Biphasic occurrence of delayed ischemia after early aneurysm surgery

Case report

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✓ An unusual case of delayed ischemia following rupture of an aneurysm of the left internal carotid artery is reported. Symptoms occurred twice after clipping the aneurysm and removing most of the subarachnoid blood on the left side the day after subarachnoid hemorrhage (SAH). Initial ischemia due to vasospasm occurred on the left side of the brain on the 8th day after SAH and responded favorably to induced hypervolemia. After complete recovery, a second episode due to vasospasm occurred on the 16th day after SAH on the right side of the brain from which the subarachnoid blood had not been removed. This caused a massive lesion and permanent severe neurological deficits. This case suggests that removal of subarachnoid blood may affect the severity and time course of vasospasm, and emphasizes the necessity of extensive removal of subarachnoid blood for prevention of severe delayed ischemic symptoms.

KEY WORDS: vasospasm • cerebral ischemia • cerebral aneurysm • subarachnoid hemorrhage

DELAYED ischemic symptoms due to vasospasm are a well recognized complication of ruptured aneurysms, and are often associated with high mortality and morbidity rates. The etiology of cerebral vasospasm is not entirely clear. A widely held view is that the subarachnoid hemorrhage (SAH) is a major etiological factor in vasospasm. The following case supports this proposition: removal of subarachnoid blood at an early stage after aneurysm rupture seemed to influence the course and the severity of delayed ischemic symptoms due to vasospasm.

Case Report

This 50-year-old woman had been in good health until 2 hours before hospitalization, when she suddenly developed severe headache and lost consciousness.

Examination. On admission, her blood pressure was 220/120 mm Hg. She was drowsy with moderate neck stiffness, but had no cranial nerve deficits or lateralizing motor or sensory findings. A computerized tomography (CT) scan demonstrated blood collected in the subarachnoid space almost symmetrically (Fig. 1 left). Four-vessel study by Seldinger’s method revealed an aneurysm of the left internal carotid artery (Fig. 2A). No aneurysms were found arising from the right carotid artery distribution (Fig. 2B).

Operation. On the day after SAH, the aneurysm was clipped via a left pterional approach. The first clip did not completely obliterate the aneurysm, and

FIG. 1. Left: Preoperative computerized tomography (CT) scan showing a diffuse symmetrical blood collection in the subarachnoid space. Right: Postoperative CT scan showing absence of the previously visualized blood collection except within the right insular cistern. The white spot in the left Sylvian fissure is the clip.
Biphasic ischemia after early aneurysm surgery

Postoperative Course. The patient’s level of consciousness gradually improved. On the 8th day after SAH, she developed mild hemiparesis in the right upper limb, and angiography showed vasospasm, more severe on the left side (Fig. 2C) than the right side (Fig. 2D). The ischemic symptoms cleared with induced hypervolemia, which was discontinued after 3 days. No abnormal CT findings were observed during this period.

She was quite well until the 16th day after SAH, when she suddenly developed left hemiplegia and became lethargic. A CT scan showed an obscure low-density lesion in the right hemisphere. Emergency right carotid angiography showed severe vasospasm of the middle cerebral artery (Fig. 2E). Induced hypervolemia and hypertension were effective only for a short period immediately after the onset of the symptoms. A catheter was introduced into the frontal horn of the right lateral ventricle to monitor intracranial pressure (ICP).

On the 17th day after SAH, the patient deteriorated

FIG. 2. A and B: Carotid angiograms performed on the day of subarachnoid hemorrhage (SAH) demonstrating an aneurysm of the left internal carotid artery (A), and no aneurysms on the right side (B). C and D: Carotid angiograms performed on the 8th day after SAH demonstrating vasospasm, more severe on the left side (C) than the right side (D). E: Right carotid angiogram performed on the 16th day after SAH demonstrating severe vasospasm in the middle cerebral artery. F: Computerized tomography scan performed on the 17th day after SAH demonstrating a large infarction in the right cerebral hemisphere. The white spot in the frontal horn of the right lateral ventricle is the cannula.
further, becoming stuporous with dense paresis of the left upper and lower extremities. Repeat CT scanning revealed a large well defined low-density lesion in the right hemisphere (Fig. 2F). On the 18th day after SAH, the ICP gradually increased and was difficult to control with hyperventilation and mannitol administration. The patient became comatose with fixed and dilated pupil on the right side. Right temporal lobectomy was carried out, resulting in some improvement in the patient's condition. Six months after SAH, she could occasionally respond to verbal commands, but she still remained totally dependent.

Discussion

Removal of subarachnoid blood clots before the onset of vasospasm has been suggested as a promising method to prevent delayed ischemia after rupture of cerebral aneurysms. However, there is no published evidence that development of delayed ischemic symptoms could be eliminated by surgery performed shortly after SAH. On the contrary, an increased tendency for development of delayed ischemia has been found in patients who underwent early surgery.

In our case, early removal of subarachnoid blood did not prevent the occurrence of vasospasm; however, vasospasm and delayed ischemic symptoms were milder on the left side where the subarachnoid blood was almost completely removed. The most severe vasospasm was observed in the middle cerebral artery in the right insular cistern where the postoperative CT scan clearly showed the presence of blood. The fact that vasospasm was more severe on the side contralateral to the aneurysm in this case would be unusual without considering the effect of removal of subarachnoid blood, because vasospasm is usually worse on the side of aneurysm. The surgery did not prevent vasospasm in the arteries around which blood clots had been believed to be completely removed. However, the vasospasm was not severe enough to cause serious cerebral ischemia. Consequently, extensive removal of subarachnoid blood including the blood in the right insular cistern might have resulted in a better outcome. To achieve good results with early aneurysm surgery, it seems to be necessary to remove the subarachnoid blood demonstrated in CT scan as completely as possible.

The interval between SAH and the onset of ischemia was different in the right and left sides in our patient. We do not know whether surgery hastened the appearance of vasospasm on the side of operation or delayed it on the opposite side. In either case, it seems reasonable to assume that surgical removal of subarachnoid clots was a factor in the different timing of the appearance of delayed ischemia in the right and left sides.

This case strongly suggests that removal of subarachnoid clots affects the time course of vasospasm and is a protective measure against severe vasospasm.

References


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