Angiographic changes to induced hypertension in cerebral vasospasm

Case report

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A case of cerebral vasospasm complicating intracranial aneurysm surgery is presented. Angiographic findings under hypertension and normotension revealed a paradoxical response of involved vessels suggesting that normal autoregulation is either lost or overcome by spasm.

KEY WORDS • vasospasm • autoregulation • cerebral angiography

Cerebral vasospasm following craniotomy for intracranial aneurysm remains a significant neurosurgical problem. Postoperative neurological deficits associated with spasm are thought to be caused by cerebral ischemia. Diminished oxygenation of brain tissue is believed to result from decreased cerebral blood flow (CBF) secondary to arterial constriction and increased vascular resistance.

Induced hypertension for treatment of cerebral vasospasm was originally carried out by Farhat and Schneider. In recent years it has been used with encouraging results. The rationale for this therapy is based on clinical evidence of increased blood flow under hypertension.

The present report deals with a reversal by induced hypertension of neurological complications associated with cerebral vasospasm after craniotomy for intracranial aneurysm. Instead of the anticipated vasodilatation, further increase of vasospasm was seen in the cerebral angiogram when the blood pressure was allowed to drop to normal values. This case is, to the best of our knowledge, the first angiographic demonstration of the effects of induced hypertension on cerebral vasospasm.

Case Report

This 45-year-old right-handed man entered Mount Sinai Hospital in January, 1977, after the sudden onset of headache and stiff neck. General physical and neurological examinations were normal. Lumbar puncture revealed grossly bloody cerebrospinal fluid with xanthochromic supernatant. The opening pressure was 330 mm H₂O. Nine days after admission, bilateral carotid and left vertebral angiography revealed a lobulated aneurysm of the A₁ segment of the left anterior cerebral artery. There was mild vasospasm of the supraclinoid portion of both internal carotid arteries, the left A₁ segment, the left M₁ segment (Fig. 1), the basilar artery, and both posterior cerebral arteries. Seventeen days after the original hemor-
rhage, the aneurysm was uneventfully clipped by microscopic technique (S.A.H.). Following surgery, the patient was neurologically intact. Laboratory tests were normal except for a fibrinogen elevation, which was treated with small doses of heparin.

On the ninth postoperative day, the patient suddenly developed a right hemiplegia and global aphasia. Blood pressure at that time was 130/70 mm Hg. Review of the patient's chart showed that systolic blood pressure varied between 110 and 140 mm Hg for the 12 hours preceding the postoperative complication. Approximately 1½ hours after the onset of neurological symptoms, his blood pressure spontaneously rose to 170/100 mm Hg. At that time, the right hemiplegia improved; he was able to follow simple commands, although he still had difficulty in responding to complex orders and was unable to utter intelligible words. A computerized tomography scan was negative.

Over the next 6 hours, the neurological status stabilized. Systolic blood pressure remained approximately 170 mm Hg without any treatment. The blood pressure then dropped to 136/70 mm Hg, and the hemiparesis and aphasia worsened. A presumptive diagnosis of cerebral vasospasm was made and hypertensive therapy was instituted. A right atrial catheter was inserted for the administration of levarterenol and volume expanders. Systolic blood pressure, continuously monitored by an ascending aorta catheter, was maintained between 200 and 220 mm Hg.

Thirty minutes after hypertensive therapy was started, the right hemiparesis completely resolved. Over the next 3 hours, the aphasia improved to the degree that the patient was able to follow complex commands and point to named objects, although nominal aphasia was still present. Seven hours after the onset of induced hypertension, he could name objects but was unable to form sentences or use simple phrases. Over the next 48 hours, his speech continued to improve with hypertensive therapy: During this period, systolic blood pressure dropped below 160 mm Hg on three occasions, each accompanied by hemiparesis and worsening of the aphasia. The deficits were promptly improved by increasing the systolic blood pressure to 200 mm Hg.

A left common carotid angiogram was performed via a right femoral arterial approach 72 hours after the acute onset of neurological symptoms. First an angiographic series at a blood pressure of 180/100 mm Hg was done. The tip of the catheter was anchored in the
FIG. 2. Postoperative left common carotid angiograms at a blood pressure of 180/100 mm Hg (left) and 90 mm Hg systolic (right). Note the difference in caliber of the left middle cerebral arterial branches (arrowheads). Spasm is also seen in the pericallosal artery (arrow) but there is no difference in caliber of the anterior cerebral arterial branches at different blood pressure levels. The aneurysmal clip (X) is properly placed; its shaft causes a subtraction artifact over the supraclinoid portion of the left internal carotid artery.

proximal portion of the left common carotid artery, and 12 ml of 60% iothalamate meglumine were injected at 8 ml/second. The series demonstrated proper clipping with total obliteration of the aneurysm; there was spasm of the pericallosal artery proximal to the origin of the frontopolar branch of the left anterior cerebral artery (Fig. 2 left). Early filling veins were noted draining the left frontal opercular area, representing luxury perfusion indicating cerebral ischemia. After levarterenol infusion was discontinued and systolic blood pressure spontaneously dropped to 90 mm Hg, 12 ml of contrast material were injected at the same speed as before. At this blood pressure, angiography revealed narrowing of all middle cerebral arterial branches (Fig. 2 right).

Over the next few days, levarterenol was gradually discontinued. Systolic blood pressure drifted down to 130 mm Hg without worsening of the patient's neurological condition. He was discharged on the 24th postoperative day. At that time, his motor power was normal; there was no sensory aphasia, although a mild degree of expressive aphasia remained. Three months later, his neurological examination was normal.

Discussion

Autoregulation is an intrinsic mechanism of cerebral vessels that maintains a constant CBF over a wide range of systemic arterial pressure by altering vascular resistance. Cerebral vascular autoregulation was originally demonstrated by Fog.7 Observing pial vessels through a cranial window, Fog found that "changes in the intravascular pressure cause changes in the physiologic state (tonus) of the walls of the pial arteries: a fall in pressure causing relaxation and a rise in pressure contraction of the arterial muscle fibers." Autoregulation has also been visualized angiographically during attempts to better delineate tumor vasculature.12,18 Due to autoregulation, normal cerebral vasculature constricts in response to hypertension whereas pathological tumor vessels remain unaffected. The above phenomena can be expressed by the formula CBF = P/R, where R is cerebral vascular resistance, and P is the arteriovenous blood pressure gradient across the cerebrovascular bed.

Cerebral hypoxia, trauma, infarct, or vasospasm are some conditions which may abolish normal autoregulation.16 Clinical improvement following induced hypertension in patients with cerebral ischemia has generally been attributed to a linear increase of CBF with rise in blood pressure when vascular resistance was rendered constant by vasodilatation (strokes) or constriction (vasospasm).5,11,14,16 In trauma, hypoxia, or infarct,
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cerebral vessels may behave as rigid tubes and remain dilated\textsuperscript{4,6,15} with failure to respond to variations in blood pressure.

A paradoxical response of cerebral vessels to blood-pressure alterations in cerebral vasospasm was first suggested by du Boulay, et al.\textsuperscript{4} These authors investigated the reactivity of cerebral arteries after induced spasm in baboons. In a later paper,\textsuperscript{3} they observed that "when the blood pressure rises or is maintained at a high level this pressure may be sufficient to resist or partly overcome the vasoconstriction, but when the blood pressure falls undue vasoconstriction occurs."

The same phenomena may occur in man. Angiographic findings in our case indicate that in the area of cerebral vasospasm a decrease in blood pressure caused paradoxical constriction of the involved vessels. It is significant that the areas supplied by the vessels that responded better to induced hypertension were the ones where a better clinical response was observed. In contrast, there was much less improvement in neurological function associated with the opercular area, where ischemia was demonstrated even under hypertension. This suggests that the main factor leading to an increase in local CBF is decreased vascular resistance brought about by a rise in blood pressure. The increased arterial diameter would then result in an improved flow.

This is a paradoxical change in resistance compared to the expected response of normal vessels; it indicates, in our view, that the normal autoregulatory mechanism is either abolished or overcome by the spasm of the involved vessels.

References
7. Fog M: Cerebral circulation. II. Reaction of pial arteries to increase in blood pressure. Arch Neurol Psychiatry 41:260–268, 1939

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