Mohamad Nazri Munawar Khan[1], Muhamad Zabidi Ahmad[2]

[1]School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia.

[2]Oncological and Radiological Sciences Cluster, Advanced Medical and Dental Institute, Universiti Sains Malaysia, Bertam, 13200 Kepala Batas, Pulau Pinang, Malaysia.

Vertebrobasilar Artery Dissection – A Rare Cause of Stroke

***Abstract*— Basilar artery dissection with or without vertebral artery dissection is a rare lesion that can cause intracranial haemorrhage, infarct or mass lesion. We present a case of a middle-aged man with chronic occipital headache and acute hemiparesis. Non-contrasted CT brain shows ectatic right vertebral artery compressing the medulla oblongata and MRI shows acute pontine infarct caused by a basilar artery dissection.**

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*Corresponding author
Muhamad Zabidi Ahmad
E-mail: zabidiahmad@usm.my

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1 INTRODUCTION

Vertebrobasilar artery dissection (VBAD) is a rare disease entity with limited knowledge of its prognosis and effective treatment. Clinically manifesting as subarachnoid haemorrhage, stroke, or mass effect to the brainstem, the disease is more prevalent in the younger age group (20 to 45 years old). The overall incidence of basilar artery dissection is estimated at 0.25 per 100 000 person-year [1]. We report a case of posterior circulation stroke caused by vertebrobasilar artery dissection.

2 CASE REPORT

A 40-year-old gentleman with underlying diabetes mellitus and ex-smoker presented to the emergency department with sudden onset of left-sided body weakness associated with numbness and difficulty swallowing, associated with involuntary movement suspicious of seizure activity. No symptoms of raised intracranial pressure were noted at this point. On further questioning, he had visited Emergency department one month earlier due to worsening occipital headache, disturbing him for the past four months but was discharged home and was given an appointment for an outpatient CT scan of the brain showed no evidence of infarction. No symptoms of raised intracranial pressure were noted at the current admission. He denied hypertension, history of trauma to the neck or antecedent neck manipulation.

Physical examination revealed mild elevation of his blood pressure (165/99 mmHg) and pulse rate (115 beats per minutes) but he was otherwise pain-free. There was mild facial asymmetry noted and reduced muscle power demonstrated involving the left upper and lower limbs. Other cranial nerves were intact and the cerebellar signs including Babinski were negative.

A review of the non-contrasted CT brain during the first visit showed the ectatic right vertebral artery compressing the medulla oblongata. His capillary blood sugar, full blood count, renal profile, liver profile, coagulation profile and electrocardiogram were normal. No signs of left ventricular hypertrophy were seen. A repeat non contrasted CT scan brain during the second visit showed no additional finding, with a low NIHSS score indicating minor stroke. He was subsequently admitted and was started on double antiplatelet and lipid-lowering drugs. He was discharged after some improvement of the neurological deficits. Connective tissue disease screening was negative.

Magnetic resonance imaging (MRI) of the brain, five days later showed an area of T1 hypointense and T2 hyperintense lesion in the right side of pons with low signal intensity in ADC against the high signal in DWI consistent with an acute ischemic infarct. Time of flight magnetic

resonance angiogram showed a fusiform right vertebral artery aneurysm measuring 2.0 cm (length) x 1.0 cm (width) and a moderate to severe stenosis of the same artery immediately distal to the aneurysm. There was also basilar artery dissection with acute intramural hematoma causing thrombosis of the false lumen, which measures 2.2 cm in length (Figure 1). Urgent diagnostic cerebral angiogram similarly showed the fusiform aneurysm of the right vertebral artery (Figure 2). The basilar artery was diffusely smaller in calibre. The right vertebral artery aneurysm was successfully stented one month later.

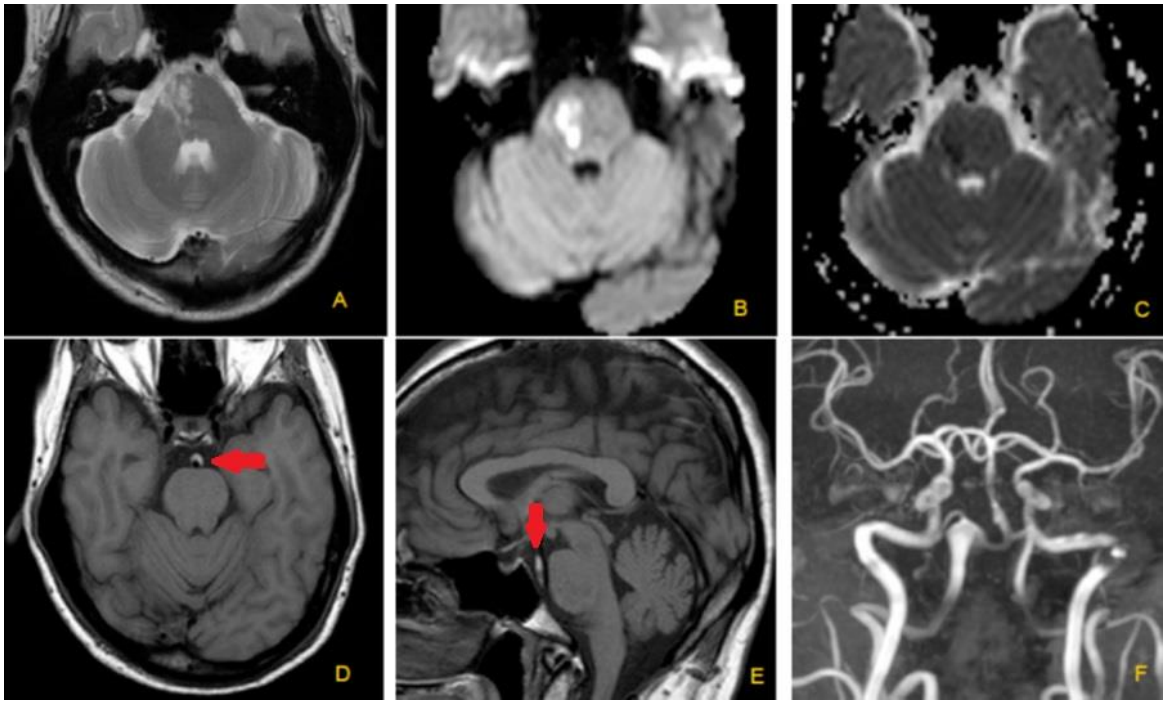

Figure 1: Hyperintense left pontine lesion with restricted diffusion suggestive of recent infarct (A – C). Axial and sagittal T1 weighted image showing crescentic basilar artery hematoma (Red arrow, D - E). Aneurysmal dilatation of the right vertebral artery seen with narrowing of the bilateral distal vertebral and basilar arteries on time-of-flight MRA (F).

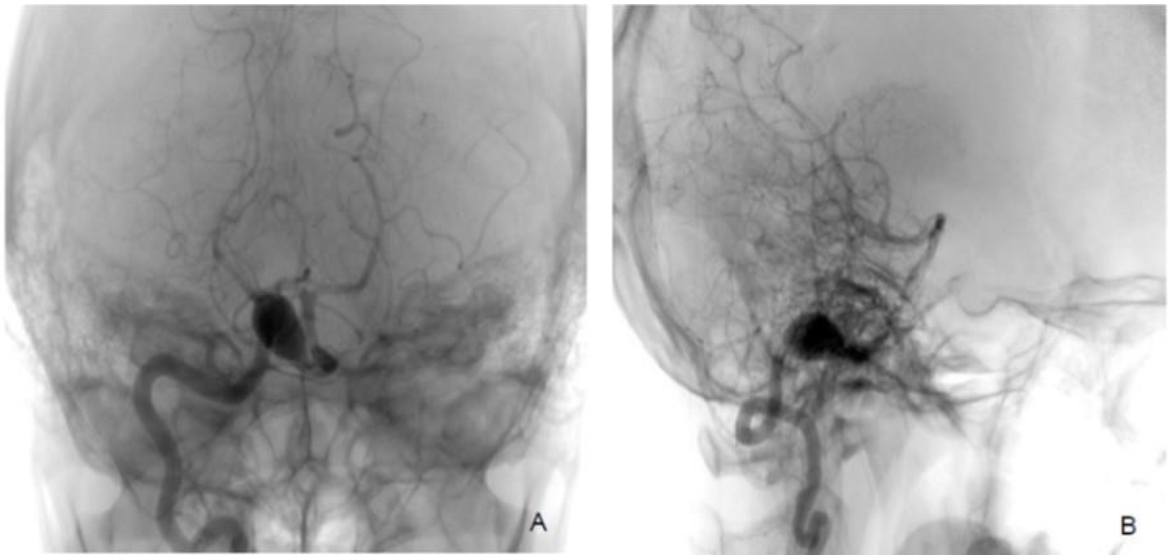

Figure 2: Coronal and sagittal view of right vertebral artery angiogram showing aneurysm of the right vertebral artery

3 DISCUSSION

Spontaneous vertebral artery dissection is increasingly recognized as a cause of posterior circulation stroke in young and middle age groups; however, spontaneous isolated basilar artery dissection is rare [2] and was reported to have a less favourable outcome. Intracranial VBAD occurs more commonly in the Asian population. Intracranial VBAD constitutes only 10% of all spontaneous VBAD and is more prevalent in males [3]. It has a higher likelihood of forming an aneurysm and more commonly complicated by bleeding compared to extracranial VBAD.

VBAD can be spontaneous or traumatic. The spontaneous cause is commonly idiopathic but underlying pathologies such as hypertension, atherosclerosis, fibromuscular dysplasia, Ehlers-Danlos syndrome type IV, Marfan syndrome, osteogenesis imperfecta type I, alfa-1-antitrypsin deficiency, cystic medial necrosis, autosomal dominant polycystic renal disease, medial mucoid degeneration, polyarteritis nodosa, Behçet's disease and migraine [3]. Traumatic VBAD is from blunt trauma and antecedent neck manipulation.

Dissection or tear of the vessel wall can be in the subintimal or subadventitial layers. In subintimal dissection, a tear occurs between the intimal and medial layers of the arterial wall, and penetrating blood between these layers leads to stenosis. In subadventitial dissection, a tear occurs between the medial and adventitial layers of the arterial wall and may cause aneurysmal dilation in the artery wall. The radiological findings of VBAD are summarized in Table I [3].

Table I: Radiological findings in craniocervical dissection.

CT and CTA	MR and MRA	DSA
Enlarged external diameter of artery Hyperdense thrombus Narrowed eccentric lumen Mural thickening Short segment stenosis Total occlusion Dissecting aneurysm Filling defect Intimal flap Focal stenosis and dilatation (string and pearl) Tapering stenosis (flame sign)	Crescent-shaped intramural hematoma Luminal stenosis Eccentric lumen Increase external diameter of artery	Pearl sign (stenosis) Pearl and string sign (stenosis and dilatation) Tapered occlusion Double lumen (intimal flap) Saccular or fusiform aneurysm

In this patient, the vertebral artery aneurysm and stenosis are likely the dissection results, which had occurred a few months ago when the patient first experienced the symptoms and the basilar artery dissection is the new/acute event. In VBAD without significant luminal stenosis, head and neck pain (occipital), pulsatile tinnitus and cranial nerve palsies may be present. In 90% of cases, the pain resolves within one week [3]. In VBAD with significant luminal stenosis, more than two-thirds of patients will commonly experience ischaemic stroke involving the brainstem and cerebellum, as in our patient. He has the classical imaging finding of acute dissection on MRI with acute mural thrombus within the false lumen causing luminal stenosis. Interestingly, this patient has underlying Fibromuscular Dysplasia, as he is not known to be hypertensive and has negative connective tissue screening. No renal angiogram was performed to exclude this possibility. The first and second CT scans were negative because they were non-contrasted, and the CT scan's sensitivity in detecting infarct in the posterior fossa is relatively low [4]. This is the inherent limitation of CT scan due to beam hardening artefact and relative time delay for infarct to appear in the white matter. MRI on the other hand readily shows acute infarct. The MRA has elegantly shown multiple arterial abnormalities: mild to moderate stenosis involving petrous part of the left internal carotid artery with irregular beaded appearance and short segment stenosis at the left intracranial segment of left vertebral artery (figure not shown). Digital subtraction angiogram (DSA) remains the gold standard for diagnosing arterial stenosis/aneurysm apart from its therapeutic ability [5]. However, as for dissection, the sensitivity of DSA varies depending on the patency of the false lumen. In addition, MRI and MRA (TOF and CE) are superior then DSA in arterial dissection. The sensitivity of DSA is slightly reduced when the false lumen is occluded.

4 CONCLUSIONS

VBAD is rare, albeit an important cause of stroke in a young patient. The timely use of MRI in this group of patients presented with stroke allows early detection and treatment of vertebrobasilar artery dissection.

ACKNOWLEDGEMENT

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CONSENT FOR PUBLICATION

Written consent was granted from the patient for the publication of this case report and the accompanying images. Institutional Review Board approval is not required at the authors' institutions for the presentation of a single case report.

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