Traumatic subarachnoid hemorrhage as a predictable indicator of delayed ischemic symptoms

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This report provides findings of an investigation of the influence of traumatic subarachnoid hemorrhage on the development of delayed cerebral ischemia caused by vasospasm. The authors prospectively studied 130 patients with closed-head trauma, who exhibited subarachnoid blood on admission computerized tomography (CT) scans. Ten (7.7%) of these patients developed delayed ischemic symptoms between Days 4 and 16 after the head injury. They consisted of three (3.0%) of 101 patients with small amounts of subarachnoid blood and seven (24.1%) of 29 patients with massive quantities of subarachnoid blood on admission CT scans. In each of the 10 patients, severe vasospasm was demonstrated by angiography performed soon after development of ischemic symptoms. There was a close correlation between the main site of the subarachnoid blood and the location of severe vasospasm. In seven of the patients, follow-up CT scans showed development of focal ischemic areas in the cerebral territories corresponding to the vasospastic arteries. These results demonstrate that traumatic subarachnoid hemorrhage, especially if massive, is a predictable indicator of delayed ischemic symptoms.

KEY WORDS • cerebral ischemia • vasospasm • subarachnoid hemorrhage • head injury • delayed ischemic deficit

It is generally accepted that subarachnoid blood from a ruptured aneurysm is responsible for subsequent development of vasospasm and ischemic symptoms directly attributable to vasospasm. In head injury, subarachnoid hemorrhage (SAH) occurs in 12% to 53% of cases, and there are several reports suggesting that subarachnoid blood is one factor in the genesis of post-traumatic vasospasm. Nevertheless, there are only a few reported cases in which a causal relationship between vasospasm following traumatic SAH and spasm-related cerebral infarction has been shown.

The purpose of this study was to correlate the presence of traumatic subarachnoid blood with the development of delayed ischemic symptoms caused by vasospasm.

Clinical Material and Methods

Patient Population

We treated 883 head-injured patients over a period of 10 years. Of these, 130 patients (14.7%), whose admission computerized tomography (CT) scans showed SAH, were enrolled in this prospective study. Three patients, in whom angiographic study confirmed the presence of a cerebral aneurysm possibly responsible for SAH, were excluded from the study. The patients selected included 98 males and 32 females ranging in age from 4 to 91 years (mean 52.2 years) at the time of treatment. At admission and after resuscitation, the patients’ Glasgow Coma Scale (GCS) scores ranged from 3 to 15 (mean 10). The GCS score was 3 to 8 in 47 patients, 9 to 12 in 34 patients, and 13 to 15 in 49 patients.

All patients received sequential neurological examinations and routine intensive management with emphasis on ventilatory support and control of intracranial pressure below 25 mm Hg, if necessary. In 49 patients, craniotomies were performed for removal of hematomas and decompression.

Patients who developed vasospasm, identified by angiography, or ischemic symptoms possibly attributable to vasospasm were treated with induced hypervolemia and dopamine-induced hypertension.

The final neurological outcome of each patient was measured according to the Glasgow Outcome Scale at 3 or more months after the trauma.

Computerized Tomography Studies

On admission, CT was performed as soon as possible after cardiopulmonary stabilization. The amount of subarachnoid blood was estimated according to a modification of the criteria of Fisher, et al., for the analysis of
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patients suffering aneurysm rupture, as follows: Group A, a diffuse deposition or thin layer of SAH with all layers of blood measuring less than 1 mm thick, with or without other intracranial lesions; Group B, localized clots and/or layers of subarachnoid blood thicker than 1 mm, with or without other intracranial lesions. Statistical analysis of the two SAH groups was done using the chi square test.

Serial CT scanning was repeated at regular intervals, in response to a reduced level of consciousness and/or focal neurological signs, and at 3 months after head injury. Particular attention was paid to identifying the appearance of a new low-density area possibly caused by vasospasm with distinct margins restricted to an arterial territory.

**Angiography and Estimation of Vasospasm**

Four-vessel angiographic study was performed soon after admission in some patients to rule out rupture of an aneurysm or other causes of the SAH. In some patients, follow-up angiography was performed between postinjury Days 7 and 11 to identify whether their vasospasm developed in accordance with the usual time course for vasospasm following aneurysm rupture.

If focal cerebral ischemia was suspected on the basis of clinical findings and/or CT scans, cerebral angiography was performed to investigate the presence or absence of vasospasm. In several patients whose vasospasm was identified on angiograms, angiography was repeated more than 3 weeks after injury to follow the course of the vasospasm.

To grade the severity of vasospasm, we used the system of Fisher, et al. Vasospasm was considered severe if the residual lumen of various artery segments measured as follows: the internal carotid and basilar arteries, less than 2 mm; the middle cerebral artery (MCA) from origin to bifurcation, 1.0 mm or less; the MCA from bifurcation to emergence from the sylvian fissure, 0.5 mm or less; the anterior cerebral artery (ACA) from the origin to the anterior communicating artery, 1.0 mm or less; the ACA from the anterior communicating artery to the genu, 0.5 mm or less; the posterior cerebral artery (PCA) proximal to the posterior communicating artery, 0.5 mm or less; and distal branches, 0.5 mm or less. Vasospasm was considered mild if it ranged from slightly more than none to borderline severe.

When arterial vasospasm was severe and the distribution of vasospasm corresponded to the clinical symptoms, the ischemia caused by the vasospasm was believed to be responsible for the patient’s clinical deterioration. Diffuse brain swelling, new hemorrhagic lesions, hydrocephalus, and extracranial factors that might cause clinical deterioration (such as disturbances of fluid and electrolyte balance) were ruled out.

**Illustrative Cases**

**Case 1**

This 47-year-old man was found unconscious under the stairs of his home. On admission, the patient’s GCS score was 8. A plain skull x-ray film disclosed a linear fracture at the right temporal bone. Admission CT scanning revealed a hematoma in the left frontal lobe with a heavy deposit of blood in the left sylvian fissure (Fig. 1 upper).

A four-vessel angiographic study was immediately performed to rule out aneurysm rupture. However, an aneurysm was not found and the main supratentorial arterial trunks seemed to constrict slightly (Fig. 2A–C). The patient was taken to the operating room for emergency evacuation of the intracerebral hematoma. A postoperative CT scan revealed successful removal of the hematoma and persistence of the blood deposit in the left sylvian fissure (Fig. 1 lower).

Postoperatively, the patient improved to some extent. However, on Day 7, the patient suddenly developed a severe right hemiparesis and deterioration of consciousness; a CT scan obtained at that time demonstrated an ill-defined small infarcted area in the left frontal lobe. Despite induced hypervolemia and hypertension therapy, slight improvement was noted only for a brief period. An angiographic study performed on Day 10 showed severe spasm of the left MCA and ACA with moderate narrowing of the distal portion of the basilar artery (Fig. 2D–F). A CT scan obtained on Day 11 demonstrated a large infarcted area in the territory of the left MCA (Fig. 3). The patient deteriorated further in spite of hyperventilation therapy and mannitol administration. He became comatose with a fixed and dilated pupil on the left side on Day 12 and died on Day 15.

**Case 2**

This 73-year-old woman fell down on the street and was found unresponsive at the scene. On admission, the patient’s GCS score was 14. The admission CT scan revealed a heavy diffuse deposit of blood in the bilateral sylvian fissures and the left ambient cistern (Fig. 4 upper). The patient became alert on the next day. However, on Day 4, the patient’s level of consciousness suddenly deteriorated, and her GCS score became 10. A follow-up CT scan obtained on Day 5 revealed a hematoma in the left temporal lobe with a heavy deposit of blood in the left sylvian fissure (Fig. 4 lower).
scan performed on Day 5 demonstrated an infarcted area in the occipital lobe. An angiographic study performed on Day 7 showed severe diffuse spasm of the left PCA. Although the patient was treated with induced hypervolemia and hypertension, her clinical condition deteriorated further and her GCS score became 7 on Day 12. A CT scan on Day 13 demonstrated cerebral infarction not only in the left occipital lobe, but also in the right parietal lobe (Fig. 4 lower). Angiography performed on Day 14 showed persistent severe spasm at the left PCA and additional severe spasm at the right MCA and mild diffuse spasm at the left MCA. The vasospasm completely disappeared on the follow-up angiography on Day 26. At 3 months, the patient remained in a persistent vegetative state.

Results

Clinical Condition Related to Amount of Subarachnoid Blood

Table 1 shows the incidence of ischemic symptoms in each of the SAH groups. Of the 130 patients with traumatic SAH, 29 (22.3%) exhibited a massive amount of subarachnoid blood (Group B) and 101 (77.7%) a small amount of subarachnoid blood (Group A) on admission CT scans. Ischemic symptoms directly attributable to vasospasm occurred in 10 patients (7.7%). In Group B, seven (24.1%) of the 29 patients developed ischemic symptoms, in contrast to three (3.0%) of the 101 patients in Group A. This difference was statistically significant (p < 0.001).

In Group A, the GCS score on admission was 3 to 8 in 36 patients, 9 to 12 in 27 patients, and 13 to 15 in 38 patients, whereas in Group B, it was 3 to 8 in 11 patients, 9 to 12 in seven patients, and 13 to 15 in 11 patients.
Craniotomy for removal of hematoma and decompression was performed in 39 Group A patients and 10 Group B patients. Differences between the two groups concerning need for craniotomy and severity of injury judged by admission GCS scores were not statistically significant. Thus, there seemed to be similar clinical conditions soon after admission in each of the two groups (Table 2).

The survival rates in the two SAH groups are shown in Table 2. The number of survivors as of Day 3 was 83 (82.2%) of 101 Group A patients and 20 (69.0%) of 29 Group B patients. No patients who died before Day 3 exhibited any evidence of focal ischemia on clinical findings and/or CT scans. The difference between the two groups was not statistically significant. However, survival rates at 1 month and at 3 months were higher in Group A than in Group B. Those differences were statistically significant (both p < 0.01).

Delayed Ischemic Symptoms

Of the 130 patients with traumatic SAH, 10 patients (7.7%) developed ischemic symptoms. The clinical and radiographic characteristics in those patients are summarized in Table 3. Two of 10 patients had SAH as the only traumatic lesion on the initial CT scans. The remaining eight patients had other intracranial lesions, such as an intracerebral hematoma, an extradural hematoma, a subdural hematoma, and/or contusion. Ischemic deficits appeared between Days 4 and 16 after injury, with the peak incidence on Days 9 and 10 (four patients). Of the 10 patients with ischemic symptoms, three died, two deteriorated to persistent vegetative state, one suffered from severe disability, and one from moderate disability. Only three patients fully recovered.

The main location of subarachnoid blood identified on CT was the sylvian fissure in all cases. In one case (Case 2), there was massive blood in the ambient cistern as well. Cerebral angiography performed soon after the development of focal cerebral ischemia demonstrated the presence of severe vasospasm, the location of which had a proximal relationship with the site of major subarachnoid blood. Exact correlation existed between the particular artery involved in the vasospasm and the ischemic symptoms. There were seven patients in whom CT scans showed development of focal infarction, which invariably corresponded to the site of severe vasospasm.

Occurrence of Vasospasm

Of the 130 patients, 10 Group A patients (9.9%) and 10 Group B patients (34.5%) underwent angiographic study more than twice. A total of 45 angiographic studies were performed for those 20 patients. Table 4 summarizes the CT findings at admission and the time course of angiography performed to measure arterial caliber in each patient. Vasospasm could not be found on the initial and follow-up angiograms in four patients, all of whom were in Group A. Vasospasm was identified at least once in all 10 Group B patients and in six Group A patients.

Angiography was performed between Days 0 and 2 in 18 patients. Vasospasm was found in two (20%) of 10 Group A patients and five (63%) of eight Group B patients. The correlation between the site of SAH and the location of vasospasm was variable. The grade of vasospasm was mild in six patients and severe in one.

In the 21 angiographic studies performed between Days 7 and 18, vasospasm appeared to be severe in 15 instances, mild in two, and absent in four. Severe vasospasm was most frequently observed in this period. The location of the vasospasm had a close relationship with the site of major subarachnoid blood. There were six patients in whom severe vasospasm occurred with no neurological symptom.

Angiography performed on or later than Day 23 in six patients showed disappearance or diminution of vasospasm that had been observed previously.
Discussion

Subarachnoid Hemorrhage and Delayed Ischemic Symptoms

We found that ischemic symptoms caused by cerebral arterial spasm following traumatic SAH in this series are comparable to those found following aneurysmal SAH: appearance of symptoms between Days 4 and 16 after injury, with the peak incidence on Days 9 and 10; close correlation between the main site of the subarachnoid blood and the location of severe vasospasm responsible for the symptoms; and a higher incidence of symptoms in patients with massive SAH than those with slight SAH. These findings provide evidence that subarachnoid blood plays an important role in the later development of vasospasm, not only following aneurysm rupture but also after head injury. Nevertheless, there is no general agreement that subarachnoid blood in head injury is an important risk factor in the development of vasospasm and ischemic brain damage.

Despite the common occurrence of SAH in head-injured patients, the presence of massive SAH (Group B), which proved to be closely related to the ischemic symptoms caused by vasospasm, was found in 22% of the patients with SAH and only 3% of all head-injured patients in this series. In addition, SAH was significantly common in head-injured patients who experienced an unfavorable outcome, and it was an indicator for the severity of the injury. Therefore, some of the patients in Group B may have died before the appearance of delayed ischemic symptoms. We found that 31% of Group B patients died between Days 0 and 3. With so few patients presenting with massive SAH and such a high risk of death in patients who do have SAH, it is not surprising that posttraumatic delayed ischemic symptoms caused by vasospasm are not frequently seen.

Traumatic Vasospasm and Ischemic Symptoms

In patients with head injury, angiographic demonstration of cerebral vasospasm has been documented by several authors. The incidence ranges from 2% to 41%. However, the incidence and clinical manifestation of ischemia that is caused by the vasospasm are not well known.

It is not exceptional to find vasospasm soon after head injury, as shown in this study and in other reports, whereas vasospasm after aneurysmal SAH is rarely seen during the first few days post-SAH and reaches its maximum between Days 7 and 12. Using transcranial Doppler ultrasound monitoring, serial measurements of

<table>
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<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Main Location of SAH; Group†</th>
<th>Other Lesions</th>
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<th>Day of Onset of Delayed Ischemic Symptoms</th>
<th>Location of Infarction on CT; Day of Angiography</th>
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* Abbreviations: ACA = anterior cerebral artery; amb = ambient cistern; CT = computerized tomography; EDH = extradural hematoma; F = frontal lobe; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; GR = good recovery; ICA = internal carotid artery; ICH = intracerebral hematoma; MCA = middle cerebral artery; MD = moderate disability; O = occipital lobe; P = parietal lobe; PCA = posterior cerebral artery; PVS = persistent vegetative state; SAH = subarachnoid hemorrhage; SD = severe disability; SDH = subdural hematoma; sylv = sylvian fissure; T = temporal lobe.
† Subarachnoid hemorrhage was judged on the CT scan using a modification of the system of Fisher, et al. For explanation of groups, see text.
‡ For explanation of severe vasospasm, see text.
flow velocity in the basal cerebral arteries have been performed in head-injured patients with and without SAH, and the time course of vasospasm has been speculated on by several authors.3,13,17,23 A maximum increase in blood flow, which might indicate maximum narrowing of the vessel, occurred on Days 2 and 3,3 or between Days 5 and 7.17,23 Those are apparently different from the time course of vasospasm following aneurysm rupture.

Although cerebral angiography was not routinely performed for evaluation of the patients suffering cranio-cerebral trauma, we occasionally found evidence of vasospasm on the angiograms obtained soon after the injury, as shown in Table 4. We did not find any clinical or CT evidence of focal cerebral ischemia in those patients. There have been no patients reported whose vasospasm was diagnosed on the basis of clinical symptoms or CT findings between Days 0 and 2. Vasospasm during the first few days after trauma might be short in duration13 and remain subclinical,17 although it might exert some unfavorable global effect on critically injured trauma patients.22

Traumatic cerebral arterial spasm could occur in patients whose CT scans do not show subarachnoid blood or whose cerebrospinal fluid obtained by lumbar puncture is clear.3,6,13,18 Some factors other than SAH may be responsible for the majority of traumatic vasospasm.26 It has been postulated that direct stretching or mechanical irritation of the cerebral arteries plays an important role in pathogenesis of vasospasm.1,2,11,19

Subarachnoid blood appears to be one factor in the genesis of traumatic vasospasm.13,15,23 because there is a close correlation between the main site of the subarachnoid blood and the location of vasospasm. The results of this study confirm that the vasospasm responsible for focal cerebral ischemia was severe and closely related to SAH. The symptoms of focal cerebral ischemia appeared only on Day 4 or later. This SAH-related traumatic spasm appeared to be identical to the spasm that occurs after aneurysm rupture. On the other hand, severe delayed vasospasm did not always manifest ischemic symptoms. Some additional pathological cerebral condition, such as impairment in cerebral perfusion pressure, might be required for vasospasm to lead to infarction.

### Treatment Efficacy

We treated the patients with symptomatic vasospasm by means of hypervolemic therapy and dopamine-induced hypertension. However, the efficacy was not generally impressive. In association with many kinds of intracranial traumatic pathologies, such as contusions, diffuse edema, or mass lesions, hypervolemic therapy may not be effective or may sometimes cause complications. There have been several reports of successful treatments for traumatic vasospasm using intraarterial papaverine infusion,22 a calcium antagonist,10 or balloon dilation angioplasty.14 Those have been successful strategies for treatment of ischemic symptoms caused by arterial spasm following aneurysm rupture. Further clinical trials are necessary to establish the efficacy of those treatments for posttraumatic vasospasm.

The clinical significance of posttraumatic vasospasm still remains a matter of speculation. However, vasospasm that occurs after SAH is clinically important because it is
an identified causative factor in cerebral infarction, which is a significant delayed complication of head injury, as demonstrated in this study. Moreover, if vasospasm following traumatic SAH has the same pathogenesis as vasospasm following aneurysmal SAH, early surgical removal of the blood deposited in the subarachnoid spaces might prove to be a prophylactic method to guard against vasospasm and to prevent or minimize delayed ischemic symptoms.  

References

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