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Restenosis of the sigmoid sinus after stenting for treatment of intracranial venous hypertension: case report

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T. Terada Department of Neurological Surgery, Wakayama Medical University, Wakayama, Japan Abstract We report what we believe to be the first case of restenosis of the sigmoid sinus after stenting, in a 42-year-old man with an arteriovenous malformation with progressive right hemiparesis secondary to venous hypertension. Angiography revealed severe stenosis of the left sigmoid sinus, which was dilated with a self-expandable stent. Six months after the procedure, however, the sinus was again severely stenosed. Intravascular sonography revealed intimal proliferation in the stented sinus. It was dilated percutaneously, and the venous pressure decreased from 51 to 33 mmHg. On

sonography, the intimal tissue decreased in thickness and the diameter of the stent enlarged a little.

Keywords Stenting · Venous sinus · Restenosis

Introduction

Stenosis or occlusion of a venous sinus is frequently encountered in cases of cerebral arteriovenous malformation (AVM) [1, 2, 3]; it sometimes causes intracranial venous hypertension with or without intracerebral haemorrhage. Treatment of the AVM may be difficult, and revascularisation of a stenosed or occluded sinus may be effective in reducing intracranial pressure.

Previously, there were no devices suitable for navigation through the tortuous sigmoid sinus/internal jugular vein junction. However, advances in stent technology have allowed the introduction of more flexible stents into the intracranial vessels. There are reports of stent placement in the intracranial internal carotid, basilar and vertebral arteries and venous sinuses [4, 5, 6, 7, 8]. Stenting of a stenosed or occluded sinus can relieve venous hypertension [6, 7, 8], although the long-term outcome is unknown. We report what we believe to be the first case of restenosis of the sigmoid sinus after stenting for treatment of venous hypertension associated with a cerebral AVM. We discuss the pathogenesis of restenosis in relation to intravascular ultrasound (IVUS) findings and the use of percutaneous transluminal angioplasty (PTA) for sinus restenosis.

Case report

A 42-year-old man with a history of convulsions 10 years previously, treated by anticonvulsants, was first admitted to our hospital 2 years ago. MRI and angiography demonstrated a left frontoparietal Spetzler-Martin grade IV AVM (Fig. 1) fed by the anterior, middle, and posterior cerebral arteries. It had three drainage routes, into the superior sagittal sinus (SSS), the sigmoid sinus via

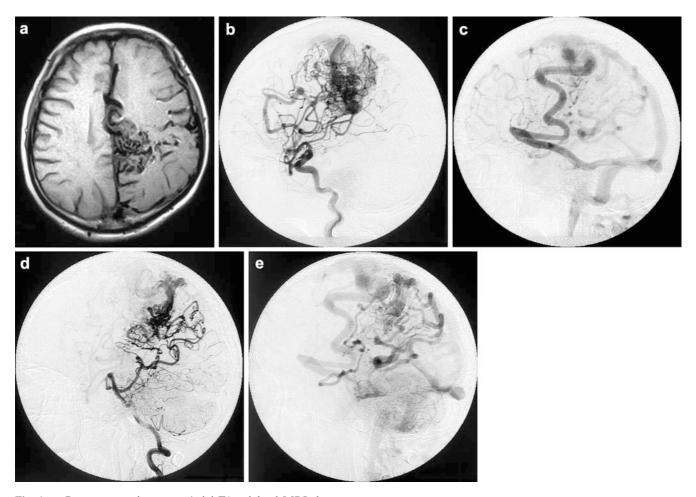


Fig. 1a–e Pre-treatment images. a Axial T1-weighted MRI showing a left frontoparietal arteriovenous malformation (AVM). b, c Left internal carotid angiogram b arterial phase, showing the anterior part of the AVM fed by the anterior and middle cerebral arteries, and c venous phase showing drainage to the superior sagittal, sigmoid, and straight sinuses. d, e Left vertebral angiogram d arterial phase, showing the posterior part of the AVM fed by the posterior cerebral artery, and e venous phase, showing major drainage to the straight sinus

the vein of Labbé, and the straight sinus via the vein of Galen. The patient was neurologically intact, and his convulsions were well controlled. We therefore decided against surgery.

He was readmitted because of uncontrollable convulsions, motor aphasia, right hemiparesis and homonymous hemianopia. CT and MRI (Fig. 2) demonstrated intraventricular blood, dense haemorrhage around flow voids above the corpus callosum, and widespread high signal in the occipital lobe. Angiography revealed occlusion of the straight sinus and severe stenosis of the sigmoid sinus; there was an intranidal varix, corresponding to the flow void on MRI. We thought that venous hypertension due to reduced drainage route led to the formation of an intranidal varix and intraventricular haemorrhage. His family declined surgical resection of the AVM, so we decided to treat the patient palliatively.

First, we embolised the intranidal varix with coils and eudragit [9] to prevent further bleeding. Immediately after embolisation, the varix was completely obliterated. We then dilated the stenosed sigmoid sinus to relieve the venous hypertension. We placed a 7 F

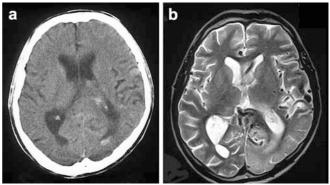


Fig. 2a, b CT and T2-weighted MRI showing intraventricular haemorrhage and white matter changes in the left occipital lobe

sheath in the left internal jugular vein and a 5 French sheath in the right femoral artery, for diagnostic angiography. Before treatment, the cerebral circulation time (from the appearance of intracranial arteries to the disappearance of the sigmoid sinus) was 6.0 s. We navigated a PTA balloon into the stenosed sigmoid sinus, and dilated it at 6 atm for 1 min, repeating this procedure so as to cover the entire stenosis. Angiography showed significant improvement in venous outflow, but the sigmoid sinus restenosed after several minutes. Finally, we deployed a 6×20 mm self-expandable Easy

Wallstent to cover the entire stenosis from the proximal transverse sinus to the sigmoid sinus (Fig. 3). Venography confirmed wide patency of the sigmoid sinus, and the cerebral circulation time fell to 4.8 s.

The patient's hemiparesis improved dramatically within a day. Ticlopidine hydrochloride (200 mg/day) was started on the night of the procedure and continued thereafter. Angiography 3 months after stenting revealed slight restenosis of the sinus, but the circulation time remained normal. However, angiography 6 months after the procedure showed severe restenosis and the circulation time was prolonged (Fig. 4). We planned to operate to prevent further neurological deterioration and rebleeding, but the family again declined surgical resection of the AVM.

The patient suddenly deteriorated 2 weeks later, becoming drowsy, with global aphasia and right hemiplegia. CT demonstrated a large haemorrhage around the nidus (Fig. 4c). We thought that venous hypertension due to restenosis had caused the haemorrhage. The family finally consented to surgical resection. To reduce the venous pressure and to operate more safely, we again

performed PTA of the restenosed sigmoid sinus. Introducer sheaths were placed as in the previous procedure. A microcatheter was navigated distal to the stenosis, where the venous pressure was 51 mmHg, while that proximal to the stenosis was 17 mmHg. An IVUS probe was used to examine the wall of the stented sinus, showing homogeneously hyperechoic tissue (Fig. 5). The diameter of the stent was 4.8 mm, but that of the most stenosed portion was 2.1 mm. Initially, we navigated a PTA balloon catheter into the stenosed sinus. After confirming an appropriate position, the stenosis was dilated at 6 atm for 90 s. Angiography demonstrated significant improvement in venous outflow (Fig. 5). After the procedure, the venous pressure at the distal end of the stent had decreased to 33 mmHg. IVUS showed that the hyperechoic tissue around the imaging catheter was less thick and the diameter of the stent was slightly larger, at 5.3 mm (Fig. 5d). We subsequently resected the AVM and evacuated the haematoma, without producing further neurological deficit.

Discussion

Fig. 3a-c Left internal carotid angiography. **a** Venous phase before stenting, showing severe stenosis of the left sigmoid sinus. **b**, **c** After stent deployment the sinus is widely patent

Venous sinus stenosis with AVMs is probably related to the endothelial damage derived from turbulent, unstable and irregular flow [10, 11]. Whatever the mechanism, it

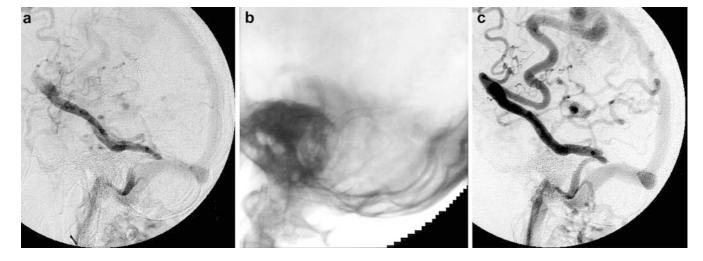
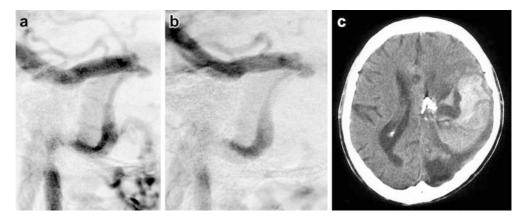


Fig. 4a-c Follow-up. a, b Left internal carotid angiogram, venous phase a 3 months after stenting, showing slight restenosis and b 6 months after the procedure showing severe restenosis of the stented sinus. c CT showing massive intracerebral haemorrhage around the nidus



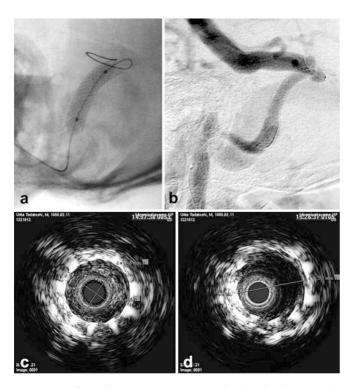


Fig. 5a-d Effects of percutaneous transluminal angioplasty (PTA). **a**, **b** Left internal carotid angiogram, venous phase showing significant improvement of restenosis. Intravascular ultrasound **c** before **d** after PTA showing intimal tissue proliferation within the stented sinus, with subsequent decrease in the thickness of the tissue and a slight increase in the diameter of the stent

can result in intracranial venous hypertension, which sometimes causes intracranial haemorrhage or neurological deficit. Our patient originally had three drainage routes, but these gradually decreased. Impairment of venous outflow caused increased pressure in the nidus, producing the varix and resulting in intraventricular haemorrhage. We therefore dilated the stenosed sigmoid sinus with a stent to improve venous hypertension. There have been three reports of stenting within a venous sinus as treatment for venous hypertension [6, 7, 8]. Two patients had dural arteriovenous fistulae [6, 7] and after stent placement, both showed antegrade venous drainage, with clinical improvement. The third had an adult pial AVM with stenosis of the SSS [8]. After embolisation of the high-flow fistula, the SSS was dilated with a 6×13 mm stent. Although the arteriovenous circulation time improved significantly, the patient died 2 weeks later from intracranial haemorrhage unrelated to the sinus stenting. Judging from these three reports and our case, stenting within a stenosed or occluded sinus appears a promising treatment for venous hypertension.

The long-term results of venous sinus stenting are unknown. Our report is the first case of sinus restenosis 6 months after stenting. In restenosis of stented coronary arteries, IVUS has shown the following: minimal chronic stent recoil; late luminal loss and in-stent restenosis because of intimal proliferation, and neointimal accumulation uniformly distributed over the length of the stent [12]. In our case, the homogeneously hyperechoic tissue was packed around the imaging catheter; it was distributed within the struts of the stent along its length, although the stent itself maintained the deployed diameter of 4.8 mm. We therefore assumed that restenosis was due to intimal proliferation, as in the coronary arteries. Restenosis of coronary arteries frequently occurs 3-6 months after stenting [13]. In our case, we saw severe restenosis 6 months after stent insertion, so that the timing of the restenosis was similar to that in coronary arteries. We did not carry out surgery after embolising the varix and stenting within the sinus. High flow shunting therefore persisted within the AVM and could have favoured restenosis of the stented segment. If we had obliterated the high-flow shunt by superselective embolisation, to reduce the pressure in the venous sinus, we might have been able to prevent the restenosis.

Several methods, such as balloon angioplasty, further stenting, cutting balloon angioplasty, rotational atherectomy and intravascular irradiation have been developed for treatment of restenosis after coronary stenting [14, 15]. Balloon angioplasty is the easiest and safest of these in the intracranial vessels. IVUS of coronary arteries after balloon angioplasty for restenosis has demonstrated that the vessel is dilated with a combination of stent expansion and tissue extrusion out of the stent [16]. In our case, the restenosed sinus could be dilated only by PTA and venous outflow was significantly improved. IVUS showed that the neointimal tissue decreased in thickness and the stent slightly expanded, These changes were therefore similar to those seen in the coronary arteries.

Development of new techniques and devices for preventing restenosis is already under way. New devices such as radioactive and drug-eluting stents will be available for the intracranial vessels [17]. A sirolimuseluting stent has already proved effective in preventing neointimal hyperplasia in coronary arteries in a multicentre, randomised, double-blind trial [18].

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