
The effect of intracerebral hematoma location on the risk of brain-stem compression and on clinical outcome

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The clinical findings and computerized tomography (CT) brain scans of 45 patients with supratentorial intracerebral hematomas were evaluated to determine the effect of hematoma location on the clinical course and outcome of the disease. The lesions were frontal in 18 patients, temporal or temporoparietal in 17, and parieto-occipital in 10. No patient with a frontal or parieto-occipital hematoma had clinical signs of transtentorial herniation at admission or subsequently, whereas seven (41%) of those with temporal or temporoparietal lesions had signs of herniation (p < 0.05); three of these seven patients had an abnormal mental status, ipsilateral anisocoria, and lateralizing motor findings at admission, and four developed these signs within 12 hours after admission, necessitating urgent surgical intervention. The mean volume of the lesions estimated from the CT scans was similar in the three groups (frontal 47 ± 28 cc; parieto-occipital 53 ± 26 cc; temporal/temporoparietal 41 ± 21 cc). None of the six patients with temporal or temporoparietal hematomas smaller than 30 cc had signs of tentorial herniation, compared with seven (64%) of 11 patients with larger hematomas (p < 0.05); in six of these seven cases, the hematoma was caused by head injury. Patients with a temporal or temporoparietal hematoma had a worse outcome than those in the other two groups, and no patient with signs of tentorial herniation had a good outcome. Patients with temporal or temporoparietal hematomas appear to be at greater risk of brain-stem compression, especially if the lesion is larger than 30 cc and caused by head injury, than are those with hematomas in other sites. In such cases, prompt surgical intervention should be considered.

KEY WORDS • intracerebral hematoma • head injury • hypertensive hemorrhage • tentorial herniation

INTRACEREBRAL hematomas may be caused by head injury or arise as a complication of hypertension, rupture of an aneurysm or arteriovenous malformation, anticoagulation therapy, bleeding disorders, or intracranial tumors. Most traumatic hematomas develop 6 hours or more after injury, causing further depression of consciousness, failure to improve, or new focal neurological findings. Spontaneous intracerebral hematomas usually result in the sudden onset of headache, focal neurological deficits, and depression of consciousness. In both groups, loss of consciousness and brain-stem compression markedly worsen the prognosis regardless of treatment. Although it is well known that cerebellar hematomas may rapidly lead to brain-stem compression, and prompt surgical removal is recommended, previous studies have not suggested a similar concern with supratentorial hematomas. This report describes a series of patients with supratentorial hematomas and minimal other intracranial abnormalities in whom brain-stem compression was commonly associated with temporal or temporoparietal lesions.

Clinical Material and Methods
The medical records and computerized tomography (CT) brain scans of all patients admitted to the neurosurgical service at San Francisco General Hospital between July 1, 1983, and August 1, 1987, with the diagnosis of supratentorial intracerebral hemorrhage were reviewed. Patients who had a significant neurological deficit or who were in a coma before developing an intracerebral hematoma were excluded to avoid confounding the effects of the mass lesion with the effects of brain contusion or direct axonal or brain-stem injury.
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FIG. 1. Axial computerized tomography scans obtained without contrast enhancement showing a large (79-cc) frontal hematoma in a patient with no signs of brain-stem compression (left), a large (89-cc) parieto-occipital hematoma in a patient with no signs of brain-stem compression (center), and a small (43.5-cc) temporal hematoma in a patient who developed signs of tentorial herniation 8 hours after admission (right).

Also excluded were patients whose CT scans showed intracranial pathology other than a supratentorial hematoma localized primarily to a single lobe.

Each patient underwent cranial CT scanning at admission or at the time of clinical deterioration. The volume of the hematoma was estimated by calculating the product of the anteroposterior, medial-lateral, and superoinferior diameters of the lesion on axial CT scans. The maximal midline shift was also measured. A craniotomy was performed to evacuate the hematoma if there was a focal neurological deficit, neurological deterioration, or failure to improve. The outcome was measured according to the Glasgow Outcome Scale at the time of discharge.

The unpaired t-test was used to determine the statistical significance of differences in hematoma size and midline shift between groups, and the correlation coefficient was calculated to evaluate the association between these two variables. The chi-square test was used to compare frequency differences between groups.

Results

Patient Population

Forty-five patients met the entry criteria. There were 34 males and 11 females, ranging in age from 6 to 89 years (mean 47 years). The patients were separated into three groups according to the location of the hematoma. The hematomas were located in the frontal region in 18 patients, in the temporal or temporoparietal region in 17, and in the parieto-occipital region in 10 (Fig. 1). These lesions occurred after head injury in 32 patients; they were discovered at admission in 28 cases and developed later in four. Thirteen patients had nontraumatic hematomas, which were related to hypertension in five, bacterial endocarditis in two, embolic events in two, and rupture of a middle cerebral artery aneurysm in one. In three patients, the cause of the hematoma was unknown despite cerebral arteriography. The three groups were well matched for age and for mechanism of injury, although the group with parieto-occipital hematomas had fewer traumatic lesions than the other two groups.

Clinical Presentation

The initial scores on the Glasgow Coma Scale (GCS) and the frequency of hemiparesis at admission in each group are summarized in Table 1. The differences in the GCS scores and in the frequency of hemiparesis between groups were not statistically significant.

No patient with a frontal or parieto-occipital hematoma had abnormal brain-stem reflexes or the clinical triad of tentorial herniation (anisocoria, hemiparesis, and a decreased level of consciousness) at admission or developed them later. Among patients with temporal or temporoparietal hematomas, ipsilateral clinical signs of tentorial herniation were present at admission in three and developed within 12 hours after admission in four (p < 0.05). Six of seven patients with temporal or

<table>
<thead>
<tr>
<th>Hematoma Location</th>
<th>No. of Cases</th>
<th>GCS Score &gt; 8</th>
<th>≤ 8</th>
<th>Hemiparesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>frontal</td>
<td>18</td>
<td>14 (78%)</td>
<td>4 (22%)</td>
<td>6 (33%)</td>
</tr>
<tr>
<td>parieto-occipital</td>
<td>10</td>
<td>9 (90%)</td>
<td>1 (10%)</td>
<td>3 (30%)</td>
</tr>
<tr>
<td>temporal or temporoparietal</td>
<td>17</td>
<td>15 (88%)</td>
<td>2 (12%)</td>
<td>3 (18%)</td>
</tr>
</tbody>
</table>

*GCS = Glasgow Coma Scale. Numbers in parentheses are percentages of the total cases in each group.
temporoparietal hematomas and signs of brain-stem compression had a head injury.

**Computerized Tomography Findings**

Evaluation of the CT scans showed that the mean volume (± standard error of the mean) of temporal and temporoparietal hematomas (41 ± 21 cc) was not significantly different from that of frontal (47 ± 21 cc) or parieto-occipital (53 ± 26 cc) hematomas. In the temporal/temporoparietal group, none of the six patients with hematomas of 30 cc or smaller had signs of brain-stem compression at admission or subsequently, compared with seven of 11 patients with larger hematomas (p < 0.05). There was no correlation between the hematoma size and midline shift on the CT scan in patients with frontal (r = 0.426) or parieto-occipital hematomas (r = 0.24). Among patients with temporal or temporoparietal hematomas, however, the correlation was much stronger (r = 0.829) (Fig. 2).

**Surgical Treatment**

Eight of 18 patients with frontal hematomas underwent a craniotomy (two within 6 hours after admission, four within 6 to 12 hours, one on the 2nd day, and one approximately 1 week after admission); in two patients, a catheter was placed to monitor intracranial pressure (ICP). Among the 10 patients with parieto-occipital hematomas, one had a craniotomy and one a ventriculostomy 12 hours after admission, and three had craniotomies within 4 to 7 days. Ten of 17 patients with temporal or temporoparietal hematomas underwent surgical treatment, including five craniotomies within the first 6 hours, three within 3 to 16 hours after admission, and one on the 4th day; one patient had a catheter placed to monitor ICP.

Two of the three patients with signs of tentorial herniation at admission underwent immediate hematoma evacuation and temporal lobectomy. The third patient, who had anisocoria but bilaterally reactive pupils, a mild hemiparesis, and changes in mental status, had a very small temporal hematoma (31.5 cc) that improved with conservative treatment (mannitol and dehydration). In all four patients who developed signs of tentorial herniation during the first 12 hours after admission, a hematoma was diagnosed by CT scanning and was evacuated immediately.

**Outcome**

The outcome at the time of hospital discharge was much worse among the 17 patients with temporal or temporoparietal hematomas than among those in the other two groups (Table 2). Only three (18%) of these 17 patients had a good outcome (p < 0.05 vs. the frontal or parieto-occipital group); seven were moderately disabled, and two were severely disabled. All five deaths in the temporal/temporoparietal group were due to the hematoma, compared with only two of four in the frontal hematoma group and none of those in the parieto-occipital hematoma group. No patient with signs of brain-stem compression had a good outcome; two were moderately disabled, two were severely disabled, and three (43%) died.

**Discussion**

There has been continued controversy over the surgical treatment of intracerebral hematomas. In the past, most spontaneous supratentorial hematomas not due to rupture of an arteriovenous malformation or aneurysm were treated conservatively, partly as a result of the report by McKissock, et al., that patients treated surgically did no better than those treated medically. Currently, the only clear indication for surgical treatment of spontaneous supratentorial hematomas is a lobar hemorrhage that causes a progressive neurological deficit despite medical therapy. The criteria for removal of traumatic hematomas include depressed consciousness or focal neurological deficit associated with a large hematoma in an anatomically appropriate location, a deteriorating or unimproved neurological status, intractable

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**TABLE 2**

**Outcome at the time of hospital discharge**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Hematoma Location</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Frontal</td>
</tr>
<tr>
<td>good</td>
<td>8 (44%)</td>
</tr>
<tr>
<td>moderately disabled</td>
<td>3</td>
</tr>
<tr>
<td>severely disabled</td>
<td>3</td>
</tr>
<tr>
<td>dead</td>
<td>4 (22%)</td>
</tr>
<tr>
<td>total cases</td>
<td>18</td>
</tr>
</tbody>
</table>

*Outcome was assessed by the Glasgow Coma Scale. Numbers in parentheses are percentages of the total cases in each group.

†Statistical significance: p < 0.05 (chi-square test) versus the parieto-occipital group.
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...elevation of ICP, or a significant degree of midline shift on the CT scan. Cerebellar hematomas can lead rapidly to brain-stem compression. Urgent evacuation has been recommended if there is clinical evidence of brain-stem dysfunction or altered consciousness, or if the hematoma is larger than 3 cm in diameter or 30 cc in volume as estimated from the CT scan. Similar recommendations have not been made for patients with supratentorial hematomas.

In our series, patients with a temporal or temporoparietal hematoma were at greater risk of transtentorial herniation from compression of the upper brain stem than were those with frontal or parieto-occipital hematomas of similar volume (41% vs. 0%, respectively); as a result, outcome in these cases was the worst. Temporal or temporoparietal hematomas probably cause brain-stem compression because the lesion is contained by the middle fossa anteriorly, laterally, and inferiorly and by a large mass of normal brain superiorly and posteriorly. Therefore, the path of least resistance may be medial into the ambient cistern and toward the brain stem. In addition, the temporal lobe is closer to the midbrain than are the frontal or parieto-occipital regions. Last, whereas the falc cerebri may prevent significant acute midline shift by even a large frontal or parieto-occipital hematoma, no such barrier exists for hematomas within the temporal lobe. Indeed, the correlation between midline shift and the size of the hematoma was much stronger in patients with temporal or temporoparietal lesions than it was in those with frontal or parieto-occipital lesions. Marshall (unpublished data, 1985) reported that patients with temporal lobe mass lesions are at greater risk of brain-stem compression despite normal ICP than are patients with mass lesions in other locations.

Transtentorial herniation occurred only in patients with temporal or temporoparietal hematomas larger than 30 cc. Nearly two-thirds of such patients developed brain-stem compression, compared with none of those with smaller lesions. This finding agrees, coincidentally, with that previously reported for cerebellar hematomas and suggests that temporal or temporoparietal hematomas larger than 30 cc should also be evacuated promptly.

Head injury was the cause of the hematoma in six of the seven patients who developed brain-stem compression. This may simply reflect the predominance of head injury in this group or, alternatively, may indicate that traumatic hematomas are more likely to enlarge and compress the brain stem. This complication has not been commonly reported in patients with spontaneous supratentorial hematomas. Ropper and Davis noted that seven of eight patients with spontaneous temporal lobe hematomas had focal neurological deficits but no signs of brain-stem compression; one patient was admitted in a coma. In the series of Kase, et al., two of eight patients with spontaneous temporoparietal lobe hemorrhages were comatose at admission and subsequently died. In neither report was it stated whether the comatose patients had brain-stem compression.

The mortality rate after spontaneous lobar hematomas in all sites is reported to be low (between 11.5% and 21%). While a specific association between the location of traumatic hematomas and brain-stem compression has not previously been established, Solomonik, et al., reported that traumatic hematomas in the temporal lobe resulted in a higher mortality rate than those in other sites (57% vs. 37%, respectively). Owing to the poor outcome of patients with brain-stem compression, the highest mortality rate in our series was among patients with temporal or temporoparietal hematomas.

In summary, patients with hematomas in the temporal or temporoparietal region are more likely to have signs of tentorial herniation at admission or to develop them within 12 hours than are patients with frontal or parieto-occipital hematomas. Patients with temporal or temporoparietal hematomas larger than 30 cc, particularly after head trauma, are at greater risk for brain-stem compression and must be considered for early evacuation of their mass lesions.

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