After recanalization of the SSS, however, it may re-occlude as in the present case. This is a rare case in which surgically recanalized SSS re-occluded during the postoperative course. One needs to know that re-occlusion of the SSS may occur possibly due to a change in the surrounding structure and hypercoagulopathy.

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A giant fusiform basilar aneurysm treated by bilateral vertebral artery occlusion

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Summary Objective and importance. Fusiform aneurysms of the vertebrobasilar arteries that progressively enlarge causing symptomatic brainstem compression are dangerous and their treatment is difficult. A patient with such an aneurysm treated successfully with staged, microsurgical occlusions of the proximal vertebral arteries is described, and the literature pertaining to this rare condition is briefly reviewed. Clinical presentation. A 48-year-old man with a fusiform basilar trunk aneurysm of uncertain etiology presented initially with transient ischemic attacks (TIAs) of the posterior circulation that ceased with anticoagulation. Four years later he presented again with progressive ataxia, dysphagia and dysphonia due to considerable enlargement of the aneurysm causing brainstem compression. Intervention. Staged microsurgical vertebral artery occlusions proximal to the aneurysm were performed. The second (left) vertebral artery was clipped only after the patient passed its temporary occlusion with an endovascular test balloon. The aneurysm subsequently thrombosed, the distal basilar artery kept patent by a single (left) posterior communicating artery. The patient's clinical condition improved markedly over a number of months as the aneurysm mass atrophied. Conclusion. Giant vertebrobasilar aneurysms are rare but treacherous lesions, sometimes justifying aggressive management. Carefully selected patients with progressive and severe symptoms due to brainstem compression may tolerate proximal vertebral artery occlusions, provided there is adequate collateral flow to the basilar termination and all of its perforating branches. © 2003 Elsevier Ltd. All rights reserved.

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INTRODUCTION

It has been estimated that as many as 80% of patients with symptomatic and untreated giant aneurysms become severely disabled or die within 5 years of presentation.¹ Presenting most commonly with either brainstem compression (including blockage of cerebrospinal fluid [CSF] flow and hydrocephalus) or ischemia,² these aneurysms can also rupture.³

Elimination of these aneurysms is difficult because their fusiform morphology usually prevents maintenance or reconstruction of the parent vessel, leaving as the only option proximal parent arterial occlusion (sometimes referred to as "Hunterian ligation" in older literature).³ We present the case of a man who had become unable to walk, maintain a safe airway, or swallow due to severe brainstem compression by a large and growing vertebrobasilar fusiform aneurysm, who was treated successfully with bilateral vertebral artery occlusion.

CASE PRESENTATION

A 48-year-old Caucasian male presented in 1998 after four discrete episodes of ataxia combined with left-sided weakness, each attack lasting minutes and resolving completely. The patient was a



Fig. 1 Axial CT scan of the posterior fossa and base of the brain showing a fusiform and partly calcified aneurysmal dilatation of the basilar artery.



Fig. 2 Digital subtraction right vertebral angiogram showing an irregular fusiform aneurysm of the proximal to mid basilar artery.



Fig. 4 Saggital T1-weighted MRI scan showing severe brain stem distortion from a giant, ventrally placed basilar aneurysm.



Fig. 3 CT angiogram demonstrating the relationship of the elongated and aneurysmal basilar artery to the skull base.

reformed cigarette smoker and had recently begun treatment for hypertension, but his past health was otherwise unremarkable. There was no history of connective tissue disease or systemic vasculopathy. A computed tomographic (CT) scan suggested ectasia of the vertebrobasilar arteries (Fig. 1), and a large fusiform aneurysm of the basilar artery was confirmed by digital subtraction angiography and CT-angiography (Figs. 2 and 3). It was noted that the patient had a dominant left-sided posterior inferior cerebellar artery (PICA), supplying both the left and right inferior cerebellar hemispheres. The clinical diagnosis was transient brainstem ischemia resulting from intraaneurysmal thrombosis or thromboembolism, and the patient was treated with coumadin.

The patient remained well for 4 years, but presented again in 2002 following several months of worsening balance and dysphagia. On examination the patient demonstrated severe truncal



Fig. 5 Axial T2-weighted MRI scan again showing severe brain stem distortion from a now giant fusiform aneurysm. The presence of laminated intraluminal thrombus is seen as layers of mixed signal intensity.

ataxia, moderate dysarthria and impaired gag reflexes. Magnetic resonance imaging (MRI) (Figs. 4 and 5) showed considerable aneurysm enlargement, to over 3 cm in diameter. The aneurysm contained laminated thrombus and was causing severe brainstem compression and obstructive hydrocephalus (Fig. 6) Despite ventriculoperitoneal shunting, the patient's condition deteriorated. Over several weeks he acquired aspiration pneumonia requiring mechanical ventilation and tracheostomy. A percutaneous enteral feeding tube was also necessary.

An "Alcock Test" was performed to test for angiographic patency of the posterior communicating arteries. This test involves



Fig. 6 CT scan prior to insertion of ventriculoperitoneal shunt, showing lateral ventriculomegally.

manual compression of each cervical carotid artery during contrast injection of a vertebral artery; if present and of sufficient size (i.e., >1 mm in diameter) the posterior communicating artery will be visualized during this maneuver supplying contrast to the supraclinoid carotid artery and its branches.¹ The presence of a posterior communicating artery suggests the possibility that the basilar termination and its branches will be provided collateral



Fig. 7 Left vertebral angiogram following left vertebral artery occlusion immediately distal to dominant left posterior inferior cerebellar artery supplying the inferior aspect of both cerebellar hemispheres (arrows).



Fig. 8 Left carotid angiogram showing filling of the terminal basilar artery in both posterior cerebral arteries via the left posterior communicating artery (arrow).

blood flow from the carotid in the event of deliberate proximal basilar artery or bilateral vertebral artery occlusion. Our patient had a patent left posterior communicating artery, which opacified the left middle cerebral artery on temporary manual occlusion of the left common carotid artery and vertebral artery contrast injection.

Due to the patient's severe clinical deterioration, an attempt at staged bilateral vertebral artery occlusions was recommended in the hope that aneurysm thrombosis and gradual shrinkage would decompress the brainstem. A posterior fossa exposure revealed ectatic vertebral arteries, and the left vertebral artery was occluded with a clip just distal to the dominant PICA and proximal to the visualized fusiform aneurysm. Endovascular permanent balloon occlusion of this vertebral artery was not performed because it was not considered possible to maintain patency of the PICA with this technique. The patient tolerated the procedure without neurological change.

Several days later an endovascular test balloon occlusion of the right vertebral artery was performed with the patient awake and under full heparinization. The patient remained neurologically unchanged over 15 min of right vertebral artery occlusion, at which time the test balloon was deflated and withdrawn. It therefore appeared that the patient would tolerate hemodynamic loss of both vertebral arteries proximal to the giant basilar aneurysm. The following day the patient was returned to the operating room where clip ligation of the right vertebral artery just distal to a large brainstem perforator (appreciated at the time of his first surgery) was performed. Postoperative angiography showed occlusion of the vertebral arteries proximal to the basilar aneurysm, continued filling of the dominant PICA (Fig. 7), and filling of the distal basilar artery and both posterior cerebral arteries via the left internal carotid artery and its posterior communicating artery branch (Fig. 8).

Follow-up CT scanning has shown aneurysm thrombosis and shrinkage. Over 8 months of follow-up to the time of this writing the patient has had his tracheostomy discontinued, has resumed normal feeding, has returned home and is ambulatory. Clopidogrel, which had been commenced prior to vertebral artery occlusions, was changed to aspirin (325 mg twice daily) 3 months following his surgery.

DISCUSSION

Fusiform aneurysms comprise less than 1% of vertebrobasilar aneurysms in some series,^{4,5} however the majority of *giant* aneurysms in this location are fusiform in shape.³ In many cases no identifiable underlying cause such as atherosclerosis or connective tissue disease is found.⁶ Pathological studies show attenuation of the smooth muscle layer of the arterial wall, with fragmentation of the elastic lamina.⁷ A similar disruption of the internal elastic lamina occurs in atherosclerosis.⁸ Acute and chronic inflammatory changes and atheromatous degeneration in the walls of some aneurysms have been documented.⁹ Despite this, many dispute a connection between atherosclerosis and aneurysm formation in all cases.^{2,10} Some authors have postulated the existence of an arteriopathy sharing the characteristic defect in the elastic lamina, but etiologically distinct from atherosclerosis.¹¹

Intrinsic congenital defects in the internal elastic membrane and absence of the tunica media may play a role, especially in the pediatric population.¹² De novo formation of fusiform vertebrobasilar aneurysms has been described in three children.¹³ Conditions which weaken the vessel wall such as collagen vascular disease,³ fibromuscular dysplasia,¹⁴ or maltase deficiency¹⁵ may also contribute. Hypertension is commonly associated with these lesions,¹⁶ and arterial dissection is presumed to play a role in the pathogenesis of some.¹⁶ Radiological studies of the natural history of incidentally discovered fusiform vertebrobasilar aneurysms have shown that hemorrhage into the vessel wall is associated with enlargement.¹⁷

In general, the natural history of giant aneurysms is that of progressive morbidity and mortality.¹⁸ Yasui et al. describe four patients with incidentally discovered fusiform aneurysms followed for a mean of 3.5 years. Two of the aneurysms increased in size, both associated with MRI-demonstrated hemorrhage into the vessel wall. When ischemic symptoms are present, anticoagulation is commonly used.^{5,19} Some authors advocate antiplatelet agents for all patients with fusiform vertebrobasilar aneurysms, regardless of the presence of ischemic symptoms.²⁰ Many surgical strategies have been attempted including wrapping of the aneurysm wall with various materials to reinforce it, vertebral artery occlusion (both single and bilateral), basilar artery occlusion, aneurysm resection with basilar artery reconstruction and trapping of the aneurysm (occlusion of the parent vessel proximal and distal to the aneurysm).^{1,3,16} Another technique used is extracranial-intracranial bypass followed by delayed endovascular occlusion of the parent artery.²¹ Sullivan et al.²² described trapping a giant basilar aneurysm followed by partial basilar resection and a cervical internal carotid-distal basilar saphenous vein graft, during profound hypothermia and circulatory arrest. Endovascular balloon occlusion, either alone,²³ or in combination with surgical clipping, stenting and aneurysm coiling^{24,25} have been tried. Placement of a porous stent across a fusiform aneurysm has also been successfully performed.²⁵ Finally, vessel transposition has been used to treat lower cranial nerve palsies due to compression.¹⁶ Results using any of these procedures have been highly variable.

The benchmark case-series of giant fusiform VB aneurysms is that of Drake and Peerless.³ Their series of 120 surgically treated aneurysms contained 32 patients with basilar artery aneurysms. Bilateral VA occlusion was performed on three patients; one patient had an excellent outcome and two died. Another 17 patients underwent basilar occlusion; 12 had excellent outcomes, three had good outcomes and two died. Twenty-five percent of patients treated in this manner suffer a major neurological deficit within the first 4 weeks.¹ Anson et al. treated 13 basilar, six vertebral and one PICA fusiform aneurysm, with 65% of patients experiencing good outcomes.

Proximal parent artery occlusion usually leads to obliterative thrombosis of the aneurysm,³ usually within 1 month.¹ The closer the site of occlusion is to the aneurysm, the more likely complete thrombosis will occur.¹ The risk of infarction increases with the length of basilar artery involved,³ and with aneurysms more distally located along the vertebrobasilar complex.¹

Tolerance of proximal arterial occlusion depends on collateral flow to distal branches. The posterior communicating artery size is an important predictor of success of basilar occlusion, ^{1,3,26} with a posterior communicating artery diameter greater than 1 mm significantly related to tolerance of basilar artery occlusion (p =0.004), and a favorable long-term clinical outcome.¹ Other predictors of tolerance include normal baseline cerebral blood flow and tolerance of balloon occlusion.²⁷ Perforating vessels from the basilar artery supplying the pons have traditionally been thought of as end-arteries.²⁸ The fact that brainstem infarcts can be avoided when perforating vessels arising from the walls of aneurysms are thrombosed have led to the speculation that collateral vessels exist within the brainstem.¹ It may be that fusiform basilar aneurysms which involve the entire circumference of the vessel and contain laminated thrombus have developed collateral flow, since the ostia of perforating vessels have already been obliterated with thrombus. In contrast, eccentrically positioned aneurysms with areas of native vessel wall still exposed to the lumen may pose greater risks for ischemic complications, since the perforating vessels are still patent and collateral flow has not been recruited.

In the past, brainstem compression was treated by aneurysm trapping and resection of either the aneurysmal segment or thrombus contained within it.¹ More recently it has been appreciated that reversal of compressive symptoms can result from proximal occlusion or trapping alone, due either to elimination of the pulsatile "water-hammer" effect of the aneurysm mass on adjacent neural structures or to a delayed and progressive shrinkage of the thrombosed aneurysm.^{1,24}

Endovascular techniques have gained an important role in the management of intracranial aneurysms.^{27,32-34} Temporary test balloon occlusion can assess collateral flow beyond an aneurysm and predict the hemodynamic consequences of parent artery occlusion, although not the thromboembolic risks.¹ Permanent occlusion using detachable balloons or detachable coils are useful, noninvasive methods of achieving parent artery occlusion, providing the balloon or coil mass (normally a number of millimeters in length) does not cover and occlude important branch vessels. Detachable coils are less useful within partially thrombosed aneurysms because of their tendency to sink within intra-aneurysmal clot over time, allowing recanalization.²⁹⁻³¹ In the management of the patient described in this report, microsurgical clip occlusion was selected rather than endovascular vertebral artery occlusion in order to accurately preserve the dominant left PICA and a maximum number of perforating vessels arising from the distal vertebral arteries.

CONCLUSION

A patient with a giant fusiform basilar aneurysm causing hydrocephalus and progressive brainstem compression was treated by staged bilateral microsurgical vertebral artery occlusions leading to aneurysm thrombosis. This was possible because of the presence of a posterior communicating artery adequate to supply the basilar termination, as well as intra-aneurysmal clot that had already occluded perforating branches arising from the aneurysm wall.

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Primary central nervous lymphoma presenting as bilateral cerebellopontine angle lesions: a rare case report

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Summary The authors describe a rare case of primary central nervous system (CNS) lymphoma presenting as bilateral cerebellopontine angle lesions. Imaging showed bilateral cerebellopontine angle lesions and also a small mass in right lateral ventricle and anterior third ventricle region. The right cerebellopontine angle mass was surgically excised and radio- and chemotherapy given for the remaining lesions. This is the second case reported in the world literature. The case is discussed and the literature is reviewed.

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