A Dissecting Aneurysm of the Posterior Inferior Cerebellar Artery

A Case Report

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Summary

Dissecting aneurysms occur when blood extrudes into the wall of a vessel. Posterior circulation dissections are recognised as an important cause of cerbral infarction and subarachnoid haemorrhage(SAH), however posterior inferior cerebellar artery (PICA) aneurysmal dissections are rare. A 49-year-old man who presented with SAH was found to have a left PICA dissection on cerebral angiography. The lesion was treated with surgical clipping proximal to the dissection and a distal PICA to PICA anastomosis. The pathology, diagnosis, presentation and treatment of these difficult lesions is discussed.

Introduction

A 49-year-old man presented to the emergency room with confusion, hypertension and meningism. He was unable to give a history but according to his wife he had a mild headache and upper respiratory tract infection symptoms for the preceding three days.

He was not known to be hypertensive but had inflamatory bowel disease that was controlled by salazopyrine.

On the evening before presentation he had drunk a few tots of alcohol before going to bed. His wife heard him get up to go to the bathroom at 4am and when he did not return she went looking for him.

She found him in a confused state complaining of occipital headache and thinking he was intoxicated helped him into bed. In the morning he was difficult to rouse and an ambulance was called. On arrival at hospital his Glasgow Coma Score was 12/15, temperature was 38.5 C and he had no cranial nerve impairment or limb weakness.

A CT scan was performed which showed diffuse subarachnoid haemorrhage with a predominance of blood in the posterior fossa (figure 1).

The cisterna magna was filled with blood and there was a collection seen to the left of the brainstem. Early hydrocephalus was also evident.

The patient was transferred to the Toronto Western Hospital where he was noted to have pupils that were 3mm and sluggishly reactive to light although his GCS was unchanged.

He was intubated and a ventricular drain was inserted. Investigation of clotting parameters and a full blood count revealed no bleeding diathesis.

Cerebral angiography on the day of admission revealed a small fusiform dilation of the left lateral medullary segment of the PICA (figure 2). Because of its location and fusiform nature this was thought to represent a dissection of the artery.

The patient remained hypertensive but his

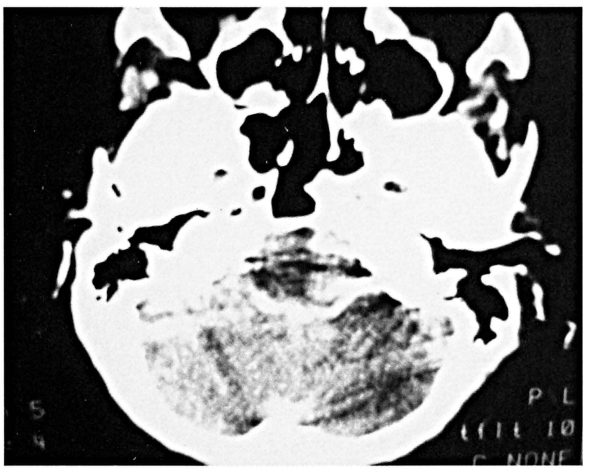

Figure 1 A CT scan on admission showing posterior fossa subarachnoid haemorrhage with clot in the left cerebello medullary cistern.

temperature settled and his level of consciousness improved. One day after admission he was extubated.

Treatment

On day 2 after admission a suboccipital craniectomy extended on the left to include the posterior third of the occipital condyle was performed. On opening the dura there was a large amount of organising haematoma in the cisterna magna which was cautiously removed to expose the lower brainstem with both tonsillomedullary segments of the PICA.

The vessels were mobilised without compromising any branches or perforators and a side to side PICA anastomosis was performed (figure 3).

The flow through the anastamosis appeared good on release of the temporary clips. Dissection of the left PICA was continued proximally to the vessel origin from the vertebral artery. A discoloured fusiform dilation was seen in the lateral medullary segment with adherent fibrinous material presumably covering the site of rupture (figure 4).

Brainstem perforators were noted to leave the PICA in the region of the dissection therefore trapping was not attempted but a proximal clip was placed at the origin of the dissection.

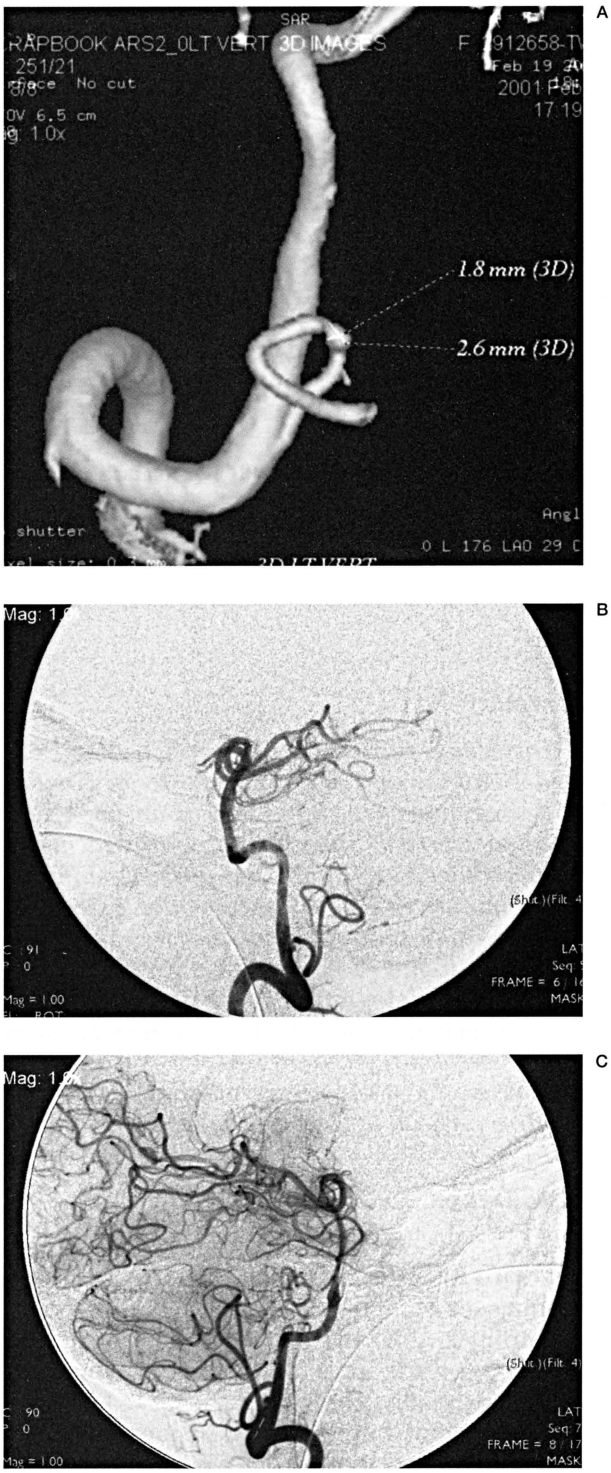

Figure 2 A) A 3D reconstruction of the left vertebral angiogram showing a small fusiform dilatation on the lateral PICA suggestive of a dissection. B) A left vertebral subtracted angiogram showing some PICA narrowing before a small fusiform dilatation of the lateral medullary segment. C) A right vertebral angiogram showing a dominant PICA suitable for anastamosis.

Discussion

Pathology

There are 21 cases of isolated PICA dissection in the literature including this case [1-4,5,7,12,15-19]. The reported frequency has increased in recent years and this presumably is due to improved imaging techniques and increased awareness of this problem. The percentage of PICA aneurysms due to dissection is unknown however Yanaura reported that 28% of all aneurysms in a series of 86 posterior circulation aneurysms were due to dissection [20]. The dissection would appear to be spontaneous in almost all cases although one patient has been described with a PICA dissection associated with Giant cell angiitis [16]. This is in contrast to extracranial carotid or vertebral artery dissection where there is often a preceding history of trauma, atherosclerosis, Marfan's syndrome, fibro-muscular dysplasia or angiitis.

Extracranial dissections typically occur in the outer two thirds of the tunica media, but intracranial dissections have been previously described as occurring in two sites. Type I dissections occur between the internal elastic lamina and media resulting in parent vessel occlusion and an ischaemic event. Type II haemorrhages occur within the media or adventitia and result in vessel rupture and SAH [21]. It would seem logical that intracranial dissection should occur between the internal elastic lamina and media as these vessels lack vasa vasorum, which are implicated in rupture of the media layer [4].

They also have areas of focal deficit in the media and where this happens the elastic layer plays an important role in vessel reinforcement. Should this layer also be weak dissection could occur. It would also seem likely that the posterior circulation harbours some other unique factor either related to flow or vessel wall integrity that predisposes to dissecting aneurysm's, as the incidence is higher than in the anterior circulation. There have been two case reports where histology of PICA dissections was reviewed. In one post mortem study haemorrhage was seen throughout the vessel walls in a bilateral PICA dissection and no defect was noted in the elastic layer [15]. In another examination of an excised aneurysm the dissection had ruptured the internal elastic layer and lay between this and the media, supporting the contention that dissection starts here but may progress to the adventitia resulting in SAH [12].

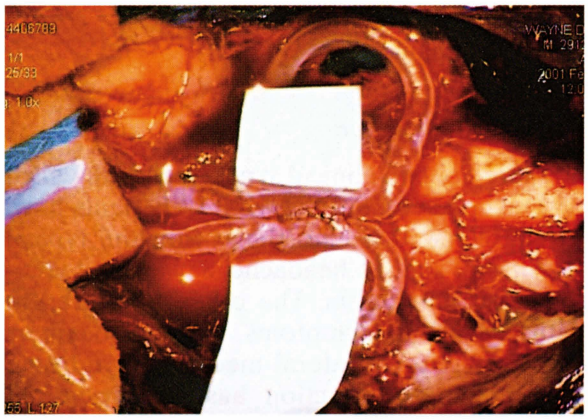

Figure 3 Intra-operative photograph of PICA to PICA anastomosis using the posterior medullary segments.

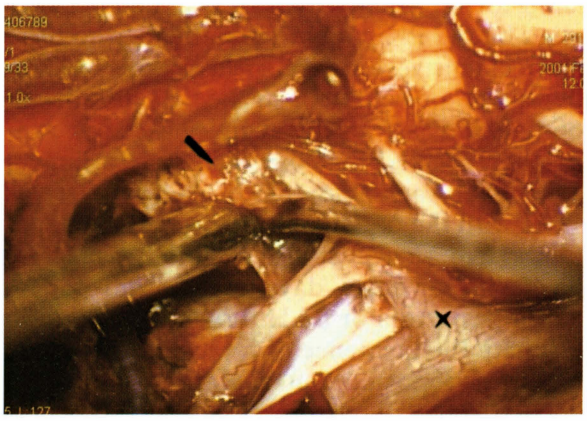

Figure 4 Intra-operative photograph showing the origin of the PICA from the vertebral artery (star) and the site of vessel dissection with discoloration and fibrinous covering (arrow).

Unlike saccular aneurysms which may occur distally on the PICA vessel at a site of branching dissecting aneurysms occur in either the anterior, lateral or posterior medullary segments of the vessel. As in this case the location may help in making the diagnosis. Isolated PICA dissections also need to be differentiated from vertebral artery dissections which expand to include the PICA origin, these may easily be confused with PICA origin saccular aneurysms.

Macroscopically these lesions have a similar appearance when viewed in the acute phase. There is a sausage like dilatation of the vessel with purple or red discoloration and even blistering of the adventitia. This represents the

mural haematoma in various phases of coagulation or organisation [1,3,11,16,19].

Clinical Presentation

There are two clinical syndromes with which patients present. Fifty percent of patients have a subarachnoid haemorrhage with the typical findings of sudden headache, nausea and vomiting and meningism. The other 50% present with ischaemic symptoms without haemorrhage, most often lateral medullary syndrome but cerebellar infarction has also been described [15].

The patients with ischaemia tend to have improvement in their condition presumably related to the smaller area of the medulla supplied by the PICA than the patients who have more extensive vertebral artery occlusion [6]. Patients who present with stroke may also develop haemorrhage in the acute phase so it cannot be expected that they will have a benign course not warranting treatment [15].

As with the patient described here most patients experience some occipital pain and headache for a few days prior to developing either ischaemia or SAH. It is not possible to estimate the incidence of rebleeding from the few cases reported, but Yamaura has indicated that rebleeding from vertebral artery dissections is as high as 24% within the first month [20].

Age of patients varies from 20 to 60 years with most in the 40th decade of life. Hypertension is not reported in all cases but occurs in many and may be a predisposing factor [19]. The distribution between sexes would appear to be equal.

Diagnosis

Computed tomography often shows the effects of vessel dissection, either subarachnoid haemorrhage or cerebellar infarction [15]. The pattern of SAH may show an increased amount of blood in the posterior fossa cisterns or fourth ventricle, but the definitive investigation is cerebral angiography.

The angiographic appearance of posterior circulation dissections have variously been reported as a string or pearl sign, double lumen, fusiform dilatation or vessel occlusion [2,21]. The most common finding however as with this case is a fusiform dilatation of one of the first three parts of the PICA that may be associated with proximal or distal vessel narrowing.

Dilatation distal to the PICA vertebral junction but proximal to branching of the vessel should lead to diagnostic suspicion. MRI has been reported to be useful in the diagnosis of vertebral artery dissection often showing wall haematoma, loss of flow void and even a double lumen however it is unlikely to be of use in smaller vessels [14]. It may be of value though in patient's who have ischaemic symptoms in defining the area of infarction [6].

Treatment

The cerebral angiogram in this patient showed bilaterally large PICA vessels with PICA dominance over AICA in supply of the cerebellum and presumably the lateral brainstem too. Because the dissection was in a segment of the vessel where lateral medullary perforators originate, proximal vessel occlusion or aneurysm trapping alone were not considered to be good therapeutic options.

This is in contrast to treatment of vertebral artery dissection where endovascular or surgical proximal occlusion close to the area of dissection has had good results [9]. In these patients reversal of flow through the artery has resulted in angiographic resolution of the dissection and maintenance of perforator and PICA blood supply from the opposite vertebral artery.

Endovascular and surgical occlusion of the involved PICA segment alone have also been described as successful treatments. However this strategy relies on AICA – PICA anastamosis to re-establish distal PICA flow and must increase the risk of lateral medullary infarction. Conservative therapy as advocated by some authors was also thought to be unsuitable because of the risk of re-bleeding in the acute phase [13].

This left two possible options. Firstly aneurysm reinforcement by encirciling clip as used by Jafar, although this approach depends on the origin of perforating vessels which may be caught in the clip and could inadvertently occlude the vessel [1]. It also does not offer any resistance to the distal elongation of the dissection past the clip.

The second option was proximal vessel oc-

clusion and reversal of flow through the left PICA by performing a PICA to PICA anastamosis. Occipital artery to PICA anastomosis has been described twice previously and would also have been possible but the bilaterally large PICA vessels in this patient favoured the anastamosis performed [5,11].

Anticoagulation with heparin is a mainstay of therapy in the treatment of extracranial arterial dissection in order to limit thrombo-embolism. It has not been advocated for use in intracranial dissection because of the risk of SAH in both patient's who have presented with haemorrhage and ischaemia.

Conclusion

A patient presenting with subarachnoid haemorrhage from a left PICA dissection was treated with proximal PICA clipping and PICA to PICA anastamosis in order to spare brain stem perforating vessels. Follow-up angiography shows resolution of the dissection and patency of the anastamosis.

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