Tissue Microvenography of the Adrenal in Cirrhosis with Portal Hypertension

NOBUAKI SASANO, NORIKO HORIKAWA and KATSUHIRO NAKAMURA

Department of Pathology, Tohoku University School of Medicine, Sendai

SASANO, N., HORIKAWA, N. and NAKAMURA, K. Tissue Microvenography of the Adrenal in Cirrhosis with Portal Hypertension. Tohoku J. exp. Med., 1974, 112 (3), 197-203 — The adrenal venous system demonstrated by the postmortem tissue microvenography varied with the length of clinical course of portal hypertension. Veins passing vertically through the cortex, e.g., the emissary and corticomedullary veins, increased remarkably in number without noticeable dilatation in chronic cases. The venous tree dilated and proliferated in the periadrenal tissue communicating with the capsular vein. In fulminant cases, the original emissary and corticomedullary veins dilated severely and irregularly without increase in number. Signs of the hepato-adrenal collateral circulation were severer in the right gland than in the left. — emissary veins; corticomedullary veins; hepato-adrenal collateral circulation

A considerable number of reports dealing with adrenal changes associated with liver cirrhosis have taken almost no notice of the venous communication between adrenals and liver. This venous relation, however, is conceived to be significant in exerting a vital influence on the adrenal circulation and function as a whole. The human adrenal venous system is different from that of dog in the following features (Dempster 1974); 1) the presence of an emissary vein, 2) the muscular content, and 3) a venous communication with the liver. Sasano et al. (1971) have demonstrated the above venous communication in the right adrenal gland. This study concerns postmortem microvenography of the adrenals in cases with liver cirrhosis, in an attempt to throw light on the adrenal-liver communication with special reference to changes in the adrenal venous system.

MATERIALS AND METHODS

Materials were the adrenal glands obtained from 6 patients with liver cirrhosis within 3 postmortem hours (Table 1). All but one with alcoholic cirrhosis were associated with splenomegaly of about 300 g and ascites of over 1,000 ml as undoubtful evidence of portal hypertension. Other 6 autopsy cases without any of these pathological findings were used as controls.

The adrenals were removed together with sufficient periadrenal tissue including the contiguous part of liver tissue to the right gland. These were perfused with physiological
TABLE 1. Cirrhosis cases

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Clinical course</th>
<th>Liver weight (g)</th>
<th>Spleen weight (g)</th>
<th>Ascites (ml)</th>
<th>Type of cirrhosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>19</td>
<td>F</td>
<td>9 months</td>
<td>950</td>
<td>230</td>
<td>3,000</td>
<td>Postnecrotic</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>M</td>
<td>2 years</td>
<td>1,030</td>
<td>360</td>
<td>2,500</td>
<td>Postnecrotic</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>F</td>
<td>8 years</td>
<td>650</td>
<td>340</td>
<td>5,500</td>
<td>Posthepatic</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>M</td>
<td>?</td>
<td>3,080</td>
<td>290</td>
<td>1,000</td>
<td>Postnecrotic with hepatoma</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
<td>M</td>
<td>5 years</td>
<td>1,480</td>
<td>250</td>
<td>2,000</td>
<td>Postnecrotic</td>
</tr>
<tr>
<td>6</td>
<td>64</td>
<td>M</td>
<td>14 years</td>
<td>1,110</td>
<td>100</td>
<td>—</td>
<td>Alcoholic</td>
</tr>
</tbody>
</table>

saline from the opening of the suprarenal vein. Then 3-5 ml of 6% gelatine solution, containing dissolved Micropaque in a proportion of 60%, were injected at a pressure of 60 mmHg. After fixation in formalin, an ultrasoft X-ray picture of the whole adrenal was taken by “Softex.” Then the gland was made into cross-sectional serial slices 1 mm in thickness and again radiographed of its each slice. The slices were made in paraffin sections for roentgenographic interpretations.

RESULTS

The whole picture of adrenal microvenography usually revealed arborescent veins distinctly even in the terminal. These veins drained into the two thick medullary veins, which converged into the central vein and further formed the extraadrenal suprarenal veins. Such features were indistinguishable between cirrhosis and controls.

On the cross sections of the slices, the control group showed that contrast medium remained mainly in the medulla, infrequently entering the cortex. But, as special veins from the superficial cortical layers to the central vein, the corticomedullary vein was observed along with the emissary vein connecting the central vein with periadrenal veins. In control cases, these special veins were generally a few in number almost with constant thickness and rarely with large one. In the right adrenal en bloc with part of liver tissue, the contrast medium from the adrenal central vein usually demonstrated the central vein of the hepatic lobule.

In cases with portal hypertension, the adrenal venous system was modified in different manners depending on the duration of clinical courses. In chronic cases, the periadrenal veins, showing prominent vascular arborization, were linked with many intracortical veins which run vertically to drain into the central vein (Fig. 1). As compared with the original corticomedullary and emissary veins, the intracortical veins were numerous, particularly in the ala and the tail of the gland (Figs. 2 and 3). But each individual vessel was thin in general in contrast to the thick periadrenal vein. In the right gland en bloc with liver tissue, the venous plexus between the liver and the adrenal was conspicuously demonstrated by detailed tracing of venous communication between the liver and the adrenal parenchyma (Fig. 4).

In acute cases of portal hypertension with shorter but severer clinical courses, veins passing vertically through the cortex showed no increase in number, while
Fig. 1. The periadrenal venous tree (T) originated from the capsular vein (P) proliferates and dilates remarkably. (Case 6)

Fig. 2. Venous drainages from the central vein (C) straightly into the capsule (P) do not show significant dilatation but increase in number. (Case 3)

Fig. 3. The capillary network in the cortex draining into the capsular vein (P) is finely demonstrated and particularly dense in the deeper cortex (D). (Case 3)
Fig. 4. The contrast medium filling the capsular and periadrenal veins (P) in the right adrenal further demonstrates the fine capillary network in the contiguous liver tissue (L). (Case 3)

Fig. 5. The contrast medium filling the emissary (E) and capsular (P) veins drains into the periadrenal venous tree (T). (Case 1)
the original periadrenal, emissary and corticomedullary veins dilated abnormally (Fig. 5). Frequently they enlarged 4 to 5 times the size in controls (Fig. 6). The dilated emissary veins, which were linked with the periadrenal veins, formed occasionally a ball-like dam in the cortex (Fig. 7).

The findings in the adrenal venous system were generally more conspicuous in the right gland than in the left.

**DISCUSSION**

1. *Hepato-adrenal venous communication*

It is generally accepted that the steroid-producing endocrine glands are excluded from the area of portal circulation. The adrenal glands seem to be partly
against this rule having venous communication with the portal system.

The evidence for communication of blood flow between adrenals and liver was first obtained in the dog by Donath (1957), who demonstrated that a fluorescent substance injected into the adrenal appeared in the portal vein in the liver. In the human adult adrenals, the right gland usually intrudes superiodorsally into the liver and the parenchymal fusion may occur, though rarely, between these two organs without any intervening capsular tissue (Dolan and Janovski 1968). Shdanow and Saapin (1968) observed that the network of adrenal capsular veins was drained in two opposite directions: one into veins of the diaphragm, loin, kidneys and retroperitoneal tissue and finally into the inferior caval vein, and one through veins of the spleen and pancreas into the portal vein and directly into veins of the hepatic capsule and parenchyma. In our present experiment, the contrast medium injected via the right suprarenal vein entered into the central veins of the hepatic lobules contiguous to the adrenal.

These experimental results suggest that the venous communication between adrenals and liver through which the two opposite directions of blood flow can occur has some distinctive functional significances.

**Blood flow from the adrenals to the liver:** Since the discovery of the emissary veins, it has been known that the medullary and central veins can partly drain into the capsular veins. Moreover, direct venous communication between adrenals and liver has been verified particularly on the right side. Thus it is quite likely that various adrenocortical and medullary hormones enter directly into the liver.

However, there still remains a question what proportion of the adrenal blood can flow into the liver. For drain into the liver, the venous pressure in the adrenal central vein must be higher than that in the portal system. The blood flow from the adrenal to the portal system in the dog was only possible when the drain into the suprarenal vein was blocked (Donath 1957). In human cases, the pathologic conditions that cause the block of drain into the suprarenal vein are extremely rare.

**Blood flow from the liver to the adrenals:** Venous blood in the adrenal capsule from the zona glomerulosa or via the emissary vein from the medullary or the central vein will naturally flow back toward the central vein when the venous pressure in the capsule rises above normal under some pathologic conditions. The capsular-portal venous communication will play a role of collateral circulation in cases of liver cirrhosis and other diseases associated with portal hypertension.

2. **Microvenography in the adrenals with liver cirrhosis**

In cases of chronic hepatitis or liver cirrhosis, Demura (1962) noted proliferation of the capsular veins of the adrenals which was associated with dilatation of the intracortical veins and hypertrophy of the smooth muscle bundles of the medullary veins. On the basis of these histological findings he suggested blood flow from the capsular to the central vein to be increased. The present authors demonstrated communications between the capsular and the central vein by tissue microvenography.
Our results suggest that the portal hypertension directly causes venous dilatation in the periadrenal tissue, and that this induces abnormal enlargement of the emissary and corticomedullary veins which connect the periadrenal veins with the central and medullary veins. Some artifactual causes can be conceived for the occurrence of dams of the contrast medium in the emissary veins. But such phenomena were extremely rare in the control cases of our series. This finding also suggests the formation of collateral circulation in portal hypertension. In cases with chronic portal hypertension, the venous plexuses grew conspicuously in the periadrenal tissue. They were linked with the central vein via intracortical veins densely distributed everywhere.

These changes in the adrenal venous system were observed more prominent in the right gland than in the left. The reasons for it are probably explained by 1) closer topographical relationship of the right gland to the liver, and 2) more direct venous flow to the inferior caval vein from the right gland. Accordingly, the right adrenal plays more significant roles in collateral circulation of the portal vein than the left gland.

CONCLUSION

In cases of liver cirrhosis with clinical and anatomical signs of portal hypertension, the adrenal venous system participates in the hepato-adrenal collateral circulation. The microvenographic findings of this participation is more conspicuous in the right gland than in the left. The resultant increase in the adrenal blood flow may partially be responsible for adrenal changes such as secondary aldosteronism.

References