Case report: Intra-procedural aneurysm rupture during endovascular treatment causing immediate, transient angiographic vasospasm

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Abstract

Introduction—Cerebral vasospasm is a major cause of delayed ischemic cerebral injury, typically occurring 3–14 days after subarachnoid hemorrhage (SAH). Ultra-early vasospasm is defined as angiographic vasospasm observed *within 48 h* of SAH onset. Immediate vasospasm at the time of aneurysmal rupture has been suspected, but has not been previously reported. We describe a case of immediate, transient vasospasm following intra-procedural aneurysmal rupture.

Methods—A 55-year-old woman presented with SAH from a ruptured anterior communicating artery aneurysm. Subsequent coil embolization was complicated by an intra-procedural rupture following placement of the initial coil. A follow-up angiogram obtained after 9 min demonstrated moderate-to-severe vasospasm in the A2 segments of both anterior cerebral arteries.

Results—A repeat angiogram 20 min later demonstrated complete resolution of the vasospasm. The aneurysm was successfully obliterated with coil embolization. Post-procedure, the patient manifested no clinical vasospasm and made a good neurological recovery.

Conclusion—We document a case of ultra-early cerebral vasospasm that occurred immediately after an intra-procedural aneurysmal rupture. Catheter-induced vasospasm from mechanical manipulation of extracranial vasculature is well described. However, immediate vasospasm related to extravascular blood has never before been reported. This finding suggests that extravascular blood can have a *local* direct effect (presumably mechanical) on cerebral blood vessels, and may be an important mechanism for vasospasm.

Keywords

Vasospasm; aneurysm rupture; endovascular embolization; coil embolization

Introduction

Ultra-early neurological injury after aneurysmal subarachnoid hemorrhage (SAH) is the most important determinant of death and disability in SAH patients. The mechanisms that result in ultra-early neurological injury are poorly understood, particularly with a relatively poor correlation with mass effect of hemorrhage within the subarachnoid space.[20] Evidence of regional and global ischemia in the ultra-early period of SAH has been noted in anecdotal reports.[21] Cerebral vasospasm, manifesting as a delayed ischemic neurologic deficit, is an important cause of morbidity and mortality in patients with SAH.[2]

Cerebral vasospasm was first described in 1951,[9] and is commonly known to occur after day 3 of aneurysmal rupture.[8] Approximately 30%–70% of angiograms performed around the 7th day following a SAH demonstrate evidence of angiographic vasospasm.[1,3,15,16] Up to 50% of these patients will eventually suffer delayed ischemic cerebral injury as a result of vasospasm.[17,18] Ultra-early vasospasm, defined as angiographic vasospasm observed within 48 h of SAH onset, has raised the possibility that vasospasm may occur early and contribute to the ultra-early neurological injury.[6] Immediate vasospasm within minutes of aneurysm rupture has been suspected but has never been described before in litera-

Figure 1. Non contrast head CT scan demonstrating diffuse SAH with intraventricular blood (modified Fisher grade 4).

ture and may represent a mechanism for ultra-early neurological injury in SAH patients. We describe a case of immediate, transient vasospasm, which occurred within 10 min of an intra-procedural aneurysm rupture during endovascular treatment of a ruptured anterior communicating artery aneurysm.

Case report

The patient was a 55-year-old woman who presented to an outside hospital with severe headache, neck pain, and photophobia. She was transferred to our facility after a non-contrast head CT scan demonstrated diffuse SAH originating from an anterior communicating artery aneurysm, intra-ventricular hemorrhage and obstructive hydrocephalus (modified Fisher grade 4, Figure 1).

Upon physical examination, she was mildly confused and found to have nuchal rigidity. No other deficits were noted on a detailed neurological examination. The patient was then admitted to the intensive care unit and external ventricular drain was inserted to treat hydrocephalus. She was then electively intubated for a cerebral angiogram with intention to treat the ruptured aneurysm.

Procedure technique: Left internal carotid artery angiogram performed through a transfemoral route demonstrated a 3.9 mm × 4.1 mm anterior communicating artery (ACOM) aneurysm (Figure 2).

A 6 French cook shuttle® (Shuttle-SL; Cook, Bloomington, Ind.) was advanced into the left common carotid artery. An SL-10 microcatheter was advanced into the aneurysm sac over a Transend™ Ex microwire (Boston Sci. Ntick, MA) microwire. Through the microcatheter, a Cosmos Complex 3 mm × 6 cm coil (MicroVention, CA, USA) was advanced into the aneurysm without difficulty. At this point, the patient's intracranial pressure increased from 7 to 23 mmHg. Her blood pressure also increased by 20% of baseline. Immediate post-event angiogram demonstrated an intra-procedural rupture and contrast extravasation (double arrows, Figure 3) into the subarachnoid space.

The initial coil was immediately detached and the patient given 1 g/kg dose of intravenous mannitol. A second coil (Hydrosoft 2 mm × 2 cm, MicroVention, CA, USA) was expediently placed within the first framing coil mass. A second angiogram 9 min later demon-
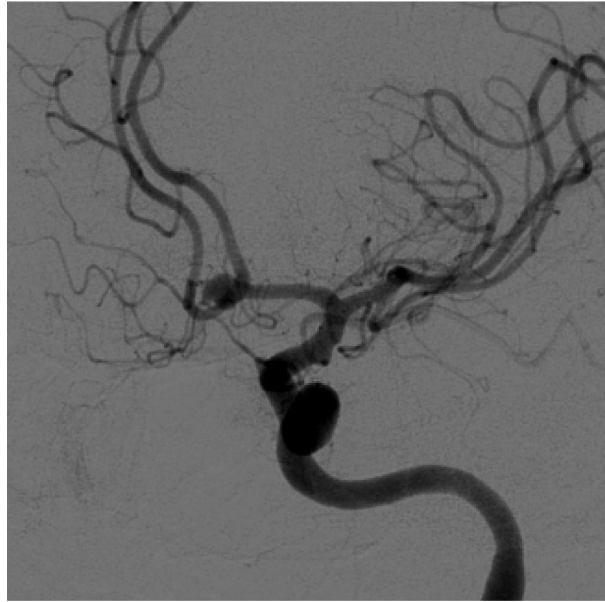
Figure 2. Left internal carotid artery angiogram demonstrating a 3.9 mm × 4.1 mm anterior communicating artery aneurysm (Acom) with normal caliber anterior cerebral arteries.
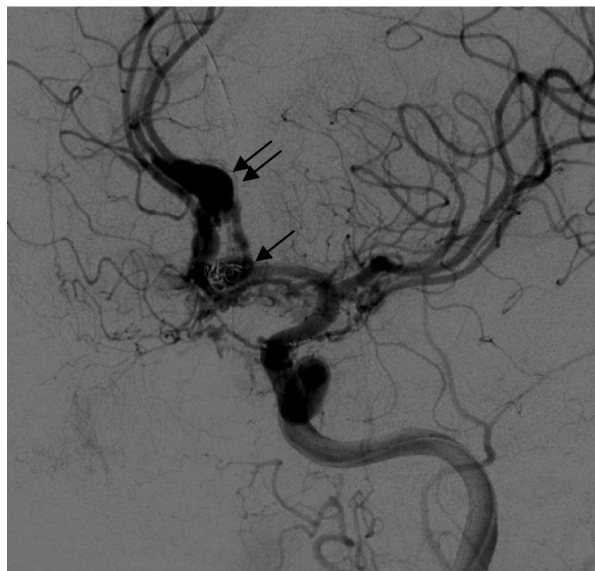

Figure 3. Left internal carotid artery angiogram demonstrating intra-procedural rupture with contrast extravasation (double arrows) into the subarachnoid space after the first coil placement (single arrow).

strated moderate to severe vasospasm in the A2 segment of both anterior cerebral arteries (double arrows, Figure 4).

There was also persistent but minimal contrast extravasation (single arrow, Figure 4). Subsequently, six more Hydrosoft coils (MicroVention, CA, USA) were placed inside the original coil mass, resulting in complete obliteration of the aneurysm. Follow up angiogram after the third coil (14 min after the intra-procedural rupture) showed mild improvement in vasospasm in both anterior cerebral arteries, with no evidence of continued contrast extravasation. Twenty minutes later (after the fourth coil
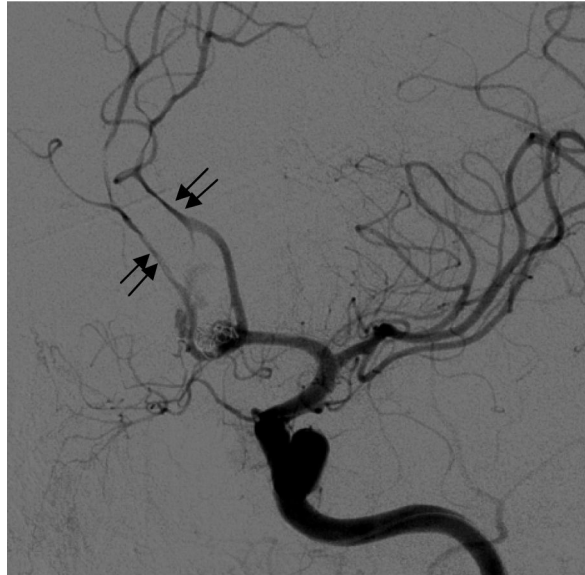
Figure 4. Left internal carotid artery angiogram demonstrating moderate to severe vasospasm (double arrows) involving both anterior cerebral arteries.
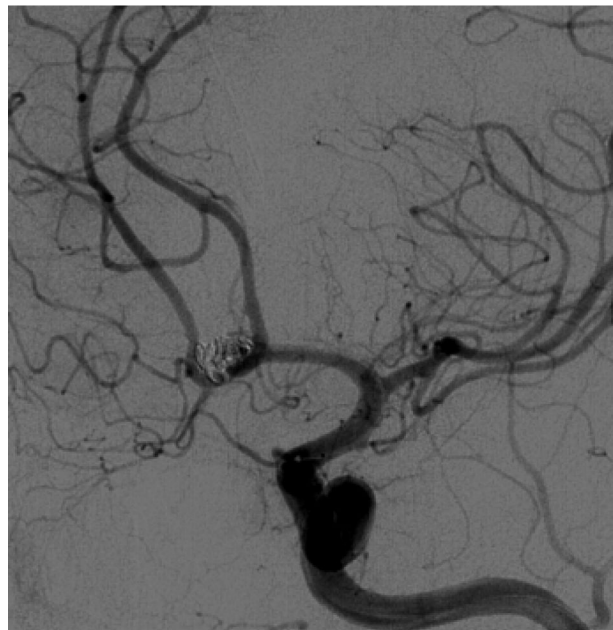

Figure 5. Left internal carotid artery angiogram shows complete resolution of bilateral anterior cerebral artery vasospasm at 20 min post aneurysmal rupture.

placement), the angiogram showed complete resolution of the vasospasm (Figure 5).

After placement of a total of eight coils, the final angiogram demonstrated complete obliteration of the aneurysm without any evidence of vasospasm.

Hospital course: Post-procedure non-contrast CT scan of the head showed increased hyperdensity in the anterior inter-hemispheric fissures and subarachnoid space. The exact contribution of blood and contrast within the hyperdense regions could not be determined. The patient's intracranial pressures remained under 20

mmHg after the first intra-procedural mannitol dose. On post-procedure day 1, patient was successfully extubated. She did not have any neurological deficit upon examination. She was monitored in the intensive care unit over the next 14 days with serial daily transcranial Doppler ultrasound studies. She did not show any evidence of radiographic or clinical vasospasm. Two weeks later, she underwent ventriculoperitoneal shunt placement for persistent hydrocephalus. On day 17, she was discharged to a short term rehabilitation facility with no focal neurological deficits.

Discussion

Cerebral vasospasm is a consequence of breakdown products of erythrocytes and subsequent production of vasoactive and spasmogenic agents, particularly oxyhemoglobin.[14, 19] Oxyhemoglobin has been shown to cause prolonged vascular smooth muscle cell constriction when injected adjacent to the middle cerebral artery in primates.[11, 12] Calcium channel blockers act by partially reversing the oxyhemoglobin mediated contraction of smooth muscle cells.[14] Concentrations of Endothelin 1, another important regulator of vascular smooth muscles, causes a dose-dependent, long-lasting vasoconstriction when applied to the adventitial side of blood vessels in experimental models.[4,5] Changes in the vessel wall including increased inflammatory cells,[13] free radicals,[14] muscle necrosis, endothelial swelling, and opening of tight junctions have also been observed.

Occurrence of vasospasm in a time frame inconsistent with blood product breakdown also supports other mechanisms that lead to cerebral vasospasm. A study was conducted on ultra-early vasospasm (within the first 48 h) among 296 patients with SAH.[6] Cerebral angiograms were performed within 48 h of the SAH onset. Thirty-seven patients (13%) patients had evidence of ultra-early vasospasm. Vasospasm was mild (minimal vessel change) in 29, moderate (vessel narrowing of 25%–50%) in six, and severe (narrowing of greater than 50%) in two patients. There was an independent effect of ultra-early angiographic vasospasm on both symptomatic vasospasm and 3-month outcome.

We report a case of ultra-early cerebral vasospasm that occurred immediately after an aneurysmal rupture. The vasospasm spontaneously resolved within 20 min. There is evidence to support that mechanical perturbation of cerebral arteries can lead to vasospasm. Direct manipulation of cerebral vasculature during aneurysm surgery, especially if it occurs between days 4 and 10, has also been shown to increase the incidence of cerebral vaso-

spasm.[7] Catheter-induced vasospasm from mechanical manipulation of extracranial vasculature is well described; however, immediate vasospasm related to extravascular blood has not been documented. One explanation could be "vessel hypersensitivity" due to already elevated circulating levels of spasmogenic agents after the first rupture; however, the vasospasm was only seen in the anterior cerebral arteries, which were in the immediate vicinity of the contrast extravasation. This observation suggests that local direct effect of extravascular blood (presumably mechanical) on cerebral vessels may also be an important mechanism for vasospasm. We hypothesize that this form of immediate transient vasospasm is a poorly recognized entity that might cause immediate neurological injury in certain poor grade SAH patients. A large number of these patients have neither hydrocephalus, nor hemorrhage with mass effect to provide an explanation for the etiology of their coma. Further studies need to identify the exact prevalence of such immediate vasospasm following aneurysm rupture and its clinical significance.

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