The minor leak preceding subarachnoid hemorrhage

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Thirty-four of 87 consecutive patients with subarachnoid hemorrhage from a cerebral aneurysm had a premonitory minor leak. There were 12 men and 22 women, aged 25 to 73 years (mean 44.4 years). Twenty-two had a small and 12 had a large aneurysm located on the internal carotid artery (17 cases), anterior communicating artery (10 cases), middle cerebral artery (five cases), and pericallosal artery (two cases). Fifty-two percent of patients with a minor leak from an internal carotid artery aneurysm had ipsilateral, hemicranial, hemifacial, or periorbital pain. Half of the patients initially saw a physician, but in no case was the correct diagnosis made. Twenty-five patients had a major rupture within 24 hours to 4 weeks after findings suggesting a minor leak, with a mortality rate of 53%. Nine other patients were diagnosed by lumbar puncture or computerized tomography (CT) scanning after initial misdiagnosis and were operated on, without mortality, before a major rupture could occur. The CT scans were negative in 55% of patients with a minor leak, but lumbar puncture, when performed, was always positive.

A minor leak prior to major aneurysmal rupture is a common occurrence and, if unrecognized, is associated with a high mortality. Computerized tomography scanning is unreliable in diagnosing this event, and lumbar puncture is the examination of choice once intracranial hypertension has been ruled out.

KEY WORDS • subarachnoid hemorrhage • cerebral aneurysm • premonitory bleed

It is estimated that subarachnoid hemorrhage (SAH) from a ruptured cerebral aneurysm occurs in 28,000 people per year in North America and is associated with death or permanent disability in close to two-thirds of patients. The classic presentation of aneurysmal SAH is that of a very severe headache associated with stiff neck, photophobia, nausea and vomiting, and often with transient loss of consciousness. With such a presentation the diagnosis of SAH is usually obvious. Neurologists and neurosurgeons, however, have become aware that this presentation is often heralded by a more minor hemorrhage that can precede a major rupture by hours, days, or weeks. The significance of such a minor leak is often unrecognized by the physician, common misdiagnoses being migraine and neuralgia. It has been the impression that patients who sustain a major rupture after a minor leak have a worse prognosis than patients who do not have a premonitory minor leak. If the minor leak is diagnosed before a major rupture, surgery can be performed on patients in excellent clinical condition with low mortality and morbidity rates.

Although the importance of recognizing the minor leak is obvious, only a few studies have attempted to delineate this condition. The present study was undertaken to determine the characteristics of the minor leak and of the patients who experience it, and to compare the outcome of these patients to a concurrent group of patients with SAH who did not sustain a minor leak.

Clinical Material and Methods

The medical records of 87 consecutive patients with SAH from a ruptured cerebral aneurysm proven radiologically, surgically, or at autopsy were retrospectively reviewed. Those with a clear-cut premonitory event suggestive of a minor leak were analyzed according to the character, distribution, duration, and accompanying symptoms of the associated headache. The time interval from the minor leak to the major rupture or to the diagnosis of SAH, the clinical condition of the patients on admission, subsequent complications, and outcome were also considered. Patients with an aneurysm and other pathology, such as arteriovenous malformation, as well as patients with an unruptured aneurysm were excluded. In 13 patients with multiple aneurysms only the aneurysm that was proven at surgery or at autopsy to have bled was considered.

Patients were considered in three groups for compar-
FIG. 1. Histological section of the right temporal lobe of a 46-year-old woman with a history of right facial pain of 4 weeks duration diagnosed as sinusitis. On the day of admission, 4 weeks after onset, the pain suddenly became worse and she lapsed into a coma. Angiography demonstrated an aneurysm of the right posterior communicating artery. Craniotomy was performed the same day and the patient died 3 days later. Gross and microscopic pathological examination demonstrated the ruptured aneurysm and recent acute subarachnoid hemorrhage. Clusters of iron-laden macrophages (blue) are seen in the basal meninges of the right incisura temporalis, indicating a subarachnoid hemorrhage at least 2 weeks prior to the bleed that caused her death. Similar changes were absent on the left side. Gomori's iron stain, × 50.

Results

Of 87 consecutive patients with SAH from a ruptured cerebral aneurysm, 34 (39%) had a minor leak. There were 12 men and 22 women, aged 25 to 73 years (mean 44.4 years). Seven had a history of arterial hypertension and three of recurrent headaches. Seventeen aneurysms were on the right side, seven were on the left, and 10 were in the midline. They were located at the origin of the posterior communicating artery (PCoA) in 13 cases, elsewhere on the internal carotid artery (ICA) in four cases, at the anterior communicating artery (ACoA) complex in 10 cases, on the pericallosal artery in two cases, and at the middle cerebral artery bifurcation in five cases. Twenty-two aneurysms were smaller than 12.0 mm and 12 were larger. There was no difference in the location and size of aneurysms of patients without a minor leak, and a proportionally similar number suffered from hypertension and chronic headache.

Headache was the event associated with a minor leak in 33 of 34 cases. The remaining patient had what was interpreted by a physician as transient cerebral ischemia, and postmortem examination demonstrated acute and remote subarachnoid hematomas without evidence of extra- or intracranial atherosclerosis. The headache was characterized as unusual in severity and location and was so exceptional in 17 cases that medical advice was sought. Onset was sudden and the headache was most often unremitting, lasting either until a subsequent major rupture occurred or up to 2 weeks. The location of the headache is detailed in Table 1. Aneu-
rysms originating from the ACoA complex were associated with bifrontal headache radiating bi-occipitally in four (40%) of 10 cases. Ipsilateral periorbital, hemi-
cranial, or hemifacial headache was present in 53% of patients with a PCoA or ICA aneurysm. The headache was accompanied by transient nausea, vomiting, and neck pain in six cases each. Photophobia was initially
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TABLE 1

<table>
<thead>
<tr>
<th>Location of Headache</th>
<th>ICA &amp; ACoA</th>
<th>ACoA</th>
<th>MCA</th>
<th>A3</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of cases</td>
<td>17</td>
<td>10</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>frontal &amp; bifrontal</td>
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<td>4</td>
<td>2</td>
<td>1</td>
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<tr>
<td>bi-occipital</td>
<td>2</td>
<td>4</td>
<td>0</td>
<td>0</td>
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<tr>
<td>bitemporal</td>
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<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>hemicranial, hemifacial,</td>
<td>9</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
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<tr>
<td>diffuse</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

* A3 = pericallosal artery; ACoA = anterior communicating artery; ICA = internal carotid artery; MCA = middle cerebral artery; PCoA = posterior communicating artery.

absent in all cases. Confusion, lethargy, malaise, weakness, ataxia, and third cranial nerve palsy were seen in one case each.

In Group A patients, the interval from the event suggesting a minor leak to major rupture was less than 24 hours in six cases, 2 to 14 days in 13 cases, and 15 to 30 days in four cases (Fig. 2). In two cases the interval to rebleeding was unknown. Ten patients (40%) were seen by a physician before the major rupture. In no case was the correct diagnosis made. The clinical impression was of migraine in two cases and of meningitis, neuralgia, diabetes mellitus, anemia, and otitis media in one case each. In four cases no diagnosis was formulated but the patients were treated with analgesics. After the major rupture, patients were in Hunt and Hess Grades III to V in 60% of cases (Table 2). Vasospasm was demonstrated angiographically in 12 patients and was associated with cerebral infarction in six. Computerized tomography scanning demonstrated ventricular dilatation in seven patients and intracerebral and intraventricular hemorrhage in four (Table 3). Thirteen patients (52%) died, two were disabled, and 10 were independent at the time of discharge. These mortality and morbidity rates are significantly higher than those of patients with a minor leak operated on before a major rupture could occur (Group B) or those of patients without a minor leak (Group C) (Table 4).

Seven of the nine patients with a history suggesting a minor leak who were treated before a major rupture could occur (Group B) initially sought medical advice. They were at first misdiagnosed as having gastroenteritis, migraine, vasculitis, or hysteria or were undiagnosed. The eventual diagnosis of minor SAH in these patients was made by lumbar puncture or CT scanning done as part of an investigation for persistent headache. Computerized tomography scanning was performed in all cases; it demonstrated the presence of blood in the subarachnoid space in only four cases. Lumbar punc-
ture was performed in seven cases and yielded xanthochromic cerebrospinal fluid (CSF) in all. Four patients had a negative CT scan and a positive lumbar puncture. The patients were admitted in Grades I or II in 89% of cases (Table 2). Vasospasm was confirmed angiographically in six patients and was associated with clinically silent but CT-demonstrable cerebral infarction in two (Table 3). There were no deaths in this group and eight of the nine patients were independent at the time of discharge (Table 4).

Fifty-three patients seen concomitantly with the preceding groups had the classic presentation of a ruptured aneurysm that was not preceded by a minor leak (Group C). This group comprised a proportionally larger number of patients in Grades I and II and a proportionally smaller number of patients in Grades III to V than patients whose rupture was preceded by a minor leak (Table 2 and Fig. 3). In 17 patients, cerebral vasospasm was seen on angiography, and 13 had cerebral infarcts. Computerized tomography scanning demonstrated seven intracerebral and 13 intraventricular hematomas of varying size and clinical importance. Ventricular dilatation was noted in 13 patients and often resolved without specific treatment (Table 3). Six patients had CT-proven rebleeding during their hospitalization. Twelve patients (22.6%) died, and seven were disabled at the time of discharge. There were significantly lower (p < 0.05) mortality and morbidity rates in this group than in Group A (Table 4).

Discussion

The rupture of a cerebral aneurysm produces the most severe of headaches, meningismus, and photophobia, and is often associated with collapse, prostration, or a depressed level of consciousness. It has been suggested that some cerebral aneurysms are subject to recurrent small leaks,11 and Gillingham6 coined the term "warning leak" to describe this situation. He observed that the warning leak produces a moderately severe headache with minor or no accompanying features and can precede the major rupture by 10 days to 4 weeks. Ball5 has provided histological evidence that "sentinel headaches" that precede the lethal rupture of an aneurysm can be due to leakage of blood into the subarachnoid space.

With present techniques, the mortality associated with the surgery of intracranial aneurysms can be very low, the outcome depending upon the condition of the patient on admission to the hospital. The importance of diagnosing patients with a minor leak is therefore obvious because they are usually in excellent condition and their aneurysms can be treated with minimal mortality and morbidity, as was the case in our Group B patients. If the minor leak is unrecognized and a subsequent major rupture occurs, the SAH is more severe, the clinical condition of the patient is more precarious, and the risk of dying is higher than for other patients. Significant improvement in the treatment of cerebral aneurysms can, therefore, be achieved by diagnosing and treating patients before they sustain a major rupture. It is noteworthy, in this regard, that the headache associated with a minor leak was subjectively unusual in all cases. In half the cases it was so unusual that medical advice was sought, and in some cases the physician was concerned enough to have investigative procedures performed, including CT scanning. However, CT scanning is unreliable in demonstrating the subarachnoid blood produced by a minor leak.4 In some cases, persistence of the headache led to the performance of a lumbar puncture. This invariably demonstrated xanthochromic staining of the CSF. Especially interesting is the hemicranial, hemifacial, or periorbital location of the pain in 53% of patients with an ICA or PCoA aneurysm who had a minor leak. In many cases the aneurysm ruptured more than 24 hours after the minor leak, affording enough time to diagnose and treat the aneurysm if the minor leak is recognized.

We conclude that a minor leak preceding the major rupture of a cerebral aneurysm is a common occurrence. It is associated with a high mortality if it is unrecognized. Computerized tomography scanning is unreliable in diagnosing this condition. A minor leak is suspected in those patients without a history of chronic headache who present with severe, unremitting, unusual head or face pain, particularly if it is hemicranial, hemifacial, or periorbital. Other signs of an aneurysm such as a third cranial nerve palsy5 may also occur. In such a circumstance, lumbar puncture is the examination of choice once CT scanning has ruled out an intracranial mass lesion or intracranial hypertension.
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xanthochromic staining of the CSF is observed or if the CSF is bloody following atraumatic puncture, then angiography should be performed promptly and followed by surgery as soon as possible if an aneurysm is demonstrated.

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References


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