Renovascular hypertension is counted among the surgically curable hypertensions, and occupies from 5 to 20 per cent of all hypertensions (UENO et al. 1965). Its mechanism is the same as the Goldblatt hypertension (GOLDBLATT et al. 1934), whose vascular lesion can gradually advance, until it becomes refractory to all surgical procedures, if it is left untreated. These circumstances will be greatly clarified by the histological studies of the renal biopsy specimens.

Materials and Methods

Bilateral renal wedge biopsy specimens were obtained from seven renovascular hypertensive patients, including four cases of unilateral, two cases of bilateral main renal artery stenosis and one case of renal infarction. These three kinds of lesions can be diagrammatically shown as in Figure 1. Each of them is the case operated upon in our clinic, and the lesions were certified directly under vision during operation. Treatment consisted of renal revascularization or resection of infarction. The specimens were immediately fixed in 10% formalin.

The control renal materials were obtained regardless of side from those autopsy cases, that were considered free from both circulatory and renal diseases, and had been studied formerly by the author in the clinicopathological studies on the aneurysm of the abdominal aorta (SEKI 1964). They range from newborn to eighth decade of age, and each decade contains four cases. The specimens, thirty-six in all, were fixed in Zenker-formol.

Paraffin sections, both clinical and control, were cut at 3.5 μ, and stained with hematoxylin-eosin and elastica-Masson. Studies were made of the external and internal diameter of the renal cortical artery or arteriole and of its histological state of the intima, the internal...
elastic membrane and the medial muscular layers. At least forty arteries per section were examined.

Results

The external and internal diameter of normal renal cortical arteries are graphically shown in Fig. 2. They tend to be distributed along straight line with definite gradient of about 50 degrees, and can be divided into several groups according to their histological constituents. These groups are encircled by a line in the Figure. Group A has only scanty elastic tissue and one layer of medial muscle. Group B has one layer of internal elastic membrane and sometimes two layers of medial muscle. Group C has a firm elastic layer and sometimes three layers of medial muscle. Group D often shows duplication of elastic membrane and has three or more medial muscular layers.

Because of the size of the biopsy specimens, studies were restricted to those arteries measuring up to about 100µ in external and 70µ in internal diameter. Group A seems to

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**Fig. 2.** External and internal diameter of normal renal cortical arteries. Note the distribution along straight line with definite gradient. The explanation of the encircled groups is in the text.
correspond to the arterioles, and the other groups to the interlobular arteries and their branches. The diameter of the arteries in collapsed state in this study differs greatly from that in expanded state of More and Duff’s study on the renal arterial neoprene casts (More et al. 1951), who showed the width of the lumen of the afferent arteriole and the interlobular artery to be 29.8 μ and 60.7 μ respectively. This difference can be attributed to the methods preparing the materials. The histological state of the arteries revealed the following facts. The renal cortical arteries develop until the third decade of age, and then degenerate gradually, showing slight disruption of the internal elastic membrane in the fifth decade of age, which becomes evident in the seventh decade of age. A little intimal thickening is seen in the sixth decade of age, but rather of rare occurrence. In the eighth decade of age, the changes are accompanied by elastic duplication and medial muscular atrophy. These observations are generally more obvious in the interlobular arteries than in the arterioles.

The results obtained from the biopsy specimens of renovascular hypertensive patients are shown in the same way in Figures 3, 4 and 5. Each clinical data are summarized in Table 1.

Table 1. Clinical data of renovascular hypertensive patients.

<table>
<thead>
<tr>
<th>Case</th>
<th>Disease</th>
<th>Age</th>
<th>Sex</th>
<th>Duration of Hypertension</th>
<th>Chief Complaint</th>
<th>Blood Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Preop.</td>
</tr>
<tr>
<td>1</td>
<td>Unilateral main renal artery stenosis</td>
<td>23</td>
<td>F</td>
<td>More than 4 months</td>
<td>Headache, Nausea</td>
<td>180/130</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>25</td>
<td>F</td>
<td>More than 16 months</td>
<td>Tinnitus, Diminution of vision</td>
<td>200/120</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>27</td>
<td>F</td>
<td>More than 20 months</td>
<td>Headache</td>
<td>230/140</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td>25</td>
<td>M</td>
<td>More than 26 months</td>
<td>None</td>
<td>184/128</td>
</tr>
<tr>
<td>5</td>
<td>Bilateral main renal artery stenosis</td>
<td>16</td>
<td>F</td>
<td>More than 12 months</td>
<td>Diminution of vision</td>
<td>220/100</td>
</tr>
<tr>
<td>6</td>
<td></td>
<td>27</td>
<td>F</td>
<td>More than 84 months</td>
<td>Palpitation</td>
<td>144/92</td>
</tr>
<tr>
<td>7</td>
<td>Renal infarction</td>
<td>26</td>
<td>M</td>
<td>More than 12 months</td>
<td>Headache</td>
<td>176/92</td>
</tr>
</tbody>
</table>

* Operative death.

It is clear that in the cases of unilateral main renal artery stenosis (Fig. 3), there is more or less difference between the histological pictures of the left and right renal cortex. They distribute along straight lines with different gradient according to side. Namely in the stenosed side the gradient is about 50 degrees, and in the unstenosed side the gradient is about 55 degrees or more, indicating that the latter has a tendency of luminal narrowing or thickening of the vascular wall. The histological picture of the renal cortical arteries is also different according to side. In the stenosed side they show no significant change, but in the unstenosed side precocious arteriosclerotic changes of Heptinstall’s I or II group can be recognized (Heptinstall 1954). These changes are also more obvious in the interlobular arteries than in the arterioles, and consist of intimal thickening, elastic disruption (Cases 1, 3 and 4) or, in the case with the longest duration of hypertension, hyalinized lesions of the arterioles and glomeruli (Case 4). The change may be limited only to medial muscular hypertrophy, which can
Case 1. Group A' has little change, but group B' shows elastic duplication and disruption.

Case 2. Groups A', B', C' and D' show only medial muscular hypertrophy.

Case 3. Group A' has little change, but group B' shows intimal thickening and slight elastic disruption.

Case 4. Group A' shows sometimes hyaline degeneration of the arteriole. Groups B', C' and D' show intimal thickening, elastic duplication and disruption.

Fig. 3. External and internal diameter of renal cortical arteries in unilateral main renal artery stenosis. The groups of stenosed side are almost normal in all cases. Solid circles bounded by solid line: Stenosed side. Open circles bounded by interrupted line: Unstenosed side.

hardly be distinguished from normal state in smaller specimens, and which can be reversible and respond well to surgery (Case 2). In all cases no renal parenchymal change was recognized.

In the cases of bilateral main renal artery stenosis (Fig. 4), on the contrary, no difference can be seen according to side, and their histological picture was almost normal with no parenchymal change whatever. This is very surprising, considering that the patient had hypertensive retinopathy (Case 5) or long duration of hypertension (Case 6).

In the case of renal infarction (Fig. 5) the renal cortical arteries show occlusion and recanalization in the infarcted side and elastic duplication and disruption in the other side,
so that the distribution curve shows narrowing tendency of either side.

Discussion

Vertes and his collaborators (1963, 1964 and 1965) suggested in their studies the importance of renal biopsy in the treatment of renovascular hypertension. The findings of the present study indicate that the renal cortical arteries of hypertensive patients due to main renal artery stenosis are almost normal in the stenosed side and more or less arteriosclerotic in the unstenosed side. Similar facts were mentioned by Saphir and his collaborator (1940) or Aronson and his collaborator (1951) on the autopsy cases and by Morris and his collaborators (1960) or Thal and his collaborators (1963) on the clinical cases. These characteristics seem to be attributed to the protective action of the stenosis against the blood pressure, and can
produce some discrepancy between the results of renal split function test and the true localisation of main renal artery stenosis. Considering that the kidney of stenosed side can become better one, and that the histological changes of the renal cortical arteries in the unstenosed side may be so slight as reversible, renal revascularization is, if possible, recommended, and the postoperative changes of the blood pressure should be observed. But there can be also rare exceptional cases in the above-mentioned characteristics (ALLEN 1951) or complicated cases with renal thrombosis or infarction, so the diagnosis of renovascular hypertension must be made cautiously referring to the clinical history, physical examination, laboratory data, renal angiography or split function studies (MELTZER 1965).

Summary

Histological studies on the renal cortical arteries of renovascular hypertensive patients, including four cases of unilateral, two cases of bilateral main renal artery stenosis and one case of renal infarction, were made on wedge biopsy materials, referring to thirty-six normal cases. The renal cortical arteries proved to be almost normal in the stenosed side and more or less arteriosclerotic in the unstenosed side. These characteristics can disturb the results of renal split function test or others. The changes are generally more obvious in the interlobular arteries than in the arterioles, and may partly be reversible. Moreover because there can be rare exceptional or complicated cases, the diagnosis and treatment of renovascular hypertension must be made cautiously.

Grateful acknowledgement is made to Dr. Akira UENO for his suggestions in this investigation.

References


