The Lymphatic Drainage in Portal Hypertension

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- Ascites
- Hypertension portal
- Liver cirrhosis
- Lymph
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- Thoracic duct

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It is now well known that the lymphatic drainage of the liver goes through the thoracic duct, the pressure of which reflects faithfully the condition of the portal system. Extensive experimental work and its clinical application demonstrated a definite correlation between hypertension of the portal vein in cirrhotic patients and increased pressure in the thoracic duct.

In liver cirrhosis there is an increased production of lymph and the thoracic duct becomes enlarged. At its junction with the left jugulo-subclavicular angle there is a valvular mechanism which stops the lymphatic flow, and therefore the pressure in the thoracic duct increases. The flow tries to bypass the normal lymphatic pathways, goes to the subclavicular spaces, and from there drips into the peritoneal cavity to form ascites. The work of Dumont, New-York, has shown that by external diversion of the lymph through a catheter introduced in the thoracic duct, there was a drop in the portal pressure, a diminution of ascites and in case of hemorrhage a stop of the bleeding.

Up to now, there has been a general agreement upon the surgical treatment of portal hypertension.

The porto-caval shunt is generally accepted as being the procedure of choice in bleeding oesophageal varices either as an emergency procedure or as a planned operation assuming that the patient’s general condition is satisfactory. For poor risk patients we had nothing to offer until recently. The lymphatic derivation opens a new field and offers a good chance to these patients.

The lympho-venous anastamosis seems to be better than the external lymphatic fistula. It brings the same advantages as the external drainage but prevents the complications such as hypovolemia due to the high quantity of diverted liquids, hypoproteinemia, lymphopenia and electrolytic imbalance.

The operation is performed under general anesthesia, involves minimal operative risk, is quickly performed and consists of an anastomosis between the terminal portion of the thoracic duct and the internal jugular vein.
In a recent publication, H. W. Schreiber from Hamburg, reports his first results which confirm the findings of Dumont and others:

The bleeding stops.
The ascites decreases.

We have personally tried this procedure in 2 cases which were in acute intractable hemorrhage due to ruptured esophageal varices. Both had ascites, and were in a precomatous stage. The first patient stopped bleeding immediately after the operation. The abdominal wall, which was distended by ascites prior to operation became markedly softer within a few hours after surgery. She recovered, regained consciousness and was discharged 2 months later with a stabilized ascites and a normal blood count.

The second case was even worse: dramatic hemorrhage, ascites, jaundice and evidence of encephalopathy. Shortly after surgery, the bleeding subsided for 2 days. We had to monitor very carefully the central venous pressure which increased markedly. This is a very important point which should be emphasized. The blood volume is expanded by the sudden and massive flow of lymph in the venous system and this may lead to cardiac insufficiency unless the central venous pressure is carefully monitored. If necessary, one must not hesitate to perform a venisection, which we did in our second case.

In conclusion, one may say that the lymphatico-venous anastomosis in portal hypertension is worth trying in patients whose general condition prevents extensive surgical procedure, such as portocaval shunts.

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