Vertebral Artery Compression of the Medulla

Sean I. Savitz, MD; Michael Ronthal, MD; Louis R. Caplan, MD

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.

Arch Neurol. 2006;63:234-241
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-
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 patients8-21 (Table 3). 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 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). 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.20 Anterolateral compression of the lower cranial nerves or the nucleus ambiguus could explain the vocal cord paralysis in patient 7 and dysphagia and dysphonia seen in previously described patients.14,21 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 hypertension22,23 and sleep-disordered breathing,10 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.

**VESSEL DISEASE**

The intracranial posterior circulation arteries often show regions of dolichoectasia, which can stretch cranial nerves, compress the brainstem, and cause brain infarcts.6 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.24,25 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.5,26 Transcranial Doppler studies of patients with dolichoectasia have shown abnormal flow patterns. Blood

<table>
<thead>
<tr>
<th>Patient Age, y/ 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>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>Left elongated VA crossed ventral surface</td>
<td>Quadriparesthesia and bilateral sensory loss</td>
<td>MVD</td>
<td>Slight improvement</td>
<td>9</td>
</tr>
<tr>
<td>5/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>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>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>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>Tortuous right VA on ventrolateral surface</td>
<td>Progressive right hemiparesis and hypoalgesia, dysphagia</td>
<td>MVD</td>
<td>Gradual improvement</td>
<td>14</td>
</tr>
<tr>
<td>47/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>15</td>
</tr>
<tr>
<td>54/F  74/F</td>
<td>Left lateral surface</td>
<td>Hypertension, hyperekplexia, hemiparesis</td>
<td>MVD</td>
<td>Blood pressure normalized, startle responses disappeared</td>
<td>16</td>
</tr>
<tr>
<td>47/M</td>
<td>Left ectatic VA compressing inferior olive</td>
<td>Palatal myoclonus</td>
<td>1 Patient: MVD</td>
<td>Cured</td>
<td>17</td>
</tr>
<tr>
<td>51/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>18</td>
</tr>
<tr>
<td>53/F</td>
<td>Elongated and curved left VA pressing ventrolateral surface</td>
<td>Right hemiparesis and hemihypesthesia</td>
<td>MVD</td>
<td>Gradual improvement</td>
<td>19</td>
</tr>
<tr>
<td>70/M</td>
<td>Left dolichoectatic VA pressing on cervicomedullary region</td>
<td>Rapid progression of cranial nerves III-XII, quadriparesthesia, titubation, respiratory distress</td>
<td>MVD</td>
<td>Respirations normalized; cranial nerves III, VI, VII, and VIII resolved in 6 months; quadriparesthesia persisted</td>
<td>20</td>
</tr>
<tr>
<td>54/M</td>
<td>Right ectatic VA on lateral surface</td>
<td>Left hypoalgesia and hypothermesthesia</td>
<td>None</td>
<td>Slight improvement</td>
<td>21</td>
</tr>
<tr>
<td>63/M  58/M  55/F  67/M  73/M</td>
<td>Ectatic VAs</td>
<td>Dysphagia (3), dysphonia (1), hemiparesis (1), quadriparesthesia (3)</td>
<td>MVD (5)</td>
<td>4 Patients almost asymptomatic, 1 patient with slight improvement</td>
<td>22</td>
</tr>
</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.

**REFERENCES**


