Very Late-onset Symptomatic Cerebral Vasospasm Caused by a Large Residual Aneurysmal Subarachnoid Hematoma

—Case Report—

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Abstract

A 70-year-old female developed delayed ischemic neurological deficits at 35 days after subarachnoid hemorrhage (Hunt and Kosnik grade III, Fisher group 4) caused by a ruptured aneurysm of the left middle cerebral artery. Angiography indicated late-onset cerebral vasospasm probably due to the mass effect of a large hematoma remaining in the sylvian fissure and an intracerebral hematoma after surgery. Patients with a large subarachnoid hematoma after subarachnoid hemorrhage should receive therapy to prevent cerebral vasospasm until the mass effect of the hematoma has diminished.

Key words: subarachnoid hemorrhage, vasospasm, delayed ischemic neurological deficit

Introduction

Cerebral vasospasm usually occurs between 4 and 16 days after subarachnoid hemorrhage and persists for about 2 weeks.1,6,8,12 Delayed onset of symptomatic cerebral vasospasm is rare but may be encountered unexpectedly. We treated a patient who developed delayed ischemic neurological deficit at 35 days after subarachnoid hemorrhage.

Case Report

A 70-year-old female receiving long-term therapy for hypertension presented at the emergency room of our hospital on May 20, 1998 with sudden onset of headache followed by nausea and somnolence. On admission, her blood pressure was 240/120 mmHg. Her Glasgow Coma Scale score was 12 points (E3V3M6), indicating disturbance of consciousness. Localized neurological deficits were absent and the auditory brain stem response was normal. Brain computed tomography (CT) findings were consistent with Fisher group 4 subarachnoid hemorrhage (Fig. 1). Cerebral angiography showed a ruptured, broad-necked aneurysm measuring about 8 mm in diameter projecting anterosuperiorly at the bifurcation of the left middle cerebral artery and extrav-
sation of contrast medium (Fig. 2). Brain CT repeated immediately after cerebral angiography showed an increase in the size of the subarachnoid hematoma as well as intracerebral hemorrhage (Fig. 3). By this time, her neurological abnormalities progressed to left mydriasis, right-sided paresis, and a further decrease in the level of consciousness.

The patient underwent emergency craniotomy to clip the neck of the aneurysm and remove the hematomas. However, the hematoma in the sylvian fissure was very firm and inaccessible, and was not removed to preserve the surrounding brain tissue. The operation was completed after placing three drains in the cistern and the ventricle. Postoperative brain CT showed large residual hematomas (Fig. 4 left). Cisternal perfusion with 6000 IU/500 ml urokinase solution a day was continued for 5 days after surgery combined with normovolemic hypertensive therapy until hospital day 14. Urokinase infusion achieved almost complete lysis of the hematoma in the interpeduncular cistern, but had little effect on the large hematoma in the sylvian fissure.

Continuous transcranial Doppler (TCD) sonography showed that the mean blood flow in the left and right middle cerebral arteries was 50–60 cm/sec during the first 14 hospital days, suggesting that vasospasm had not occurred. During this period, her level of consciousness and neurological deficits did not worsen and she regained the ability to make simple daily conversation. Ventriculoperitoneal shunting and cranioplasty were performed on hospital day 21 and then rehabilitation was commenced. However, total aphasia and right hemiplegia suddenly developed on hospital day 35.

Cerebral angiography showed vasospasm causing more than 50% stenosis of the temporal and parietal rami of the left middle cerebral artery (Fig. 5), near the site of the massive residual hematoma in the sylvian fissure. Brain CT visualized cerebral infarcts
caused by the vasospasm (Fig. 4 right). Normovolemic hypertensive therapy was repeated, but did not improve the neurological deficits. Her activities of daily living grade decreased from III to IV.

Discussion

The present patient suffered very delayed onset of symptomatic cerebral vasospasm at 35 days after subarachnoid hemorrhage due to a large subarachnoid hematoma with mass effect. Previous reports of late symptomatic vasospasm occurring more than 15 days after subarachnoid hemorrhage have suggested that sepsis, meningitis, and intracerebral hematoma may be associated with increased risk of late-onset vasospasm.23 About 5% of symptomatic cerebral vasospasm following subarachnoid hemorrhage showed such late onset, so the specific hemodynamic changes following withdrawal of intravenous hydration for postoperative sepsis might provoke late symptomatic cerebral vasospasm.10 Our patient had no specific factors other than the large residual subarachnoid hematoma.

Histological studies have shown that cerebral vasospasm following subarachnoid hemorrhage is often attributable to vascular endothelial cell damage,47 platelet adhesion to the vascular intima and thrombosis, fibrous intimal hypertrophy, and infiltration of inflammatory cells into the tunica externa.9 Vasospasm does not occur when the subarachnoid hematoma is removed within 24 hours after hemorrhage.9 These findings suggest that vasoconstrictive substances spontaneously released from the subarachnoid hematoma and/or generated in the arterial walls by mechanical stimulation from the hematoma may induce cerebral vasospasm. The most important neuroimaging risk factors for cerebral vasospasm following subarachnoid hemorrhage are CT findings of large and extensive subarachnoid hematoma.11 Low blood flow in the superficial sylvian vein on the side from which the subarachnoid cavity is usually reached at surgery has also been associated with an increased risk of symptomatic cerebral vasospasm.3 Therefore, the current understanding is that several factors collaborate to induce severe cerebral vasospasm and that vasoconstrictors released from residual subarachnoid hematoma and the mass effect of hematomas are important in the etiology of vasospasm following subarachnoid hemorrhage.

We speculate that asymptomatic vasospasm occurred in the usual period, and consider that the cause of further progression of vasospasm is the large residual hematoma. In our patient, the presence of a large hematoma with mass effect and intracerebral hematomas may have caused marked mechanical stress resulting in arterial stretching or twisting around the arachnoid bands. Such mechanical stress combined with vascular traction, impaired venous return due to hydrocephalus and cerebral edema, and denervation hypersensitivity may all promote vasospasm. Continued release of vasoconstrictive substances from the residual large hematoma might have been the direct cause of the very late onset of vasospasm. We should remove residual aneurysmal subarachnoid hematoma as possible. However, patients with subarachnoid hemorrhage and a large hematoma that cannot be removed by surgery, like the present patient, should be monitored by TCD sonography and other methods for early detection of the indicators of vasospasm and should continue to receive therapy to prevent vasospasm for much longer than usual.

References

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