

Delayed Posttraumatic Middle Cerebral Artery Vasospasm Demonstrated by Magnetic Resonance Angiography: Case Report

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OBJECTIVE AND IMPORTANCE: Magnetic resonance (MR) angiographic diagnosis is a noninvasive method having high sensitivity and specificity in the detection of various cerebrovascular disorders. This is the first report of MR angiographic detection of delayed posttraumatic middle cerebral artery vasospasm, the occurrence of which has been rarely described.

CLINICAL PRESENTATION: A 42-year-old man sustained head trauma in a traffic accident, which caused a right subdural hematoma. Even though the hematoma was irrigated through one burr hole on Day 10, the patient subsequently developed left hemiparesis in association with dysarthria 4 days after surgery. MR angiography demonstrated decreased flow signal in the right M1 and M2 portions, suggestive of vasospasm.

INTERVENTION: The patient underwent intravascular volume expansion/hemodilution therapy for 3 days.

CONCLUSION: After the therapy, the ischemic symptoms completely disappeared. Follow-up study confirmed resolution of the flow signal in the right middle cerebral artery. It is suggested that MR angiography is a useful noninvasive method in the evaluation of posttraumatic cerebrovascular disorders, which constitute important secondary insults in head trauma. (Neurosurgery 43:153–156, 1998)

Key words: Cerebral ischemia, Head injury, Magnetic resonance angiography, Middle cerebral artery, Subdural hematoma, Vasospasm

Magnetic resonance (MR) angiographic diagnosis has been demonstrated to have high sensitivity and specificity in the detection of various cerebrovascular disorders, including cerebral aneurysm, arteriovenous malformations, and cerebral arterial stenosis (1, 7, 9–11, 18, 25, 26, 32). Although MR angiography still poses some limitations in comparison with intra-arterial angiography (25, 26), it offers a great advantage in the view of the noninvasive

method (26). We report a case of traumatic subdural hematoma and delayed middle cerebral artery vasospasm, which was promptly detected with MR angiography and was successfully treated.

CASE REPORT

A 42-year-old man tumbled down on an asphalt street when he tried to step aside from a bicycle on February 3, 1997,

which caused head trauma on the right side. He did not experience loss of consciousness. After the incident, he suffered from sustained headache; however, he did not seek medical treatment until he consulted Kumamoto Neurosurgical Hospital on February 10, 1997. At the time of his admission, he complained of unbearable nausea as well as severe headache, but he was alert and did not exhibit any other neurological deficits. The x-ray films disclosed no cranial fractures. Computed tomography revealed a subdural hematoma widely distributed over the right cerebral hemisphere, but deposition of blood in the subarachnoid space or in the cerebral parenchyma was not evident (Fig. 1, left).

While he received conservative treatment for 3 days, the patient manifested delirious behavior. A follow-up computed tomographic (CT) scan demonstrated increased size of the hematoma, accompanied by marked ventricular shift (Fig. 1, center). However, motor weakness was not present. Emergent one-burr hole surgery was performed on February 13, 1997. The dark brown clotty hematoma was irrigated through the burr hole made in the right parietal bone. One day postoperatively, a CT scan demonstrated that the subdural hematoma and the ventricular shift were satisfactorily improved (Fig. 1, right),

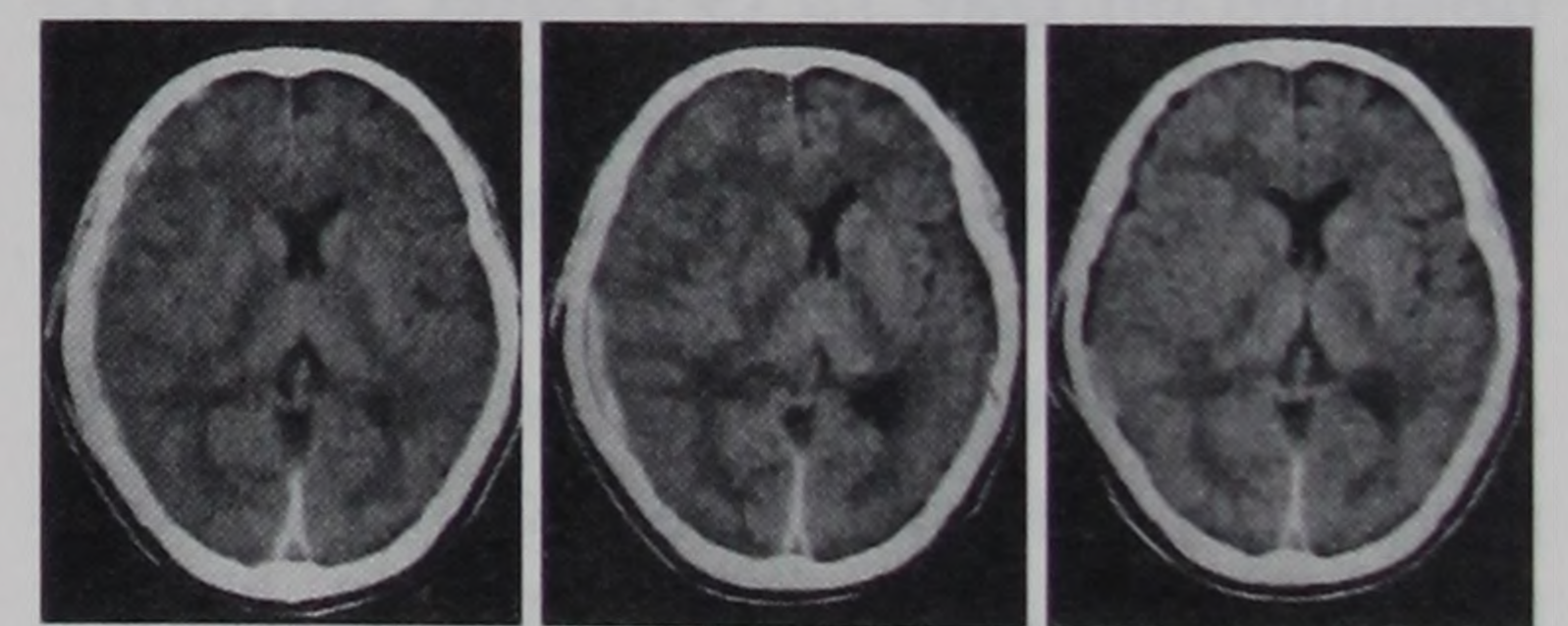


FIGURE 1. Left, CT scan obtained 7 days after head injury (at the time of admission), revealing a subdural hematoma distributed over the right side. Center, preoperative CT scan obtained on Day 10, showing increased size of the hematoma accompanied by marked ventricular shift. Right, postoperative CT scan obtained on Day 11, disclosing reduction in the hematoma size and improvement of the mass effect.

and the psychiatric symptoms had disappeared. However, the patient subsequently developed moderate left hemiparesis and mild dysarthria 4 days after undergoing surgery (14 d after incurring trauma). Although there was no evidence suggestive of increased size of the hematoma or ischemic infarction on the MR images (Fig. 2A), MR angiography clearly demonstrated a diffuse reduction in the flow signal in the right M1 portion (Fig. 2C, arrowheads) and segmental narrowing of the proximal right M2 portion (Fig. 2C, arrow). In addition, there was also mild narrowing of the A1 segments bilaterally, as well as more minimal narrowing of the left M1

segment. The flow signals in the distal branches were, however, relatively well preserved. The patient underwent intravascular volume expansion/hemodilution therapy using dextran 40/lactate Ringer's solution for 3 days, by which time the left motor weakness and dysarthria were fully resolved. The follow-up study conducted 15 days after the onset of the ischemic symptoms (29 d after incurring trauma) demonstrated consequent resolution of the arterial stenosis (Fig. 2D). MR images obtained at the same time revealed no sign of the hematoma (Fig. 2B). The patient was discharged on March 14 without any neurological deficits.

DISCUSSION

Recent clinical studies (22, 24) have provided strong evidence that ischemia is a potentially treatable secondary insult after head injury. Ischemic brain damage has been observed in 91% of the patients who died as a result of their head injury, and its distribution often conforms to the regional patterns characteristic of occlusive arterial disease (6). Therefore, subsequent to head injury, the patient should be cautiously monitored not only for subdural, epidural, or intraparenchymal hematoma or cerebral contusion but also for secondary arterial stenosis. In the present case, arterial stenosis was most evident in the right M1 and M2 portions, but mild degree of narrowing was observed in the bilateral A1 as well as in the left M1 segments. This may suggest that head injury exerts a considerable influence on widely distributed cerebrovascular systems extending over bilateral hemispheres. Thus, early detection and appropriate therapies are essential for patients with posttraumatic arterial stenosis to prevent the spreading of cerebral ischemia.

Although intra-arterial conventional and/or digital subtraction angiography is considered to be the standard of reference to depict such intracranial vessel abnormalities, the present report suggested that MR angiography is a useful noninvasive method in the evaluation of posttraumatic arterial stenosis. If available, transcranial Doppler ultrasonography may greatly support the MR angio-

graphic findings and is noninvasive (17, 30).

Relatively few cases of posttraumatic middle cerebral artery stenosis have been reported (2-4, 8, 13, 14, 20, 29), and the pathogenesis is not clearly understood. Although the MR angiographic findings in the present case were suggestive of arterial vasospasm (15), several other postulated mechanisms, including dissecting aneurysm, emboli from the cervical portion of the internal carotid artery, and thrombus formation, may also possibly be involved (8). Subarachnoid blood has been intimately related to the development of delayed posttraumatic vasospasm (12, 17, 23, 27, 30). Although in the present case, computed tomography disclosed no evident subarachnoid hemorrhage, it is possible that minor bleeding into the subarachnoid space occurred at the acute stage and did not become detectable until 7 days after the trauma was incurred. Interestingly, Freidenfelt and Sundström (5) reported that spasm was demonstrated even in the absence of associated intracranial bleeding. Biomechanical injury directly related to the initial impact may have been contributory in these cases without subarachnoid hemorrhage (16, 21, 23, 31). Intracranial hypertension, as well, has been postulated to produce arterial narrowing after head injury (19); however, when the ischemic symptoms developed in our patient, the subdural hematoma and associated cerebral edema showed successful improvement after surgery. It is unlikely that the surgical intervention produced any mechanical effects on the surrounding vasculature because it involved only minimally invasive surgery with irrigation through one burr hole.

A routine therapy for posttraumatic arterial occlusion has not yet been established. Although for posttraumatic vasospasm, it seems reasonable to consider the use of calcium channel blockers, papaverine, hypervolemia/hemodilution/hypertension, or balloon dilatation angioplasty (17, 23, 28), the safety and efficacy of these treatments in head-injured patients is a complex issue. For example, hyperdynamic volume expansion therapy, which we used in the present patient, may be somewhat hazardous if high intracranial pressure is

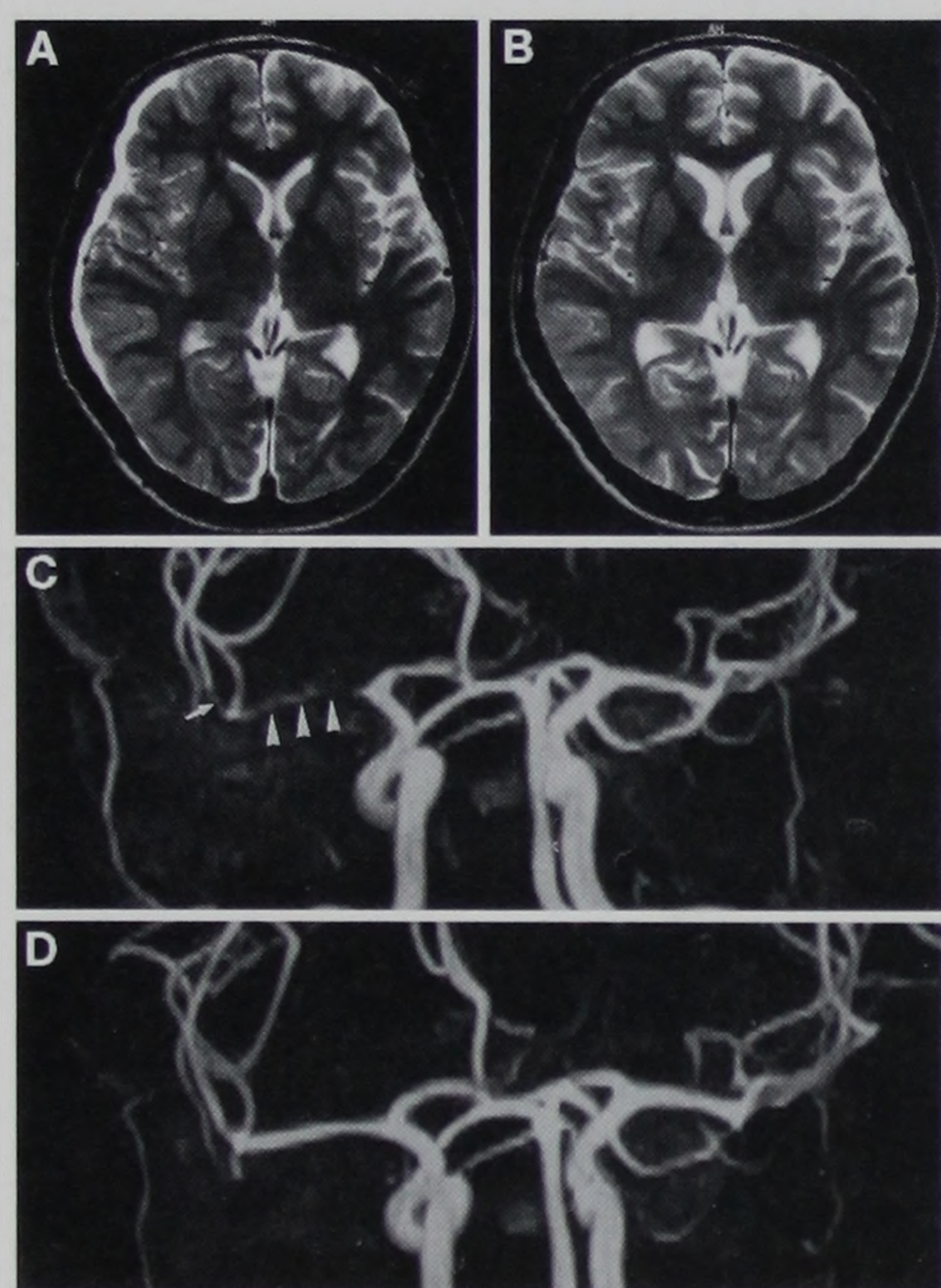


FIGURE 2. A, T2-weighted MR image obtained on Day 15 (5 d after surgery), disclosing no abnormal findings except for a small residual hematoma in the right subdural space. B, T2-weighted MR image obtained on Day 29, revealing no evidence of the subdural hematoma. C, MR angiogram (Day 15) demonstrating decreased flow signal in the right M1 portion (arrowheads) and segmental stenosis of the right M2 portion (arrow), suggestive of vasospasm. There is also mild narrowing of the A1 segments bilaterally, as well as more minimal narrowing of the left M1 segment. D, MR angiogram (Day 29) demonstrating complete restoration of the flow signal in the right M1 and M2 portions, indicating resolution of vasospasm.

present. Thus, care should be taken to examine whether the other intracranial abnormalities exist when such therapies are used. The combination of MR imaging and MR angiography holds the potential for the noninvasive detection, at one examination, of both intracranial parenchymal and vascular lesions, providing information that will be helpful in treating delayed cerebral ischemia subsequent to head injury.

ACKNOWLEDGMENTS

We are very grateful to Dr. Masaki Miura, M.D., Ph.D., of the Department of Neurosurgery in Kumamoto City Hospital for essential advice and to Hirohisa Futami of the radiological section in Kumamoto Neurosurgical Hospital for help with MR angiography and MR imaging.

Received, March 27, 1997.

Accepted, February 19, 1998.

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COMMENTS

One of the most critical issues in the successful management of patients with brain injury is the prompt recognition and treatment of symptoms of secondary ischemic insult. In this case report, the authors present a patient who developed a traumatic subdural hematoma, which was evacuated by burr hole drainage. The deterioration revealed by his neurological examination was not explainable by conventional computed tomography, which showed resolution of the preoperative mass effect. A magnetic resonance (MR) angiogram was obtained and confirmed the clinical suspicion that the patient's neurological deficits were caused by arterial spasm. This noninvasive study clearly demonstrated the presence of impressive segmental narrowing of the right M1 and M2 branches. The patient was treated with volume expansion and achieved an excellent neurological recovery. Follow-up MR images and MR angiograms showed no evidence of ischemia and complete resolution of the vasospasm.

This is an interesting and important study that serves principally to remind us that secondary ischemia after head injury may be the result of delayed vasospasm, and that MR angiography may be a useful noninvasive means of confirming this diagnosis. Surprisingly, the initial computed tomographic (CT) scan did not demonstrate significant

subarachnoid hemorrhage (SAH), but posttraumatic vasospasm has been reported previously in the absence of SAH. The authors correctly point out that the treatment of vasospasm with volume expansion may be problematic in patients with brain swelling secondary to trauma. The MR angiogram in this setting was useful in establishing the diagnosis that led to appropriate treatment.

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Gary K. Steinberg
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The authors present a case of a 42-year-old man who, after an accident, developed a subdural hematoma. The hematoma enlarged slightly during several days, with significant midline shift, and the hematoma was drained through a burr hole. The CT scan showed resolution, for the most part, of the midline shift. On Day 15 after the accident, the patient worsened, and MR angiography demonstrated reduced flow in the middle cerebral artery on the right side, consistent with the patient's symptoms. The patient was treated with hyperdynamic therapy, and by Day 29, MR angiography showed resolution of the narrowing of the middle cerebral artery vessel. Although a CT scan from the time immediately after the patient's head injury is not available, one suspects that SAH was present around the middle cerebral artery. This would then lead to a delayed reversible arterial narrowing. It has been documented that traumatic SAH occurring around the appropriate vessels can produce blood flow changes. The authors' argument

would be more convincing if they had obtained transcranial Doppler ultrasonographic data after determining transcranial Doppler ultrasonographic narrowing with resolution or if they had obtained an initial CT scan demonstrating blood around the middle cerebral artery. Unfortunately, neither studies are available for this particular patient. We do know whether traumatic SAH around the basal cisterns may cause these patients to develop late vasospasm identical to that observed after aneurysmal SAH. If there is hemorrhage in a specific sylvian fissure, patients must be watched for late spasm even if they are recovering well from the head injury. More distal subarachnoid blood over the convexity of the hemisphere is typically less problematic regarding the incidence or severity of true vasospasm.

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Posttraumatic arterial narrowing is a common pathological phenomenon, causing various temporary or permanent symptoms. Yamada et al. present a case in which MR angiography clearly showed arterial narrowing at the middle cerebral artery and follow-up study confirmed complete resolution.

MR angiography is a useful study of choice in such a case because the symptoms are atypical. If MR angiography were used more often in cases of more severe injury, it would likely reveal more phenomena that we do not know of at present.

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