Arterial Pathology in Cerebellar Infarction

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We reviewed 88 pathologically proven cerebellar infarcts, examining the entire thoracocervical arterial system (including the spinal part of the vertebral artery, the intracranial arteries, and the heart) in all cases. For 50 infarcts, we found an arterial occlusion. The intracranial part of the vertebral artery was involved in 38 infarcts (76%), the basilar artery in 20 infarcts (40%), and a cerebellar artery in 12 infarcts (24%). Eight infarcts were due to bilateral distal vertebral artery occlusions. For the 50 infarcts, the cause of the occlusion was atherosclerosis in 28 (56%) and a cardiac embolism in 12 (24%). For the remaining 38 infarcts, we found no arterial occlusions on postmortem examination. Of these 38 infarcts, 26 (68%) were associated with a cardiac source of emboli. For the entire group of 88 infarcts, atherosclerosis was the cause in 31 (35%), while a cardiac embolism was proven or presumed in 38 (43%). Dissecting aneurysms, atherosclerotic artery-to-artery embolism, or hemodynamic mechanisms could have been responsible for a few infarcts. We conclude that cerebellar infarcts often arise from cardiogenic embolism. (Stroke 1990;21:1299–1305)

Data on the mechanisms of cerebellar infarction are scarce. A few detailed clinico-pathologic reports based on single cases or short series are available.\(^1 - \text{9}\) In a larger series of 28 acute massive cerebellar infarcts Sypert and Alvord\(^10\) found 31 arterial occlusions. The most common site of occlusion (18 of 31) was the distal vertebral artery (VA), but causes of the occlusions were not specified. Thompson\(^11\) studied five cases of superior cerebellar artery (SCA) infarction and emphasized a cardiac embolism. Rodda\(^12\) studied postmortem arteriograms of patients with 21 cerebellar infarcts; 19 infarcts were < 1.5 cm in diameter. Twelve patients had a stenosis of the VA or basilar artery (BA); 10 of these 12 also had a significant stenosis of the cerebellar arteries, and Rodda suggested that hemodynamic factors are frequently involved in cerebellar infarction. Seven other patients were free of arterial stenosis, and five of these seven had a cardiac source of emboli. In a thorough study of 44 arterial occlusions in the verteobasilar system, Castaigne et al\(^13\) pointed out the high frequency of thrombotic occlusions. Embolism was more frequent in the distal part of the BA and in the posterior cerebral artery (PCA) than in the VA. We report the results of a pathologic study of the heart and the extracranial and intracranial arteries of 56 patients with a total of 88 cerebellar infarcts.

Subjects and Methods

Among pathologic records of 190 cerebellar strokes from the files of the Raymond Escourolle Laboratory of Neuropathology, 28 (15%) were due to a cerebellar hemorrhage and the remaining 162 (85%) to infarcts > 2 cm in diameter. Based on the presence of detailed clinical notes, a complete autopsy including a study of the heart, and a thorough study of the arterial supply of the brain from the heart, including the cervical part of the VAs, we selected 56 patients with cerebellar infarcts for a study of the intracranial arteries. Data from macroscopic and microscopic studies of the cerebral hemispheres, brain stem, and cerebellum and the clinical correlations have been reported.\(^14 - 16\) Using a technique described elsewhere,\(^17,18\) we dissected the aorta and its cervical branches and excised and decalcified the cervical spine and base of the skull. In each patient, the arterial pattern and the site and nature of the arterial lesions were noted.\(^19\) The degree of stenosis was graded as 0, no stenosis; 1, stenosis of <75%; 2, stenosis of >75%; or 3, occlusion as previously reported.\(^17\) To ascertain the nature of the occlusions, segments of the occluded arteries were embedded in paraffin and stained with hematoxylin and eosin and phosphotungstic acid and hematoxylin. Diagnostic criteria for embolism included occlusion without an arterial wall lesion or the absence of occlusion in the entire arterial supply, particularly in the case of hemorrhagic infarcts. Criteria for cardiac embolism were the presence of a cardiac source of emboli and infarcts in other terri-
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FIGURE 1. Diagrams of arterial pattern, sites of occlusions, and sites of atherosclerotic stenoses in 32 patients with 50 cerebellar infarcts and arterial occlusion at postmortem examination. Site and side of cerebellar infarcts and presence (+) or absence (-) of cardiac source of emboli (CSE) are given within diagrams. Notches in aortic arch represent ulcerative plaques. Degrees of atherosclerotic stenosis are graded as 0, 1, 2, or 3. SCA, superior cerebellar artery; AICA, anterior inferior cerebellar artery; PICA, posterior inferior cerebellar artery; R, right; L, left.

In the 56 patients, there were 88 infarcts, 36 in the SCA territory, 33 in the posterior inferior cerebellar artery (PICA) territory, and 19 in the anterior inferior cerebellar artery (AICA) territory. We found 25
We found 93 anatomic developmental variations was present in six patients (11%), hypercholesterolemia in five (9%), and arteriopathy of the lower limbs in four (7%). No significant differences concerning age, risk factors, or stroke etiology were noted among the groups of patients with recent and old infarcts.

Additional infarcts in the PCA territory. Thirty-two cerebellar infarcts occurred together with tonsillar herniation and brainstem compression. There were 11 hemorrhagic infarcts, eight in the SCA and three in the PICA territories. Sixty-eight infarcts were recent, that is, the patients died <3 months after the stroke, while the other 20 were old infarcts, that is, the patients died >3 months after the stroke.

**Results**

Our 56 patients comprised 36 men and 20 women aged 27–88 (median 67, mean ± SEM 65 ± 13) years; more than half were 60–80 years old at death. Hypertension was present in 36 patients (64%); 22 (39%) had experienced a previous stroke. Ischemic heart disease was present in 19 patients (34%) (nine had acute myocardial infarction), 11 (20%) had atrial fibrillation, six (11%) had intracardiac thrombus (either mural or atrial), six (11%) had valvular disease, two (4%) had endocarditis, and one had non-ischemic dilated cardiomyopathy. Diabetes mellitus was present in six patients (11%), hypercholesterolemia in five (9%), and arteriopathy of the lower limbs in four (7%). No significant differences concerning age, risk factors, or stroke etiology were noted among the groups of patients with recent and old infarcts. We found 93 anatomic developmental variations from the “normal” arterial pattern of the vertebrobasilar system among the 56 patients (Table 1).

We found 50 cerebellar infarcts among 32 patients with arterial occlusion. Figure 1 shows the pathologic vascular anatomy in these 32 patients, and Figure 2 lists the causes of occlusion. Atherothrombotic infarcts included those due to a thrombus superimposed on atheromatous stenosis (26 infarcts) and those resulting from artery-to-artery embolism due to severe (≥90%) atherosclerotic stenosis of the VA ostium (two infarcts). For seven infarcts, the source of the emboli (six) or thrombus (one) remained undetermined. Three other infarcts from the earliest patients of this series were due to a dissecting VA aneurysm secondary to traumatic VA puncture during angiography. The sites of the occlusions noted in Figure 1 are summarized in Table 2. For only six infarcts a single occlusion of one cerebellar artery occurred; the six other infarcts with cerebellar artery occlusion were associated with VA or BA occlusions. Among the 38 infarcts associated with VA occlusion, the vessel was occluded only in the intracranial part in 31 instances, in both the intracranial and intraspinal parts in two instances, along the entire length in three instances, and along the entire length with an old occlusion of the contralateral ostium in two instances. Eight of the 38 infarcts were due to occlusion of both VAs, six of the eight to bilateral distal VA occlusion. Among the 38 infarcts, the VA was occluded below the PICA ostium in seven, at it in 21, and above it in eight; in the remaining two infarcts, the occlusion involved the VA below the PICA ostium on one side and above it on the other side. Among these 38 infarcts, the eight involving only the AICA territory were more often due to occlusion above the PICA ostium (seven infarcts) than to occlusion at the ostium (one infarct with an old occlusion); the nine involving only the PICA territory were often due to VA occlusion at the PICA ostium (six infarcts); the three infarcts involving only the SCA territory were due to occlusion of the distal VA, below the PICA ostium sometimes in association with a contralateral healed occlusion at the VA ostium. The sites of the occlusions are given in Table 3 by cause.
Two reasons may account for this discrepancy. Between cases with occluded and stenotic cerebellar arteries. In the 28 cases with cerebellar infarcts associated with occluded cerebellar arteries (12 of 88) was smaller than that found by Rodda (12 of 21) based on postmortem angiograms of the main trunks, branches, and anastomotic network. Two reasons may account for this discrepancy. In addition to the shortcomings of postmortem angiography, Rodda made no clear-cut distinctions of arterial occlusion and at least one occlusion is severe atherosclerosis of the intracranial vessels (stenosis of ≥75%). Of the nine remaining infaracts, five from the earliest patients of this series were due to a dissecting VA aneurysm secondary to traumatic VA puncture during angiography and four had no recognized cause.

In summary, for the 88 cerebellar infarcts, atherosclerosis was the cause in 31 (35%), a proven or presumed cardiac embolism in 38 (43%), and a dissecting aneurysm in eight (9%). For 11 infarcts (13%), no significant conditions other than atherosclerosis and a cardiac source of emboli were found.

**Discussion**

Cerebellar infarcts were predominantly associated with occlusions of the VA and BA. Our percentage of cerebellar infarcts associated with occluded cerebellar arteries (12 of 88) was smaller than that found by Rodda (12 of 21) based on postmortem angiograms of the main trunks, branches, and anastomotic network. Two reasons may account for this discrepancy. In addition to the shortcomings of postmortem angiography, Rodda made no clear-cut distinctions between cases with occluded and stenotic cerebellar arteries. In the 28 cases with cerebellar infarcts reported by Sypert and Alvord, the VA was occluded in 18 (66%) and the PICA was occluded in 10 (35%), but the BA was not occluded in any patient.

In our series, the VAs were most often (82%) occluded only in their intracranial part. These results are in accordance with those of most series, except the series of Hutchinson and Yates, who emphasized the predominant involvement of the cervical segment of the VA in a pathologic study of four cases with cerebellar infarcts. In our patients, whatever the topography of the cerebellar involvement (rostral or caudal), the VA was occluded more often below or at the PICA ostium (28 of 38) than above it (eight of 38). Thus, some infarcts could have a hemodynamic mechanism, although embolism cannot be completely excluded. The consequences of bilateral distal VA occlusions could also support the hemodynamic hypothesis since in eight instances one of the two occlusions was located below the PICA ostium, causing SCA infarcts and AICA infarcts. There were also five cerebellar infarcts among the six patients with bilateral distal VA occlusions reported by Caplan, all of whom had at least one occlusion below the PICA ostium. It is possible that when both VAs are occluded and at least one occlusion is located below the PICA ostium, the decrease in blood flow may not be compensated for by the anterior circulation. This is not the case when occlusion occurs above the PICA ostium. This explains

**Table 3. Site of Arterial Occlusion by Cause for 38 of 50 Cerebellar Infarcts for Which the Cause Was Determined**

<table>
<thead>
<tr>
<th>Cause</th>
<th>n</th>
<th>Distal vertebrobasilar artery</th>
<th>Basilar artery</th>
<th>Proximal</th>
<th>Entire length</th>
<th>Distal</th>
<th>PICA/SCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerotic thrombus</td>
<td>26</td>
<td>12</td>
<td></td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Cardiac source of emboli</td>
<td>12</td>
<td>6</td>
<td></td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

PICA/SCA, posterior inferior cerebellar artery/superior cerebellar artery.

For the 38 infarcts in the remaining 24 patients, there was no arterial occlusion in the vertebrobasilar system at postmortem examination (Figure 2), but 26 infarcts (68%) were associated with a cardiac source of emboli (Figure 2, Table 4) and three (8%) with severe atherosclerosis of the intracranial vessels (stenosis of ≥75%). Of the nine remaining infarcts, five from the earliest patients of this series were due to a dissecting VA aneurysm secondary to traumatic VA puncture during angiography and four had no recognized cause.

**Table 4. Nature of Cardiac Source of Emboli for 26 Cerebellar Infarcts Without Arterial Occlusion at Postmortem Examination**

<table>
<thead>
<tr>
<th>Cardiac source of emboli</th>
<th>SCA (n=16)</th>
<th>AICA (n=2)</th>
<th>PICA (n=8)</th>
<th>Total (n=26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AF</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Acute MI</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Acute MI+ECA embolism</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Acute MI+mural thrombus</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Atrial thrombosis</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Mitral stenosis+AF</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Calcified aortic stenosis+AF</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Aortic disease+AF+atrial thrombosis</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Aortic prosthesis+ICA embolism</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Endocarditis+MCA embolism</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Left ventricular enlargement+ICA embolism+hemorrhagic infarction</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

SCA, superior cerebellar artery; AICA, anterior inferior cerebellar artery; PICA, posterior inferior cerebellar artery; AF, atrial fibrillation; MI, myocardial infarction; ECA, external carotid artery; ICA, internal carotid artery.
why the rostral cerebellum may be spared in cases of BA occlusion such as in the series of Kubik and Adams,26 in which only nine of 18 patients had cerebellar involvement.

Although a hemodynamic mechanism could be involved in some cases, we found no watershed cerebellar infarcts14-16 as reported by others,27-29 which may be due to the size of the infarcts selected in our series (>2 cm). The 19 cerebellar infarcts <1.5 cm in diameter reported by Rodda12 lay in the border zones of either the SCA or PICA or the left and right SCAs. The size of the infarcts, the pattern of the occlusions, and the mechanism of infarction in our series were quite different from those in the series of Rodda.12

In cases with unilateral or bilateral VA occlusion associated with infarction in the territories of the SCA (three infarcts) or AICA (eight infarcts) or the SCA and PICA (13 infarcts), there was no evidence of artery-to-artery embolism to the SCA or AICA. However, this mechanism cannot be excluded since embolism from the occluded VA or lower BA was found in four associated PCA infarcts and was possible in eight others. This is in accordance with several reports.13,30-32 Among the six cerebellar infarcts associated with ulcerative plaques of the aortic arch, there was a vertebrobasilar arterial occlusion in four. Two had atherosclerotic arterial thrombosis and the other two had an embolic outcome without a cardiac source of emboli. These cases could have been due to embolism from thrombus in the aortic arch. In the two other cerebellar infarcts associated with ulcerative plaques of the aortic arch, we found a cardiac source of emboli but no arterial occlusion. Eight cerebellar infarcts were due to dissecting VA aneurysms following angiography. Other mechanisms of arterial dissection associated with cerebellar infarcts have been reported including traumatic dissection33-35 and spontaneous dissection secondary to fibromuscular dysplasia.36,37

We would like to emphasize the high frequency of a cardiac source of emboli in our series, which has been suggested by various others.11,38-40 This could be due to the overrepresentation of some lethal heart diseases, such as endocarditis or myocardial infarction, in our autopsy series. However, our percentage of cases with a proven cardiac embolism (24%) was higher than that found by Castaigne et al13 (9.2%) in a postmortem study of nonselected arterial occlusions in the vertebrobasilar system. In our series, whatever the criteria chosen for cardiogenic embolism (proven [24%] or proven plus presumed [43%]), this mechanism seemed to be more frequent for cerebellar infarction than for other vertebrobasilar infarctions.13,19

We found numerous abnormalities of the vertebrobasilar arterial anatomy (Table 1). There was frequently an anatomic basis for failure of the collateral blood supply from the carotid circulation to the posterior circulation through the posterior communicating arteries and the P1 segment of the PCA. VA abnormalities, such as hypoplasia, origin from the aortic arch, or termination in the PICA, were more frequent than abnormalities of the BA or the cerebellar arteries. On the whole, an abnormal arterial disposition was found for 49 of 88 infarcts. Although this high frequency may deserve mention, we do not know of a similar study in age- and sex-matched patients without cerebellar infarction.

In accordance with previous reports,13-26 we found a good correlation between the site and cause of arterial occlusions (Table 3); involvement of the proximal BA was due only to atherosclerotic thrombus, and there was no atherosclerotic occlusion of the distal BA. Emboli were arrested in the distal VA or the distal BA. On the other hand, the distal VA was the site of half of the atherosclerotic and embolic occlusions. Occlusion of the cerebellar arteries was mainly due to atherosclerosis. Almost half of the multiple cerebellar infarcts were due to embolism and a quarter to atherosclerosis.

We conclude that cerebellar infarcts are mainly due to occlusions of first, the intracranial VA and second, the BA. The mechanism of occlusion is more often cardiogenic embolism than atherosclerosis. This might help in planning investigations of cerebellar infarction.

References


**KEY WORDS** • cerebral arteries • cerebral infarction • cerebellum
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