Appendix 1 (as supplied by the authors): Additional images and details on the surgical procedure of a 45-year-old man with a giant intracranial aneurysm

Surgical treatment of a giant intracranial aneurysm

When our patient was admitted, immediate and significant concern centred on the possibility of a cerebrovascular cause for his presentation. Urgent radiologic investigations were arranged to assess the brain and the cerebrovascular circulation of the patient. Both computed tomography (CT) and magnetic resonance imaging (MRI) of the brain showed a giant intracranial aneurysm (Figure 1A, B and D). Maximal dimensions of the aneurysm measured 6.3 cm x 5 cm in transverse and AP diameter. The aneurysm displayed a thick rim and scattered coarse internal calcification together with significant vasogenic edema. Conventional contrast cerebral angiography showed that the partially thrombosed aneurysm originated from the right internal carotid artery (ICA) bifurcation (Figure 1C). It also revealed an incomplete circle of Willis with a virtually absent proximal right anterior cerebral artery and a small posterior communicating artery (Figure 2).

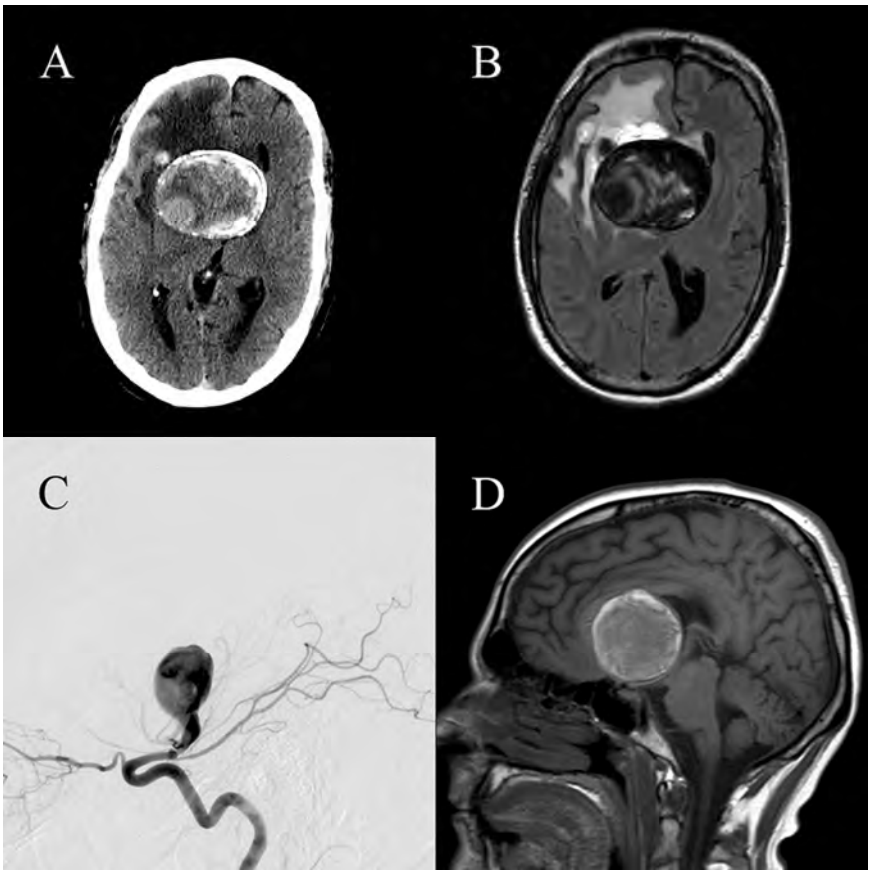

Figure 1: (A, B) Axial noncontrast computed tomography and axial fluid attenuated inversion recovery sequence magnetic resonance image (MRI), respectively, of the head of a 45-year-old man, showing a large supratentorial midline mass with thick rim of calcification and prominent vasogenic edema in right frontal lobe. (C) Conventional cerebral angiogram confirmed a giant partially thrombosed aneurysm at the origin of the right internal carotid artery bifurcation. (D) Sagittal T_1 sequence MRI of the head showing the large midline aneurysm compressing the thalamic and hypothalamic structures, as well as the superior anterior aspect of the brainstem.

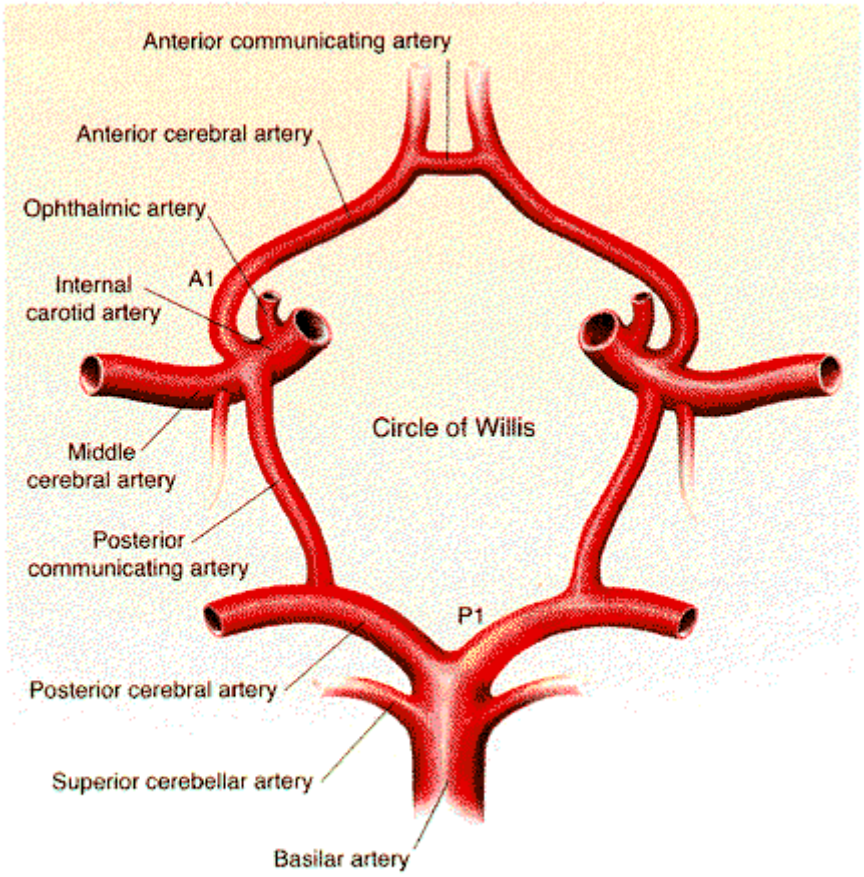

Figure 2: Circle of Willis. Reproduced with permission from Schomer DF, Marks MP, Steinberg GK, et al. The anatomy of the posterior communicating artery as a risk factor for ischemic cerebral infarction. *N Eng J Med* 1994;330:1565-70. ©1994 Massachusetts Medical Society. All rights reserved.

The patient was taken to the operating room where temporary ligation of the extracranial ICA demonstrated immediate cessation of pulsation within the aneurysm. Intraoperative micro-Doppler studies were used to assess the flow through the superficial temporal artery and to assess flow within the proximal right middle cerebral artery and right intracranial ICA with and without extracranial ICA occlusion. With extracranial ICA occlusion, the amplitude of flow within the proximal right middle cerebral artery was decreased. Because of this and the close proximity of the anterior choroidal artery to the aneurysm, as well as the somewhat isolated blood flow to the right hemisphere, we chose to obliterate the right extracranial ICA. A small amount of blood flow persisted through the right extracranial ICA supplying the nearby origin of the anterior choroidal artery. To establish collateral blood flow, we then performed an extracranial to intracranial (EC/IC) bypass with a graft from the superficial temporal artery of the external carotid artery to a temporal lobe branch of the middle cerebral artery. Intraoperative micro-Doppler studies demonstrated that flow within the middle cerebral artery branches was adequate (Figure 3).

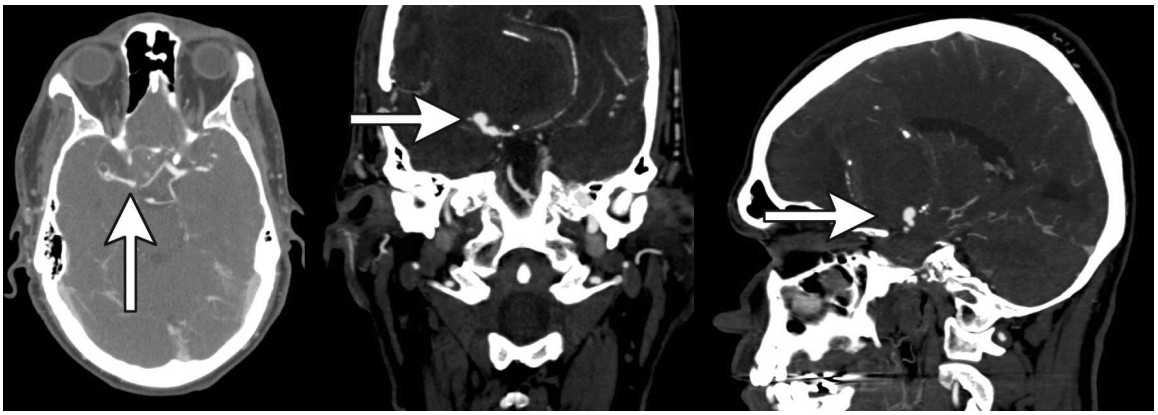

Figure 3: Postoperative computed tomographic angiogram (axial, coronal and sagittal views) showing near complete obliteration and thrombosis of the aneurysm (arrows).

Considerations behind our surgical approach

Consideration was given to more conventional treatment such as directly approaching the aneurysm through the right frontal lobe, removing clot and calcified material, and reconstruction of the right carotid bifurcation. However, given the significant amount of cerebral swelling and neurological deficit that already existed, this approach would have been accompanied by a markedly increased risk of further neurological injury and death.[1] Furthermore, intraoperative inspection of the aneurysm confirmed eccentric calcified. Therefore without entering the aneurysm to remove clot and calcium, complete clip obliteration of the proximal internal carotid artery would have resulted in distal occlusion of the ICA. Lastly, the anterior choroidal artery had its origin at the intracranial-ICA aneurysm junction. Placing the clip proximal to the anterior choroidal artery would have put this artery at risk. Over time, increasing collateral network to his right middle cerebral artery from the bypass, leptomeningial collaterals, and collateral blood flow from the anterior and posterior communicating arteries may facilitate complete occlusion of the affected aneurysmal ICA without compromise to the nearby anterior choroidal artery.

We also considered grafting of the bypass to an even more proximal segment of the middle cerebral artery. This would have provided a more robust supply of collateral blood flow; however, we felt this would have also been at the expense of significantly increased risk to the patient, given the existing degree of cerebral swelling and neurological deficit.

Treatment of giant intracranial aneurysms must be individualized and is dependent on such factors as aneurysm location, characteristics and geometry, as well as the potential for collateral flow, should an indirect approach be undertaken.

Reference

1. Choi IS, David C. Giant intracranial aneurysms: development, clinical presentation and treatment. *Euro J Radiology* 2003;46:178-94.