

The Role of Lymphatics in the Formation of Ascites Complicating Schistosomal Hepatic Fibrosis

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Summary

The total protein content in plasma, ascitic fluid, thoracic duct lymph, hepatic and intestinal lymph was studied in a series of 15 patients suffering from schistosomal hepatic fibrosis and intractable ascites. Pure schistosomal cases with presinusoidal resistance to portal blood flow have excessive thoracic duct lymph low in protein. The main source of such excess lymph is the extra-hepatic portal bed. Ascitic fluid in such patients has a low protein content and has the character of a transudate. The bulk of such peritoneal fluid seems to originate largely from the excess extrahepatic portal lymph.

Introduction

Ascites is not a simple hydrodynamic effect of portal hypertension as other factors are concerned in its development and includes hypoproteinaemia, salt and water retention, and renal haemodynamic changes (2, 18, 23, 30). In addition, excessive hepatic lymph may initiate or perpetuates ascites in cases of *Laennec's* cirrhosis (12, 14, 19, 37).

Schistosomal hepatic fibrosis carries a presinusoidal obstruction (5, 24, 28) as judged from the intrasplenic and wedged hepatic vein pressures and liver biopsy. Excess thoracic duct lymph was found in patients with such a lesion (26). *Aboul-Enein* and *Ismail* (1) have found some contributory effect from the congestive spleen to the excess lymph production in schistosomal patients with presinusoidal block.

The aim of the present work is to study the possible role of the sources of excessive lymph production in ascites formation in patients having schistosomal hepatic fibrosis.

Material and Method

Fifteen patients suffering from schistosomal hepatic fibrosis and intractable ascites were studied. All were males aged 24 to 56 years. The liver pathology was evidenced by 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. Portal vein occlusion was excluded in all cases by splenoportography. The total protein content in plasma, ascitic fluid, and thoracic duct lymph was determined in all patients. Hepatic and intestinal lymph protein was measured in only 8 cases. The total protein content of these different fluids was measured by the salt fractionation Biuret method. Lymph was obtained from the neck during thoracic duct cannulation for diagnostic or therapeutic purposes. Hepatic lymph was taken from the periportal lymphatics and intestinal lymph from the mesenteric lacteals during laparotomy or at post-mortem. The study also included 5 normal controls with no hepatic disease and all were males aged 20 to 52 years.

Results

Tables 1 and 2 show the data obtained in 15 schistosomal patients and 5 control subjects. The total protein content in thoracic duct lymph in schistosomal cases with presinusoidal block-pure schistosomal hepatic fibrosis – was significantly lowered (1.4 to 3.4 gm per 100 ml), and the total protein content of ascitic fluid in such cases was also low (16.7 percent of plasma). In all patients, thoracic duct lymph flow and pressure were significantly increased.

Table 3 shows the mean values and the percent. Plasma level of the total protein content in hepatic and intestinal lymph in the eight patients studied. In all schistosomal patients, there was a significant decrease in the total protein content in intestinal lymph. The values in control group were slightly lower than those reported by *Courtice* (11); and *Witte* et al. (37) – hepatic lymph 87.7 percent of plasma and intestinal lymph 58.3 percent.

Table 1 Total protein content in plasma, T.D.L., and ascitic fluid in 15 schistosomal patients and 5 control subjects; T.D. flow and pressure data obtained in these cases. (Mean values \pm S.E.)

Type of cases	No.	Plasma		Thoracic duct lymph			Ascitic fluid		Portal pressure	
		Total protein gm%	Total protein gm%	Flow cc/min.	Occlusive pressure cm saline	Lateral pressure cm saline	Total protein gm%	Splenic mm saline	W.H.V.P. mmHg	
Controls	5	7.2 \pm 0.15	4.1 \pm 0.53	1.9 \pm 0.03	10.2 \pm 1.5	–	–	130 \pm 5.5	6.2 \pm 0.37	
Schistosomal hepatic fibrosis	15	7.04 \pm 0.21	2.5 \pm 0.27	7.7 \pm 0.10	40.2 \pm 7.7	12.1 \pm 1.21	22 \pm 0.10	294 \pm 17.0	6.9 \pm 0.40	
		P > 0.05	P < 0.01	P < 0.001	P < 0.001			P < 0.001	P > 0.05	

T.D.L. = Thoracic duct lymph

W.H.V.P. = Wedged hepatic venous pressure

Table 2 Total protein content of thoracic duct lymph and ascitic fluid expressed as per cent plasma level (Mean values)

	Thoracic duct lymph	Ascitic fluid
Controls	57.20	–
Schistosomal Hepatic fibrosis	34.70	16.7

Table 3 Total protein content in plasma, hepatic, and intestinal lymph in 8 schistosomal patients and 5 controls (Mean values \pm S.E.). The percent plasma level (Mean values).

Type of cases	No	Plasma		Hepatic lymph		Intestinal lymph	
		Total protein gm%	Total protein gm%	Percent plasma level	Total Protein gm%	Percent plasma level	
Controls	5	7.2 \pm 0.15	6.3 \pm 0.15	87.70	4.2 \pm 0.14	58.3	
Schistosomal hepatic fibrosis	8	7.0 \pm 1.0	5.9 \pm 0.06	84.28	1.1 \pm 0.09	15.7	
		P > 0.05	P > 0.05		P < 0.001		

Discussion

Thoracic duct lymph originates almost entirely from the liver and the extrahepatic portal bed (7). The liver is the principle source of increased lymph production in *Laennec's* cirrhosis and in experimental constriction of the thoracic inferior vena cava (6, 13-15, 20, 22, 32). Such excessive lymph has a high protein content (35-40) as the liver sinusoids are freely permeable to proteins (21). Pure schistosomal hepatic fibrosis with presinusoidal obstruction has no analogous mechanism to that found in *Laennec's* cirrhosis. The present work shows that in patients with such a lesion, the total protein content of intestinal lymph and thoracic duct lymph is significantly low. Similar results were found in patients and animals with presinusoidal portal hypertension (35, 36). As lymph arises from plasma by capillary filtration regulated largely by hydrostatic and osmotic pressures on either side of the capillary membrane (25), the sustained rise in portal venous pressure leads to elevation of the hydrostatic pressure in the intestinal capillaries and increased filtration of water and freely diffuse solutes (34). So that the protein content of small intestinal lymph and hence thoracic duct lymph falls (17, 21, 31, 35). The total protein content of ascitic fluid in schistosomal patients with presinusoidal block was low (1 to 2.1 gm per 100 ml), and nearly similar to that found in experimental animals after prolonged extrahepatic portal congestion (36). As peritoneal fluid reaches osmotic equilibrium with the extra-hepatic portal bed (9, 10, 16), the ascitic fluid protein content was used to estimate the splanchnic tissue colloid osmotic pressure (8), and to provide a clue to the nature of the splanchnic circulatory disturbances. In the absence of nephrotic syndrome or other conditions associated with marked hypoproteinaemia, a peritoneal transudate is virtually diagnostic of portal system obstruction regardless of the underlying diseases (42). Since the peritoneal cavity is regarded as a specialized tissue space and ascites constitutes a subcompartment of the expanded splanchnic lymph volume, the bulk of the low protein ascitic fluid in cases with presinusoidal block was suggested to originate from the extra-hepatic portal bed (41). According to such authors, ascites will be aggravated by factors which either stimulate splanchnic lymph formation or impair splanchnic lymphatic drainage.

Disparity between lymph production and its drainage along the thoracic duct proved to exist in *Laennec's* cirrhosis and schistosomal hepatic fibrosis (12, 26, 29, 43). The damping back of lymph leads to oedema of the bowel as was noted on X-ray (3), intestinal biopsy (4, 27), and during portacaval shunt and splenectomy operations (27, 33). The undrained low protein lymph seems to weep from the serosal surfaces of the intestine and mesentery into the peritoneal cavity.

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