Dolichoectatic Basilar Artery: A Review of 23 Cases

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SUMMARY The dolichoectatic basilar artery was found in 23 cases during a 10-year period. The 19 males and 4 females ranged in age from 30 to 69 years (mean: 55 years). Hypertension was noted in 17 patients. In seventeen (74%) of the present cases this anomaly could be visualized with CT scan. Seven patients (30%) presented with pontine infarction, which was identified on CT scan in all cases. Vertebro-basilar insufficiency was found in four patients. One patient had transient ischemic attacks. There were facial spasms in four patients and impairment of the lower cranial nerves in one. One patient exhibited cerebellar hemorrhage. In two patients this anomaly was found incidentally. Associated intracranial aneurysms were identified in seven patients, including fusiform aneurysms in 4 and saccular aneurysms in 3. Three patients had an accompanying hydrocephalus. The dolichoectatic basilar artery is associated with various consequences especially in relation to the pathogenesis of brainstem infarction. When this anomaly is diagnosed by CT findings, even if it is clinically asymptomatic, it may be better to treat these patients with medical therapy used to prevent ischemic stroke.

THE BASILAR ARTERY at times is the site of marked elongation, widening, and tortuosity. This is usually called a dolichoectatic basilar artery or a mega-dolichobasilar anomaly. There are many reports of complications due to the compression of the brainstem or cranial nerves. Information about ischemic changes in the patients with this anomaly is scarce. The object of this paper is to study the relationship between this anomaly and cerebral ischemia, especially brainstem infarction, and to discuss the significance of the clinical entity.

Patients and Methods

Twenty-three patients with a dolichoectatic basilar artery were seen during a 10-year period from 1976 to 1985. The 19 male and 4 female patients ranged in age from 30 to 69 years (mean: 55 years).

The diagnosis of this anomaly was based on radiological findings. The dolichoectatic basilar artery of this series was defined as one longer that 21 mm in distance between the upper bifurcation of the basilar artery and the dorsum sellae. Computed tomography (CT) with and without contrast material, and four vessel angiography were performed in all except one patient.

Results

A summary of the 23 patients in the present series is shown in table 1. There was a male predominance (19 patients). Hypertension was a preexisting condition in seventeen patients (74%). The dolichoectatic basilar artery could be visualized with CT scan in 17 patients (fig. 1).

The clinical features are divided into four groups and are summarized in table 2. Among ischemic signs, 7 (30%) of 23 patients presented with pontine infarction, which was identified on CT scan in all cases (fig. 1).

2. Vertebro-basilar insufficiency, with such symptoms as persistent vertigo, tinnitus, and headache, was found in four patients (case 2, 16, 17, 23). One patient had transient ischemic attacks with episodes of hemiparesis on the left side lasting from 30 minutes to one hour. Four patients had a history of cerebral infarction, including multiple "lacunar" infarctions (case 13, 20) (fig. 3) and left temporal infarction in two (case 5, 10).

As evidence of cranial nerve compression, facial spasm was noted in 4 patients and impairment of the ninth, tenth, and twelfth nerves in one patient.

Seven of the patients had associated intracranial aneurysms: 4 had fusiform aneurysms, three of whom also had multiple giant fusiform aneurysms of both internal carotid arteries. One had a fusiform aneurysm at the junction of internal carotid artery and posterior communicating arteries. Three patients had a saccular aneurysm. Two of 3 patients with a saccular aneurysm presented with subarachnoid hemorrhage. One had a completely thrombosed aneurysm 2 cms in diameter, involving the left vertebral artery-basilar artery junction. Another with a 10 year history of pulseless disease had a 2 cm saccular aneurysm at the tip of the basilar artery. One patient with a 4.5 cm partially thrombosed aneurysm of the left vertebral artery-basilar artery junction suffered dysarthria and cerebellar incoordination due to mass effect at the cerebello-pontine angle (fig. 3).

One patient with a history of hypertension had a small hemorrhage in the cerebellar vermis. There was no lesion detected by angiography to account for this cerebellar hemorrhage. In two patients the dolichoectatic basilar artery was found incidentally. Three patients had an accompanying asymptomatic hydrocephalus (fig. 3).

Nineteen patients, including two without symptoms, had received conservative therapy. Four patients with facial spasm were surgically treated. The follow-up period varied from 6 months to 5 years. In two (case 16, 20) of 19 patients with medical therapy the symptoms improved slightly. The others had no improvement, but did not deteriorate. There were four deaths in this series: two (cases 12, 14) died of rupture of the aneurysmal sac, with massive subarachnoid hemorrhage. One patient with a fusiform aneurysm had subarachnoid hemorrhage. One patient with a saccular aneurysm had subarachnoid hemorrhage.
Table 1  Summary of 23 Patients with a Dolichoectatic Basilar Artery

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Hypertension</th>
<th>Clinical Presentation</th>
<th>Visualized DEBA</th>
<th>Other Findings except DEBA</th>
<th>Associated Intracranial Aneurysms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>43</td>
<td>+</td>
<td>vertigo, rt. hemiparesis rt. hyperreflexia, rt. Babinski sign</td>
<td>-</td>
<td>lt. pontine infarction</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>54</td>
<td>-</td>
<td>vertigo</td>
<td>-</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>30</td>
<td>-</td>
<td>lt. facial spasm</td>
<td>-</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>43</td>
<td>+</td>
<td>lt. facial spasm</td>
<td>+</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>43</td>
<td>+</td>
<td>ataxic gait</td>
<td>+</td>
<td>cerebellar hemorrhage, lt. temporal infarction</td>
<td>both ICA giant fusiform aneurysms</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>47</td>
<td>+</td>
<td>dysarthria, lt. hemiparesis lt. hyperreflexia, lt. Babinski sign</td>
<td>+</td>
<td>rt. pontine infarction</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>50</td>
<td>-</td>
<td>lt. facial spasm</td>
<td>-</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>52</td>
<td>+</td>
<td>impairment of the IX, X, XII cranial nerves</td>
<td>+</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>65</td>
<td>+</td>
<td>deep stupor, rt. hemiparesis rt. hyperreflexia, rt. Babinski sign eyes: conjugate deviation to the left</td>
<td>+</td>
<td>lt. pontine infarction</td>
<td>none</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>69</td>
<td>+</td>
<td>vertigo, lt. hemiparesis lt. hyperreflexia, lt. Babinski sign</td>
<td>+</td>
<td>rt. pontine infarction, lt. temporal infarction</td>
<td>both ICA giant fusiform aneurysms</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>69</td>
<td>-</td>
<td>lt. hemiparesis, lt. hypotension lt. hyperreflexia, lt. Babinski sign, horizontal nystagmus</td>
<td>+</td>
<td>rt. pontine infarction</td>
<td>none</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>56</td>
<td>+</td>
<td>sudden unconsciousness stiff neck</td>
<td>+</td>
<td>subarachnoid hemorrhage, lt. ponto-cerebelo junction high dense mass</td>
<td>Lt. V-B junction complete thrombosed aneurysm</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>66</td>
<td>+</td>
<td>dysarthria, dysphagia, lt. hemiparesis, lt. hyperreflexia, lt. Babinski sign, eyes: conjugate deviation to the left</td>
<td>+</td>
<td>rt. pontine infarction</td>
<td>both ICA giant fusiform aneurysms</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>56</td>
<td>+</td>
<td>sudden unconsciousness stiff neck</td>
<td>+</td>
<td>subarachnoid hemorrhage, lt. thalamic high dense mass</td>
<td>basilar tip aneurysm</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>52</td>
<td>-</td>
<td>lt. facial spasm</td>
<td>+</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>62</td>
<td>+</td>
<td>vertigo, headache, vomiting</td>
<td>-</td>
<td>none</td>
<td>rt. IC-PC fusiform aneurysm</td>
</tr>
<tr>
<td>17</td>
<td>M</td>
<td>60</td>
<td>+</td>
<td>vertigo, tinnitus</td>
<td>+</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>68</td>
<td>+</td>
<td>drowsiness, lt. hemiparesis lt. hyperreflexia, lt. Babinski sign</td>
<td>+</td>
<td>rt. pontine infarction</td>
<td>none</td>
</tr>
<tr>
<td>19</td>
<td>M</td>
<td>46</td>
<td>+</td>
<td>incidentally</td>
<td>+</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>20</td>
<td>M</td>
<td>58</td>
<td>+</td>
<td>dysarthria, lt. cerebellar sign</td>
<td>+</td>
<td>lt. cerebello-pontine angle high dense mass, multiple small cerebral infarctions, hydrocephalus</td>
<td>Lt. V-B junction aneurysm</td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>59</td>
<td>-</td>
<td>incidentally</td>
<td>-</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>22</td>
<td>M</td>
<td>53</td>
<td>+</td>
<td>transient ischemic attacks</td>
<td>+</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>23</td>
<td>M</td>
<td>66</td>
<td>+</td>
<td>vertigo</td>
<td>+</td>
<td>none</td>
<td>none</td>
</tr>
</tbody>
</table>

DEBA = dolichoectatic basilar artery; ICA = internal carotid artery; V-B = vertebral artery-basilar artery; IC-PC = internal carotid artery-posterior communicating artery.

Discussion

The dolichoectatic basilar artery is relatively rare and generally asymptomatic. The etiology is unclear. Occurrence in younger people has been reported\textsuperscript{2-5} and some congenital factors may contribute to its development. However, severe arteriosclerotic changes in the basilar artery were noticed on angiography with associated hypertension in 74% of twenty-three patients in this series.\textsuperscript{6,6} The diagnosis of a dolichoectatic basilar...
artery must be confirmed by angiography, but the anomaly has been identified by CT scan. In seventeen (74%) of our cases this abnormality was visualized with CT scan.

Most reports have related symptoms and neurologic deficits due to compression to the brainstem or cranial nerves. In our study, facial spasm occurred in four patients (17%), and impairment of the lower cranial nerves (ninth, tenth, and twelfth nerves) in one. Other authors have reported involvement of the third, fourth, fifth, sixth, eighth, and eleventh cranial nerves.

The relationship between intracranial ischemic changes and this abnormality has been described occasionally. Pontine infarction was found in seven patients (30%) in this study. One other report of brainstem infarction with a dolichoectatic basilar artery is available in the English literature.

We assume that brainstem infarction in these patients relates to the distortion of the paramedian branches of the basilar artery resulting from the elongation and tortuosity of the basilar artery. In addition to the displacement of these paramedian branches severe arteriosclerosis develops in the basilar artery and causes reduction of the blood flow. Presumably occlusion of the paramedian branches occurs at the site of the displacement and sharp angulation where they originate from the basilar artery. In this study vertebro-basilar insufficiency was found in 4 patients and one patient had transient ischemic attacks with episodes of left-sided hemiparesis. Transient ischemic attacks in patients with a dolichoectatic basilar artery may forewarn of brainstem infarction.

Hemiparesis in patients with this abnormality has been reported. In the present study four patients had a history of cerebral infarction. There were multiple lacunar infarctions in two patients and left temporal infarction in two. The lacunar infarctions have been described to be most frequent in patients who have had long-sustained hypertension. Dolichoectasia involves the carotid system less frequently.

Seven (30%) of the 23 patients had associated intracranial aneurysms. Fusiform aneurysms were seen in 4 patients, in three of whom they were multiple and giant. Fusiform aneurysms have been reported more frequently in older age groups, more often in males, and occur generally in severely atherosclerotic basilar arteries. In our study the mean age was 55 years,
males were predominant, and severe arteriosclerotic changes were seen in the basilar artery. Although saccular aneurysms have been reported to be uncommon by contrast with fusiform aneurysms in these patients, three of the 8 patients had saccular aneurysms. It is interesting that all of the saccular aneurysms were large and were either completely or partially thrombosed.

Rupture of the dolichoectatic basilar artery is considered unlikely. In this study one patient presented with cerebellar hemorrhage. The angiographic findings in this patient showed no vascular malformation and were diagnosed as afflicted with a hypertensive cerebellar hemorrhage due to rupture of a microaneurysm or of a necrotic arteriolar wall secondary to hypertensive atherosclerotic degeneration.

Three patients had accompanying hydrocephalus. The mechanism of hydrocephalus in these patients is most likely a combination of increased cerebrospinal fluid (CSF) pulse pressure and impairment of outward CSF flow by "countercurrent pulsations" of the basilar artery. In other words, a basilar artery extending into the floor of the third ventricle exerts a water-hammer pulse transmitted toward the foramina of Monro resulting in an impairment of outflow from the lateral ventricles.

When a dolichoectatic basilar artery is found by CT scan, full investigation by four vessel angiography or digital subtraction angiography may be considered in view of the association with aneurysm. It may be wise to treat these patients with platelet antiaggregate therapy to prevent brainstem or cerebral infarction, even in the absence of clinical evidence of ischemia.

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