MEDIAL DEFECTS IN THE MENINGEAL ARTERIES

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The meningeal arteries are of clinical interest from several points of view. In cases of head trauma they may rupture, and epidural or, more rarely, subdural hemorrhages may arise. As they belong to the most sensitive structures of the skull, the meningeal arteries have received much attention in the discussion of the etiology of some types of headache. When the internal carotid artery or any of its main branches (most commonly the middle cerebral artery but also the anterior and posterior cerebral arteries) are occluded, a collateral circulation through small anastomoses (mainly frontal and temporal) to the meningeal arteries may be extremely important for the nutrition of the cerebral parenchyma. In cases of meningioma these arteries supply the tumor with a close network of branches, making operation difficult. The meningeal arteries can be ligated without any complications. Where the middle meningeal artery passes the foramen spinosum and enters the cranial cavity its histological appearance changes from that of a muscular artery to that of an intracranial (cerebral) artery. The most proximal intracranial segment is a most common site for atheromatous changes.

MATERIAL AND METHODS

The middle meningeal artery with its major intracranial branches was investigated bilaterally in 45 necropsies, 5 from each age decade over a lifespan of 90 years. In 9 of the cases, 1 from each decade, the frontal and occipital meningeal arteries also were removed. Dissection took place within 36 hours post mortem. The dura mater in the immediate neighborhood was taken with the arteries, which were fixed in Bouin’s solution. Arterial specimens originating from individuals showing advanced atheromatous changes in the aorta, the coronary or the cerebral arteries were subjected to decalcification. Such preparations were placed in a mixture of formic acid and sodium citrate for 48 hours, whereupon they were returned to a 4 per cent neutral solution of formaldehyde for 48 hours.

After fixation the arteries were divided into segments 2 cm. long and embedded in paraffin. Serial sections (20 μ thick) were cut, treated with various stains for elastin (orcein, aldehyde-fuchsian, or resorcin-fuchsin) and counterstained with a general stain (Ludewig, azan, van Gieson, nuclear-fast red, hematoxylin-eosin).

Every medial defect encountered during examination was reconstructed graphically. The area of the defect thus could be easily estimated. If it exceeded 0.3 mm.², the defect was classified as large. If the area was between 0.03 and 0.3 mm.², it was classified as small. Defects with an area less than 0.03 mm.² were excluded.

RESULTS

As shown in Fig. 1 a total of 19 large and 45 small medial defects were found, using the criteria set up under Methods.

The distribution among individuals of different age groups is given in Table 1. The defects were distributed similarly in relation to sex. In 6 of the individuals with large or small medial defects a weight of heart exceeding 400 g. and a statement of enlarged left side of the heart were recorded in the postmortem report. No relationship could be detected between the presence of defects and clinical signs, with the exception of hypertonia which seemed to be commoner in individuals with a defect than in those with none. All defects but one were located at points of branching and at the presum-
TABLE 1

Distribution of medial defects among different age groups

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th></th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0-30</td>
<td>31-60</td>
<td>61-90</td>
<td></td>
</tr>
<tr>
<td>Large defects</td>
<td>5</td>
<td>7</td>
<td>9</td>
<td>19</td>
</tr>
<tr>
<td>Small defects</td>
<td>12</td>
<td>16</td>
<td>17</td>
<td>45</td>
</tr>
<tr>
<td>At least one large defect</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>13</td>
</tr>
<tr>
<td>At least one small defect, but no large one</td>
<td>3</td>
<td>8</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>No large or small defect</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Total number of individuals</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>45</td>
</tr>
<tr>
<td>Large and small defects per individual</td>
<td>1.0</td>
<td>1.5</td>
<td>1.7</td>
<td>1.4</td>
</tr>
</tbody>
</table>

Fig. 1. Diagram to illustrate the middle meningeal artery, showing the locations of classified medial defects in 45 subjects. The total number of defects is given before the parentheses, in which the number of large defects is added to the number of small defects.

ably congenitally weak line of junction between the trunk and the branch. Most of the defects were located at the distal carina.

The edge of the media was rounded off gradually, as reported in the cerebral arteries by previous investigators. The media showed generalized moderate fibrosis, which was not accentuated in the neighborhood of

the defect. The maximum diameter of the largest defect was slightly more than 1.5 mm. At the edge of one defect a clump of eosinophilic substance was observed.

The intima over the media-defective areas of the wall was often thickened considerably (Fig. 2) and contained an abundance of chromatotropic substance. The elastic lamina sometimes was thickened, with increased staining properties and fragmentation. Windows were observed in the lamina over the medial defects. The adventitia often showed increased thickness over the medial defect, and its collagenous fibres also seemed to be more densely packed over the defect. Thus, in most cases, the arterial wall was neither thinner over the medial defect,
nor did it seem to be weakened. There were some exceptions, however, in which the intima was not thickened and/or the adventitia was thinner than the surrounding parts of the wall, so that the total thickness of the arterial wall was diminished considerably over the medial defect. Three medial defects showed the extreme thinning out of the arterial wall commonly seen in the cerebral arteries.

Some of the thin-walled defects faced the dura mater or the bone but others gave upon the subdural space, which was the case with two of the three most thin-walled defects mentioned above. These defects were separated from the subarachnoid space only by a thin, loose layer of collagenous connective tissue. At the site of the medial defects the values recorded of the total thickness of the arterial wall ranged between 30 and 600 μ.

DISCUSSION

Medial defects in the cerebral arteries have been described by several investigators. The etiology of these defects is not known. When Forbus\textsuperscript{3} first described them he believed that they were congenital and many authors after him have accepted this view. On the other hand Glynn\textsuperscript{5} as well as Stehbens\textsuperscript{13} observed a much lower incidence of medial defects in children than in adults. Hassler\textsuperscript{7} thought that the defects probably grow during life and suggested that Glynn's and Stehbens' lower figures of incidence might be explained by a strong postnatal growth of exceedingly small congenital defects.

Even supposing the defects of the cerebral arteries are formed prenatally, the way in which they arise is not known. Bremer\textsuperscript{4} suggested that no media is formed around those points of bifurcation where the adjacent walls of the branches support each other. Instead, the defects develop when the branches spread out in connection with the growth of the brain. This hypothesis has been questioned by Krauland\textsuperscript{8} and others, who pointed out that the defects do not occur exclusively in the acute angle at the point of bifurcation. However, the complicated embryogenesis and postnatal growing process of the brain probably allow for further speculation concerning a developmental origin of the medial defects. The findings of medial defects in the meningeal arteries, which have another pre- and postnatal development but almost the same histological appearance, may be of interest in this connection.

The pathological significance of the medial defects in the cerebral arteries lies to a large extent in the fact that the common saccular aneurysms develop at their sites. But saccular aneurysms are extremely rare in the meningeal arteries, although, as is shown by the present investigation, medial defects are common at this location. There is thus a distinct difference between the medial defects of the cerebral arteries and those of the meningeal arteries concerning their tendency to form aneurysms. The main reason for this probably lies in the fact that the arterial wall at the site of the defect generally is thicker in the meningeal arteries than in the cerebral arteries. The existence of firmer tissues around the meningeal arteries than around the cerebral arteries also is probably significant. However, these explanations do not account for the extremely thin-walled defects separated from the subarachnoid space only by a very thin, loose collagenous layer. Another contributory factor may be that the meningeal arteries are situated further distally from the heart and have a slower blood flow than the cerebral arteries. The meningeal arteries also may have still weaker pulsations than the cerebral arteries because they are branches from muscular arteries.

When the etiology of unexplained cases of epidural, subdural and subarachnoid hemorrhage is discussed the possibility of a rupture of a thin-walled medial defect in the meningeal arteries ought to be borne in mind.

SUMMARY

In 45 necropsies medial defects were found to be common in the meningeal arteries. Usually the arterial wall over the defects was not very much thinned out, but in some cases it was almost as thin as in the most thin-walled medial defects of the cerebral
arteries. On the basis of the findings the etiology of medial defects is discussed as well as the reason why sacculare aneurysms do not form in the medial defects of the meningeal arteries, which have almost the same general histological appearance as the cerebral arteries. The somewhat greater thickness of the arterial wall over the medial defect and the fact that the surrounding tissues provide a stronger support of the arterial wall are considered to be of great importance, but probably are not the whole explanation. Ruptures of thin-walled medial defects in the meningeal arteries may cause the bleeding in etiologically unexplained cases of epidural, subdural and subarachnoid hemorrhage.

REFERENCES