Posttraumatic cerebral arterial spasm: transcranial Doppler ultrasound, cerebral blood flow, and angiographic findings

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Thirty patients admitted after suffering closed head injuries, with Glasgow Coma Scale scores ranging from 3 to 15, were evaluated with transcranial Doppler ultrasound monitoring. Blood flow velocity was determined in the middle cerebral artery (MCA) and the intracranial portion of the internal carotid artery (ICA) in all patients. Because proximal flow in the extracranial ICA declines in velocity when arterial narrowing becomes hemodynamically significant, the extracranial ICA velocity was concurrently monitored in 19 patients. To assess cerebral perfusion, cerebral blood flow (CBF) measurements obtained with the intravenous $^{133}$Xe technique were completed in 16 patients. Vasospasm, designated as MCA velocity exceeding 120 cm/sec, was found in eight patients (26.7%). Severe vasospasm, defined as MCA velocity greater than 200 cm/sec, occurred in three patients, and was confirmed by angiography in all three. Subarachnoid hemorrhage (SAH) was documented by computerized tomography in five (62.5%) of the eight patients with vasospasm. All cases of severe vasospasm were associated with subarachnoid blood. The time course of vasospasm in patients with traumatic SAH was similar to that found in patients with aneurysmal SAH; in contrast, arterial spasm not associated with SAH demonstrated an uncharacteristically short duration (mean 1.25 days). suggesting that this may be a different type of spasm. A significant correlation ($p < 0.05$) was identified between the lowest CBF and highest MCA velocity in patients during the period of vasospasm, indicating that arterial narrowing can lead to impaired CBF. Ischemic brain damage was found in one patient who had evidence of cerebral infarction in the territories supplied by the arteries affected by spasm.

These findings demonstrate that delayed cerebral arterial spasm is a frequent complication of closed head injury and that the severity of spasm is, in some cases, comparable to that seen in aneurysmal SAH. This experience suggests that vasospasm is an important secondary posttraumatic insult that is potentially treatable.

KEY WORDS - cerebral blood flow - cerebral ischemia - head injury - transcranial Doppler ultrasound - vasospasm

When cerebral angiography was routinely performed for the evaluation of patients suffering cranioencebral trauma, cerebral arterial spasm was identified in 5% to 10% of severe cases. Since angiography was generally performed only as an initial diagnostic study and not necessarily repeated during the patient's hospital course, it might be expected that the actual incidence of delayed cerebral arterial spasm is significantly higher than that determined by these earlier reports. However, when angiography became a routine diagnostic test, treatment had not yet been developed for vasospasm, and it was regarded only as a potentially important pathophysiological factor rather than a focal point of diagnostic or therapeutic efforts. With the advent of computerized tomography (CT), angiography has been performed much less frequently in the evaluation of patients with head injury, and vasospasm as a complication of trauma has been neglected.

The recent development of transcranial Doppler ultrasound (TCD) monitoring has made it possible to diagnose intracranial arterial spasm noninvasively. This test is ideally suited for the intermittent monitoring of critically ill patients because the apparatus is portable, it is easily repeated, and it poses no risk. As the current of vasospasm following aneurysmal subarachnoid hemis
orrhage (SAH). We describe eight cases of post-traumatic cerebral arterial spasm identified by TCD studies in a series of 30 head-injured patients. This report correlates clinical features, radiological findings, and cerebral blood flow (CBF) measurements in patients with vasospasm identified by TCD.

Clinical Material and Methods

Patient Population

We studied 30 patients (21 males and nine females) who were admitted to the University of California at Los Angeles Medical Center suffering various degrees of closed head injury and who had undergone at least one TCD recording during their hospital stay. Their ages ranged from 10 to 84 years (mean 35.6 years). At admission and after resuscitation, the patients' Glasgow Coma Scale (GCS) scores ranged from 3 to 15 (mean 7.6).

Management Protocol

Following CT or surgery, the patients were admitted to the intensive care unit. All received routine intensive management, including intracranial pressure (ICP) monitoring when clinically indicated. Intracranial hypertension was treated by intubation and hyperventilation, supplemented by sedation, mannitol, or barbiturate administration if necessary. Transcranial Doppler ultrasound examinations were performed shortly after admission at the patient's bedside in the intensive care unit. Treatment of vasospasm in patients with TCD evidence of arterial narrowing was individualized. Three patients were treated with hypervolemia, which was augmented by the administration of nimodipine in one case and dopamine-induced hypertension in two cases.

Transcranial Doppler Ultrasound

Recordings were made of mean blood flow velocity in the intracranial internal carotid artery (ICA) and middle cerebral artery (MCA) using a 2-MHz probe. These vessels were sonicated through the temporal window as previously described by Aaslid, et al. All measurements were performed using a TCD apparatus.* Systemic blood pressure, hematocrit, PaCO2, and ICP were recorded simultaneously.

Transcranial Doppler ultrasound studies were performed as soon as possible following admission (mean time of first study 2.2 days after admission). Early in this study it was not uncommon for only a single TCD examination to be performed; however, later in the study serial evaluations were routine. Patients who had TCD evidence of vasospasm underwent serial TCD examinations until velocities decreased to normal values. The majority of patients underwent multiple studies (mean 4.1 studies) between Days 1 and 25 following head injury.

* Transcranial Doppler ultrasound monitoring device, Model TC2-64, manufactured by Eden Medical Electronics, Kent, Washington.

Velocities exceeding 120 cm/sec have been associated with arterial narrowing induced by aneurysm rupture, and were used to define vasospasm in our study group. Velocities above 200 cm/sec are associated with a greater than 50% reduction in vessel diameter, and this value was used to designate severe vasospasm. Lindgaard, et al.,12 found angiographic evidence of MCA narrowing induced by SAH to be associated with a ratio of MCA velocity to extracranial ICA velocity (V_mca/ V_eica) of greater than 3. This ratio is intended to compensate for CBF alterations which, in addition to vessel diameter, also affect TCD velocity recordings. In 19 of our patients, flow velocities were recorded from the extracranial ICA just below the skull base.

Cerebral Blood Flow Measurements

Cerebral blood flow studies were completed in 16 patients using the intravenous 133Xe method described by Agnoli and coworkers. A total of 35 examinations were performed. The patient's systemic arterial pressure, hematocrit, ICP, and PaCO2 were recorded. In patients with TCD evidence of vasospasm, CBF studies were completed during episodes of arterial narrowing and prior to the development (or after the resolution) of vasospasm. Cerebral blood flow studies were also completed in eight patients without vasospasm. For CBF measurement, intravenous injections of 133Xe dissolved in saline ranged in concentration from 20 to 30 mCi. A cerebrograph with five detectors monitoring each hemisphere was used for all studies. The values reported are hemispheric CBF obtained by averaging each set of five probes.

Analysis of CBF was based on CBF15, a mathematical model that is the calculated mean blood flow in the fast (gray matter)- and slow (white matter)-clearing compartments. The CBF15 analysis is more stable than compartmental parameters in the presence of severe pathology and reduces contamination from the extracerebral circulation. Because of variations in arterial CO2 tension, CBF values were reported both as they were recorded (without correction) and as adjusted to a normative PaCO2 of 34 mm Hg. The correction factor used was a 3% change in CBF for each 1-mm Hg change in PaCO2.

Computerized Tomography

Computerized tomography was performed on admission in all patients. Admission CT scans were reviewed retrospectively and analyzed for the presence of traumatic lesions (epidural hematoma, subdural hematoma, intracerebral hemorrhage, SAH, intraventricular hemorrhage, or cerebral contusion). To evaluate the role of SAH severity, which has been associated with the vasospasm of aneurysm rupture, patients were classified into four groups based on the presence and amount of subarachnoid blood. We used a modification

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of the system devised by Fisher, et al., for the analysis of patients suffering aneurysm rupture: Group 1, normal CT scan without evidence of SAH; Group 2, diffuse deposition or thin layer of SAH with all layers of blood less than 1 mm thick, with or without other intracranial lesions; Group 3, localized clots and/or layers of subarachnoid blood thicker than 1 mm, with or without other intracranial lesions; and Group 4, no SAH, but having evidence of epidural hematoma, subdural hematoma, intracerebral hemorrhage, intraventricular hemorrhage, or cerebral contusion.

Angiography

Digital subtraction angiography with injection of both carotid and vertebrobasilar circulations was performed in three patients with TCD velocities greater than 200 cm/sec. These patients were chosen for angiographic evaluation because this degree of vasospasm is associated with a high risk of cerebral ischemia and because therapy directed at the vasospasm was contemplated. No patient suffered complications due to angiography.

Clinical Outcome

The final neurological outcome was assessed in all patients at 3 months or later based on the Glasgow Outcome Scale (GOS). Most patients or their families were contacted directly by telephone; however, four could not be located. In these cases, outcome was determined by questioning the rehabilitation therapist involved in their extended care or by analyzing discharge reports from their rehabilitation facility.

Statistical Analysis

A chi-squared analysis was used to test the association between vasospasm and the admission GCS score, the GOS score, the CT findings, and the presence of intracranial hypertension. The Pearson linear regression analysis was utilized to compare the highest TCD velocities with the corresponding lowest CBF values in patients with vasospasm. The mean time-course plots of TCD velocities in patients with and without vasospasm (Fig. 1) were generated using a least-squares polynomial curve-fitting process with a polynomial degree of 3.

Results

Incidence and Time Course of Vasospasm

The clinical, CBF, and radiographic characteristics of patients with vasospasm are summarized in Table 1.

![Graph showing mean time course of middle cerebral artery (MCA) velocities](image)

FIG. 1. Graphs showing the mean time course of middle cerebral artery (MCA) velocities (triangles) and extracranial internal carotid artery (ICA) velocities (squares) in patients who developed vasospasm (MCA velocity > 120 cm/sec) (upper) and in those without vasospasm (MCA velocity < 120 cm/sec) (lower). The points are generated by averaging the MCA or ICA velocity for each postinjury day.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Patient's Age (yrs)</th>
<th>Admission GCS Score</th>
<th>SAH Group</th>
<th>Period of Vasospasm (days)</th>
<th>Maximum MCA Velocity (cm/sec)</th>
<th>Lowest CBF (cc/100 gm/min)</th>
<th>GOS Score</th>
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<td></td>
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<td>2</td>
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<td>30 (27) 32 (29)</td>
<td>MD</td>
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<td>13</td>
<td>3</td>
<td>6-16</td>
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<td>51 (49) 62 (61)</td>
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</tr>
<tr>
<td>3</td>
<td>28</td>
<td>6</td>
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<td>38 (42) 35 (38)</td>
<td>MD</td>
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<tr>
<td>4</td>
<td>29</td>
<td>3</td>
<td>2</td>
<td>2-16</td>
<td>262 (6.5) 172 (4.8)</td>
<td>26 (32) 27 (33)</td>
<td>PVS</td>
</tr>
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<td>31</td>
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<td>2</td>
<td>5</td>
<td>118 (2.6) 156 (3.7)</td>
<td>28 (30) 27 (30)</td>
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<td>4-22</td>
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<td>3</td>
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<td>55 (47) 56 (47)</td>
<td>GR</td>
</tr>
<tr>
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<td>21</td>
<td>9</td>
<td>1</td>
<td>2-3</td>
<td>138 (NA) 134 (3.2)</td>
<td>45 (49) 51 (55)</td>
<td>MD</td>
</tr>
</tbody>
</table>

* Abbreviations: CBF = cerebral blood flow; GCS = Glasgow Coma Scale; SAH = subarachnoid hemorrhage; MCA = middle cerebral artery; GOS = Glasgow Outcome Scale; GR = good recovery; MD = moderate disability; SD = severe disability; PVS = persistent vegetative state.
† As judged on computerized tomography using a modification of the system of Fisher, et al. For explanation of groups, see text.
‡ Numbers in parentheses indicate the Lindegaard ratio of MCA velocity to extracranial internal carotid artery velocity. NA = not available.
§ Numbers in parentheses indicate the CBF corrected to a normative PaCO₂ of 34 mm Hg using a 3% change in CBF for each 1-mm Hg change in PaCO₂.
During arterial spasm, TCD velocities increased in proportion to the reduction in vessel cross-sectional area. Velocities greater than 120 cm/sec, consistent with arterial narrowing, were found in eight (26.7%) of 30 patients. The highest mean MCA velocity recorded in patients with vasospasm ranged from 130 to 292 cm/sec (mean 183 cm/sec). Three of these patients were classified as having severe vasospasm (MCA velocity > 200 cm/sec). Four patients had bilateral evidence of vasospasm, while four had unilateral arterial spasm. Seven patients in our study with an MCA velocity greater than 120 cm/sec had concurrent assessment of extracranial ICA velocity. Six demonstrated \( \frac{V_{MCA}}{V_{ICA}} \) ratios greater than 3. This ratio is intended to compensate for CBF alterations which, in addition to vessel diameter, also affect TCD velocity recordings. A ratio over 3 suggests that the high MCA velocity is due to arterial narrowing rather than to hyperemia.

The mean time course of both MCA and extracranial ICA velocities in patients with and without vasospasm are shown in Fig. 1. On average, elevated MCA velocities consistent with vasospasm first appeared 2 days after injury and reached a maximum between Days 11 and 13. Middle cerebral artery velocities decreased over the ensuing 6 days, but did not fall to normal baseline values. This may be attributed to the fact that TCD studies were terminated when vasospasm began to resolve. One patient exhibited delayed and prolonged vasospasm that lasted almost 3 weeks after admission (Case 6, Table 1). This case causes the upward slope seen at the terminal portion of the MCA velocity curve for vasospasm patients. The mean extracranial ICA velocities increased during the initial 2 to 4 days after admission, then fell over the next 2 or 3 days to normal or slightly subnormal values. The drop in extracranial ICA velocity between Days 4 and 6 corresponds temporally to rising MCA velocities, consistent with a reduction in volume flow in the ICA due to progressing MCA vasospasm.

The MCA velocity in patients without vasospasm increased slightly from baseline during the first 6 post-injury days, but then remained at fairly constant values (Fig. 1 lower). The ICA velocity followed the general pattern of the MCA velocity suggesting an initial increase followed by a subsequent decline in CBF. This observation was generally confirmed by \(^{133}Xe\) studies.

Correlation With Admission GCS Scores and Neurological Outcome

Figure 2 shows the number of patients with and without vasospasm for each GCS score. These scores ranged from 3 to 15 in patients who developed vasospasm following head injury; however, vasospasm was more common in patients suffering severe injuries.

Table 2 summarizes GOS scores for patients with and without vasospasm. Sixteen patients underwent CBF studies, and Table 2 is further stratified according to the lowest CBF measurement. Although a statistically significant association was not found between vasospasm and clinical outcome, 12 (54.5%) of 22 patients without arterial spasm had a good recovery compared to only two (25%) of eight vasospasm cases. Furthermore, when patients who died within 5 days of admission (which is typically prior to the onset of arterial spasm) were not included in the analysis, there was a closer association between vasospasm and clinical outcome. All three patients who developed vasospasm and who had a CBF less than 30 cc/100 gm/min had a poor outcome (severe disability, persistent vegetative state, or dead), whereas all five vasospasm patients who had a CBF greater than 30 cc/100 gm/min had a good outcome (good recovery or moderate disability). This suggests that arterial narrowing in conjunction with low CBF adversely affects outcome. One vasospasm patient (Case 6, Table 1), who died 1 month after injury, had postmortem evidence of cerebral infarction in territories supplied by the vessels that were severely constricted (anterior cerebral artery (ACA) and MCA watershed territory bilaterally).
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Fig. 4. Graph showing the distribution of patients with severe vasospasm, moderate vasospasm, and no vasospasm with respect to the classification of subarachnoid blood visualized on computerized tomography. For explanation of subarachnoid hemorrhage (SAH) groups, see text.

Correlation With CT Findings

Twenty-four patients (80%) had at least one lesion (epidural hematoma, subdural hematoma, intracerebral hemorrhage, SAH, intraventricular hemorrhage, or cerebral contusion) present on their initial CT scan (Fig. 3). The association between CT lesions and arterial spasm was variable. Subarachnoid blood was present in five (62.5%) of the eight patients who developed vasospasm and in 12 (54.5%) of the 22 in whom arterial spasm did not occur. When patients were classified into groups based on the appearance of subarachnoid blood as previously described (modified Fisher grade), vasospasm patients were identified in groups with and without SAH (Fig. 4). However, all patients who developed severe or prolonged arterial narrowing had subarachnoid blood visualized on their initial CT scan. The three patients with an MCA velocity greater than 120 cm/sec and no CT evidence of subarachnoid blood had an unusually short duration of elevated TCD velocities, which lasted no more than 2 days. With the exception of one case, patients who developed vasospasm associated with SAH had an extended course of vasospasm, lasting an average of 11.2 days.

Correlation With Angiographic Findings

The three patients with TCD evidence of severe vasospasm had angiographic confirmation of vessel narrowing (Figs. 5–7). Two patients demonstrated se-

TABLE 2

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Glasgow Outcome Scale Score &amp; rCBF (cc/100 gm/min)†</th>
<th>GR</th>
<th>MD</th>
<th>SD</th>
<th>PVS</th>
<th>Dead</th>
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<td>1</td>
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<tr>
<td>rCBF &lt; 30</td>
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<td>2</td>
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</tr>
</tbody>
</table>

*Abbreviations: rCBF = regional cerebral blood flow; GR = good recovery; MD = moderate disability; SD = severe disability; PVS = persistent vegetative state.
†Sixteen of the 30 total patients analyzed underwent CBF studies. The rCBF used was the lowest hemispheric CBF, value obtained.

Fig. 5. Case 6. The presence of bilateral severe vasospasm was predicted by middle cerebral artery (MCA) velocities > 200 cm/sec on transcranial Doppler ultrasonography. The patient had concomitant blood flow values below 20 cc/100 gm/min and died 1 month after injury. Postmortem examination revealed bilateral watershed infarctions in the distribution of the vessels affected by vasospasm. Left: Right angiogram, anteroposterior view, demonstrating severe vasospasm of the suprachiasmatic segment of the internal carotid artery (ICA) and MCA (arrows). Right: Left angiogram, anteroposterior view, demonstrating severe ICA and MCA spasm (arrows).

Fig. 6. Case 4. Transcranial Doppler ultrasound velocities suggested moderate to severe vasospasm in this patient and were coupled with low cerebral blood flow values. Left: Right angiogram, anteroposterior view, demonstrating moderate internal carotid artery (ICA) and mild middle cerebral artery (MCA) spasm (arrows). Right: Left angiogram, anteroposterior view, showing severe spasm of the ICA and moderate MCA spasm (arrows). The patient remained in a persistently vegetative state.
vere segmental vasospasm affecting the supraclinoid segment of the ICA, the MCA, and in one case the proximal ACA. Both of these patients also had radiographic documentation of severe diffuse verteobasilar vasospasm. Another patient had angiographic evidence of unilateral MCA vasospasm that was consistent with TCD findings of elevated velocities in the right MCA only (Fig. 7).

Correlation With CBF

The patients with vasospasm had an average ipsilateral hemispheric CBF of 38.9 cc/100 gm/min during TCD evidence of MCA narrowing. Three patients demonstrated CBF below 30 cc/100 gm/min and had poor neurological outcomes. In contrast, the vasospasm patients had a mean hemispheric CBF of 48 cc/100 gm/min at times when TCD velocities were within the standard range (before vasospasm developed or after vasospasm resolved). This value was essentially the same as the average CBF of patients who did not have vasospasm (47.9 cc/100 gm/min).

A significant correlation (p < 0.05) was identified between the maximum mean MCA velocity and the lowest ipsilateral hemispheric CBF level during vasospasm in patients with TCD evidence of arterial narrowing (Fig. 8), documenting that vasospasm induced by craniocerebral trauma is associated with a reduction in CBF.

Correlation Between ICP and Vasospasm

Twenty-three patients underwent ICP monitoring, and transient elevations above 20 mm Hg occurred in 15. No significant difference in the incidence of intracranial hypertension was found when comparing the groups with and without vasospasm. Elevations in ICP above 20 mm Hg persisting longer than 12 hours were seen in four patients, two of whom developed vasospasm. Intracranial hypertension was present during only two of the 61 TCD studies performed in patients with vasospasm. Therefore, no association was found between intracranial hypertension and elevated blood flow velocity.

Discussion

Role of Ultrasound and CBF Studies

In the past, cerebral angiography was a primary diagnostic modality for the evaluation of head-injured patients. In addition to demonstrating traumatic mass lesions, angiographic studies on occasion defined narrowing of the intracranial arteries. Several reports have focused on posttraumatic angiographic vasospasm, with the incidence ranging from 2% to 41%.

In the largest angiographic study to date, Suwanwela and Suwanwela found narrowing of one or more of the intracranial arteries in 65 (18.6%) of 350 patients with moderate to severe head injury. With the advent of TCD monitoring, which allows repetitive noninvasive assessment of flow velocity (and therefore narrowing) in the basal cerebral arteries, a more thorough study of posttraumatic vasospasm has become possible. Weber, et al., performed serial measurements of blood flow velocity in the MCA’s and the extracranial ICA’s in 35 patients with severe closed head injury. These investigators used the Lindegaard ratio (the ratio of blood flow velocity in the MCA to blood flow velocity in the extracranial ICA) to distinguish cerebral arterial spasm from posttraumatic hyperemia. Patients with a Lindegaard ratio of greater than 3 were assumed to have arterial spasm. They found that 14 (40%) of the 35 patients had blood flow velocities consistent with MCA spasm. In only one case was the ultrasound diagnosis of MCA spasm confirmed angiographically. Conventional CBF measurements, which would support the diagnosis of vasospasm and rule out hyperemia, were not obtained as a part of this study.
Posttraumatic vasospasm

The present study has confirmed that cerebral arterial spasm occurs in a significant proportion of head-injured patients. Furthermore, the angiographic findings and CBF measurements reported in this study refute the notion that elevated MCA velocity in head-trauma patients is due to hyperemia rather than arterial narrowing. Figure 8 demonstrates that the highest MCA velocities are associated with the lowest ipsilateral hemispheric blood flow measurements, as would be expected if elevated MCA velocity is due to arterial narrowing rather than hyperemia.

A review of the three patients with the most severe vasospasm, as evidenced by the highest MCA velocities, is instructive. Cases 4 and 6 had the highest recorded MCA velocities and the lowest hemispheric CBF (Table 1). In both cases, angiography at the time of the highest velocity recordings demonstrated bilateral MCA spasm (Figs. 5 and 6). Transcranial Doppler ultrasound recordings in Case 2 demonstrated right but not left MCA spasm (Table 1); an angiographic study performed shortly after this TCD evaluation confirmed the finding of right MCA spasm and showed a nonspastic left MCA (Fig. 7). This patient’s left hemisphere had the highest CBF measurement of any patient with elevated MCA velocities. Cerebral blood flow in the left hemisphere was 62 cc/100 gm/min, and the flow velocity in the left MCA, which was proven by angiography not to be in spasm, was only 98 cm/sec. In contrast, the CBF in the right hemisphere was lower (51 cc/100 gm/min), but flow velocity in the narrowed right MCA was 228 cm/sec. These findings clearly indicate that arterial spasm, rather than high CBF, is the primary cause of high MCA flow velocities in patients with head trauma. On the other hand, Case 7 may very well represent a case of hyperemia, rather than MCA spasm (this patient was placed in the “vasospasm” group because the MCA velocity on the left was greater than 120 cm/sec, which was the criterion for vasospasm used in this study). The CBF in both hemispheres was slightly above normal (50 cc/100 gm/min), and the V_MCA/V_ICA ratio was less than 3. This may be an example in which the high velocity was due to high volume flow through the MCA secondary to elevated CBF, rather than arterial narrowing. Although one cannot be sure that this patient did not have both hyperemia and mild MCA spasm, this case illustrates the importance of measuring some index of total MCA flow (hemispheric CBF or ICA velocity) in order to interpret MCA flow velocity correctly.

Subarachnoid Hemorrhage in Traumatic Vasospasm

The arterial spasm that follows head injury is similar in a number of ways to vasospasm following aneurysm rupture. Weber and associates27 found a statistically significant relationship between MCA spasm and the presence of CT-visualized subarachnoid or intracerebral blood. Although we found that cerebral arterial spasm could occur in patients whose CT scans did not show subarachnoid or intracerebral blood (Fig. 3), severe arterial spasm was invariably associated with CT-visualized SAH (Fig. 4). Weber, et al., defined a time course for posttraumatic vasospasm that was somewhat different from that for vasospasm following aneurysm rupture. In their experience, vasospasm was identified as early as 48 hours after injury and peaked between 5 and 7 days posttrauma. Several patients described in our report had the onset of elevated MCA velocity on the 2nd day after injury, but the patients with a more severe degree of vasospasm showed a rise and subsequent fall in MCA velocities that was very similar to the picture reported for aneurysmal SAH (Table 1 and Fig. 1 upper). The angiographic findings in Cases 2, 4, and 6 emphasize the similarity between posttraumatic vasospasm and vasospasm following aneurysm rupture (Figs. 5–7). The arterial segments affected by spasm in these cases included the suprachordoid segment of the ICA, the proximal MCA and ACA, and the basilar trunk, all areas typically affected by aneurysmal SAH. These findings provide qualitative evidence that cerebral arterial spasm after head injury is closely related to vasospasm after aneurysm rupture, and reinforce the pathophysiological linkage of vasospasm to SAH, no matter what the cause.

The frequency of posttraumatic cerebral arterial spasm is not surprising; SAH is a common CT finding in head-injured patients. In a study of patients with severe head injury from the National Institutes of Health Traumatic Coma Data Bank, Eisenberg, et al., found CT evidence of SAH in 39% of 1030 patients. Among the patients reviewed in our report, 57% showed some degree of SAH. Because of the frequency of trauma, head injury is almost certainly the most common overall cause of SAH. The CT findings in the Traumatic Coma Data Bank study7 also showed that the degree of midline shift, the presence of an extracerebral mass, and the status of the mesencephalic cisterns were all powerful predictors of death. Traumatic SAH was also found to be a significant independent predictor of death. This finding led the investigators to propose that SAH is a pathophysiological determinant of outcome, possibly due to cerebral ischemia related to arterial spasm.

Clinical Implications

Recent clinical studies, notably those of Robertson, et al.,22 and Muizelaar,15 have provided strong evidence that ischemia is a potentially treatable secondary insult following head injury. Ischemic brain damage has been found in 90% of patients dying from head injury, and often conforms to regional patterns characteristic of occlusive arterial disease (MCA and ACA territories).8 Although diffuse cerebral ischemia, and possibly watershed distribution ischemia, in head-trauma patients can be explained by impairment in cerebral perfusion pressure due to elevated ICP or systemic arterial hypotension, regional posttraumatic ischemic lesions may be related to focal arterial spasm.

Intracranial hypertension has been postulated to be

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responsible for arterial narrowing after head injury,\textsuperscript{14} however, increased ICP was present on only two occasions during all of the TCD studies performed in patients with vasospasm. It seems unlikely that elevated ICP is the cause of posttraumatic vasospasm.

Maclerhorn and Graham,\textsuperscript{15} in a study of angiograms performed on 96 patients who subsequently died from head trauma, found a significant correlation between angiographic arterial narrowing and the postmortem finding of ipsilateral ischemic hemispheric damage. The findings reported in the current study demonstrate that the severity of posttraumatic arterial spasm correlated with the degree of reduction in CBF. In fact, Case 6 had severe bilateral ICA and MCA spasm associated with postmortem evidence of watershed infarctions in areas supplied by the affected vessels.

There are additional reasons to believe that posttraumatic cerebral arterial spasm is an important secondary insult in head trauma. First, the degree of arterial spasm, as defined by TCD and angiographic studies, is as severe as that seen in aneurysmal SAH patients who develop delayed ischemic neurological deficits. Transcranial Doppler ultrasound studies of aneurysmal SAH patients have found that an MCA velocity greater than 200 cm/sec is associated with ischemic neurological deficits,\textsuperscript{23} and this degree of vasospasm was seen in three of our patients. The degree of arterial narrowing seen on the angiograms performed on the head-injured patients in this study is comparable to that seen in aneurysmal SAH patients who developed an ischemic deficit. It seems reasonable to assume that trauma patients with this degree of severe cerebral arterial narrowing will be prone to ischemic brain damage. Second, all three of the patients in this study who had markedly low CBF (<30 cc/100 gm/min) associated with arterial spasm had a poor outcome (Table 2). Finally, Newell, et al.,\textsuperscript{16} reported a patient who developed a delayed focal neurological deficit associated with TCD-defined MCA spasm following traumatic SAH. This patient experienced neurological improvement after balloon dilatation angioplasty. The evidence, taken as a whole, makes a compelling argument that posttraumatic cerebral arterial spasm is an important pathophysiological factor in head-injured patients.

Physiological manifestations such as arterial hypertension, hypervolemia, hemodilution, pharmacological therapy with calcium antagonists, and balloon dilatation angioplasty have been successful strategies for the treatment of ischemic neurological deficits due to aneurysm-induced arterial spasm.\textsuperscript{24,6,11,16,17,21} Because of the potential for treatment-related complications, none of these therapies can at the present time be advocated for routine use in head-injured patients. However, the evidence that posttraumatic cerebral arterial spasm can cause secondary brain injury, combined with the positive experience using these therapeutic modalities in patients with aneurysmal SAH, suggests that these treatments should be studied in head-injured patients. Given the current state of knowledge, it is reasonable to consider the use of nimodipine, hypervolemia/hemodilution/hypertension, or balloon dilatation angioplasty in the trauma patient who develops severe arterial spasm documented by TCD or angiographic studies concurrent with progressive neurological deterioration not due to any other identifiable factor. Careful reports of such cases and carefully conducted clinical trials of the available therapeutic modalities used for aneurysmal vasospasm will establish whether cerebral arterial spasm is an important and treatable complication of head injury.

References


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