Lymphographic Studies on Protein-losing Enteropathy

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Summary

Lymphographic studies were made on diseases of protein-losing in 19 cases, which include 9 cases of diseases of the digestive canal, 2 cases of constrictive pericarditis, and 8 cases of protein-losing enteropathy. Lymphographically in the diseases of the digestive canal no abnormal findings were detected, but in the constrictive pericarditis remarkable dilatation and kinking of the thoracic duct with relatively poor congestion of the lumbar lymphatic vessels were observed.

In the intestinal lymphangiectasia dysplasia of the lymphatics, passage disturbance of the thoracic duct with the congestive lumbar lymphatics, reflux to abdominal organs, oozing of contrast medium into the intestinal lumen were disclosed.

Also experimentally lymphographic studies in thoracic duct-ligated dogs revealed the reflux to the mesenterial lymphatic system causing protein-losing into the intestine.

It is well known that the protein loses from the gastrointestinal tract in some kinds of the gastric and intestinal diseases (7) and constrictive pericarditis (8). The protein-losing enteropathy of unknown etiology has been strongly suspected to be due to abnormalities of the lymphatic system (1, 2, 3, 6).

Therefore lymphographic studies were made on the relationship of the lymphatic system and the diseases with protein-losing in 19 cases, which were 8 protein-losing enteropathy with unknown etiology, 2 constrictive pericarditis, 6 non-specific multiple ulcers, 2 gastric giant rugae and one regional ileitis. They had some symptoms of hypoproteinemia, positive Gordon's test (131 I-PVP absorption test), ascites, edema and intestinal lymphangiectasia (Table 1).

No abnormal change was disclosed in gastric giant rugae, regional enteritis and non-specific intestinal ulcers, which is the ulcer due to unknown etiology except tuberculosis and malignant tumors, but marked dilatation and kinking of the thoracic duct with the increased lumbar lymphatics was observed in constrictive pericarditis.

In the protein-losing enteropathy with unknown etiology (Table 1), two cases were suspected as systemic lymphatic dysplasia due to poor development of the lymphatics of the lower extremities. In one of them an injection of the contrast medium was performed into an inguinal node because the lymphography was impossible due to unvisualized of the lymphatic vessel by color dye.

Stenosis of the thoracic duct proved by lymphography and also verified by autopsy caused remarkable reflux to the mesenteric lymphatics in 4 cases, in one of which the reflux was so remarkable even to the hepatic and the intestinal lymphatics that leaked contrast medium is clearly shown in jejunum (Fig. 1).

In another case with chylothorax and ascites was also suspected stenosis of the thoracic duct due to the dilated lumbar lymphatic vessel. In the other case no apparent abnormal change was observed because of stenotic change of the intestinal lymphatic trunk, which was proved by autopsy but was not stained by ordinary lymphography.

In the thoracic duct ligated dogs lymphogram revealed remarkable congestion of the retroperitoneal lymphatics and reflux to the mesenteric, the hepatic and the renal lymphatics two days after the ligation. Radiogram of the removed intestine revealed clearly the contrast medium in the intestinal lymph vessel. The reflux with congestion was scarcely observed two weeks after the ligation (5).

In conclusion passage disturbance of the thoracic duct and the intestinal trunk due to either congenital or acquired etiology is very important to cause protein-losing enteropathy. It may be classified to non-lymphogenic protein-losing enteropathy secondary to the intestinal disease and lymphogenic protein-losing enteropa-
Table 1 Protein-losing enteropathy with unknown etiology

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Ser. Protein g/dl</th>
<th>I-PVP %/4 ds</th>
<th>Intestinal Ly-ectasia</th>
<th>Ascites</th>
<th>Edema</th>
<th>Lymphographic findings</th>
<th>Thoracic duct Passage</th>
<th>Lumbar Lymphatics</th>
<th>Reflux</th>
<th>Dysplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. K.I.</td>
<td>2 M</td>
<td>3.7</td>
<td>12.3</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Decrease</td>
<td>-</td>
<td>Extremities upper lower Systemic?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. O.I.</td>
<td>32 M</td>
<td>4.0</td>
<td>6.4</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Lower</td>
<td>Increase</td>
<td>-</td>
<td>Lower extrem. Systemic?</td>
<td></td>
</tr>
<tr>
<td>3. H.M.</td>
<td>25 M</td>
<td>3.0</td>
<td>15.4</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Lower</td>
<td>Increase</td>
<td>+</td>
<td>Mesenterial Hepatic Intestinal</td>
<td></td>
</tr>
<tr>
<td>4. T.M.</td>
<td>52 M</td>
<td>2.8</td>
<td>7.0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Lower</td>
<td>Increase</td>
<td>+</td>
<td>Mesenterial</td>
<td></td>
</tr>
<tr>
<td>5. T.K.</td>
<td>38 M</td>
<td>4.0</td>
<td>12.6</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Upper</td>
<td>Increase</td>
<td>+</td>
<td>Mesenterial</td>
<td></td>
</tr>
<tr>
<td>6. T.Y.</td>
<td>21 F</td>
<td>5.2</td>
<td>3.2</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Lower</td>
<td>Increase</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>7. M.S.</td>
<td>21 F</td>
<td>3.6</td>
<td>9.0</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. I.S.</td>
<td>60 M</td>
<td>6.1</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>Obstruc.</td>
<td>Decrease</td>
<td>+</td>
<td>Mesenterial</td>
<td></td>
</tr>
</tbody>
</table>
pathy, which is further subclassified to the primary congenital lymphogenic and the secondary lymphogenic protein-losing enteropathy.

Lymphography is necessary to determine the pathogenesis of the protein-losing enteropathy.

Fig. 1 Remarkable reflux to the mesenteric, the hepatic, and the intestinal lymphatics with leaked contrast medium in the jejunum immediately after the lymphography

References
1 Bismuth, U.: Lymphointestinal fistula defined by lymphography during a hypoproteinemnic syndrome with steatorrhea. J. Radiol. Electr. 46 (1965) 295
7 Steinfeld, J.L., J.D. Davidson, R.S. Gordon Jr., F.E. Greene: Mechanism of hypoproteinemia in patients with regional enteritis and ulcerative colitis. Am. J. Med. 29 (1960) 405

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