Vertebral Artery Compression of the Medulla

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Background: Intracranial arteries in the subarachnoid space may compress the brain parenchyma and cranial nerves. Most arterial compressive lesions have been attributed to dolichoectasia in the vertebral-basilar system, and prior reports have concentrated on the pressure effects of basilar artery ectasia. Much less is known about vertebral artery compression of the medulla.

Objective: To describe a series of patients with vertebral arteries compressing the medulla oblongata.

Design: Prospective case studies.

Setting: Tertiary care center.

Patients: Nine symptomatic patients, 4 men and 5 women, between the ages of 32 and 79 years.

Main Outcome Measures: Clinical phenomena, radiographic findings, treatment, and outcomes.

Results: We found that compression most commonly occurs at the ventrolateral surface. The clinical features can be transient or permanent and are predominantly motor and cerebellar or vestibular, but a poor correlation exists between the clinical findings and the severity or extent of impingement. The vertebral arteries were angulated, tortuous, or dilated but not necessarily dolichoectatic to cause obvious indentation. Seven patients were treated with antiplatelets and anticoagulants or analgesics, whereas 2 underwent microvascular decompression, resulting in temporary or no relief. One surgical patient developed cranial nerve complications. Among the medically treated patients, none had progression of deficits, and those with single episodes had no recurrence of symptoms.

Conclusion: This study is the largest collection, to our knowledge, of patients with medullary vascular compression. Further studies are needed to estimate its frequency, natural course, and preferred management.

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Cerebral arteries in the subarachnoid space may generate pressure and distortion of the brain parenchyma and stretching of the cranial nerves. Most intracranial arterial compressive lesions have been attributed to dolichoectasia, which refers to dilation, enlargement, and tortuosity of vessels.1 Within the cervicocranial arteries, dilative arteriopathy preferentially involves the vertebrobasilar system. Past reports have emphasized basilar artery ectasia compressing the pons and cranial nerves exiting the pons, causing trigeminal neuralgia and hemifacial spasm6,9 and also causing pontine infarcts.5,9 Other reports6,7 have described the general features and clinical symptoms of vertebrobasilar dolichoectasia. Compression of the medulla by dilated and/or tortuous vertebral arteries is less well known. We report herein a series of patients with vertebral arteries compressing the medulla oblongata.

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Methods:
From 1998 to 2004, we prospectively collected information on 9 symptomatic patients evaluated by at least 1 of the authors, including clinical phenomena, radiographic findings, treatment, and outcome. All patients underwent magnetic resonance imaging (MRI) and magnetic resonance angiography. The main inclusion criterion was obvious medullary compression by a vertebral artery, which was ectatic, tortuous, or dilated. Patients were excluded if they had dolichoectasia of the basilar artery or if they had other brain or vascular imaging findings that better explained their symptoms and signs. None of the patients had vascular occlusive lesions above the vertebral arteries in the posterior or anterior circulation.

Results:
The clinical characteristics of the 9 patients are summarized in Table 1. There were 4 men and 5 women. Ages ranged from 32 to 79 years. There was a bimodal age distribution at initial symptom presentation. Four patients developed their first symptoms in their 30s, and 5 patients first presented at older than 60 years. There were 8 white individuals and 1 African American individual. None of the patients had large artery occlusive lesions.

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Demographics

The clinical characteristics of the 9 patients are summarized in Table 1. There were 4 men and 5 women. Ages ranged from 32 to 79 years. There was a bimodal age distribution at initial symptom presentation. Four patients developed their first symptoms in their 30s, and 5 patients first presented at older than 60 years. There were 8 white individuals and 1 African American individual. None of the patients had large artery occlusive lesions.
CLINICAL PHENOTYPES

The symptoms and signs of each patient at initial evaluation are summarized in Table 1. Three patients had a single episode of symptoms that did not recur, 4 patients had multiple recurrent episodes, and 2 patients sustained permanent deficits. Three patients presented with motor limb weakness, 2 ipsilateral and 1 contralateral to the side of compression; 3 patients had vertigo or gait ataxia; 1 patient had hoarseness, vocal cord paralysis, and abnormal palate elevation ipsilateral to the side of compression; 1 patient had isolated tinnitus; and 1 patient had only throbbing headaches.

BRAIN IMAGING

Both MRI and magnetic resonance angiography were performed in all 9 patients. The findings are summarized alongside the clinical features in Table 1. Compression was present mostly along the lateral surface and involved the pyramids in all patients but the tegmentum in only 1 patient (Figures 1, 2, 3, 4, 5, and 6). All except 1 patient had compression by the left vertebral artery, indenting on the left surface of the medulla. Only 1 patient (patient 5; Figure 4) had a right vertebral artery that compressed the right medullary surface. This patient also had increased signal on T2-weighted imaging studies within the right medial medulla, representing either wallerian degeneration or damage from branch artery occlusion or compression (Figure 4C). Of note, 3 patients had MRIs that showed enlarged cisterns (Figures 3, 5, and 6).

TREATMENT AND CLINICAL COURSE

Six patients were treated conservatively with analgesics, antiplatelets, and anticoagulants, whereas 2 patients had decompressive surgery (patients 7 and 9 in Table 2). Patient 7 had slight postoperative improvement of her hoarseness but developed cranial nerve complications and occipital neuralgia. Patient 9 had temporary relief of symptoms, but episodes recurred 4 months after surgery. The 3 patients with single transient episodes treated conservatively have not had recurrences to date.

REPORT OF CASES

We describe 2 patients to illustrate different clinical features.
Patient 4

A 58-year-old man with hypertension suddenly lost his balance while standing near his desk at work and felt his body suddenly being directed to the right. He sensed that the ground was moving underneath him. The episode lasted approximately 20 seconds and did not recur. His neurologic examination results were normal. An MRI was obtained the following day (Figure 3), which showed an ectatic left vertebral artery severely compressing the anterolateral medulla. The MRI showed no acute infarcts on diffusion-weighted imaging and no hemorrhages on sus-

Figure 2. A 35-year-old woman developed throbbing headaches, although the results of a neurologic examination were normal. Magnetic resonance imaging showed impingement of the left anterolateral surface (A) by an angulated left vertebral artery (B).

Figure 3. A 68-year-old man developed sudden onset of ataxia, veering to the right, and vertigo for 20 seconds. Magnetic resonance imaging showed severe indentation (A) and displacement to the right of the medulla (B).
ceptibility scans. The T2-weighted imaging results were normal. He was prescribed warfarin sodium and has had no further episodes.

**Patient 5**

A 63-year-old man with hypertension and diabetes felt a prickling sensation in his left leg. An hour later, he had difficulty controlling this leg and the left hand. Results of a computed tomogram of the head that day and 2 days later were normal. While in Turkey during the next 3 weeks, his arm and leg became progressively weaker to the point where he could not move his hand or push down on the clutch pedal of his car. There was no involvement of his face. The weakness then stabilized and slowly improved during the next 2 months, after which he was examined (L.R.C.). On neurologic examination, no cranial nerve abnormalities were apparent. Fine finger movements were slow using the left hand, but his proximal strength was normal. There was a left foot drop and weakness of the hamstrings and anterior tibialis with brisk reflexes and a left extensor plantar response. An MRI showed a tortuous right vertebral artery compressing the anterolateral medulla, particularly at its basal portion near the pyramidal tract, and an increased T2 signal in the pyramid itself (Figure 4A-C). The vertebral artery was patent. He was prescribed aspirin and a generous fluid regimen. Two years later, he was walking several hours per day with a short leg brace and had only slight weakness of the leg extensors.

**COMMENT**

Medullary compression by the vertebral artery is a little-known clinical entity in the medical literature. There are 14 prior case reports, totaling 19 patients\(^8\text{--}^{21}\) ([Table 3](#table3)). We now describe the largest series, to our knowledge, of patients with this condition. From this collection, the major findings were as follows: patients can present with transient symptoms or permanent deficits, motor and vestibular or cerebellar features are the most common clinical presentations, there is a poor correlation between radiographic features and symptoms, and surgery may provide only temporary symptom relief and cause other complications.

**CLINICAL AND RADIOLOGIC FEATURES**

In our series, the clinical symptoms were variable but mostly consisted of motor and cerebellar or vestibular symptoms and signs. Compression typically occurred along the anterolateral surface, consistent with prior reports ([Table 3](#table3)). The lateral segments of the medulla contain the corticospinal tracts, which when damaged can cause either contralateral or ipsilateral findings depending on the rostral-caudal location of the compression. Two of our patients with hemiparesis had ipsilateral findings, and 1 patient had contralateral findings. In the lateral corticospinal tract, the
fibers innervating the lower extremities are located laterally. In patients with transient leg weakness, the clinical features correspond to the topographic distribution of these fibers (Figure 1). Neighboring tracts, such as the spinothalamic and spinocerebellar pathways, did not appear to be involved in our patients, but hypoalgesia and hypothermesthesia were reported in 1 patient with vertebral artery lateral medullary compression. Anterolateral compression of the lower cranial nerves or the nucleus ambiguous could explain the vocal cord paralysis in patient 7 and dysphagia and dysphonia seen in previously described patients. Aural symptoms may be explained by impingement on the cochlear nuclei or the eighth cranial nerve exiting the medulla. Bulbar compression has also been suggested as a possible cause of refractory hypertension and sleep-disordered breathing, but we did not see these features in any of our patients.

In some of our patients, the clinical features did not match the radiographic findings. In particular, patients with transient symptoms such as ataxia or limb weakness had severe medullary compression (Figure 3 and Figure 5) and yet no significant deficits. Patients who had nausea and vertigo did not show compression of the dorsolateral medulla or periventricular nuclei such as the area postrema. However, the possibility exists that ventrolateral impingement could lead to distortion of the dorsolateral areas. Seven of 9 patients had MRIs that showed pyramidal tract involvement at the ventral surface of the medulla, and only 2 patients had pyramidal signs on examination. Overall, we found a poor correlation between symptoms and signs and the extent and severity of compression.

### Table 2. Management and Subsequent Clinical Course

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Therapy</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Aspirin</td>
<td>Tinnitus persists</td>
</tr>
<tr>
<td>2</td>
<td>Aspirin and dipyridamole</td>
<td>No episodes in 1 year</td>
</tr>
<tr>
<td>3</td>
<td>Analgesics</td>
<td>Headaches responsive to analgesics</td>
</tr>
<tr>
<td>4</td>
<td>Warfarin sodium</td>
<td>No episodes in 1 year</td>
</tr>
<tr>
<td>5</td>
<td>Aspirin</td>
<td>Significant improvement at 3 years; uses a leg brace</td>
</tr>
<tr>
<td>6</td>
<td>Aspirin</td>
<td>No further deficits at 1 year</td>
</tr>
<tr>
<td>7</td>
<td>Decompression</td>
<td>After surgery, hoarseness improved; occipital neuralgia, left ear deafness, oscillopsia, vertigo, and weak tongue</td>
</tr>
<tr>
<td>8</td>
<td>Aspirin</td>
<td>No further episodes in 4 years</td>
</tr>
<tr>
<td>9</td>
<td>Decompression</td>
<td>Episodes disappeared for 4 months, then recurred; magnetic resonance imaging showed displacement of left tegmentum and base</td>
</tr>
</tbody>
</table>

Vessel Disease

The intracranial posterior circulation arteries often show regions of dolichoectasia, which can stretch cranial nerves, compress the brainstem, and cause brain infarcts. However, in our series, we found that the vertebral arteries were sometimes tortuous, angulated, and/or dominant but not necessarily elongated and dilated (Figure 2B).
Dolichoectasia is therefore not required for the vertebral artery to impinge on the medulla. Several factors likely contribute to the anatomical variations. We found that most of our elderly patients had hypertension and diabetes, which may cause progressive vascular wall damage. Younger patients may have genetic predispositions. For example, dolichoectasia occurs in young patients with Marfan syndrome, acquired immunodeficiency syndrome, sickle cell disease, and Fabry disease. The young patients in our study (patients 3, 6, 7, and 9) had negative evaluation results for these conditions.

**MECHANISMS OF BRAIN INJURY**

Vertebral arteriopathies cause neurologic symptoms through multiple mechanisms. Direct medullary compression is the likely cause for certain patients with gradual and persistent symptoms. External compression of brain structures often causes symptoms away from the local region of compression. This may explain why in some patients, the clinical symptoms did not match the location of medullary compression, similar to an expanding extraparenchymatous mass lesion such as a subdural hematoma or congenital anomaly such as Arnold-Chiari malformation. Compression may be gradual, allowing for adaptation, which may reduce the risk of damaging respiratory and autonomic centers in the medulla.

Another potential mechanism is ischemic injury. Compression could generate pressure on perforating branches from the vertebral artery. Traction on these arteries could disrupt blood flow and cause small-vessel infarcts or migrainelike headaches, as seen in patient 3. Elongation and angulation of the intracranial arteries can stretch and distort the orifices of arterial branches. Dilatative arteriopathy, for example, can lead to decreased blood flow in penetrating branches of the basilar artery and cause pontine infarcts. Transcranial Doppler studies of patients with dolichoectasia have shown abnormal flow patterns. Blood

<table>
<thead>
<tr>
<th>Patient Age, y</th>
<th>Sex</th>
<th>Radiographic Findings</th>
<th>Clinical Findings</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>53/M</td>
<td>M</td>
<td>Left tortuous VA pressing on lateral surface</td>
<td>Progressive left hemiparesis and left cranial nerve XII</td>
<td>MVD</td>
<td>Improvement</td>
<td>8</td>
</tr>
<tr>
<td>30/M</td>
<td>M</td>
<td>Left elongated VA crossed ventral surface</td>
<td>Quadriparesis and bilateral sensory loss</td>
<td>MVD</td>
<td>Slight improvement</td>
<td>9</td>
</tr>
<tr>
<td>5/M</td>
<td>M</td>
<td>Left VA loop compressing ventral surface</td>
<td>Central sleep apnea</td>
<td>Acetazolamide</td>
<td>No change</td>
<td>10</td>
</tr>
<tr>
<td>38/M</td>
<td>M</td>
<td>Right VA loop compressing cervicomedullary junction/cranial nerve XI</td>
<td>Torticollis, vertigo, vomiting, arrhythmias</td>
<td>MVD</td>
<td>Cervical stiffness improved</td>
<td>11</td>
</tr>
<tr>
<td>30/M</td>
<td>M</td>
<td>Elongated left VA</td>
<td>Progressive hemiparesis</td>
<td>MVD</td>
<td>No change; sectioning of VA led to improvement in weakness</td>
<td>12</td>
</tr>
<tr>
<td>56/M</td>
<td>M</td>
<td>Ectatic left VA compressing ventral cervicomedullary junction</td>
<td>Gradual left hemiparesis</td>
<td>None</td>
<td>Not reported</td>
<td>13</td>
</tr>
<tr>
<td>36/M</td>
<td>M</td>
<td>Tortuous right VA on ventrolateral surface</td>
<td>Progressive right hemiparesis and hypoaesthesia, dysphagia</td>
<td>MVD</td>
<td>Gradual improvement</td>
<td>14</td>
</tr>
<tr>
<td>47/M</td>
<td>M</td>
<td>Tortuous left VA on ventrolateral surface of pontomedullary junction</td>
<td>Left ear deaf, gait ataxia</td>
<td>MVD</td>
<td>Ataxia improved</td>
<td>14</td>
</tr>
<tr>
<td>54/F</td>
<td>F</td>
<td>Left lateral surface</td>
<td>Hypertension, hyperekplexia, hemiparesis</td>
<td>MVD</td>
<td>Blood pressure normalized, startle responses disappeared</td>
<td>15</td>
</tr>
<tr>
<td>74/F</td>
<td>F</td>
<td>Left ectatic VA compressing inferior olive</td>
<td>Palatal myoclonus</td>
<td>MVD</td>
<td>Cured</td>
<td>16</td>
</tr>
<tr>
<td>47/M</td>
<td>M</td>
<td>Bilateral VA compression of ventrolateral surface</td>
<td>Left hemiparesis and hemisensory loss</td>
<td>MVD of both arteries</td>
<td>Relief of symptoms</td>
<td>17</td>
</tr>
<tr>
<td>51/M</td>
<td>M</td>
<td>Bilateral VA compression of ventrolateral surface</td>
<td>Right hemiparesis and hemihypesthesia</td>
<td>MVD</td>
<td>Gradual improvement</td>
<td>18</td>
</tr>
<tr>
<td>70/M</td>
<td>M</td>
<td>Left dolichoectatic VA pressing on cervicomedullary region</td>
<td>Rapid progression of cranial nerves III-XII, quadriparesis, titubation, respiratory distress</td>
<td>MVD</td>
<td>Respiration normalized; cranial nerves III, VI, VII, and VIII resolved in 6 months; quadriparesis persisted</td>
<td>19</td>
</tr>
<tr>
<td>54/M</td>
<td>M</td>
<td>Right ectatic VA on lateral surface</td>
<td>Left hypoalgesia and hypothermesthesia</td>
<td>None</td>
<td>Slight improvement</td>
<td>20</td>
</tr>
<tr>
<td>63/M</td>
<td>M</td>
<td>Ectatic VAs</td>
<td>Dysphagia (3), dysphonia (1), hemiparesis (1), quadriparesis (3)</td>
<td>MVD (5)</td>
<td>4 Patients almost asymptomatic, 1 patient with slight improvement</td>
<td>21</td>
</tr>
<tr>
<td>58/M</td>
<td>F</td>
<td>Ectatic VAs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>55/F</td>
<td>F</td>
<td>Ectatic VAs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>67/M</td>
<td>F</td>
<td>Ectatic VAs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>73/M</td>
<td>M</td>
<td>Ectatic VAs</td>
<td></td>
<td></td>
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</tbody>
</table>

**Abbreviations:** MVD, microvascular decompression; VA, vertebral artery.
flow is often to and fro within the dilated artery, causing reduced antegrade flow in the vertebrobasilar system. Blood flow insufficiency under these circumstances can lead to transient ischemic attacks and therefore may be the cause of the transient symptoms seen in some of our patients. Reduced flow can also lead to thrombus formation within the dilated segments, obstruction of penetrating branches by the thrombus, or embolization of clot fragments into the small-vessel perforators. Alternatively, atherosclerotic plaques may form along the dilated vessel wall and obstruct the arterial branches.

Patient 5 had an angulated right vertebral artery (Figure 4B) pressing on the lateral surface of the medulla (Figure 4A), and MRI showed a T2 hyperintensity in the medial medulla (Figure 4C). Although the lesion could represent an infarct, a 3-week history of progressive symptoms is atypical for the development of a stroke. The lesion could represent either an unusual “slow stroke” mediated by altered flow in a branch of the vertebral artery or wallerian degeneration.

**TREATMENT AND OUTCOME**

The best treatment of patients with bulbar compression by a vertebral artery is unknown. In all prior reports except for 3, the patients were treated surgically (Table 3). Almost all of these surgical cases reported significant improvement of deficits after microvascular decompression, although the preoperative and postoperative neurologic examination results and extent of improvement were not reported in detail. The 2 patients we referred for surgery had only slight or temporary improvement and developed complications from new cranial nerve lesions. The 7 other patients were treated conservatively with antiplatelets, anticoagulants, or analgesics, and none had progressive or new symptoms. On the basis of our series of patients, we are reluctant to recommend surgery.

**STUDY LIMITATIONS**

One of the main limitations of this report is that we cannot be certain that the symptoms in each patient were caused by the radiographic findings; 3 patients (3, 6, and 9) had symptoms that could have been part of a migraine disorder. It would be helpful to compare our symptomatic group with an asymptomatic population with vertebral artery compression of the medulla. We have encountered patients with apparently asymptomatic compression but have not systematically studied this phenomenon, and there is no literature on asymptomatic compression. We now are embarking on a prospective blind study looking at MRIs to attempt to answer this query.

In conclusion, medullary compression from a vertebral artery can cause a wide spectrum of clinical presentations, from sudden transient events to persistent deficits and from lower cranial nerve findings to respiratory and autonomic changes. Contrary to prior reports, microvascular decompression does not always relieve symptoms. Future studies are needed to estimate the true frequency of medullary vascular compression, its natural history, and its preferred treatment.

**References**


Announcement

Trial Registration Required. In concert with the International Committee of Medical Journal Editors (ICMJE), Archives of Neurology will require, as a condition of consideration for publication, registration of all trials in a public trials registry (such as http://ClinicalTrials.gov). Trials must be registered at or before the onset of patient enrollment. This policy applies to any clinical trial starting enrollment after July 1, 2005. For trials that began enrollment before this date, registration will be required by September 13, 2005, before considering the trial for publication. The trial registration number should be supplied at the time of submission.

For details about this new policy, and for information on how the ICMJE defines a clinical trial, see the editorial by DeAngelis et al in the January issue of Archives of Dermatology (2005;141:76-77). Also see the Instructions to Authors on our Web site: www.archneurol.com.