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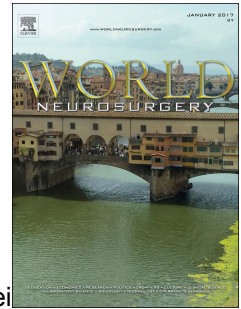
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**Successful treatment by microvascular decompression surgery of a patient with hemiparesis due to vertebral artery compression of the medulla oblongata: A case report and literature review**

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**Running title:** hemiparesis due to vertebral artery compression

**Abstract**

Little literature is available on hemiparesis caused by vascular medullary compression, which may occur as a result of dolichoectasia of the vertebrobasilar arterial system. In this paper, we report a case of vertebral artery compression of the medulla oblongata in a 67-year old woman. The patient was hypertensive and developed hemiparesis and intermittent spasms for five years, which became worsened for one year. Cranial nerve MRI showed compression of the medulla oblongata by the left vertebral artery. Motor evoked potential (MEP) examination showed abnormal conduction of MEPs of bilateral toe abductors. The patient underwent microvascular decompression surgery under general anesthesia via the retrosigmoid keyhole approach, which led to relief of vascular compression and symptomatic improvement. Our case suggests that detailed history, imaging studies and electrophysiological studies help lead to a correct and early diagnosis of hemiparesis due to vascular compression of the rostral ventrolateral medulla. MVD surgery improves patient symptoms and intraoperative electrophysiological monitoring helps avoid injury to important adjacent nerves.

**Key words:** hemiplegia; microvascular decompression; medulla oblongata; vascular compression; primary hypertension; literature review

## Introduction

Cranial neuralgias are characterized by pain in the distribution of a cranial nerve. Pain arising from cranial neuralgias can be severe and often poses a significant health burden to the patient. Vascular compression against a cranial nerve has been widely accepted as a main cause of cranial neuralgias [1] and may be an underlying cause of trigeminal neuralgia, hemifacial spasm, gloss pharyngeal neuralgia, or other cranial neuralgias[2]. Vascular compression of the medulla oblongata may occur as a result of dolichoectasia of the vertebrobasilar arterial system or rarely due to the tortuous course of an intracranial vessel, which may cause primary hypoglossal nerve palsy[3], hypertension[4], dysphagia, dysarthria, gait ataxia, quadriplegia and other symptoms[5-9]. Other documented contributory vascular anomalies include a fusiform aneurysm[10] and a persisting trigeminal artery[11].

Little literature is available on hemiparesis caused by vascular medullary compression. In 1985, Kim *et al.* described the first case of progressive left hemiparesis due to vascular compression of the medulla oblongata, which was relieved by microvascular decompression (MVD)[12]. Since then, only anecdotal cases have been reported. In this paper, we report a case of vertebral artery compression of the medulla oblongata which caused hemiparesis and possibly primary hypertension. The patient was successfully managed by MVD, which helped relieve the symptoms. A literature review was also undertaken of 15 patients with vascular medullary compression that had resulted in hemiparesis or quadriparesis.

## Case Report

A 67-year old female patient was admitted to our hospital in March 3, 2016 because of worsening of right limb weakness and intermittent spasms for one year. In January, 2011, the patient began experiencing weakness of the right upper and lower extremities and intermittent spasms for no obvious reason. The onset of weakness of the upper extremity preceded that of the lower limb and limb weakness became gradually worsened over time. The patient did not seek treatment because of its insidious course. In February, 2015, limb weakness became noticeably exacerbated without any apparent causes and interfered with her daily activities. The patient had a 15-year history of hypertension, with the highest blood pressure at 180/110 mmHg. She took oral nifedipine sustained release tablets (30 mg, 2 times/day), and irbesartan (0.15 g, once daily) and her blood pressure declined to 140/100 mmHg. A head MRI scan at a local hospital suggested white matter atrophy, and cervical MRI and head CT scan showed ossification of posterior longitudinal ligament, cervical spinal stenosis, and cervical spondylotic myelopathy.

The patient sought treatment at our hospital on March 3, 2016. The patient was alert. Physical examination revealed no deformity of the spine. Mild tenderness was noticed at the C<sub>4</sub>-C<sub>7</sub> spinous process. There was no irradiating pain in both the upper and lower extremities. The muscle power of the right upper and lower extremities was grade 4 and that of the left upper and lower extremities was normal. Both lower limbs had no deformity and no muscle atrophy. The right limb muscle tone was slightly increased while the left limb muscle tone was normal. The biceps and triceps reflex

were hyperactive, and right Hoffmann's sign was positive (+). The knee tendon reflex and Achilles tendon reflex were hyperactive, and right Babinski sign was positive (+).

On admission, hypertension (160/100mmHg) was noted. Cranial nerve MRI showed compression of the medulla oblongata by the left vertebral artery (Figure 1). Brainstem auditory evoked potential (BAEP) examination revealed bilateral normal conduction from the auditory nerve to the brainstem. Somatosensory evoked potential (SEP) examination showed normal conduction of deep sensation from bilateral upper extremities to the cortex, but abnormal conduction from the lumbar spinal cord to the cortex [criteria for abnormal SEPs: N13–N20, >6.5 ms; P14–N20, >5.5 ms; interside latency (either N13–N20 or sCCT), >1.5 ms] while peripheral conduction was normal. Motor evoked potential (MEP) examination showed abnormal conduction [criteria for abnormal MEPs: mCCT of >6.5 ms and mCCT interside latency of >1 ms (abductor pollicis brevis); mCCT of >15 ms and mCCT interside latency of >2.3 ms (tibialis anterior)] of MEPs of bilateral toe abductors, especially the right toe abductor. The patient received an admission diagnosis of vascular medullary compression syndrome (right hemiparesis).

The patient underwent MVD under general anesthesia via the retro sigmoid keyhole approach. At the same time, the use of electrophysiological monitoring, assures the safety of the operation. The cranial content was explored from the lateral cerebellum to the left vertebral artery under the microscope. The left vertebral artery was found to be ectatic (the diameter of the vertebral artery was greater than 4.5 mm) and tortuous,

forming an angle and compressing the medulla oblongata with an obvious indentation. The intraoperative findings confirmed preoperative cranial nerve MRI findings (Figure 2). The left vertebral artery was determined to be the culprit vessel, and was separated from the medulla oblongata by inserting soft Teflon felt for full decompression. The operation was uneventful, with no active bleeding. Intermittent spasms of the right extremities disappeared postoperatively, and limb weakness was gradually eased. One month after the discharge from the hospital, cranial nerve MRI showed no compression of the medulla (Figure3). At the 6-month follow up visit, arterial pressure was 130/80 mmHg; irbesartan was discontinued and oral nifedipine was reduced to 30 mg, once daily. At the 12-month followed up visit, right limb weakness was noticeably relieved, and the muscle power was grade 5- and no spasms recurred. Blood pressure was well controlled within the normal range. (The video episode for the surgery is available here).

## **Discussion**

In the current report, we have described a case of hemiparesis due to compression of the medulla oblongata by an ectatic and tortuous vertebral artery. The patient was also hypertensive, highlighting a role of the rostral ventrolateral medulla in mediating central pressor response. Hemiparesis and hypertension of the patient were markedly attenuated with successful MVD surgery.

Vascular compression of the brainstem, even with severe distortion, is rarely associated with overt clinical manifestations though subtle subclinical dysfunctions

may be frequently present. So far, fewer than twenty cases of vascular medulla compression syndrome have been reported that had overt hemiparesis or quadriparesis (Table 1). Vascular medullary compression is more common in the vicinity of the pyramidal decussation and is often unilateral. Among these anecdotal cases, ten cases including the current case (10/19, 52.6%) had an anomalous left vertebral artery including an ectatic and tortuous left vertebral artery or an elongated and curved left vertebral artery. In four cases (4/19, 21.1%), both left and right vertebral arteries were involved. The right vertebral artery was less frequently involved (5/19, 26.3%). Interestingly, thrombosis was demonstrated in the right vertebral artery in both cases. The culprit vessel(s) compresses against the corticospinal tract superior to the medulla oblongata, impairing neural conduction at the compression site, and at the same time compromising hemodynamics, leading to regional medulla ischemia and contralateral hemiparesis. In the previous literature, unilateral paralysis, contralateral paralysis and even bilateral paralysis may have a certain relationship between the occurrence of paralysis and the site of vascular compression of the medulla oblongata. Pyramidal decussation may be an important site of vascular compression, which is related to paralysis. It is speculated that vascular compression of the medulla oblongata may have caused local anatomical changes, such as reduction in blood flow or distortion and regurgitation in some perforators caused by pressure on perforating branches of the vertebral artery[13].

Our patient had a history of cervical spondylotic myelopathy, which may also cause hemiparesis. However, when severe compression of the spinal cord occurs, cervical



spondylotic myelopathy can cause injury to the corticospinal tract and anterior horn neurons. Furthermore, sensory abnormalities below the level of lesions are present on top of paralysis or paraplegia. The current case had no sensory abnormalities below the level of lesions. The nerve fibers controlling the upper extremities are located laterally while those controlling the lower extremities are located medially in the corticospinal tract superior to the pyramidal decussation. The culprit vessel compresses the lateral surface of the medulla oblongata, involving the lateral nerve fibers in the corticospinal tract first, thus causing the appearance of symptoms in the upper limb to precede that of the lower limb. This is consistent with the disease pattern in our patient; therefore, the patient's hemiplegic symptoms were not likely caused by cervical spondylotic myelopathy, but rather due to vascular compression of the rostral ventrolateral medulla. Furthermore, there was no parenchymal lesion of the medulla and the course of symptoms and signs in our patient was insidious; these together implicate direct mechanical compression rather than ischemia as a possible cause of the clinical findings.

Detailed preoperative neurological examination including electrophysiological studies, high-resolution MRI scans, and angiographic studies are important for a prompt and correct diagnosis of vascular medullary compression syndrome, as illustrated in our current case. MEP allows examination of conduction from the cortex to the muscles, shedding light on the synchronicity and integrity of the corticospinal tract. We documented abnormal conduction of MEPs of bilateral toe abductors, especially the right toe abductor, lending further support to the diagnosis of vascular medulla

compression syndrome.

There has not been a consensus on the treatment of vascular medullary compression syndrome. It has been reported that microvascular compression can be relieved by MVD surgery, resulting in symptomatic improvements. Our patient showed moderate improvement following MVD surgery. Electrophysiological monitoring was undertaken intraoperatively, which has the advantage of ensuring the safety of adjacent cranial nerves, greatly increasing the feasibility and safety of MVD surgery. Vascular medullary compression syndrome patients are also managed conservatively such as with anti-platelet drugs and anticoagulants or analgesics. Currently, there is lack of clinical evidence on whether MVD leads to a superior outcome compared with conservative therapy. Different from our surgical approach, Choudhri *et al.* [14] and Lin *et al.* [15] used a sling technique to tether the vertebrobasilar artery aneurysm with the help of clips to the cliva dura for macrovascular decompression. The technique achieved anteromedial transposition of the vertebrobasilar artery aneurysm from an inferolateral surgical approach with sling fixation to the clival dura using aneurysm clips, which may be worthwhile for further exploring for use in macrovascular decompression.

Given the role of the rostral ventrolateral medulla in mediating central pressor response, vascular compression of the rostral ventrolateral medulla has been reported to be associated with refractory hypertension [16]. Jannetta *et al.* hypothesized that vascular compression of the left root entry zone of the gloss pharyngeal and vagus nerves by looping arteries may play an important role in the pathogenesis of essential

hypertension[2].The rostral ventrolateral medulla is a major centre of the sympathetic system and is implicated in regulating the cardiovascular system. Vascular ventrolateral medulla compression was also documented in hemifacial spasm[17-19] patients with arterial hypertension than without arterial hypertension. Our patient also had hypertension, which was partially relieved following MVD surgery.

In conclusion, detailed history, imaging studies and electrophysiological studies help lead to a correct and early diagnosis of hemiparesis due to vascular compression of the rostral ventrolateral medulla. MVD surgery relieves vascular compression and improves patient symptoms and intraoperative electrophysiological monitoring helps avoid injury to important adjacent nerves.

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**Table 1 Clinical characteristics of reported cases of quadriplegia or hemiparesis due to vascular compression against the medulla oblongata**

Authors	Patient No.	Age, years /gender	Culprit vessel	Imaging studies	Treatment	Outcome	
<b>Kim et al.</b> [12]	1	53/M	Progressive left hemiparesis, CN XII	the left vertebral artery	Metrizamide computerized tomography reveals that the left vertebral artery compresses and distorts the left lateral surface of the medulla	MVD	Improved
<b>Dembo et al.</b> [20]	2	55/M	vertigo, nystagmus, and gait ataxia followed by left hemiparesis (Opalski syndrome); hypertension (164/104 mmHg)	The left vertebral artery	T2W MRI and diffusion tensor imaging; Magnetic resonance angiography (MRA) and 3-dimensional computed tomography angiography reveal a tortuous and dolichoectatic left vertebral artery	Conservative treatment	Recovered
<b>Kobayashi et al.</b> [21]	3	39/F	Hemiparesis	bilateral vertebral arteries	MRI reveals Chiari malformation and compression of the medulla oblongata by both vertebral arteries	MVD following posterior fossa decompression	Improved
<b>Shiraishi et al.</b> [10]	4	61/M	consciousness disturbances, left hemiplegia, and	fusiform right vertebral artery aneurysm with	CT, MRI and digital subtraction angiogram show partial	Intra-aneurysmal embolization	Improved

			subsequent respiratory arrest	partial thrombosis	thrombosis and bulbar with Guglielmi detachable coils		
<b>Takano et al.</b> [22]	5	53/F	severe occipital-nuchal headache and nausea with vomiting, left facial hyperalgesia, right hemihypesthesia and mild right hemiparesis, hoarseness, hypertension (180/100 mmHg)	Elongated and curved V4 portion of the left vertebral artery with normal diameter	Coronal T2W MRI and metrizamide CT scan	MVD	Improved
<b>Tomasello et al.</b> [23]	6	58/M	Dysphagia, weakness of left arm and both lower limbs	Left ectatic VA	N/A	MVD	Improved
	7	55/F	Dysphagia, weakness of the right limbs	Right ectatic VA	coronal T2-weighted MRI scan demonstrating a right ectatic VA with compression of the medulla oblongata	MVD	Improved
	8	67/M	Weakness of both upper and lower limbs	Right ectatic VA	axial T2-weighted MRI scan demonstrating a right ectatic VA with compression of the medulla oblongata	MVD	Improved
	9	73/M	Weakness of both upper and lower limbs	Left ectatic VA, hypoplasia right VA	N/A	MVD	Improved
<b>Sadashiva et al.</b> [24]	10	36/M	left hemiparesis	Right ectatic VA	T2-weighted images showing compression of anterolateral medulla by ectatic vertebral artery	MVD	Cured



<b>Hongo et al.</b> [13]	11	30/M	Right hemiparesis, gait ataxia	The left vertebral artery	MRI reveals a tortuous vertebral artery compressing the medulla oblongata	MVD	Improved
<b>Hongo et al.</b> [5]	12	36/M	Right Hemiparesis	The right vertebral artery	MRI indicating a tortuous right vertebral artery that was causing severe indentation at the medulla	MVD	Improved
<b>Salvi et al.</b> [25]	13	54/F	Hypertension, hemiparesis	The left vertebral artery	MRI showed impingement of the vertebral artery on the left lateral medulla	MVD	Improved
<b>Koyama et al.</b> [26]	14	51/M	Quadriparesis and unconsciousness, hyper-reflexia of his left lower extremity and moderate decrease of sense of pain, temperature, and tactile sensation in his left extremities and trunk	bilateral vertebral artery	MRI reveals a flow-void area in the craniocervical junction and marked narrowing of the medulla oblongata and upper cervical cord by compression of the vertebral arteries. CT myelography shows the compression and narrowing of the spinal cord. Vertebral angiography demonstrates symmetrical running course of the arteries, which curve medially at the level of craniocervical junction.	MVD	Improved
<b>Maruyama et al.</b> [27]	15	30/M	gait disturbance and numbness of both arms and legs, mild	the left vertebral artery	CT and MRI scan show that the left vertebral artery extends from	MVD	Progressed after initial

			right facial hemiatrophy, bilateral pyramidal tract signs, muscular weakness of all extremities predominant of the right side, spastic gait and sensory disturbance		the left side of the medulla oblongata to the midline and distorts it by compression. Vertebral angiography reveals that the elongated left vertebral artery crosses the ventral aspect of the medulla oblongata		slight improvement
<b>Murata et al.</b> [28]	16	58/F	progressive tetraparesis and sensory disturbance, sensory disturbance in all modalities below the level of C2, onion-skin pattern sensory disturbance of the face, and motor weakness of the sternocleidomastoid muscles	bilateral vertebral arteries	MRI and vertebral angiography demonstrate ventrolateral compression of the medulla oblongata by bilateral tortuous vertebral arteries.	Suboccipital craniectomy, C1.4 laminectomy, and decompression of the medulla oblongata	Improved
<b>Kamada et al.</b> [29]	17	58/M	weakness of both legs and gait disturbance, lower-limb-dominant spasticity in all four extremities, lower-limb weakness, hyperreflexia in all extremities with positive Wartenberg's, Babinski's and Chaddock's signs, mild hypesthesia and hypopallesthesia in both lower	bilateral vertebral arteries	Brain MRI and magnetic resonance angiography demonstrated bilateral tortuous vertebral arteries compressing the medulla oblongata	Neurovascular decompression of the right vertebral artery	Improved

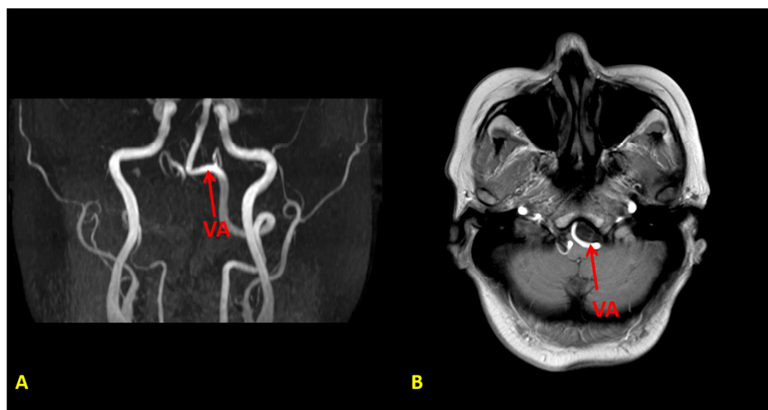
			limbs, and spastic gait						
<b>Ubogu et al.</b> [9]	18	70/M	partial paralysis, quadriparesis, and areflexia	bilateral abduction progressing to ataxia, and	the left vertebral artery	an ectatic left vertebral artery	emergency posterior fossa neurovascular decompression	Mild improvement	
<b>The current report</b>	19	67/F	right intermittent hypertension	hemiparesis and spasms;	the left vertebral artery	Cranial nerve MRI reveals a tortuous and dolichoectatic left vertebral artery compressing the medulla oblongata	MVD	Improved	

**Figure legends**

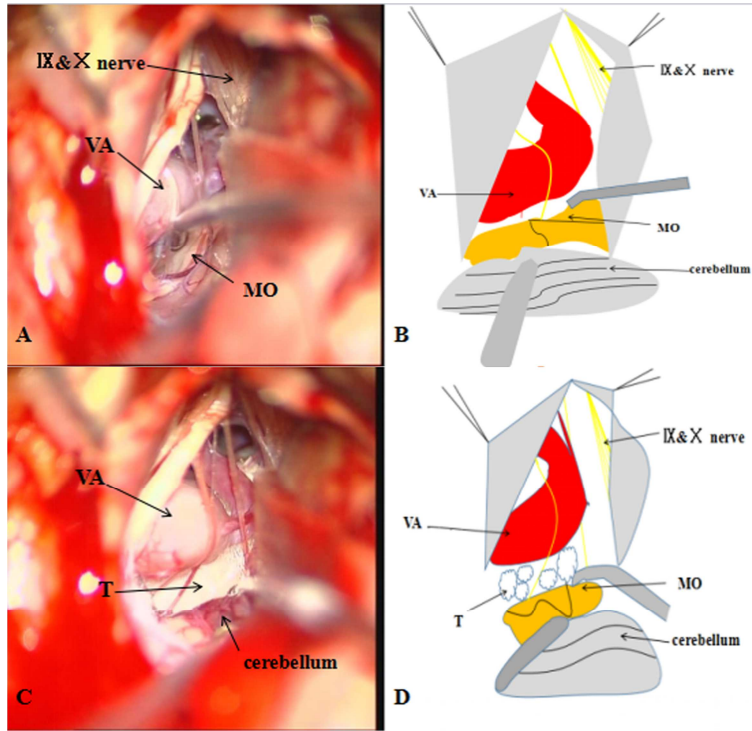
**Figure 1** (A) MRA obtained on admission shows the tortuous left vertebral artery (*Red arrow*) and (B) transverse T1-weighted MR image demonstrates intimate relation between the medulla oblongata and the vertebral artery (*Red arrow*). VA: the vertebral artery.

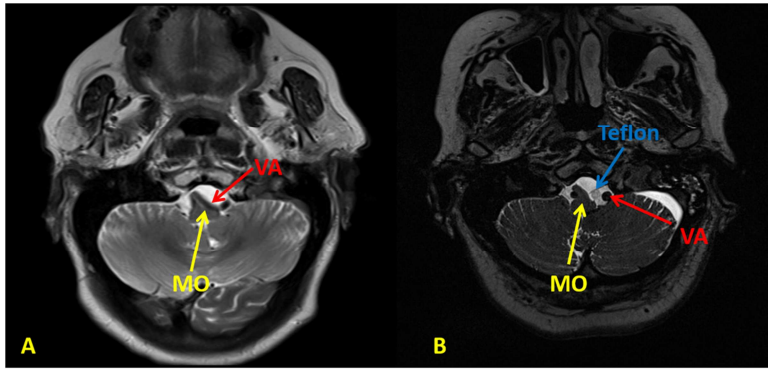
**Figure 2** (A) and (C) Intraoperative view shows the vertebral artery and the medulla. (B) and (D) The corresponding schematic representation of (A) and (C). MO: the medulla oblongata; T: Teflon felt; VA: the vertebral artery.

**Figure 3** (A) Preoperative transverse T2-weighted MR image clearly shows compression of the medulla oblongata (MO) (*Yellow arrow*) by the vertebral artery (VA) (*Red arrow*). (B) Postoperative transverse T2-weighted MR image reveals decompression of the medulla oblongata by the Teflon pad cotton (Blue arrow) between the vertebral artery and the medulla oblongata.



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**Highlights**

1. Little literature is available on hemiparesis caused by vascular medullary compression.
2. In this paper, we report a case of vertebral artery compression of the medulla oblongata which caused hemiparesis and possibly primary hypertension.
3. The patient was successfully managed by MVD, which helped relieve the symptoms.



**Abbreviations**

Magnetic Resonance Imaging (MRI); Motor evoked potential (MEP); microvascular decompression (MVD); Brainstem auditory evoked potential (BAEP); Somatosensory evoked potential (SEP)

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**Conflict of interest**

All the authors declare that they have no conflict of interest.

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