POLISH MEDICAL JOURNAL, Vol. VI, No. 6/1967 (Translated from Polski Przegląd Chirurgiczny, Vol. XXXIX, No. 2/1967)

W. OLSZEWSK1

EXPERIMENTAL LIVER LYMPHOGRAPHY IN ASCITES

Department of Experimental Surgery, Polish Academy of Sciences, Department I of Surgery, Medical Academy in Warsaw

http://rcin.org.pl

The author performed liver lymphography in dogs with ascites, which was induced experimentally by means of narrowing of the supradiaphragmatic segment of the inferior vena cava. Contrast medium was injected into lymphatic vessels of porta hepatis and it filled all intrahepatic lymphatic vessels, flowing along the branches of portal vein and subcapsular vessels, with simultaneous insufficiency of valves. Permeation of the contrast medium through the liver capsula proves that the liquor within peritoneal cavity, present in experimentally induced ascites, originates from subcapsular hepatic vessels.

A profuse amount of lymph of hepatic origin is produced in the course of liver cirrhosis. This results in an increase in lymph flow rate and rise in lymph pressure within the intrahepatic lymphatic ducts, *cisterna chyli* and thoracic duct. As results from previous studies, the lymphatic vessels of the porta hepatis become distended, the diameter of the thoracic duct increases 2—4 times, lymph pressure in it rises to 15—70 mm H₂O, and the flow rate augments 3—12 times (2). The excess of lymph, which cannot escape through the overfilled thoracic duct to the subclavian vein, penetrates through the wall of the subcapsular and portal lymph vessels into the abdominal cavity, thus giving rise to ascites. Intraparenchymatous pressure in the liver also increases in such instances, resulting in additional impairment of portal blood circulation. Methods of visualization of intrahepatic lymphatic ducts in humans as well as in animals have up till now not been reported in medical literature.

The present studies were undertaken with the aim of developing a method of visualization of intrahepatic lymphatic and efferent ducts, carrying lymph from the liver. Lymphography of such a type is necessary for: a) studying ascites and b) search for an operative method, suitable for decompression of lymphatic ducts in ascites.

The lymph in normal conditions flows from the liver to its porta, and further through the lymph nodes in the hepato-duodenal ligament to the *cisterna chyli* and thoracic duct. This direction of lymph flow is conditionned by the valves, present in the lymphatic vessels. These valves may become insufficient owing to the engorgement of the lymphatic vessels occurring in ascites. In the case of cirrhosis and overfilled and distended lymphatic ducts within the hepatoduodenal ligament it seemed to be possible to introduce contrast medium flowing towards the liver and to visualize in this way the intrahepatic lymphatic ducts.

METHODS

Investigations were carried out in two groups of dogs (15 animals), weighing from 15 to 18 kg: a) in healthy dogs and b) in dogs with experimentally induced ascites, following partial stricture of the inferior vena cava over the diaphragm. After laparotomy, one lymphatic vessel of the porta hepatis was prepared out and connected by means of a cannula to a syringe containing an 80-0 percent aqueous solution of contrast medium (Uromiro, Biligraphin). Prior to injection of contrast medium, the juxtaduodenal part of the hepato-duodenal ligament was slightly clamped with a rubber band. A total of 10—12 ml of contrast medium was injected within 10 minutes, the rubber band being repeatedly released for 15—20 sec. Radiograms were taken several times in the same dog after administration of 2, 4, 8 and 12 ml of contrast medium.

RESULTS

In the first group (healthy animals) in no case did the contrast medium pass into the liver, but it visualized the efferent lymphatic ducts. Injection of contrast medium under high pressure led to its extravasation into the hepato-duodenal ligament (Fig. 1).

In the second group (animals with experimentally induced ascites) already after injection of 2 ml of contrast medium the distended lymphatic ducts in the porta hepatis were visualized. Following the injection of the next 2-ml portion of contrast, the wide intrahepatic lymphatic vessels, concomitant with the branches of the portal vein became visible (Fig. 2). Injection of a successive 4-ml dose of contrast medium resulted in the filling of small peripheral lymphatic vessels. A magnified lymphographic picture revealed the presence of a distinctly visible fine network of lymphatic vessels, twining around even the smallest intrahepatic branches of the portal vein (Fig. 3). Upon administration of 12 ml of contrast medium the distended subcapsular lymphatic vessels became apparent. X-ray pictures demonstrated the penetration of contrast through the walls of the subcapsular vessels and liver capsule, with formation of large drops on the liver surface (Fig. 4).



Fig. 1. Healthy dog. Extravasation of contrast medium injected into lymphatic vessels of liver porta, into hepato-duodenal ligament. Efficient valves do not permit filling of intrahepatic lymphatic vessels.



Fig. 2. Dog with experimentally induced ascites. Intrahepatic lymphatic vessels accompanying branches of the portal vein were visualized following injection of 6 ml of contrast medium.



Fig. 3. Magnified radiogram. A network of very fine lymphatic vessels twining around branches of the portal vein is visible.



Fig. 4. Dog with experimentally induced ascites. Even most peripherally situated lymphatic vessels are visualized following injection of 12 ml of contrast medium. Drops of contrast medium, penetrating through the liver capsule begin to appear on the liver surface.

W. Olszewski

DISCUSSION

The investigations demonstrated that, in experimentally induced ascites also intrahepatic lymphatic vessels undergo considerable enlargement and their valves become insufficient. As shown by the enlarged radiograms, lymphatic vessels accompany the branches of the portal vein and even twine around them in a dense network. The evident penetration of contrast medium through the walls of subcapsular vessels and through the liver capsule confirms the results of other studies, carried out in this Department by *B. Szczygiel* (5) who demonstrated that the ascitic fluid in experimental ascites is lymph of hepatic origin.

CONCLUSION

The method of liver lymphography, described above, might be utilized in human patients suffering from liver cirrhosis. Clinical application of this method might perhaps contribute to the explanation of the pathomechanism of ascites.

Translated by M. Schmidt, M. D.

REFERENCES

- 1. Brauer, R. W., Holloway, R. J., Leong, G. F.: Changes in the liver function and structure due to experimental passive congestion under controlled hepatic vein pressure. Am. J. Physiol., 1959, 197, 681.
- 2. Dumont, A. E., Mulholland, J. H.: Alteration in thoracic duct lymph flow in hepatic cirrhosis. Ann. Surg., 1962, 156, 668.
- 3. Nielubowicz, J.: Quelques problemes de physiopathologie portale. La Presse Med., 1965, 73, 185.
- Olszewski, W., Szyfelbejn, S., Nielubowicz, J.: Doświadczalne nadciśnienie wrotne i wodobrzuszne w następstwie częściowego zwężenia przewodu żółciowego wspólnego (Experimental portal hypertension and ascites caused by partial constriction of the choledochus). Pol. Tyg. Lek., 1963, 18.
- 5. Szczygiel, B.: Badania doświadczalne nad mechanizmem powstawania puchliny brzusznej (Experimental studies on the mechanism of ascites formation). Pol. Tyg. Lek., 1963, 18 (Pol. Med. J. 1963, 4, 519).