showed a left temporal intracerebral haematoma.

Case 5
A 28-year-old man fell from a bus and sustained a craniocerebral injury. Initial GCS score was 7.

At 2 h after the injury a head CT scan showed extensive occipital and skull base fractures, diffuse cerebral swelling and mild bifrontal contusions. A large right temporal intracerebral haematoma was revealed on CT scan 17 h later.

Case 6
An 18-year-old boy was knocked off his bicycle by a car and sustained a head injury, with an initial GCS score of 10, deteriorating over a span of 2 h to a score of 6. A head CT scan, 4 h after injury revealed a left temporoparietal extradural haematoma (Fig.5). This was evacuated via a temporoparietal craniotomy and the patient was kept ventilated in view of the delayed treatment. Repeat CT scan at 30 h after injury showed a left temporal intracerebral haematoma that was not present initially (Fig.6).

Results
Out of thirty patients reviewed, six developed delayed intracerebral haematomas. The details of the six patients are given in the case reports. These six patients had a younger mean age and a higher mean Glasgow Coma Score (GCS) when compared with the group as a whole. However, their Glasgow Outcome Score (GOS) was lower (Tables I, II). All patients underwent craniotomy for evacuation of the delayed haematomas. Patient 1 had a CT-guided stereotactic burr hole evacuation. The average time lapse from injury until diagnosis was 20 ± 3 h (range 12–30 h). The mean intracranial pressure (ICP) was 28 ± 4 mmHg. The mean cerebral perfusion pressure (CPP) was 58 ± 5 mmHg. The average time response to osmotic diuresis was 3 h. The average time for recurrence of intracranial hypertension was 4 h. Two patients (4 and 5) out of the six, died.

The average Glasgow Outcome Score (GOS) for the six cases was 2.8 ± 0.7, including the two mortalities.

There was a positive linear correlation between the CPP and the GOS ($r = 0.97, P < 0.01$), (Fig.7).

Discussion
Our findings suggest an incidence of 20% of delayed intracerebral haematoma in young adults with moderate to severe head injuries, and a mortality of 33%. Cerebral contusions are associated with delayed deterioration, and can be used as predicting markers (8). Five of our patients developed delayed intracerebral haematomas without showing any contusions on initial scanning. Delayed haematomas may also occur at sites other than
Table I. Comparison of age, sex, Glasgow Coma Scale (GCS) and Glasgow Outcome Score (GOS) between patients with delayed haematoma and the whole patient population

<table>
<thead>
<tr>
<th>Total patients</th>
<th>n</th>
<th>%</th>
<th>Sex</th>
<th>Mean age (years)</th>
<th>Mean GCS (3-15)</th>
<th>Mean GOS (3-5)</th>
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<tr>
<td></td>
<td>18</td>
<td>60</td>
<td>M</td>
<td>35.7</td>
<td>6.0</td>
<td>3.22</td>
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<tr>
<td></td>
<td>12</td>
<td>40</td>
<td>F</td>
<td>27.4</td>
<td>6.83</td>
<td>3.25</td>
</tr>
<tr>
<td>Delayed haematoma</td>
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<td>22.2</td>
<td>M</td>
<td>23.0</td>
<td>7.5</td>
<td>3.25</td>
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<td>16.6</td>
<td>F</td>
<td>20.5</td>
<td>6.5</td>
<td>2.5</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>20.0</td>
<td>—</td>
<td>22.2</td>
<td>7.2</td>
<td>3.0</td>
</tr>
</tbody>
</table>

Table II. Patients’ findings: time lapse between injury and diagnosis, initial GCS, perfusion pressure and outcome

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>GCS 3-15</th>
<th>Time (h)</th>
<th>ICP (mmHg) ± s.e.m.</th>
<th>CPP (mmHg) ± s.e.m.</th>
<th>GOS 3-5</th>
</tr>
</thead>
<tbody>
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<td>M</td>
<td>16</td>
<td>7</td>
<td>22</td>
<td>27 ± 2</td>
<td>62 ± 3</td>
<td>3</td>
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<tr>
<td>2</td>
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<td>7</td>
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<td>57 ± 2</td>
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<td>3</td>
<td>M</td>
<td>30</td>
<td>9</td>
<td>21</td>
<td>21 ± 2</td>
<td>65 ± 2</td>
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<td>F</td>
<td>23</td>
<td>5</td>
<td>12</td>
<td>42 ± 3</td>
<td>41 ± 4</td>
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<tr>
<td>5</td>
<td>M</td>
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<td>7</td>
<td>28</td>
<td>37 ± 2</td>
<td>48 ± 2</td>
<td>1</td>
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<tr>
<td>6</td>
<td>M</td>
<td>18</td>
<td>6</td>
<td>30</td>
<td>14 ± 2</td>
<td>72 ± 2</td>
<td>5</td>
</tr>
</tbody>
</table>

ICP, intracranial pressure ± s.e.m., mmHg
CPP, cerebral perfusion pressure ± s.e.m., mmHg (gradient of intracranial and mean arterial pressures)
GCS, Glasgow coma scale (3-15)
GOS, Glasgow outcome score (1-5)
n, number of recordings (30 min intervals)
s.e.m., Standard error of the mean

Those noted to be contused on preliminary scan, as shown in Case 5.

The average time between the first head CT scan and connection to the ICP monitor was approximately 60 min. All these cases showed diffuse cerebral swelling without contusions and raised intracranial pressure from the outset. Equally, all of them responded to osmotic diuretic treatment. However, all of them had recurrent intracranial hypertension within 3-4 h. The response to osmotic diuresis was progressively reduced on subsequent occasions. It was the absence of cerebral contusions which led to the possible delay in identifying the development of the haematoma. It will remain difficult to decide when such a haematoma has formed. Critical analysis of the ICP readings is essential to diagnosis. Variability of monitoring equipment (9), wide diastatic fractures, cerebrospinal fluid leakage, craniotomy, and other coexisting injuries (10) ought to be accounted for. Provided hypovolaemia, hypotension, hypoxaemia, hypercarbia, or jugular venous blockade do not exist, the length of the response to osmotic diuresis and intracranial hypertension recurrence are the factors that best

Figure 7. Correlation between cerebral perfusion pressure and Glasgow Outcome Score (r=0.97, P<0.01).
predict the development of a delayed intracerebral haematoma.

Compared with the whole series, the six patients with delayed haematoma had a younger mean age and a higher initial GCS; however, they had a lower GOS (Table I). Cerebral perfusion pressure (CPP) is directly proportional to the mean arterial blood pressure and inversely related to the intracranial pressure. Evacuation of an intracerebral haematoma, in the presence of intracranial hypertension, is essential for optimising both global and regional cerebral perfusion (11). A wide craniotomy, evacuation of the haematoma, and bone flap removal in cases of severe brain swelling will achieve adequate decompression. This may reduce the possibility of ischaemic sequela (12,13). In all these patients, surgical evacuation of the intracerebral haematoma relieved the intracranial hypertension and improved the CPP. However, the two fatalities (Cases 4 and 5) had persistent severe brain swelling, and surgical evacuation did not alter the outcome. Whether the persistent diffuse brain swelling was primarily due to the initial injury, or was secondary to the haematoma and delayed evacuation is uncertain.

The Glasgow Outcome Score (GOS), where mortality and excellent recovery, score 1 and 5, respectively, can be used as a crude, semiquantitative measure of neurological outcome to assess morbidity and mortality (7). These cases showed a significant linear correlation between the GOS and the cerebral perfusion pressure. This emphasises the importance of CPP, especially in pathological conditions where cerebrovascular autoregulation may be impaired.

Head-injured patients are often transferred back to the referring district general hospital if the head CT scan in the acute phase does not show any intracranial 'surgical pathology'. Subsequent neurological deterioration in such patients may be caused by newly developing haematomas. Efficient anticipating treatment is therefore essential to improve the outcome of severe head injuries.

During this work A H S Huneidi was supported by a grant from the Joint Research Board of St Bartholomew's Hospital Medical College.

This work was presented, in part, at the meeting of the International College of Surgeons (British Section) in May 1990, in London.

References


Received 5 December 1991

Invited comment

I consider this an important paper because it emphasises the need for repeated scanning if the clinical or physiological measurement, including that of intracranial pressure, give cause for concern. While intensive management such as this produces good long-term results in many brain-damaged patients, it has to be acknowledged that the clinical signs are abolished thereby. There is no point in waiting for a dilating pupil, which is the only hard physical sign left, since by that time surgical remedies may well be too late.

I would comment that if the initial CT scan showed some diffuse swelling with some certainty, then the brain swelling was there at that time. This raises the interesting problem of whether tamponade by an increased intracranial pressure in fact reduces the risk of development of intracerebral haematomas. It is unlikely to prevent the