An Uncommon Cause of Intracerebral Hemorrhage in a Healthy Truck Driver

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Objectives: To describe a case and review literature for intracerebral hemorrhage caused by migraine.

Design: Case report.

Setting: Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts.

Patient: A 54-year-old truck driver with a 2-year history of atypical headaches.

Results: A 54-year-old right-handed truck driver was seen in consultation with a 2-year history of atypical headaches. The headaches were dull, throbbing, gradually progressive, and limited over the left occipital area. They were accompanied by right visual field deficit, diplopia, and, at times, confusion. These headaches were notably different from the usual migraine headaches he had been having for more than 20 years. Brain imaging revealed left parieto-occipital lobar hemorrhage. Further investigations ruled out arteriovenous malformations. He did not have any vascular risk factors, including hypertension. Migraine-associated intracerebral hemorrhage was considered to be the most likely diagnosis.

Conclusions: Intracerebral hemorrhage associated with migraine is believed to result from vasoconstriction leading to ischemia of the walls of blood vessels, making them leaky and porous. It is important to be aware of this phenomenon because vasoactive medications used to treat migraine can further aggravate the vasoconstriction and hence the intracerebral bleed.

He denied any other major medical problems or drug abuse. He has never had high blood pressure and was not aware of any excess bleeding or clotting disorder. After the first episode 2 years ago, he began receiving phenytoin sodium and levetiracetam.

Examination during the consultation revealed a normal pulse at 72 beats/min, systolic blood pressure of 125 mm Hg, and diastolic blood pressure of 80 mm Hg. On neurological examination, his visual fields were normal to confrontation but he was slow in reading and made spelling and grammatical errors in writing. He had no motor, sensory, or reflex abnormalities. Follow-up imaging was performed (Figure 2).

MIGRAINE-RELATED INTRACEREBRAL HEMORRHAGE

The patient has had 2 very different types of paroxysmal attacks. One type of attack preceded and followed the imaging finding of a left posterior intracerebral hemorrhage (ICH). This type of attack was characterized by right visual field loss, abnormalities of spoken and written language, and occasionally right limb numbness. The content of the attacks was variable. The duration was 15 minutes to an hour or longer. Many were related to severe headache. In some attacks, the abnormal functioning gradually progressed rather than beginning suddenly, and one sensory modality was affected before others. Except for the 1 attack that was followed by an ICH, the other spells left no damage either clinically or by repeated brain imaging. These attacks are consistent with migraine with aura, although the almost consistent involvement of the same brain region is atypical. A vascular malformation–arteriovenous malformation and an arterial-dural fistula were considered in the differential diagnosis. A digital-catheter angiogram showed no vascular malformation, leaving migraine with aura overwhelmingly as the most likely diagnosis for the repeated attacks.

The most likely diagnosis in this case is migraine leading to ICH. Migraines have been associated with and known to predispose to vasospasm followed by reperfusion edema and intracerebral bleeding. No comprehensive review of this important neurological entity can be found in the literature in recent years. To date, 7 instances have been described in the literature and all are in women. To our knowledge, our patient is the first man with ICH as a complication of migraine.

Migraine is a form of vascular headache; various potential biological mechanisms link migraine to ischemic stroke as outlined by Kurth,1 such as direct vasoconstriction of cerebral vessels. A number of extensive population-based studies have shown an association of migraine with ischemic brain lesions. These studies also showed that these so-called silent infarcts are more common in women than men, are more frequent in smokers, seem to be predominant in patients with migraine with aura rather than common migraine, and are predominant in posterior circulation structures.2-4

PREVIOUS REPORTS

There have been few cases in the literature about association of migraine with ICH as opposed to ischemic strokes, for which there are many studies and observations as detailed before.

Hemorrhage into the brain parenchyma has also been reported after migraine attacks. The first report of this occurrence was by Dunning,5 who described a young normotensive woman who developed a right ICH on the fourth day of a severe migraine attack. Cole and Aubé6 described 3 patients who developed lobar ICHs after at-
tacks of migraine. These individuals were all young women who started having headaches only during adulthood. The migraine headache responsible was atypical and was very severe in all patients. Examination of 2 of the patients during the headache included no neurological symptoms or abnormalities. Findings on an initial computed tomographic scan (with and without contrast enhancement) taken at the emergency department visit during the headache of 1 of the patients were normal. Intracranial bleeding was delayed after the onset of headache by at least several hours. The ICH occurred when the headache had improved markedly or was nearly gone. Angiography in all 3 patients showed vasoconstriction in arteries that supplied the region of hemorrhage and no vascular malformations.

Gautier et al described an ICH that occurred during migraine in a 62-year-old normotensive woman with normal angiographic findings. Shuaib et al described a young woman with classic migraine who had delayed ICH (36 hours after the onset of headache) without any other demonstrable cause for intracranial bleeding. This observation is similar to that made by Saper in 2 patients and Furui and Iwata in another case report. In all of these patients, including ours, migraine headache was followed by a single large lobar hemorrhage. The Table records the major data from these case reports.

The association of migraine and ICH is probably underrecognized and hence underreported. The pathophysiologic mechanism involves vascular changes during migraine attacks. Vasoconstriction is known to occur during migraine attacks, which was shown by angiography in the patients described by Cole and Aubé. This vasoconstriction can cause ischemia to brain tissue but also leads to ischemic changes in the small blood vessels supplied by the constricted arteries. The vascular ischemia can affect the continence of these vessels, making them leakier for fluid and blood. When vasoconstriction abates, blood flow to the region is augmented and reperfusion can cause hemorrhage from the damaged arteries and arterioles. This is consistent with the clinical observations made earlier that the bleed is usually delayed, suggesting the role of reperfusion and ischemia of vessel walls. The mechanism is the same as that found in hemorrhage after carotid endarterectomy and in reperfusion after brain embolization.

It is interesting to consider the role of vasoactive antimigraine medications in the ICH associated with migraine. Nighoghossian et al and Páez de la Torre et al described cases of multiple intracerebral bleeds in patients receiving large doses of vasoactive medications for treatment of migraine. These medications, if given in large amounts, could augment vasoconstriction in acute migraine, thereby increasing the risk of ICH.

The episodes of brief visual and auditory perseveration likely represent focal seizures emanating from the region adjacent to the ICH. A prolonged electroencephalographic recording confirmed the clinical suspicion of an epileptic focus in the left temporal area. However, seizures are a very unlikely explanation of the prolonged multifaceted episodes. The attacks of visual and auditory perseverations stopped after prescription of anticonvulsants but the prolonged attacks were not affected, furnishing more evidence of the disparate nature of the 2 types of attacks.

Table. Previous Reports of Migraine and Intracerebral Hemorrhage

<table>
<thead>
<tr>
<th>Source</th>
<th>Age/Sex</th>
<th>Type of Migraine</th>
<th>History of Migraines</th>
<th>Interval From Headache to Hemorrhage</th>
<th>Location</th>
<th>Angiogram With Intracranial Vessel Abnormality</th>
<th>History of High Blood Pressure</th>
<th>Surgery</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dunning, 1942</td>
<td>35/F</td>
<td>Common</td>
<td>Years</td>
<td>3-4 d</td>
<td>Right frontal</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Improved</td>
</tr>
<tr>
<td>Shuaib et al, 1989</td>
<td>41/F</td>
<td>Classic</td>
<td>25 y</td>
<td>36 h</td>
<td>Left basal ganglia</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Improved</td>
</tr>
<tr>
<td>Saper, 1989</td>
<td>54/F</td>
<td>NA</td>
<td>NA</td>
<td>Several hours</td>
<td>Right frontal</td>
<td>No</td>
<td>No</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Saper, 1989</td>
<td>43/F</td>
<td>NA</td>
<td>NA</td>
<td>5 h</td>
<td>Right frontoparietal</td>
<td>No</td>
<td>No</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Cole and Aubé, 1990</td>
<td>61/F</td>
<td>Classic</td>
<td>4 y</td>
<td>6 d</td>
<td>Right frontoparietal</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Improved</td>
</tr>
<tr>
<td>Cole and Aubé, 1990</td>
<td>45/F</td>
<td>Common</td>
<td>Years</td>
<td>24 h</td>
<td>Right frontoparietal</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>NA</td>
</tr>
<tr>
<td>Gautier et al, 1993</td>
<td>46/F</td>
<td>Common</td>
<td>20 y</td>
<td>4 d</td>
<td>Left frontoparietal</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Improved</td>
</tr>
<tr>
<td>Furui and Iwata, 1993</td>
<td>47/F</td>
<td>Common</td>
<td>7 y</td>
<td>10 d</td>
<td>Left frontal</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Improved</td>
</tr>
<tr>
<td>Current study</td>
<td>54/M</td>
<td>Common</td>
<td>25 y</td>
<td>Several hours</td>
<td>Left parieto-occipital</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Improved</td>
</tr>
</tbody>
</table>

Abbreviation: NA, not available.
REFERENCES