

Case Report

Severe Symptomatic Vasospasm following Intraventricular Hemorrhage from Arteriovenous Fistula

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The authors present a rare case of severe vasospasm following the rupture of arteriovenous fistula. On initial CT scan, hematoma in the corpus callosum and left inferior frontal region with surrounding cerebromalacia and all ventricles without apparent subarachnoid hemorrhage were seen. Angiograms showed arteriovenous fistula but did not show cerebral vasospasm. Thirteen days after admission the neurological state of patient suddenly deteriorated and bilateral motor weaknesses developed. Following angiograms revealed severe narrowing on the supraclinoid portion of bilateral internal carotid arteries, bilateral anterior cerebral arteries and bilateral middle cerebral arteries. Transluminal angioplasty and intra-arterial papaverine infusion were performed. The patient remained stable with moderate neurologic deficits.

KEY WORDS : Vasospasm · Intraventricular hemorrhage · Arteriovenous fistula.

INTRODUCTION

Cerebral vasospasm after aneurysmal subarachnoid hemorrhage (SAH) occurs frequently and is often severe. Other conditions such as spontaneous SAH of unknown origin, head injury, brain operation, lumbar puncture, hypothalamic damage, and infections are also known to cause cerebral vasospasm¹². Cerebral vasospasm after ruptured arteriovenous malformation (AVM) or arteriovenous fistula (AVF) is rare because the hemorrhage is in the intraparenchymal rather than in the subarachnoid space^{9,13}.

We report a case of intraventricular and intracerebral hemorrhage from arteriovenous fistula that unexpectedly resulted in severe symptomatic vasospasm.

CASE REPORT

A 31-year-old woman was admitted to our hospital with 3 days of recurrent headache. On admission, she was in a drowsy and disoriented state in time but had no other neuro-

logic deficits. Computerized tomography scan showed hematoma in the corpus callosum and left inferior frontal region with surrounding cerebromalacia and all ventricles without evidence of SAH (Fig. 1). Angiography revealed fine networks of arteriovenous fistula in right inferior frontal and genu portion of corpus callosum that was fed by multiple small vessels from right anterior cerebral artery (Fig. 2). Thirteen days after admission the neurological state of patient suddenly deteriorated and bilateral motor weaknesses developed. Diffusion MRI showed evidence of acute infarction in the left temporal, parietal, and perioral area and in right middle frontal and postcentral gyri (Fig. 3). Immediately performed angiography (Fig. 4) revealed severe narrowing on the supraclinoid portion of bilateral internal carotid arteries (ICAs), bilateral anterior cerebral arteries and bilateral middle cerebral arteries (MCAs). Percutaneous transluminal angioplasty was performed on the supraclinoid portion of both ICAs and M1 portion of both MCAs and intra-arterial papaverine was infused into the right middle cerebral artery (Fig. 5). After the procedure, intravenous nimodipine infusion was started. After clinical deterioration, velocities of anterior circulation returned to normal within about 2 weeks. Thirty days after admission, the frontal ventriculoperitoneal shunt was performed due to progressive hydrocephalus. The patient underwent rehabilitation exercise with moderate neurologic deficit. Definitive treatment of the AVF was postponed.

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Fig. 1. Computed tomography (CT) scans on admission showing intraventricular and intracerebral hemorrhage in the corpus callosum and left inferior frontal region with surrounding cerebromalacia. A : No subarachnoid hemorrhage in the basal cistern is detected. B : Abnormal vascular structures are noted in rostrum and genu of corpus callosum on CT angiogram.



Fig. 2. The cerebral angiogram (oblique view) on admission showing fine networks of arteriovenous fistula that received multiple small feeding vessels from right anterior cerebral artery.

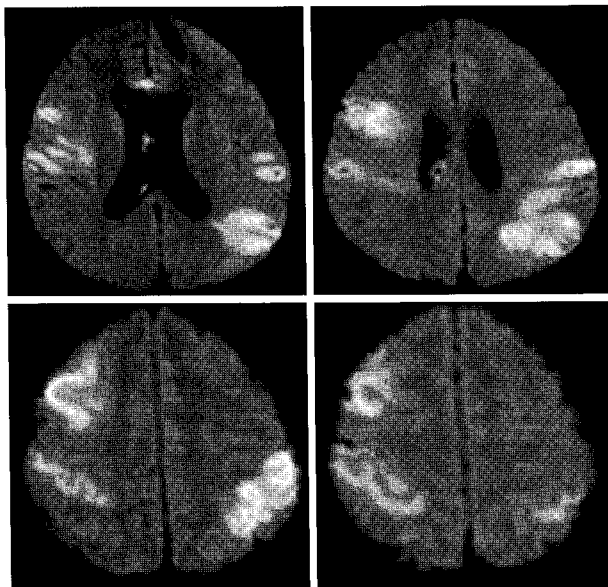


Fig. 3. Diffusion-weighted magnetic resonance images showing the evidence of acute infarction in the left temporal, parietal, and perirhinal area and in right middle frontal and postcentral gyri.

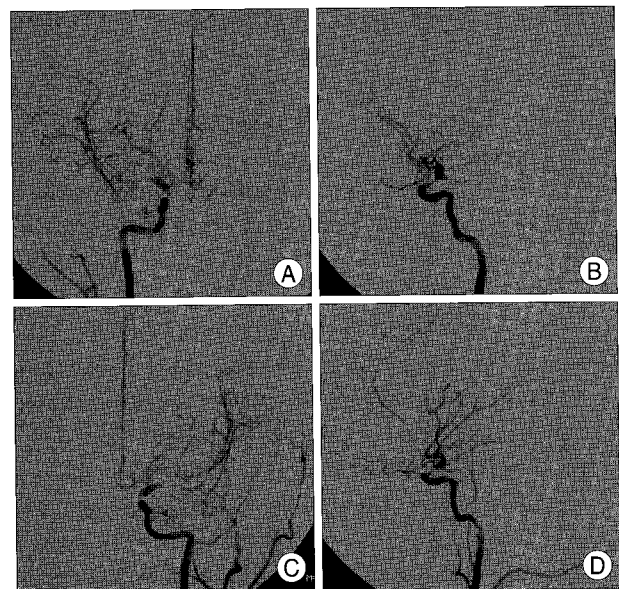


Fig. 4. Internal carotid artery angiograms on 13th day after admission. Right (A) and left (C) anteroposterior views and right (B) and left (D) lateral views reveals severe narrowing on the supraclinoid portion of internal cerebral arteries, anterior cerebral arteries and middle cerebral arteries on both sides.

DISCUSSION

The amount of blood visualized on CT scanning in the subarachnoid space in the case of a ruptured aneurysm often correlates with the severity of vasospasm^{1,3,8}. In a patient with a ruptured AVF or AVM, symptomatic vasospasm is rare^{4,5,7,13,14}. It is caused by AVM or AVF that usually rupture into the parenchyma rather than the subarachnoid space. Kurita et al.⁶ suggested characteristic features of vasospasm after solely intraventricular hemorrhage (IVH) from AVM : 1) delayed onset; 2) female predominance; 3) severely disturbed consciousness at the acute stage; and 4) localization in the internal carotid arteries on both sides. Our case demonstrated similar features in that there were delayed onset (13 days after admission), young female, and severe narrowing of the supraclinoid portion of bilateral ICAs.

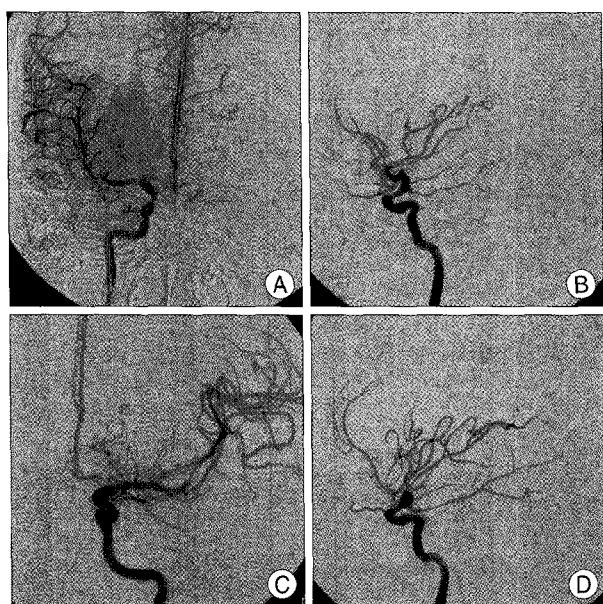


Fig. 5. Post-angioplasty internal carotid artery (ICA) angiograms showing dilatation of both ICAs and middle cerebral arteries but still narrowing of anterior cerebral arteries in anteroposterior views of right (A) and left (C) and lateral views of right (B) and left (D).

A pathophysiologic mechanism of vasospasm from solely intraventricular and intracerebral hemorrhage has not been definitely established. There are a few possibilities that explain the cerebral vasospasm to develop following rupture of an AVM or AVE. First, a factor derived from the ventricular hemorrhage and transported via the cerebrospinal fluid could be presumed. Stasis of potentially spasmogenic material due to poor clearance of CSF could develop cerebral vasospasm⁴. Second, hypothalamic dysfunction might play a role in the development of cerebral vasospasm¹¹ or functionally or structurally changed central sympathetic structures could make an influence on cerebral vessels^{2,10}. Although we often encounter hypertensive putaminal or thalamic hemorrhages with intraventricular hematoma, there is no report that patients sustain severe vasospasm without SAH. Therefore, some unknown factors may act on the development of cerebral arterial vasospasm in patients with ruptured AVM or AVE.

CONCLUSION

Vasospasm in patients with solely IVH without apparent

SAH is extremely rare. Only several cases have been reported in the literature. Although symptoms were not entirely reversed in our case, the severity of ischemia and the size of infarction were reduced. Close monitoring and early treatment of vasospasm should be considered in patients with solely IVH from vascular malformation, even if there is no SAH.

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