Dissecting aneurysm of the posterior cerebral artery: Defining the role of deliberate surgical proximal artery occlusion

Sir,

Posterior cerebral artery (PCA) aneurysms are rare and account for less than 1% among all intracranial aneurysms.[1-3] They may be either saccular or dissecting.[1]

Dissecting aneurysms are more common in the posterior circulation.[4] A 36-year-old normotensive lady presented with exacerbation of headache (present over 1 month), vomiting and drowsiness over the last 24 hours. She was opening eyes on pain, was localizing with both upper limbs and was groaning. She had right sided hemiparesis with right extensor plantar. Computed tomography (CT) scan [Figure 1a] showed left PCA territory infarct with intraventricular hemorrhage and left medial temporal bleed with effaced cisterns, cortical sulci and ipsilateral lateral ventricle and a 5 mm midline shift. 3D CT angiography [Figure 2a] showed a large upwardly and posteriorly directed aneurysm from the left P2-P3 junction. The distal PCA was not seen. Left temporal decompressive craniotomy, subtemporal approach and deliberate surgical parent artery occlusion (PAO) i.e. clipping the PCA proximal to the aneurysm and distal to the recurrent brain stem and superiorly directed branches from the P2 in the ambient cistern was done with partial removal of the left temporal clot. Postoperative CT scan [Figure 1b] showed subgaleal CSF, near complete removal of clot and left PCA territory infarct. Postoperative period was uneventful except for right homonymous hemianopia. Six months later cranioplasty was done after a CT angiogram [Figure 2b] which showed no residual aneurysm.

Intracranial dissections occur between the intima and media as opposed to extracranial dissections occurring between the media and adventitia.[5] This results in the typical angiographic features of narrowing followed by ballooning (as in our case) or of complete stenosis. Though mostly idiopathic, predisposing factors like migraine, fibromuscular dysplasia, connective tissue disorders and trauma[2,3,5] (causing shearing of the PCA along the free margin of the tentorium[2]) are associated with dissections. Unlike extracranial dissections, hypertension in not a risk factor.[5] Our patient, too, was normotensive. Further, like in our case, isolated PCA dissections have a female predilection[3-5] unlike vertebrobasilar dissections that are more common in males. The most common presenting symptom of PCA dissection is occipital headache. Aneurysm rupture can present with subarachnoid hemorrhage (SAH) and less commonly, cerebral ischemia or infarction.[3] Our patient had occipital lobe infarction, SAH and intracerebral and intraventricular hemorrhage as well. The natural history of ruptured intracranial dissecting aneurysms is reportedly unfavorable with rebleeding rates of 30%-71%,[4] hence necessitating treatment. While anticoagulation (as for extracranial dissections) has been advocated by some authors,[5] the majority favor occlusion via the endovascular route[1-4,6,7] as this location is difficult to access surgically with numerous perforators in the vicinity.[1,2] When these aneurysms are located distally, embolization of the lesion alone sparing the vessel has been highlighted as being technically difficult[4,6] and PAO by coil or glue is well tolerated due to good collateral supply.[6,7] The possible surgical strategies that can be used to treat dissecting aneurysms of PCA are trapping, excision, clipping, wrapping and deliberate PAO as well. Honda *et al*.[8] mention that approximately 20% of PCA aneurysms may require trapping or PAO (especially larger ones and those in the P2-P3 regions of the PCA). Taqi *et al*.[3] have reported four cases of dissecting PCA aneurysms from three different authors three of which underwent proximal clipping with vessel occlusion and one underwent trapping. In this case, due to the configuration and distal location of the aneurysm in the posterior circulation, it was probable that dissection was the etiology. In our case, we opted to go for a surgical intervention since our patient presented with both infarct and intracerebral hematoma causing mass effect and midline shift decompression of which would not be achieved by the endovascular route

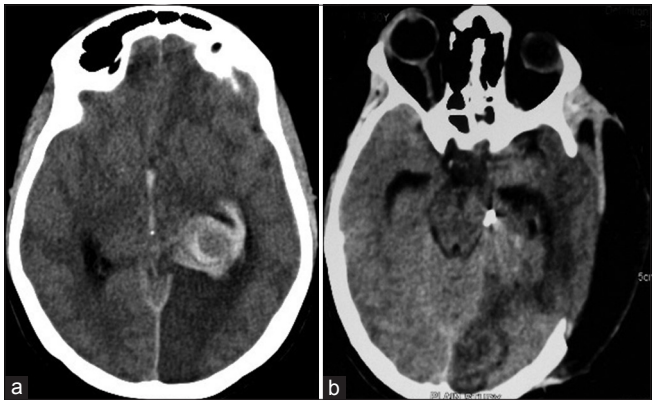

Figure 1: Axial CT (computed tomography) images showing (a) pre-operative left occipital infarct, intraventricular bleed, left medial temporal bleed, with effacement of the cisterns, cortical sulci and ipsilateral lateral ventricle and (b) postoperative established infarct, clip *in situ* and opening up of cisterns and ventricles and decrease of mass effect

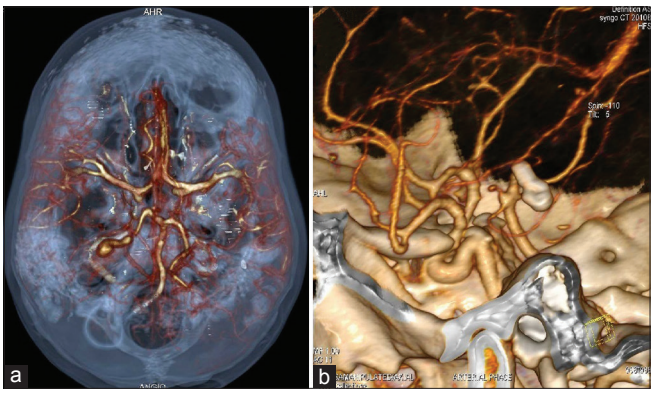

Figure 2: CT angiogram showing (a) P2-P3 junction dissecting aneurysm projecting posterosuperiorly into the brain substance. The lesion is elongated and shows stenosis followed by dilatation and (b) postoperative successful vessel occlusion by the clip with no distal flow in the vessel and no proximal reformation of neck

alone. As the observed neck in dissecting aneurysms may not represent the point of maximal weakness of the vessel wall (which may be more proximal i.e. at the site of intimal tear), clipping the neck alone does not rule out reformation of another aneurysm proximally. Hence, deliberate surgical PAO distal to all brain stem feeders was chosen. Furthermore, our patient already had an established occipital infarct and hence fresh deficits would not develop even if collaterals failed to maintain supply to the PCA territory after PAO. To conclude, surgical PAO in dissecting posterior circulation aneurysms has a definite role if the aneurysm is distal, if done in patients with an established infarct and where reduction of raised intracranial pressure is a priority.

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