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Brief Report

Vascular Compression of Medulla Oblongata by Non-Dolichoectatic Vertebral Artery

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Abstract

Neurovascular conflicts are common in the posterior fossa and basal cisterns due to coexistence of important neural and vascular structures. Neurovascular conflict arising from compression of the cranial nerves by pulsatile flow in the adjacent atherosclerotic arteries is well known and is associated with conditions like trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia. The medulla is known to be affected by dilated tortuous (dolichoectatic) vertebrobasilar arteries in the elderly or hypertensive. The vertebral artery causing the compression can be dolichoectatic or normal dominant vertebral artery or an elongated tortuous artery. Very few cases of medullary compression by non-dolichoectatic elongated tortuous or dominant vertebral artery were reported in the literature. In this article, we report three cases of medullary compression by the dominant and angulated vertebral artery. Magnetic resonance (MR) imaging with MR angiography showed indentation of the anterolateral aspect of the inferior medulla by the vertebral artery. The patients are managed conservatively and on regular follow-up.

Keywords
- vertebral artery
- medulla oblongata
- magnetic resonance angiography

Case 1

A 63-year-old male patient presented with recent-onset difficulty in walking for 6 months. He complained of giddiness and occipital headache for the past 6 months. On examination, he had dysmetria on both sides and gaze-evoked nystagmus on lateral gaze. Based on suspicion of posterior circulation stroke, magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) were done that showed dominant and angulated left vertebral artery abutting the cervicomedullary junction, crossing the midline, compressing the anterolateral aspect of the inferior medulla, and displacing the medulla to the right (►**Fig. 1**). As there were no ischemic infarcts, the giddiness and dysmetria were thought to be due to vascular indentation of the medulla. The patient was managed conservatively and improved symptomatically except for persistence of mild headache and he is on regular follow-up.

Case 2

A 59-year-old patient presented with giddiness and occasional episodes of positional vertigo for the past 15 days. He had similar complaints 3 years ago. Clinical examination showed normal motor power, reflexes, and sensation in all the four limbs. MRI showed dominant and bent left vertebral artery indenting and compressing the left lower medulla (►**Fig. 2**). No infarcts were seen. MRI spine showed mild cervical spondylotic features. As the mild cervical spine changes could not explain the patient's recurring giddiness and vertigo, symptoms were considered due to the vascular indentation. Patient was

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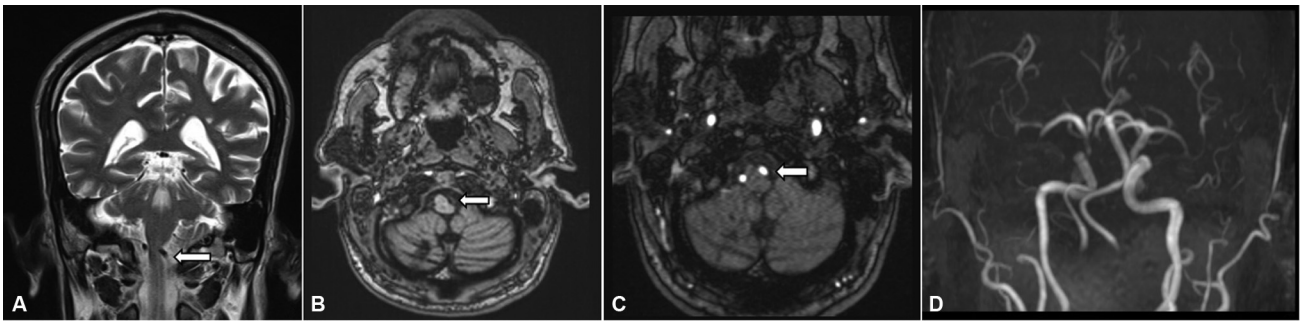

Fig. 1 T2-weighted coronal section image (**A**) showing V4 segment of the left vertebral artery flow void coursing medially from left impinging on the lower medulla. T1-weighted axial section image (**B**) shows the indentation on the left side ventrolateral aspect of medulla. Magnetic resonance angiography (MRA) axial section (**C**) shows the compression of the medulla by the left vertebral artery. MRA maximum intensity projection (MIP) coronal image (**D**) shows the medially bent and angulated left vertebral artery.

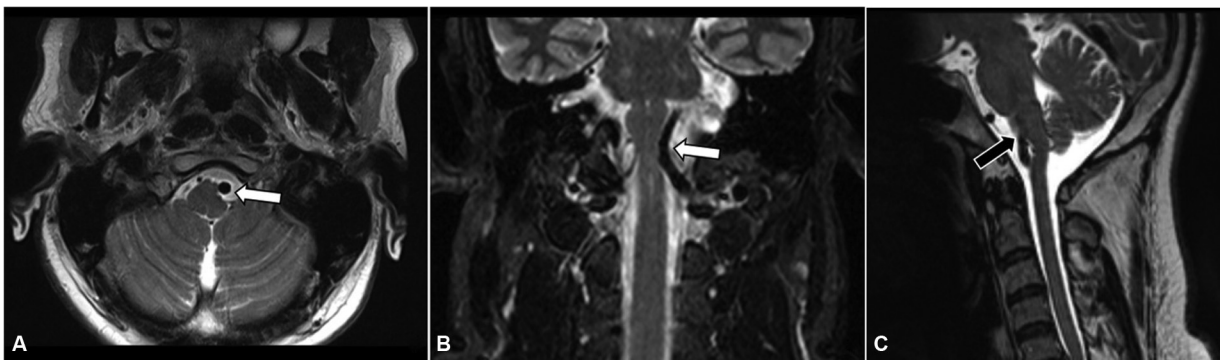

Fig. 2 T2-weighted axial (**A**), coronal (**B**), and sagittal (**C**) image sections showing dominant and angulated V4 segment of the left vertebral artery flow void indenting the anterolateral aspect of the medulla on the left side

started on anti-inflammatory and analgesic medication and showed partial resolution of symptoms over the next 4 to 6 weeks

Case 3

A 67-year-old female patient presented with progressive difficulty in speech for 6 months, complaints of dysphagia and nasal regurgitation for 2 months. Neurological examination showed uvula deviated to the right side and decreased mobility of right palatal arch, but with normal power and sensations noted in all the four limbs. MRI and MRA showed acute medial angulation of the V4 segment of the dominant left vertebral artery indenting the lateral surface of the lower medulla (►**Fig. 3**). Due to strong suspicion of posterior circulation stroke, MRI was repeated

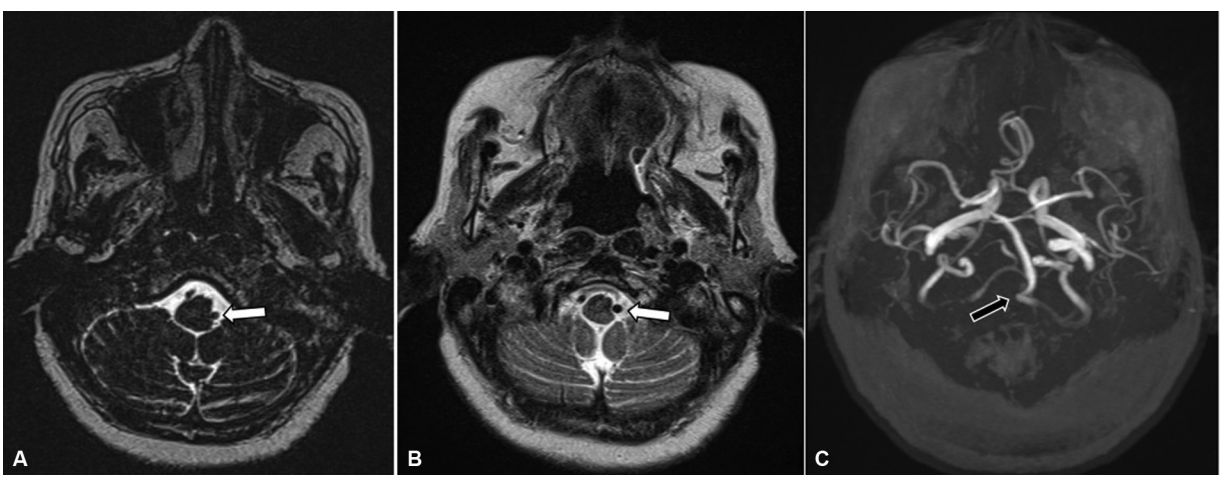

Fig. 3 Heavily T2-weighted axial section image (**A**) and T2-weighted axial section image (**B**) shows vertebral artery flow void indenting the left anterolateral aspect of the medulla. Magnetic resonance angiography (MRA) maximum intensity projection (MIP) axial image (**C**), showing dominant acutely angulated left vertebral artery.

after 1 week and showed the same findings. Her symptoms were ascribed to medullary indentation of the angulated vertebral artery. She was started on low-dose aspirin and being followed-up.

Discussion

Vascular compression of the medulla oblongata is uncommon and can occur by an elongated tortuous, dominant, or dolichoectatic vertebral artery. The prevalence of vertebral artery dolichoectasia is 0.2 to 4.4% and incidence of asymptomatic dolichoectasia about 1.3%.[1,2] Vertebral artery dominance is defined if there is difference in diameter of more than 0.3 mm between the two vertebral arteries.[3] The vascular compression of the medulla by non-dolichoectatic vertebral artery has been less reported with about 19 reported cases in 2006 to 58 cases in 2021.[4,5]

Savitz et al[4] described nine patients with medullary compression on the lateral side, involving the ventral pyramids in all cases and dorsal tegmentum in one case. The vertebral artery causing indentation is "dominant and angulated or bent, and not necessarily elongated and tortuous" as in dolichoectasia.

Li et al[6] have proposed the term "vertebral artery compression syndrome" to describe the compression of medulla or the upper spinal cord by vertebral artery in 10 cases (left side in 8 out of 10 cases). Transient or recurrent symptoms may be due to pulsatile impact or perforator branch transient ischemia. They also mentioned that ipsilateral hemiparesis may occur from compression on the lower medulla and contralateral hemiparesis, if compression at above the level of pyramidal tract decussation. As the outer nerve fibers innervate the lower extremities, motor deficits are noticed earlier in the lower extremities.

Mahrous and Busaad[7] described two cases with vertebral artery compression presenting with isolated symptom of paroxysmal dizziness and had positional nystagmus on examination. Paroxysmal symptoms provoked by physical effort or flexion can be explained by the impact of pulsatile compression. Gorton et al[8] described intractable nausea and vomiting, attributed to the distortion of the dorsolateral areas including area postrema from ventrolateral medullary impingement. As the compression is gradual, allowing for adaptation, the risk of respiratory and autonomic center damage in medulla is less. Nakahara et al[9] reported three cases with central sleep apnea from vertebral artery compression of the brainstem respiratory centers which improved after microvascular decompression (MVD). Meyer et al[10] described a case presenting with palatal myoclonus from compression of the inferior olive and Roh et al[11] reported features of lateral medullary syndrome.

A literature review by Sabet[5] revealed a total of 58 cases with medullary compression by non-dolichoectatic vertebral artery till date with compression on the left in 57%, right in 28%, and bilateral in 10%. Laterality of the vertebral artery compression is attributed to the increased velocity from the position of the heart on the left side.[12]

The vertebrobasilar system is the only place in the body where two arteries unite to form a single trunk. The vertebral artery, due to developmental and hemodynamic factors, may have changes of elongation and tortuosity, predisposing to neural compression (►**Fig. 4**). The vertebrobasilar "dolichoectasia" is defined on the basis of three criteria—diameter of the vessel (mid-basilar), laterality (in relation to clivus margins), and distal end of bifurcation (in relation of suprasellar cistern and floor of the third ventricle).[13] There can be changes in vertebral artery without basilar artery changes or fulfilling the criteria of dolichoectasia. It is not known whether the elongation and tortuosity to a stage of dolichoectasia and then further onto fusiform aneurysmal change is a continuum of progressive changes.[14]

Management is usually conservative with antiplatelets, anticoagulants or analgesics, or surgical if intractable symptoms. MVD is done by using a separating material placed between the vertebral artery and compressed brainstem, but may not be as satisfactory as vertebral artery repositioning method with anchoring/sling to the adjacent dura.[15]

Medullar compression by non-dolichoectatic vertebral artery is a diagnosis of exclusion when no incriminating lesion is found in brainstem or cranial nerve features. However, the possibility of vertebrobasilar transient ischemic attacks (TIAs) as a cause of these symptoms cannot be totally ruled out. But there were no vertebral artery narrowing or irregularity of atherosclerotic changes in the

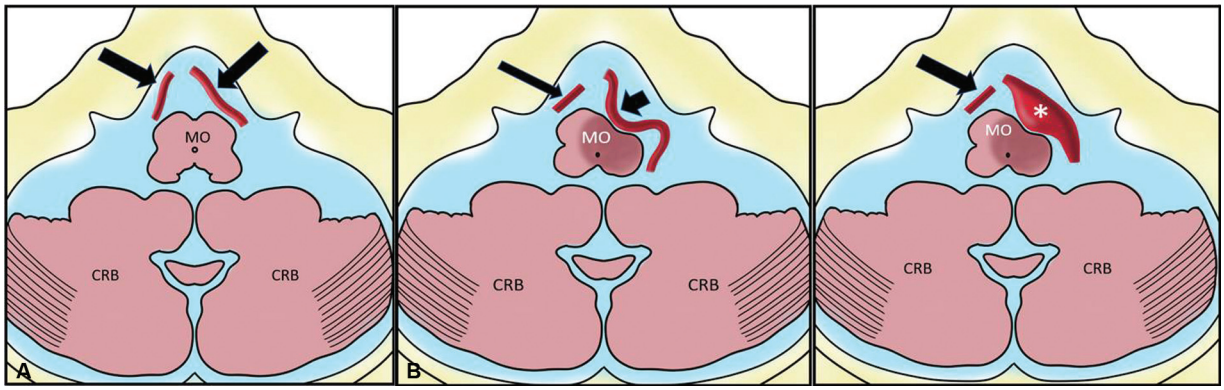

Fig. 4 Illustration showing the normal vertebral arteries in premedullary cistern (**A**), indentation by elongated or angulated vertebral artery (**B**) and vertebrobasilar dolichoectasia with medullary compression.

proximal vertebral arteries to suspect TIA. The treatment is not standardized as the use of antiplatelet/anticoagulants is considered a potential risk factor for hemorrhage in enlarging dolichoectasia. Follow-up vascular imaging with computed tomography or MRA at 6 months to 1 year is useful and necessary to ensure stability against progressive dilatation or fusiform changes.

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Conflict of Interest
None declared.

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