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 supracallosal 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 space3,13,18.

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 neurologic 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 perisylvian area and in right middle frontal and postcentral gyri (Fig. 3). Immediately performed angiography (Fig. 4) revealed severe narrowing on the supracallosal portion of bilateral internal carotid arteries (ICAs), bilateral anterior cerebral arteries and bilateral middle cerebral arteries (MCAs). Percutaneous transluminal angioplasty was performed on the supracallosal 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.
Fig. 1. Computed tomography (CT) scans on admission showing intraventricular and intracerebral hemorrhage in the corpus callosum and left inferior frontal region with surrounding cerebritis. 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 petrosectic area and in right middle frontal and postcentral gyrus.

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 reveal severe narrowing on the suprachindion 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. In a patient with a ruptured AVF or AVM, symptomatic vasospasm is rare. It is caused by AVM or AVF that usually rupture into the parenchyma rather than the subarachnoid space. Kurita et al. 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 suprachindion portion of bilateral ICAs.
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.

References

CONCLUSION

Vasospasm in patients with solely IVH without apparent