A clinicopathological study of dissecting aneurysms of the intracranial vertebral artery

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Five autopsied cases of dissecting aneurysms of the intracranial vertebral artery are reported and the literature is reviewed to clarify the clinicopathological correlations. In an autopsy series of 110 patients with subarachnoid hemorrhage (SAH), the incidence of this entity was 4.5% with all five cases progressing rapidly to death from massive SAH. Cases of intracranial vertebral dissection can be divided clearly into two groups based on the clinical and pathological features. In the first group, the dissection is confined to the vertebral artery and a massive SAH develops caused by the rupture of the arterial wall. The plane of dissection is mainly subintimal. In the second group, brain-stem infarction develops resulting from luminal occlusion by intramural hematoma. The plane of dissection is mainly subintimal with the dissection extending to the basilar artery. The condition in the second group affects patients at a younger age. If the lesion is localized within the vertebral artery and does not extend to the basilar artery, the disease seems not to be fatal. The clinical features of the vertebral dissection are largely determined by the plane and extent of dissection. Vertebral artery dissection is due to many causative factors including hypertension, congenital or degenerative changes in the arterial wall, and anatomical and pathological characteristics of the vertebral artery.

Key Words • aneurysm, dissecting • vertebral artery • subarachnoid hemorrhage • brain-stem infarction

Intracranial dissecting aneurysms have been reported with increasing frequency and are recognized as a common cause of stroke. Dissecting aneurysms of the carotid arterial system occur in young individuals and are often associated with cerebral infarction resulting from arterial stenosis or occlusion; however, those of the intracranial vertebrobasilar system, especially the vertebral artery, have been noted to produce subarachnoid hemorrhage (SAH). In recent years the angiographic diagnosis and surgical treatment of dissecting aneurysms has been discussed, but reports of autopsied cases are few, and our knowledge of these lesions is still lacking.

In this report, we present five cases of dissecting aneurysms of the vertebral artery presenting with SAH and examined at autopsy. We review the literature in order to clarify the clinicopathological correlations in these lesions. The pathogenesis of the dissection is also discussed.

Clinical Material and Methods

In our hospital, 110 patients with SAH came to autopsy between January, 1980, and December, 1990. Among these, five patients were diagnosed as having dissecting aneurysms of the intracranial vertebral artery. Therefore, the incidence of these lesions among patients with SAH was 4.5%. Of the 110 patients, 39 died soon after hospitalization and before angiography could be performed. Three of these 39 patients were found at autopsy to have vertebral artery dissection. The remaining 71 patients underwent angiography before death and two dissections were found among these. No cases with a dissecting aneurysm of the vertebral artery presenting with brain-stem or cerebellar infarction were found.

Case Reports

Case 1

This 37-year-old man with unsuspected hypertension complained of a slight headache in the occipital region that had begun on the morning of admission. During the night prior to his admission, after drinking alcohol, he felt dizzy and had a severe headache followed by loss of consciousness. On admission, he was comatose and decerebrate.

Computerized tomography (CT) scanning showed...
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Case 1

A: Computerized tomography scan revealing severe bleeding in the basal cistern. B and C: Postmortem photographs of the circle of Willis (B) showing the location and a close-up of the lesion (C). D: Photomicrograph of the lesion showing the point of rupture. Three layers of the arterial wall are destroyed at the rupture point and an intramural hematoma is seen in the outer media and/or between the media and adventitia. Toluidine blue-safranin, x 13. E: Photomicrograph showing that the media is thin at the proximal portion. Toluidine blue-safranin, x 28.

Fig. 1. Case 1. A: Computerized tomography scan revealing severe bleeding in the basal cistern. B and C: Postmortem photographs of the circle of Willis (B) showing the location and a close-up of the lesion (C). D: Photomicrograph of the lesion showing the point of rupture. Three layers of the arterial wall are destroyed at the rupture point and an intramural hematoma is seen in the outer media and/or between the media and adventitia. Toluidine blue-safranin, x 13. E: Photomicrograph showing that the media is thin at the proximal portion. Toluidine blue-safranin, x 28.

severe bleeding in the basal cistern with ventricular reflux (Fig. 1A). Following the CT scan, he suddenly stopped breathing and was intubated. He did not regain spontaneous respiration and a second CT scan taken the next day showed an increase in the SAH. He died two days after the onset of the illness.

At autopsy, the patient's heart showed mild concentric hypertrophy of the left ventricle. Hypostatic pneumonia, a fatty liver, and gastrointestinal bleeding were seen. There was mild atherosclerosis of the aorta and the coronary arteries.

The brain showed massive fresh SAH in the basal cistern with ventricular reflux and marked evidence of increased intracranial pressure (ICP); flattened gyri, closed sulci, and bilateral tonsillar herniation. Macroscopically, atherosclerosis of the circle of Willis was not seen. In the left vertebral artery, a 9-mm segment distal to the posterior inferior cerebellar artery (PICA) and 12 mm proximal to the union was dark brown and showed a fusiform distention (Fig. 1B and C). Transverse serial sections of the abnormal segment revealed an intramural hematoma with destruction of three layers of the vessel wall. The hematoma was located mainly in the outer media and/or between the media and adventitia, and ruptured into the subarachnoid space (Fig. 1D). The media was thin at the level of the intimal tear and adjacent proximal and distal portions (Fig. 1E).

Case 2

This 63-year-old man with cardiomegaly suddenly developed vertigo and headache in the occipital region while riding a bicycle. On admission, he was sleepy, but oriented. He complained of a severe headache and vomited several times. His admission CT scan showed blood in the basal cistern. Two hours after admission he suddenly became comatose and developed respiratory arrest; he was intubated and regained respirations 10 minutes later. Repeat CT showed an increase in the
SAH and his spontaneous respirations ceased again. Angiography performed under artificial respiration showed a fusiform dilatation of the right vertebral artery with luminal narrowing proximal and distal to the dilatation (Fig. 2A). Subsequently, bilateral ventricular drainage was performed, but the patient did not regain spontaneous respirations and died on the 4th day after the onset of his illness.

At autopsy, the patient’s heart showed concentric hypertrophy of the left ventricle. Hypostatic pneumonitis and mild gastrointestinal bleeding were seen and there was mild atherosclerosis of the aorta.

There was massive SAH in the basal cistern of the brain with ventricular reflux and marked evidence of increased ICP. Atherosclerosis of the circle of Willis was mild. The right vertebral artery showed fusiform distention for a distance of 23 mm. The lesion was dark brown and lay 18 mm proximal to the union and distal to the PICA (Fig. 2B and C). Histological examination showed that three layers of the vessel were destroyed and an intramural hematoma was present, mainly located in the outer media and/or between the media and adventitia (Fig. 2D). No degenerative changes in the vessel wall were observed.
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Case 3

This 53-year-old hypertensive man had complained of slight occipital pain 2 days before becoming unconscious on the toilet. After admission, he had a cardiac arrest and resuscitation was performed. Computerized tomography revealed massive SAH in the basal cistern with mild ventricular reflux. He showed no improvement, and died on the 3rd day after admission.

At autopsy, the patient's heart showed concentric hypertrophy of the left ventricle. Hypostatic pneumonia and mild gastrointestinal bleeding were observed. There was mild atherosclerosis of the aorta and the coronary arteries.

The brain showed fresh massive SAH and marked evidence of increased ICP. Atherosclerosis of the circle of the Willis was mild. A 10-mm segment of the right vertebral artery 12 mm proximal to the union exhibited fusiform distention (Fig. 3A and B). Histologically, three layers of the vessel wall were destroyed and an intramural hematoma was seen in the outer media and/or between the media and adventitia (Fig. 3C). No other abnormality was seen in the vessel wall.

Case 4

This 49-year-old man with a history of angina pectoris was found unconscious on the toilet on the morning of the day of admission. He was confused and complained of a severe headache. During neurological examination, he suddenly became dyspneic and comatose and was intubated. Computerized tomography showed blood in the basal cistern. Angiography performed under controlled respiration showed a berry aneurysm of the right vertebral artery between the PICA and the union (Fig. 4A). The lumen proximal and distal to the aneurysm was somewhat irregular and stenotic. Contrast medium was retained in the aneurysm until the venous phase. He was treated conservatively without any improvement and died on the 11th day of hospitalization.

At autopsy, the patient's heart showed concentric hypertrophy of the left ventricle and old myocardial infarct. Hypostatic pneumonia and gastrointestinal bleeding were observed. Atherosclerosis of the aorta and the coronary arteries was severe for his age.

The brain showed massive fresh SAH in the basal cistern with ventricular reflux and marked evidence of increased ICP. Atherosclerosis of the circle of the Willis was moderate. A saccular aneurysm of the right vertebral artery was seen 21 mm proximal to the union and distal to the PICA (Fig. 4B and C). A large intramural hematoma was found mainly between the media and adventitia. The adventitia was distended and three layers of the vessel were completely destroyed. Atherosclerotic thickening of the intima, disruption of the internal...
Case 4

Serial transverse sections of the distal (A) and proximal (B) portions of the lesions, and photomicrographs of the distal portion (C), point of rupture (D), and proximal portion (E) showing atherosclerotic thickening of the intima, disruption of the elastic lamina, and thinning of the media at the proximal portion. The adventitia is distended. Toluidine blue-safranin: ×6 (C), ×4 (D), ×6 (E).

Case 5

This 55-year-old hypertensive man suddenly became unresponsive while playing pinball and was transferred to our hospital. On admission, he was semicomatose and showed a right flaccid hemiparesis. Computerized tomography after neurological examination showed a severe SAH in the basal cistern. Soon after, he suddenly suffered respiratory arrest. He was intubated and treated conservatively without showing any improvement. A second CT the next day showed an increase in the SAH. He died on the 7th day after admission.

At autopsy, the patient's heart showed concentric hypertrophy of the left ventricle. Hypostatic pneumonia of the lungs and slight gastrointestinal bleeding were seen. There was mild atherosclerosis of the aorta and the coronary arteries.

The brain showed massive fresh SAH in the basal cistern with ventricular reflux and marked evidence of increased ICP. Atherosclerosis of the circle of the Willis was mild. A 9-mm dark-brown segment of the right vertebral artery lying 9 mm proximal to the union showed fusiform dilatation (Fig. 6A and B). Histologically, an intramural hematoma was seen in the outer media and/or between the media and adventitia. Three layers of the vessel wall were destroyed. Mild intimal thickening was also seen (Fig. 6C).

Discussion

Incidence

Although dissecting aneurysms of the vertebral artery producing SAH have been reported more frequently in recent years, their true incidence is unclear. In
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TABLE 1

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Side</th>
<th>Medical History</th>
<th>Clinical Symptoms &amp; Signs</th>
<th>Location of Rupture</th>
<th>Plane of Dissection</th>
<th>Condition of Vessel Wall, Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crompton, 1966</td>
<td>44.5</td>
<td>M</td>
<td>rt</td>
<td>?</td>
<td>severe headache, xanthe-chronic CSF</td>
<td>vessel wall</td>
<td>subadventitial</td>
<td>free from atheroma</td>
</tr>
<tr>
<td>Yonas, et al., 1977</td>
<td>57.5</td>
<td>M</td>
<td>rt</td>
<td>?</td>
<td>severe headache, bloody CSF, severe SAH (autopsy)</td>
<td>vessel wall media, adventitia</td>
<td>subadventitial</td>
<td>free from atheroma</td>
</tr>
<tr>
<td>Filz, 1982</td>
<td>58.5</td>
<td>M</td>
<td>lt</td>
<td>migraine</td>
<td>severe headache, coma 2 days later, SAH (autopsy)</td>
<td>3 layers</td>
<td>subadventitial</td>
<td>focal meded necrosis</td>
</tr>
<tr>
<td>Manz &amp; Luessenhop, 1983</td>
<td>57.5</td>
<td>M</td>
<td>rt</td>
<td>hypertension</td>
<td>severe headache, unresponsive, massive SAH (autopsy)</td>
<td>3 layers</td>
<td>subadventitial</td>
<td>cystic median degeneration</td>
</tr>
<tr>
<td>Friedman &amp; Drake, 1984</td>
<td>58.5</td>
<td>M</td>
<td>rt</td>
<td>?</td>
<td>unconsc, SAH on CT, rebleeding days later</td>
<td>3 layers</td>
<td>subadventitial</td>
<td>degenerative media</td>
</tr>
<tr>
<td>Shimoji, et al., 1984</td>
<td>57.5</td>
<td>M</td>
<td>rt</td>
<td>hypertension</td>
<td>severe headache, unresponsive, marked SAH on CT, rebleeding 15 days later</td>
<td>3 layers</td>
<td>subadventitial</td>
<td>disrupted IEL, mucoid degeneration &amp; intimal thickening of media</td>
</tr>
<tr>
<td>Ide, et al., 1986</td>
<td>57.5</td>
<td>M</td>
<td>lt</td>
<td>hypertension</td>
<td>severe headache, unresponsive, marked SAH on CT, rebleeding 15 days later</td>
<td>3 layers</td>
<td>subadventitial</td>
<td>disrupted IEL, mucoid degeneration &amp; intimal thickening of media</td>
</tr>
<tr>
<td>Sasaki, et al., 1991</td>
<td>37.5</td>
<td>M</td>
<td>lt</td>
<td>cardiomegaly (autopsy)</td>
<td>headache, dizziness, unconsc., 10 hrs later massive SAH on CT, rebleeding within 1 day</td>
<td>3 layers</td>
<td>intramedial, subadventitial</td>
<td>thin media</td>
</tr>
<tr>
<td>Case 2</td>
<td>63.5</td>
<td>M</td>
<td>rt</td>
<td>cardiomegaly (autopsy)</td>
<td>headache, vertigo, coma 2 hrs later, massive SAH on CT, rebleeding within 1 day</td>
<td>3 layers</td>
<td>intramedial, subadventitial</td>
<td>free from atheroma</td>
</tr>
<tr>
<td>Case 3</td>
<td>53.5</td>
<td>M</td>
<td>rt</td>
<td>hypertension, cardiomegaly (autopsy)</td>
<td>headache, coma 2 days later, massive SAH on CT, rebleeding within 2 days</td>
<td>3 layers</td>
<td>intramedial, subadventitial</td>
<td>free from atheroma</td>
</tr>
<tr>
<td>Case 4</td>
<td>49.5</td>
<td>M</td>
<td>rt</td>
<td>angina pectoris, cardiomegaly (autopsy)</td>
<td>severe headache followed by coma, SAH on CT</td>
<td>3 layers</td>
<td>intramedial, subadventitial</td>
<td>disrupted IEL, intimal thickening, thin media</td>
</tr>
<tr>
<td>Case 5</td>
<td>55.5</td>
<td>M</td>
<td>rt</td>
<td>hypertension, cardiomegaly (autopsy)</td>
<td>sudden unconsc, massive SAH on CT, rebleeding within 1 day</td>
<td>3 layers</td>
<td>intramedial, subadventitial</td>
<td>mild intimal thickening</td>
</tr>
</tbody>
</table>

* Data reflects cases in Table 2.*

**Summary of 12 reported cases of dissecting aneurysms of the vertebral artery not extending to the basilar artery (Group 1)**

**Classification of Vertebral Artery Dissecting Aneurysms**

A review of the literature has disclosed 17 histologically documented cases of dissecting aneurysms of the vertebral artery. These aneurysms can be divided clearly into two groups; in the first group (Group 1) the dissection is confined to the vertebral artery (Table 1), and in the second (Group 2) it extends into the basilar artery (Table 2). Of the 17 previously reported cases plus our five cases, 12 fell into the first group and 10 into the second.

These two groups demonstrated a variety of differences in their clinical and pathological features. Patients in Group 1 ranged in age from 37 to 63 years, with a mean of 50.4 years, which is similar to the age data of patients with saccular aneurysms; those in Group 2 tended to be younger, ranging in age from 30 to 47 years, with a mean of 38.2 years. This difference in age is of interest. Manz and Luessenhop reported that basilar dissection occurred in a relatively younger pop-
TABLE 2
Summary of 10 reported cases of dissecting aneurysms of the vertebral artery extending to the basilar artery (Group 2)*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Side</th>
<th>Medical History</th>
<th>Clinical Symptoms &amp; Signs</th>
<th>Location of Rupture</th>
<th>Plane of Dissection</th>
<th>Condition of Vessel Wall, Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scholefield, 1924</td>
<td>47, M</td>
<td>rt</td>
<td>syphilis?, hypertension?</td>
<td>dysphagia, dysnea, ataxia, lower cranial nerve paresis; clear CSF</td>
<td>vessel wall</td>
<td>vessel wall</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyland, 1933</td>
<td>42, M</td>
<td>lt</td>
<td>neurosyphilis, advanced cerebral arteriosclerosis (autopsy)</td>
<td>headache, lt hemiplegia, clear CSF</td>
<td>IEL, media</td>
<td>intramedial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wall, et al., 1967</td>
<td>30, F</td>
<td>lt</td>
<td>concussion</td>
<td>abrupt coma, clear CSF</td>
<td>intima</td>
<td>subintimal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nozicka, 1972</td>
<td>40, M</td>
<td>lt</td>
<td>hypertension, 23 yrs of headache</td>
<td>coma, tachycardia, miosis</td>
<td>intima, media</td>
<td>subintimal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Escourolle, et al., 1973</td>
<td>32, M</td>
<td>rt</td>
<td></td>
<td>headache, coma, flaccidity, clear CSF</td>
<td>intima</td>
<td>subintimal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pasquier, et al., 1976</td>
<td>43, F</td>
<td>lt</td>
<td>hypertension, glomerulonephritis</td>
<td>coma, lt hemiplegia, clear CSF</td>
<td>intima, media</td>
<td>subintimal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Takita, et al., 1979</td>
<td>33, M</td>
<td>rt</td>
<td>hypertension</td>
<td>headache, vertigo, l. weakness for 1 hr; severe headache 1 day later, clear CSF; 4 mos later l. hemiparesis, stupor, clear CSF; 48 hrs later abrupt coma (SAH)</td>
<td>3 layers</td>
<td>subadventitial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Martini, 1979</td>
<td>47, M</td>
<td>bilat</td>
<td>no hypertension</td>
<td>abrupt coma, flaccidity, clear CSF</td>
<td>intima, media</td>
<td>subintimal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kulla, et al., 1982</td>
<td>37, M</td>
<td>bilat</td>
<td>hypertension, polycystic kidney</td>
<td>coma, locked-in syndrome, clear CSF</td>
<td>intima, media</td>
<td>subadventitial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yokoyama, et al., 1984</td>
<td>31, F</td>
<td>lt</td>
<td>no hypertension</td>
<td>sudden onset of headache followed by conc dist, clear CSF, abrupt coma (brain-stem hemorrhage) 9 days later</td>
<td>intima, media</td>
<td>subintimal, intramedial</td>
<td>degeneration of media, local thickening of intima</td>
<td></td>
</tr>
</tbody>
</table>

*CSF = cerebrospinal fluid; IEL = internal elastic lamina; SAH = subarachnoid hemorrhage; 3 layers = intimal, medial, adventitial; conc dist = consciousness disturbance.

ulation than vertebral dissection, with a mean of 31.7 years, and that dissection of the carotid arterial system affected a still younger population with a mean age of 17.5 years. Analysis of these data also pointed out a male preponderance in both groups. The side of dissection also differed in the two groups. In Group 1 cases there was a ratio of 3:9 in favor of the right side. In Group 2, the ratio was 5:2 favoring the left side; the remaining three cases in this group involved bilateral vertebral artery dissection.

Intracranial dissecting aneurysms can be divided pathologically according to the plane of dissection. Generally, the dissection occurs between the internal elastic lamina and media, and an intramural hematoma occludes the lumen. In rare instances, the plane of dissection lies within the media or between the media and adventitia, and SAH often develops. In vertebral artery dissections, the plane of dissection differed between the two groups. In Group 1 with the dissection confined to the vertebral artery, an intramural hematoma was located predominantly in the media or between the media and adventitia; arterial layers were completely destroyed in all cases except for those described by Yonas, et al. On the other hand, in Group 2 with the dissection extending into the basilar artery, the plane of dissection was mainly subintimal in six cases, subadventitial in two, and intramedial in one; in one the plane of dissection was within the wall and an intramural hematoma occluded the lumen. Clinical presentations appear to be largely determined by the plane of dissection.

Clinical Presentation

A decided difference in clinical presentation also distinguishes the two groups. All the patients in Group 1 presented with SAH, usually massive; the patients in Group 2 presented with brain-stem ischemia, although they showed no difference in the severity of symptoms including various combinations of headache, vomiting, vertigo, hemiplegia, and consciousness disturbance. Our five Group 1 patients showed a rapidly progressive course. Within several hours after onset, the patients became comatoses and spontaneous respiration ceased. Rebleeding also occurred and was demonstrated in four
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of our patients. In most cases, death was caused by SAH in Group 1 patients and brain-stem infarction in Group 2. One patient in Group 2 developed SAH 4 months after his first brain-stem infarction.

Hypertension

Patients with dissecting aneurysms of the carotid arterial system are rarely hypertensive and few have a history of atherosclerosis. In contrast, most patients with a dissecting aneurysm of the posterior circulation are hypertensive. Friedman and Drake pointed out that nine of their 14 patients with a vertebrobasilar dissecting aneurysm presenting with SAH had hypertension. Most of the patients meeting Group 1 criteria had hypertension or showed cardiomegaly at autopsy, suggesting a history of hypertension while alive. Five of the 10 Group 2 patients were hypertensive. Hypertension seems to be a causative factor contributing to dissecting aneurysms of the vertebrobasilar artery.

Etiology of Dissecting Aneurysms

The etiologies of intracranial dissecting aneurysms are obscure in most reported cases, although these lesions have been associated with syphilitic arteritis, migraine, atherosclerosis, and degenerative disease of the vessel wall. Some investigations have proposed underlying mechanisms responsible for the abrupt tearing of the intima. Yonas et al. reported a case of dissecting aneurysm of the vertebral artery presenting with SAH, in which intramural hematomas were found between the media and adventitia without luminal connection. They pointed out two likely sources for the hematomas: rupture of vasa vasorum or rupture of new vessels formed in response to medial necrosis, as is seen in extracranial dissecting aneurysms. However, this explanation leaves two questions unresolved. First, in all other cases of vertebral dissection presenting with SAH, intramural hematomas were present with luminal connection and all arterial layers were completely destroyed (the case of Yonas et al., is the simple exception). Second, the media and internal layer of the intracranial arteries lack vasa vasorum, except in locations of atherosclerotic lesions, but these pathological features are rarely seen with an intracranial dissection.

The vertebral artery dissections presenting with SAH could not be attributed simply to rupture of vasa vasorum or to rupture of new vessels.

Medial defects are also proposed as causative, but these findings are common and have been found in up to 75% of cerebral arteries from normal brains. Some investigators believe that defects of the internal elastic lamina are responsible for intracranial dissecting aneurysms that occur without another apparent etiology. The internal elastic lamina, which contains the elastic tissue in the intracranial arteries, is the most important layer for determining the strength of the vessel wall. Therefore, the vessel is more prone to damage if the elastic tissue is defective, but whether the defect of the elastic layer is congenital or acquired is unknown. In the vertebral artery dissections, a variety of changes in the arterial wall were seen: defect, fragmentation, or disruption of the internal elastic lamina, medial degeneration, and intimal thickening. A weakened area in the arterial wall caused by defects of the elastic tissue, especially in association with underlying medial abnormalities, may be the origin of an intramural dissection.

It is unclear why the dissection preferentially involves the intracranial portion of the vertebral artery and often produces SAH. However, the anatomical characteristics of the vertebral artery are worthy of note. The vertebral artery joins the contralateral vertebral artery to form the basilar artery and changes in structure as it pierces the dura to enter the skull. These changes include a diminution in thickness of the adventitial and medial coat, and a very great reduction or loss of elastic fibers in the media and external lamina.

The intracranial vertebral artery is also unique pathologically. Stebbens found small fusiform aneurysms located low on the intracranial segments of the vertebral artery, with an incidence of 10.8% in 185 circles of Willis obtained from adult brains. These aneurysms are not necessarily associated with gross atherosclerosis; histologically, fibrosis of the wall, medial thinning, severe elastic-tissue degeneration with intimal thickening, and prominent vasa vasorum are seen. These changes also may be related to the dissection.

Conclusions

We have assumed that causative factors in vertebral artery dissection include various combinations of hypertension, congenital or degenerative changes in the vessel wall, and anatomical and pathological characteristics of the artery. The dissection initially occurs between the intima and media, resulting from the diversion of the arterial stream. Subsequently, all arterial layers are destroyed and SAH develops, especially in older people who have more defects in the vessel wall and a thinner adventitia. On the other hand, in younger persons the dissection tends to be confined within the arterial wall, and brain-stem ischemia develops resulting from luminal occlusion. If the lesion is localized to the vertebral artery, the condition is not fatal; if it extends to the basilar artery or the vertebral artery, death results.

References


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25. Ibid., p 353


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