

Percutaneous Trans hepatic Lymphography: Evaluation in Schistosomal Hepatic Fibrosis

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Summary

The increased interest in lymph and lymphatics has cast its mantle over the portal circulation. Lymphography has contributed greatly to our knowledge. In the present study percutaneous transhepatic lymphography showed some of the factors sharing in the production of portal hypertension in schistosomal hepatic fibrosis, and gave a further evidence that the liver is not a source of excess lymph production in hepatic lesions associated with presinusoidal block to portal blood flow.

Hepatic lymphatics were opacified in cases with mixed cirrhosis and schistosomal hepatic fibrosis as the sinusoidal pressure is elevated with subsequent cases increase in hepatic lymph production.

Introduction

Hepatic intraparenchymal deposition of contrast material proved to be a valuable mean of studying the lymphatics of the liver and the hepatic venous outflow both in experimental animals (12, 13), and man (5, 14). The advantage of this technique is its ability to demonstrate haemodynamic events rather than morphological changes. In this respect it is superior to hepatic venography in which the contrast medium is injected under pressure into the lumen of the vessel (5). The present work was undertaken mainly to study the intrahepatic lymphatics in schistosomal hepatic fibrosis.

Material and Methods

Fifteen schistosomal patients were selected. Complete clinical data and investigations were recorded. Nine were pure cases of schistosomal hepatic fibrosis and six were mixed cirrhosis. The liver pathology was evidenced by excisional or needle biopsy and the portal pressure measured by needling the spleen and using a manometer with saline solution and by wedging a catheter into an hepatic vein. Hepatic lymphography was done following the method of *Clain and McNulty* (5), through a no. 18 spinal needle. Each patients received 20 ml. of 45 percent sodium diatrizoate and serial films were taken at 2 to 4 seconds interval from the start of injection and up to 30 seconds. Thoracic duct lymph flow and pressure data were determined in all cases after thoracic duct exposure and cannulation in the left side of the neck. The study also included 4 normal controls for comparison.

Results

No serious immediate or late complications were encountered in any of the patients included in this study.

Hepatic venous radicles appeared as single smoothly outlined branches that passed medially to reach the main hepatic veins at the right para-vertebral region. The portal vein radicles were opacified through regurgitation of the contrast material from the site of deposition. They described a main stem with smaller ramifications. The criteria for designating an opacified chan-

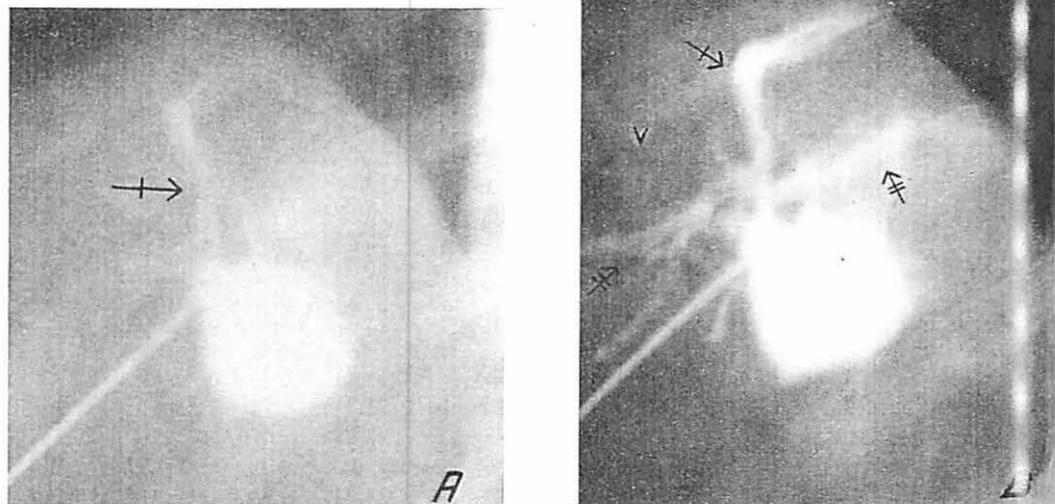


Fig. 1 Intraparenchymal deposition of contrast material in a patient with pure schistosomal hepatic fibrosis. The four seconds film (A) shows hepatic veins draining into smoothly outlined main hepatic vein (—>). Reflux of the dye into the portal vein radicles (-H→) appears in the ten seconds film (B). Note no lymphatics were opacified up to 30 seconds.

nel as being a lymphatic one were: a) tortuous course, b) beaded appearance, c) irregular calibre with no side branching or ramification, and d) direction of flow being towards the hilum of the liver.

No lymphatics were visualized in normal livers.

Findings in schistosomal hepatic fibrosis:

Most of the dye passed into the major hepatic veins from the site of injection along small radicles. The flow was rapid as evidenced by the rapid disappearance of the contrast medium from the hepatic veins at the end of injection as seen in the serial exposures.

Reflux of the contrast material into the nearby portal vein radicles was seen. Such venous radicles showed actual reduction of the minute branches and were surrounded with a rich vascular network that was filled with them (Fig. 1 and 2).

The hepatic lymphatics were not opacified in any of such cases.

Findings in the mixed cirrhosis group:

Similar results to those described by *Clain and McNulty* (5), were found. Hepatic efferent lymphatics were observed in 5 of 6 cases with mixed cirrhosis. They appeared as several fine vessels pursuing an irregular tortuous course (Fig. 3, 4, 5). The thoracic duct was opacified in one patient. It was dilated and tortuous (Fig. 4 "C").

Discussion

The demonstration of hepatic lymphatics was possible in pathological conditions of the liver associated with increased sinusoidal pressure (5, 6, 14). Schistosomal hepatic fibrosis carries a pre-sinusoidal element of obstruction (2, 15, 17), that was attributed in part to a reduction in the minute branches of the portal vein radicles secondary to thickening and fibrosis of the portal tracts (2, 17), and to a newly formed vascular network around the portal vein radicles that creates resistance to the portal blood flow (3). Both these lesions were observed in the hepatic lymphograms of such patients. Excess thoracic duct lymph was found in schistosomal hepatic fibrosis (16, Table 1). The sources of excessive lymph in cases with presinusoidal block was

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Fig. 2 (a + b) Representative ten seconds films from serial exposures of patients with pure schistosomal hepatic fibrosis showing hepatic veins (->) and portal (-#>) venous radicles with reduced terminal branches and surrounded with rich vascular formation (>). Note inferior vena cava (—>) opacified in B. Note no lymphatics were seen up to 30 seconds.

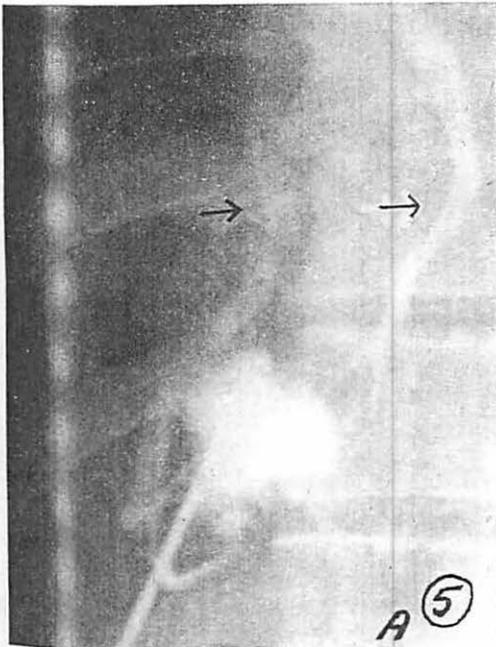


Fig. 3 Series of hepatic lymphograms of a patient with mixed cirrhosis. (A) film at four seconds showing the deposit of contrast material and small hepatic veins collecting into two large veins (->). At ten seconds (B), tortuous lymphatics are seen in the right oblique view (—>) and are still opacified in the 20 seconds film (C), while as most of the portal veins (-#>) and all hepatic veins were emptied.

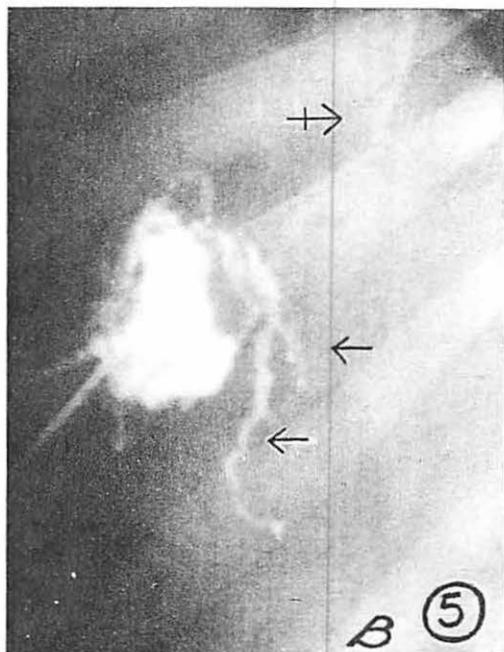


Fig. 3 B

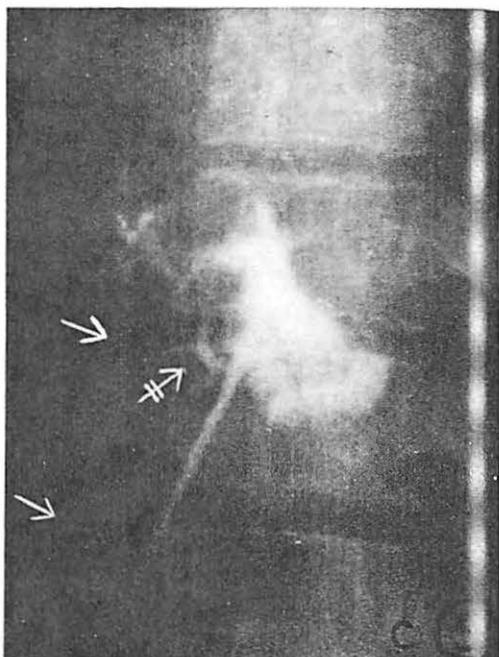


Fig. 3 C

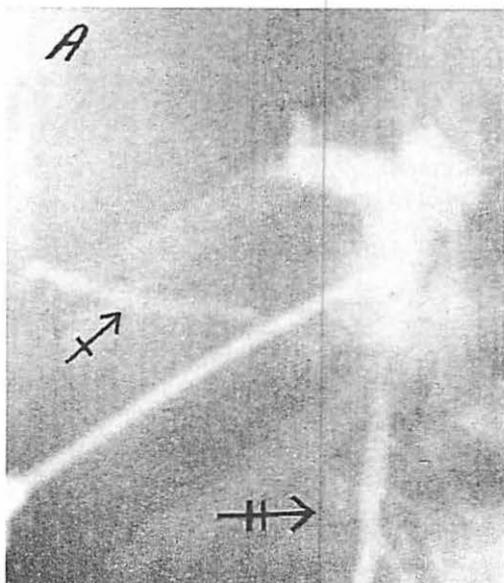


Fig. 4 A

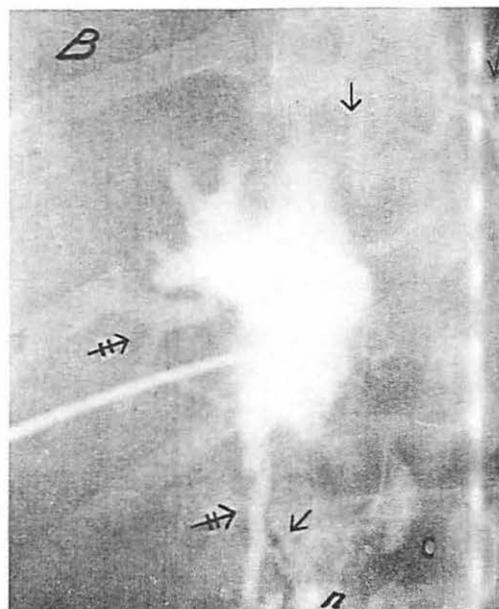


Fig. 4 B

Fig. 4 Transhepatic lymphograms of a patient with mixed cirrhosis. In the four seconds film (A), hepatic (\rightarrow) and portal (\rightarrow) venous radicles were seen and in the ten seconds film, hepatic lymphatics were opacified (\rightarrow), with a tortuous subcapsular one. Note cisterna chyli (c) and hilar lymph node (n). The thoracic duct of the same case was opacified (C), and showing dilatation, tortuosity, and a cervical reflux (\rightarrow).

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Fig. 4 C

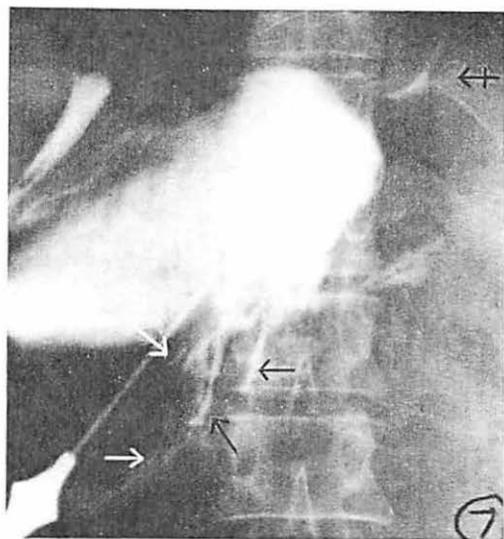


Fig. 5 Lymphogram (at ten seconds), of a patient with mixed cirrhosis. Note hepatic vein (+→) and tortuous hepatic lymphatics (→).

Table 1 Thoracic duct flow and pressure data obtained in 15 schistosomal patients and 4 controls (Mean values \pm S.E.).

Type of cases	No.	Thoracic duct lymph		Portal pressure	
		flow cc/min.	Pressure cm. saline	Splenic mm saline	W.H.V.P. mmHg
Controls	4	1.7 \pm 0.21	12.0 \pm 1.4	126.0 \pm 12.8	7.0 \pm 0.80
Schistosomal hepatic fibrosis	9	5.8 \pm 0.66 P < 0.01	31.5 \pm 4.5 P < 0.001	237.7 \pm 19.1 P < 0.001	6.8 \pm 0.93 P > 0.05
Mixed cirrhosis	6	11.0 \pm 0.89 P < 0.001	44.6 \pm 7.1 P < 0.001	280.0 \pm 40.2 P < 0.001	16.5 \pm 1.0 P < 0.001

W.H.V. P. = Wedged Hepatic Venous Pressure.

suggested to be the gastrointestinal tract and the spleen (1, 8, 18, 20). In the present study there was free and rapid flow of the contrast medium into the smoothly outlined hepatic veins as well as normal wedged hepatic venous pressure (Table 1). These observations exclude any obstruction to the outflow system, a finding which was proved before haemodynamically and pathologically (2, 15, 17). The non encroachment on the sinusoidal pressure can explain the non opacification of hepatic lymphatics and excludes the liver as a source sharing in excessive lymph production in patients with presinusoidal block to portal blood flow.

Schistosomal hepatic fibrosis may become complicated by cirrhosis due to nutritional defects or viral hepatitis (9). Six such patients were included in this study. In five of them (83.3%), the hepatic lymphatics were seen, as cirrhosis of the liver is associated with increased lymph flow (4, 7, 10, 11, 20) (Table 1), attributed to a rise in the hydrostatic forces in the highly permeable sinusoids (18). The augmentation of hepatic lymph in these cases can play a role in the

functional relief of outflow hepatic venous obstruction (5, 14), and participate in the production of ascites and thoracic duct changes (7, 10, 19).

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