Angiographic demonstration of acute phase of intracranial arterial spasm following aneurysm rupture

Case report

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A case documenting the acute phase of intracranial arterial spasm following rupture of an aneurysm arising from the left internal carotid artery is reported. The patient deteriorated due to recurrent hemorrhage while undergoing angiography 12 hours after the initial aneurysm rupture. The acute deterioration was accompanied by dilatation of the ipsilateral pupil and occurred during injection of contrast material. There was delayed filling of the middle cerebral artery complex along with this narrowing. The arterial narrowing was confirmed to have completely disappeared on an angiographic series performed 14 minutes after the first series of films. The etiology of the acute vasospasm is discussed.

KEY WORDS - vasospasm - aneurysm - subarachnoid hemorrhage

It is well recognized that cerebral vasospasm following aneurysmal subarachnoid hemorrhage (SAH) is associated with ischemic symptoms and significant rates of morbidity and mortality. Clinical ischemic symptoms due to vasospasm develop several days after SAH (4 to 16 days after SAH in our series of cases). In experimental studies, it has been observed that vasospasm is a biphasic phenomenon, occurring in the chronic phase as well as in the acute phase within minutes of SAH. However, it has not been possible to prove that an acute phase of intracranial arterial spasm occurs in man following rupture of an aneurysm. We describe the case of a patient with an aneurysm of the left internal carotid artery (ICA) in whom angiography demonstrated transient but marked narrowing of the parent artery immediately after recurrent hemorrhage.

Case Report

This 53-year-old man was well until 2 hours before admission, when he had the sudden onset of headache followed by loss of consciousness. He was drowsy on admission, but soon became semicomatose and developed a left hemiparesis. Computerized tomography (CT) demonstrated a heavy diffuse deposit of blood in the subarachnoid space and intraventricular hemorrhage (Fig. 1).

The patient received assisted ventilation while four-vessel angiographic studies were performed by the transfemoral catheter technique 12 hours after the onset of SAH. Immediately after the first injection of contrast medium for the anteroposterior series of the right carotid angiograms with left carotid compression, the patient was found to have a dilated pupil on the left side and decerebrate posturing on painful stimulation which had not been observed several minutes before.

Figure 2 illustrates the first series of angiograms obtained immediately after injection of contrast medi-

![Fig. 1. Computerized tomography scans before angiography showing blood in the subarachnoid space and in the ventricles.](image-url)
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Fig. 2. Sequential right carotid angiograms with left carotid compression, anteroposterior views. Upper Left: A film made 2 seconds after injection showing extreme narrowing of the intradural portion of the left internal carotid artery (ICA) as well as lack of filling of the distal portion of the left middle cerebral artery (MCA). Upper Right: A film made at 3 seconds showing no apparent change in the degree of the narrowing of the left ICA. There is no filling of the distal branches of the left MCA as opposed to good filling of the distal branches of the other major arteries. Lower Left: A film made at 5 seconds showing an increase in the diameter of the left ICA. Lower Right: A film made at 7 seconds showing delayed filling of the distal branches of the left MCA.

The early-phase angiograms demonstrated marked narrowing of the left ICA which filled via a hypoplastic left anterior cerebral artery. Narrowing of the ICA was demonstrated only at the intradural portion and did not involve the extradural intracavernous or the cervical portions of the vessel. However, on the film made 5 seconds after injection, the diameter of the left ICA appeared larger than on the film made 2 or 3 seconds after injection, indicating that arterial narrowing was subsiding very quickly (Fig. 2). The left middle cerebral artery (MCA) filled only at the proximal portion in the early phase, and there was delayed filling of the distal branches of the left MCA (Fig. 2). No extravasation of the contrast medium occurred.

The dilatation of the left pupil disappeared soon after a rapid intravenous infusion of mannitol was given. Oblique views of a right carotid angiogram with left carotid compression 8 minutes thereafter showed some enlargement of the left ICA and simultaneous filling of the distal branches of the MCA (Fig. 3). Anteroposterior

Fig. 3. Right carotid angiogram with left carotid compression, oblique view, 8 minutes after the first series of films which revealed arterial narrowing. The caliber of the left internal carotid artery is still small but enlarged and there is simultaneous filling of the distal branches of the left middle cerebral artery (arrows).
views of a left carotid angiogram taken 14 minutes after the first series of films did not show abnormal narrowing of the left ICA (Fig. 4 left). There was a small saccular aneurysm arising from the left ICA with a neck that was best visualized in an oblique view (Fig. 4 right). The posterior fossa angiogram was found to be normal. Repeat CT immediately after the angiographic studies revealed an increased amount of subarachnoid and intraventricular blood, providing proof of the occurrence of an aneurysm rupture during the angiographic studies (Fig. 5).

The patient’s condition slowly improved and he was able to move his left arm spontaneously the next day. On the third day after the SAH, the neck of the aneurysm was clipped via a left pterional approach and the subarachnoid blood was extensively removed. Postoperatively, the patient was stuporous for several days; his condition deteriorated thereafter due to diffuse brain swelling. The patient died 12 days after SAH; permission for an autopsy was not obtained.

Discussion

The arterial narrowing in this patient developed immediately after recurrent rupture of the aneurysm and completely disappeared soon thereafter. This transient phenomenon might be termed “acute vasospasm,” although it is unknown if this phenomenon is the same as the acute phase of vasospasm observed in experimental animals.

Vasospasm observed in clinical cases is delayed in onset for several days after hemorrhage. It has been generally believed that the extravasated blood in the subarachnoid space is related to the occurrence of this chronic phase of vasospasm. However, it is difficult to postulate that perivascular blood plays an etiological role in the development of acute vasospasm in humans, because the major vessels do not immediately contract with application of fresh blood. Vasoconstriction may be observed at or immediately after the time of rupture during aneurysm surgery. It seems that this is a reflex vasoconstriction. One cannot rule out this acute vasoconstriction of reflex origin as a possible etiology of acute vasospasm. However, this hypothesis cannot explain the simultaneous occurrence of the delayed filling in this case, because it was observed in the MCA which filled via collateral flow through the anterior communicating artery, not via the narrowed ICA.

One reasonable explanation for the arterial narrowing associated with prolonged cerebral circulation time immediately after aneurysm rupture may be an increase in the intracranial pressure. Voldby and Enevoldsen

![Fig. 4. Left: Left carotid angiogram with right carotid compression, anteroposterior view, 14 minutes after the first series of films which revealed arterial narrowing. The left internal carotid artery (ICA) is of normal size. Right: Left carotid angiogram, oblique view, obtained by rotating the head 60° to the left side, 40 minutes after the first series of films which revealed narrowing of the left ICA. The neck of the aneurysm is seen arising from the left ICA (arrow). Note that the previously narrowed artery has returned to a normal caliber.](image)

![Fig. 5. Computerized tomography scans after angiography showing an increased amount of blood, especially in the left lateral ventricle, revealing evidence of recurrent hemorrhage.](image)
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reported that recording of intraventricular pressure during recurrent hemorrhage showed a sudden steep rise, reaching a level between systolic and diastolic blood pressure in 1 to 2 minutes, followed by a gradual decrease to a steady-state level in about 10 minutes. The rise in the intracranial pressure leads to arrest of hemorrhage, but it may also cause transient arrest of the cerebral circulation. It is noteworthy in this case that a disturbance of the cerebral circulation was observed only in the area of the MCA on the side of the ruptured aneurysm and was not apparent in the territory of the other arteries, suggesting the presence of a considerable pressure gradient in the brain. In these circumstances, a major artery can narrow if the pressure of blood within it is overcome by abruptly increased extravascular pressure and by the contractile tension of its wall. It is quite possible that this pathological process was demonstrated on angiography as transient arterial narrowing and prolonged cerebral circulation time in this case.

Vasospasm produced in this way would be only transient and not last longer than seconds or minutes. Angiographic narrowing would not be demonstrated unless contrast medium had been injected during the short duration of the pressure peak following aneurysm rupture but not at the moment of rupture before the pressure would be sufficiently high to narrow the artery.

Although no satisfactory explanation as to the pathogenesis could be given for this phenomenon, this case supplies angiographic evidence for the occurrence of an acute phase of vasospasm in humans.

References


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