# Vertebrobasilar Dolichoectasia

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## ABSTRACT

Vertebrobasilar dolichoectasia (VBD) is an uncommon vasculopathy which causes enlargement, tortuosity, or elongation of vertebral and basilar arteries. It is also called dilative arteriopathy. It is a rare anomaly with an incidence of 0.06%–5.8%. Posterior circulation arteries are more commonly affected than anterior circulation arteries. More than 40% of patients with VBD are asymptomatic. It may also present as ischemic stroke, hemorrhagic stroke, compression of cranial nerves and brainstem. VBD can be due to either atherosclerosis or congenital abnormalities such as Marfan's syndrome, Ehlers–Danlos syndrome, and Fabry's disease. Here, we describe a case of a 60-year–old male who presented with an episode of generalized seizure. His metabolic workup was negative for seizure. As a part of evaluation, he underwent computed tomography (CT) scan of brain and magnetic resonance imaging of the brain with magnetic resonance angiogram (MRA). His CT/MRA revealed VBD. He was treated with phenytoin and made a good recovery. VBD presenting as stroke is well known in literature, but here we report a rare case of VBD presenting as seizure.

**Key words:** Seizure; vasculopathy; vertebrobasilar dolichoectasia

## Introduction

Vertebrobasilar dolichoectasia (VBD) is an uncommon vascular anomaly with unclear etiology characterized by dilated, elongated, and tortuous arteries. The elongated and tortuous arteries cause hemodynamic and hemostatic changes which lead to thrombosis, microembolization, brainstem, or cranial nerve compression. VBD can also be asymptomatic. VBD is a condition which may be easily missed by radiologists and neurologists owing to the normal variation of vertebrobasilar arteries in healthy individuals.[1] VBD is believed to be an independent risk factor for posterior circulation stroke irrespective of other vascular risk factors. A variety of clinical syndromes are associated with VBD, but VBD presenting as seizure has rarely been reported in literature.

## Case Report

We report a case of a 60-year-old male who came with an episode of generalized tonic-clonic seizure. The patient had tonic-clonic jerking of limbs associated with tongue bite and urinary incontinence. The jerking movements lasted for few minutes and it was followed by postictal confusion. He never had seizures before. He denied consumption of toxins or poisons. There was no history of trauma to head or fever prior to the onset of seizure. His past medical history included myocardial infarction 4 years back, hypertension for 11 years, dyslipidemia for 11 years, and diabetes mellitus for 15 years. He is on regular treatment with insulin, metformin, aspirin, statin, lisinopril, metoprolol, and nitrates.

On examination in emergency department, he was confused and disoriented. His pupil was 3 mm and normally reacting to light. There was no fever or neck stiffness. He moved all the four limbs to painful stimulus. He had no focal neurological deficits. Random blood sugar was 216 mg/dl. His vitals were within normal limits except for blood pressure which was 160/100 mm of Hg. His electrocardiogram revealed left ventricular

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hypertrophy and evidence of old anterior wall myocardial infarction. He underwent computed tomography (CT) scan of brain which revealed dilated vertebrobasilar arteries. Magnetic resonance imaging of the brain with magnetic resonance angiogram (MRA) was planned to study the vertebrobasilar arteries in detail. The imaging studies revealed dilated tortuous vertebrobasilar arteries suggestive of VBD without evidence of any acute infarct [Figures 1 and 2].

He was initially treated with intravenous phenytoin and later managed with oral phenytoin. He did well clinically and was discharged with aspirin and phenytoin.

## Discussion

VBD is an uncommon vasculopathy which causes enlargement, tortuosity, or elongation of arteries. It is also called dilative arteriopathy, megadolichoectasia, and fusiform aneurysm of the vertebral and basilar arteries. It is a rare condition with an incidence of 0.06%–5.8%. Posterior circulation arteries are more commonly affected than anterior circulation arteries. Compared to anterior circulation, posterior circulation has less sympathetic innervation and is prone to high pressure of blood flow making them ectactic in due course of time. It is a radiological diagnosis with basilar artery (BA) or vertebral artery (VA) of diameter >4.5 mm or deviation of any portion >10 mm from the shortest expected course, BA length >29.5 mm or intracranial VA length >23.5 mm.[2] VBD can be due to either atherosclerosis or congenital abnormalities. Marfan's syndrome, Ehlers–Danlos syndrome, Fabry's disease, and sickle cell disease are some of the examples of congenital anomalies associated with VBD. In general, atherosclerosis leads to VBD in patients over 40 years of age, and congenital anomalies are seen in young patients.[3] Atherosclerotic degeneration of the vascular wall, with or without arterial hypertension, is the initial pathogenetic factor in the development of this condition. VBD must be differentiated from fusiform aneurysms which can mimic them. More than 40% of patients with VBD are asymptomatic. Other clinical features include ischemic stroke, hemorrhagic stroke, compression of cranial nerves leading to hemifacial spasm, trigeminal neuralgia, diplopia, dysarthria, dysphagia, tinnitus, and vertigo. It can also cause compression of brainstem leading to ataxia, vestibular deficits, and quadriparesis. VBD causes ischemic stroke due to abnormal blood flow which results in thrombus formation. Atherosclerotic plaques can form thrombi and block the lumen of branch arteries or embolize distally causing stroke. VBD is an independent risk factor for stroke, especially in posterior circulation territory. It increases the mortality and decreases the survival, independent of other vascular risk factors.[4] Defects in internal elastic lamina lead to fragile dolichoectatic arteries and can cause intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH). In addition, use of antiplatelets and anticoagulants can increase the risk of hemorrhage in VBD. In a prospective study of 156 patients with VBD followed up for 11.7 years, 60% of them had a stroke. Fifty-nine patients had ischemic stroke, 21 had hemorrhagic stroke, 31 patients developed compressive symptoms, and 2 patients had hydrocephalus.

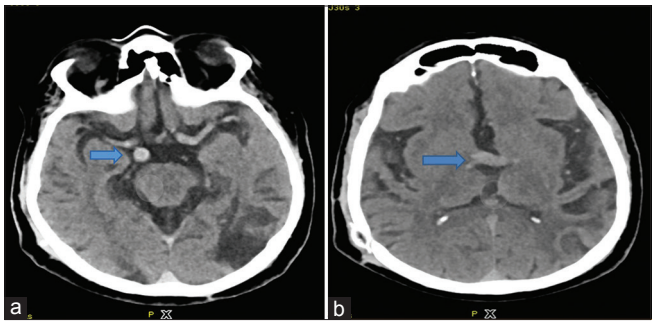


**Figure 1:** Computed tomography of the brain (arrows in a and b) shows dilated and tortuous vertebrobasilar arteries

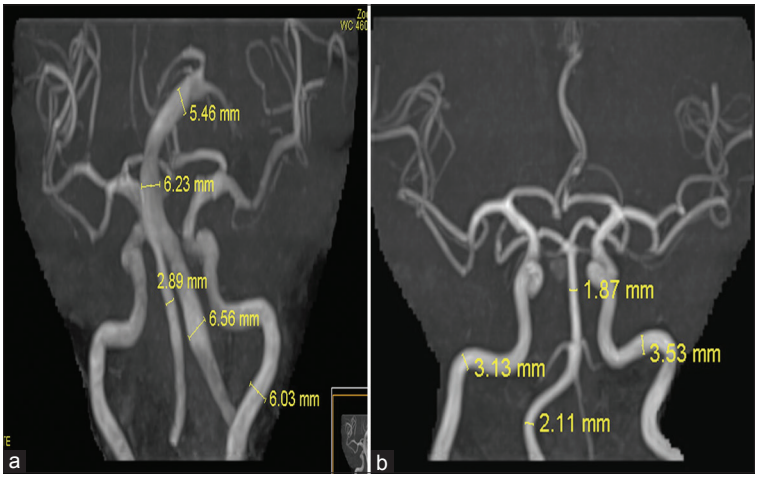


**Figure 2:** Magnetic resonance angiograms show comparison of vertebrobasilar dolichoectasia (a) with normal cerebral blood vessels (b)

The long-term prognosis depends on the severity of VBD at diagnosis and progression of VBD.[3] Seizure as a presenting feature is rarely reported in literature. Seizures are reported in about 6.1% of VBD.[5] However, seizures are usually secondary to ICH or SAH. Our patient could have had seizure secondary to the compressive effects of VBD.

VBD can be diagnosed by CT angiogram, MRA, or digital subtraction angiography. MRA is cost-effective and widely available to diagnose VBD.[2] Usually, it requires no treatment as 40% of patients are asymptomatic. However, surgical options such as resection and anastomosis are available for certain selected patients.[6]

## Conclusion

VBD is an uncommon vasculopathy which causes enlargement, tortuosity, or elongation of arteries. Posterior circulation arteries are more commonly affected than anterior circulation arteries. Although seizures are reported in about 6.1% of VBD patients, they are usually secondary to ICH. Our patient presented with seizure which may be secondary to the compressive effects of VBD.

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### Conflicts of interest
There are no conflicts of interest.

## References

1. de Oliveira Rde M, Cardeal JO, Lima JG. Basilar ectasia and stroke: Clinical aspects of 21 cases. Arq Neuropsiquiatr 1997;55:558-62.
2. Ubogu EE, Zaidat OO. Vertebrobasilar dolichoectasia diagnosed by magnetic resonance angiography and risk of stroke and death: A cohort study. J Neurol Neurosurg Psychiatry 2004;75:22-6.
3. Passero SG, Rossi S. Natural history of vertebrobasilar dolichoectasia. Neurology 2008;70:66-72.
4. Passero S, Filosomi G. Posterior circulation infarcts in patients with vertebrobasilar dolichoectasia. Stroke 1998;29:653-9.
5. Yu YL, Moseley IF, Pullicino P, McDonald WI. The clinical picture of ectasia of the intracerebral arteries. J Neurol Neurosurg Psychiatry 1982;45:29-36.
6. Ince B, Petty GW, Brown RD Jr., Chu CP, Sicks JD, Whisnant JP. Dolichoectasia of the intracranial arteries in patients with first ischemic stroke: A population-based study. Neurology 1998;50:1694-8.