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Radiographic and haemodynamic patterns of portal hypertension in hepatosplenic schistosomiasis: selection of surgical procedure

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SUMMARY Twenty-eight patients with hepatosplenic schistosomiasis and portal hypertension were studied. The transumbilical portal pressure, transsplenic portal pressure, and thoracic duct occluded pressure were measured simultaneously and thoracic duct lymph flow estimated. Changes in the splanchnic vasculature were studied radiologically by barium swallow, splenoportography, and umbilical portography. The transumbilical-transsplenic portal pressure gradient was found to be of particular value. The gradient was considered to be positive when the transumbilical portal pressure was higher than the transsplenic portal pressure, in such cases the primary generating factor of portal hypertension and/or ascites was most probably of hepatic origin. The gradient was considered to be negative when the transumbilical portal pressure was lower than the transsplenic portal pressure; in such cases the primary generating factor of portal hypertension and/or ascites was most probably of splenic or prehepatic origin. A correlation was found between the type of gradient and the radiographic pattern met with. For instance, in cases with positive gradient the hepatic blood flows, as estimated from the splenoportography, were mostly stage I or II, and showed no retrograde portal vein flow on umbilical portography. While, in cases with negative gradients, the hepatic blood flows were mostly stage III or IV, and showed retrograde portal vein flow on umbilical portography. The type of gradient, the clinicopathological stage, and the radiographic changes in the splanchnic vasculature were taken into account in selecting the surgical procedure to be used in each individual case.

Portal hypertension is a complex syndrome produced by various circulatory dynamics. Some authors believe in the forward flow theory (Ravenna, 1940; Peters and Womack, 1961), others believe in the backward flow theory (Bradley *et al.*, 1952, Kelty *et al.*, 1950), and yet others subscribe to both theories (Gitlin *et al.*, 1970).

There is no single criterion which can be used in the selection of patients for non-operative or operative therapy or in determining the type of operation to be performed (Warren *et al.*, 1967). Two major parameters were found useful, not only in selecting patients for the surgical procedure, but also in selecting the most suitable procedure for each individual patient: these were the clinicopathological stage and the haemodynamic pattern. The presence and significance of contrasting haemodynamic patterns of portal hypertension in hepatosplenic schistosomiasis has been demonstrated. These patterns in conjunction with the clinicopathological stage were found to be of value in selecting the surgical procedure that was most suited to the individual patient (El-Gendi and Gemeuh, 1977).

This paper presents the radiographic changes in the splanchnic vasculature in hepatosplenic schistosomiasis and portal hypertension at different clinicopathological stages as correlated with the haemodynamic patterns.

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Methods

Twenty-eight patients with schistosomal hepatosplenomegaly and portal hypertension were randomly picked from the medical ward for this study. They were 12 non-bleeders, six bleeders, and 10 with ascites. Diagnosis of the condition was based on the finding of schistosomal ova in the stools or rectal scrabbing and the presence of liver fibrosis, enlarged spleen, and other clinical manifestations of portal hypertension. The following procedure

was observed in each case.

Barium swallow and splenoportography were performed.

Ascites when present was gradually evacuated (for five days before operation) using a suprapubic catheter (introduced by the trocar and cannula method). The catheter was kept in place for seven days after operation.

Extraperitoneal cannulation of the obliterated umbilical vein was carried out under local anaesthesia to measure the transumbilical portal pressure and to perform portography.

The thoracic duct was cannulated in the neck to measure the thoracic duct lymph flow/min, and thoracic duct occluded pressure.

Immediately before operation the transumbilical portal pressure, transsplenic portal pressure, and thoracic duct occluded pressure were measured simultaneously and the thoracic duct lymph flow/min reestimated. All pressures were measured in cm saline and flows in ml lymph/min.

At operation, the size of the spleen, the presence of a thrill on the splenic hilum, the degree of collateral, and the state of the liver were assessed and a wedge liver biopsy was taken.

The surgical procedure was selected, guided by the clinicopathological stage, the operative findings, the vascular radiographic changes, and the haemodynamic pattern that was encountered.

The main operations used were as follows:

Splenectomy alone was performed in non-bleeders for hypersplenism, or for the mechanical effects attributed to the grossly enlarged spleen that was present in nearly all cases and/or for its possible haemodynamic role and in certain ascitic cases—namely, those associated with a hyperdynamic spleen (case nos 20, 25, and 27).

Splenectomy with gastro-oesophageal decongestion (non-shunt) for all bleeders.

Omental transposition (to the right side of the chest wall) was performed in ascitic cases with an insufficient lymphatic system and who were a poor surgical risk (case no. 19); splenectomy was added if the spleen were hyperdynamic (case nos 21, 22, and 23).

Thoracic duct-vein shunt with omental transposition was carried out in cases with an inefficient thoracic duct system (case nos 24 and 28).

Table 1 *Clinicopathological findings in 28 cases of hepatosplenic schistosomiasis and portal hypertension*

Case no.	*Liver*		*Collaterals*	*Spleen*		
	*Size**	*Biopsy*		*Size**	*Weight (g)*	*Thrill*
Non-bleeders						
1	3	—	++	6	1800	+
2	2	—	+	5	750	−
4	3	M	++	4	600	+
5	3	Sc	−	5	1200	+
7	3	M	++	10	2200	+
8	3	Sc	−	4	1250	+
9	3	M	+	9	2000	+
10	3	Sc	−	7	950	−
12	3	M	−	6	1070	+
14	2	Sc	+	4	980	+
17	3	—	+	4	900	−
18	3	—	+	7	1200	+
Bleeders						
3	3	M	−	5	700	+
6	2	M	+	8	1300	+
11	1	Sc	−	7	1500	+
13	3	—	−	10	2150	+
15	3	Sc	+	3	700	−
16	3	Sc	+	10	2100	+
Ascitics						
19	1	M	−	4	—	−
20	1	Sc	−	8	1900	++
21	1	Sc	−	4	650	+
22	—	Sc	−	4	720	+
23	1	Sc	−	10	2100	++
24	—	M	−	4	—	−
25	1	Sc	−	10	1850	++
26	1	M	+	2	—	−
27	1	Sc	−	9	1750	+
28	—	M	++	5	—	−

*Finger below costal margin.
M: Mixed cirrhosis.
Sc: Pure schistosomal fibrosis.

Portocaval shunt was done for mixed cases with positive gradient (those with superadded post-sinusoidal obstruction and in whom the spleen was not hyperdynamic), as in case no. 26.

The transumbilical portal pressure was measured daily after operation till the catheter was removed after postoperative portography on the fifth day.

Results

The results obtained are shown in Tables 1 to 4 and Figs 1 to 5. The clinicopathological findings in the 28 cases are demonstrated in Table 1 after being grouped into non-bleeders, bleeders, and ascitics.

All 28 cases were studied radiographically by barium swallow, splenoportography, and umbilical portography. These studies were carried out pre-operatively and/or postoperatively, and many tests were done repeatedly (Table 2). Technically satisfactory splenortograms were obtained in nearly all cases before operation. The site, size, and patency of the portal veins were recorded. The degree and pattern of collateral vessels were estimated. On the basis of radiographic appearance, the degree of interference with hepatic blood flow was estimated (Warren *et al.*, 1967). The appearance and degree of the hepatic blush or hepatogram on late films were used to assess the amount of portal flow going to the liver in relation to that diverted through the collateral channels. With high hepatoportal flow the blush was prominent (stage I, normal or only slightly restricted); when the flow was predominantly hepatofugal, it was poor (stage III, severe restriction); or absent (stage IV, total lack of opacification of the portal vein). Intermediary flow could also be gauged —that is, stage II, moderate reduction.

Of the 28 cases studied, four were of stage I; 15 were of stage II; four were of stage III; and the remaining five were stage IV. The non-opacified portal vein in these last five cases was proved by transumbilical portography to be patent in all except one (case no. 7).

In the remaining four cases of stage IV, the portal vein was filled in a retrograde direction till the splenic vein and marked collaterals were visualised (Fig. 1). These same cases were associated with

Table 2 *Radiographic findings in 28 cases of hepatosplenic schistosomiasis and portal hypertension at different stages*

		Splenoportography				*Umbilical portography*	
Case no.	*Barium sw. for oesoph. var.*	*Oesoph. var.*	*Degree of hepat. flow*	*Collat.*	*Portal vein BF*	*L. gastric vein vis.*	*TUPP-TSPP gradient*
Non-bleeders							
1	−	−	I	−	−	−	+18
2	−	−	II	+	−	−	+ 9
4	−	−	I	−	−	−	+20
5	−	−	II	−	−	−	+ 4
7	−	+	IV	++	−	−	−29
8	−	−	II	+	−	−	+ 5
9	−	−	II	+	−	+	+ 7
10	−	−	III	−	+	−	− 3
12	+	+	I	++	−	−	+ 5
14	−	−	II	−	−	+	+ 6
17	−	−	II	−	−	+	+ 6
18	−	−	II	−	−	+	+ 6
Bleeders							
3	+	+	II	++	−		+ 6
6	+	+	II	++	−	+	+13
11	+	+	IV	++++	+	+	− 7
13	+	+	II	++	−	+	+ 8
15	+	+	II	++	−	+	+13
16	+	+	III	+++	+		−12
Ascitics							
19	−	−	II	++	−		+ 3
20	+	+	IV	+++	+	−	−20
21	−	+	III	++	+	−	− 6
22	−	−	III	++	+	+	− 2
23	−	−	II	−	−	−	+ 6
24	−	−	II	−	−	−	+ 5
25	−	+	IV	+	+	−	−15
26	−	−	II	−	−	−	+ 6
27	+	+	IV	++	+	−	−12
28	−	−	II	−	−	−	+ 2

BF: backflow.

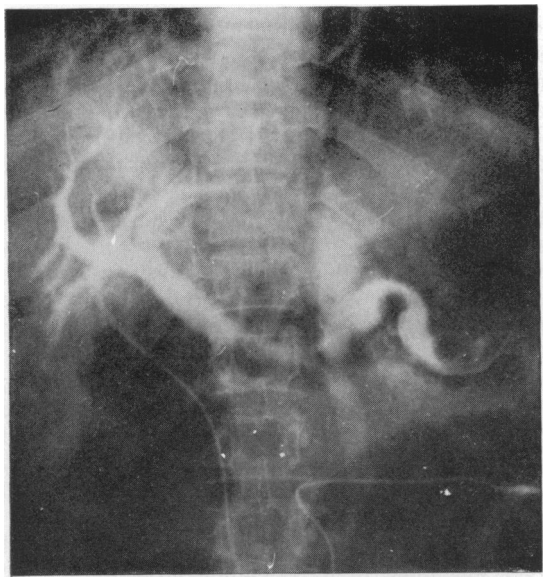

Fig. 1 *Case 16: bleeder with a negative transumbilical-transsplenic portal pressure gradient. Transumbilical portography shows (1) retrograde portal vein flow with both cephalic and caudal collaterals (the portal vein was not opacified in the splenoportogram of the same case—that is, stage IV portal blood flow); (2) the 'withered tree' appearance characteristic of advanced schistosomal liver.*

a negative gradient of transumbilical-transsplenic portal pressure. In the four cases with stage III, very faint visualisation of the portal vein was found on splenoportography, but, on transumbilical portography, retrograde flow to the portal vein was a constant finding in the four cases—two cases to the splenic vein and retroperitoneal collateral veins and in the remaining two cases the retrograde flow was mainly to the left gastric vein. In these four cases, the gradient of transumbilical-transsplenic portal pressure was also negative.

On umbilical portography in cases with stage I and II hepatic flow, as estimated by splenoportography (Fig. 2), retrograde flow into the portal vein was rarely seen to reach beyond the carina at the junction of the splenic and superior mesenteric veins (Fig. 2a). The hepatograms obtained were of value in identifying the intrahepatic portal vasculature characteristic of pure schistosomal hepatic fibrosis—namely, the portal vein, its first, second, and third order branches maintained their normal anatomical pattern—that is, no deflection, no reduction in number, no obstruction, no stenosis, no amputations—and their courses were rather straight and appeared as rigid tubes (Figs 1 and 2a). But the size of the portal venous bed showed actual diminution of many of the small fourth order branches and other branches (particularly in

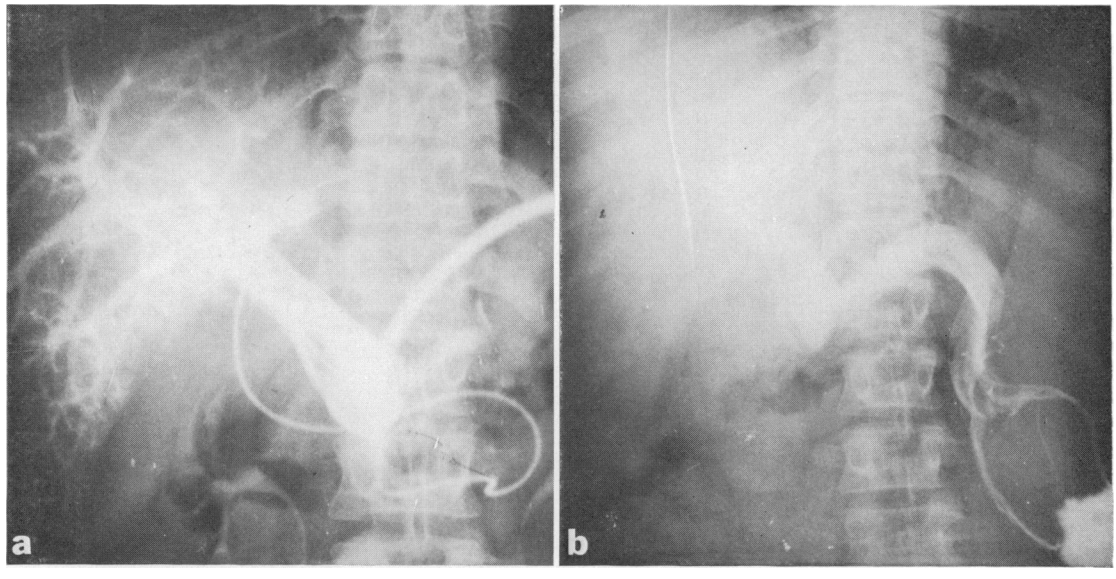

Fig. 2 *Case 10: non-bleeder with a positive transumbilical-transsplenic portal pressure gradient. Transumbilical portography shows* (a) *retrograde portal vein flow up to the junction of the superior mesenteric and splenic veins; the typical schistosomal vascular changes.* (b) *Splenoportogram demonstrates the portal vein with a near normal hepatic portal flow—that is, stage I.*

advanced cases where the liver was shrunken) giving the appearance of a 'withered tree' (Fig. 1). In some cases the main portal vein showed indentations from outside (Fig. 3). These indentations were found at operation to be due to enlarged lymph nodes surrounding the portal vein at the porta hepatis and, in one case, granulomatous nodules were considered to be responsible, as the vein was found to be patent in these cases with no evidences of thrombosis.

Splenoportographies were found to be more accurate in demonstrating the presence and degree of collateral veins and oesophageal varices than barium swallow (Fig. 4). The 28 cases were studied by both procedures, splenoportography showed oesophageal varices in 12 cases while barium swallow showed the varices in only nine of these same cases.

Haemodynamic findings are shown in Table 3. The portal pressures, transumbilical and transsplenic, were significantly higher in the bleeders than in the non-bleeders ($P < 0.005$ and < 0.001 respectively). But the differences in pressure between non-bleeders and asctic patients were not significant. In contrast, the thoracic duct lymph flow was highest in the non-bleeders and, in comparison, it was lower in the bleeders ($P < 0.01$) and least in ascitic patients ($P < 0.005$).

When these cases were analysed according to the type of gradient, the non-bleeders with a positive gradient—that is, transumbilical portal pressure higher than transsplenic portal pressure—showed higher thoracic duct lymph flow when compared with the bleeders with positive gradient ($P < 0.02$). In contrast, the pressures, transumbilical and transsplenic, showed a significant increase in the bleeders when compared with non-bleeders ($P < 0.05$ and < 0.01 respectively). Similarly, the ascitics with positive gradients showed significant diminution in the thoracic duct lymph flow when compared with non-bleeders ($P < 0.005$, Table 4).

All cases with positive gradients (non-bleeders, bleeders, or ascitics) were associated with stage I or II hepatic blood flows as estimated from splenoportography (Table 2, Fig. 2b). In contrast, all cases associated with negative gradients (non-bleeders, bleeders, or ascitics) were associated with stage 3 or 4 hepatic blood flow as estimated from splenoportography (Fig. 1). This denotes that, in cases with stage III and IV, not only was the liver markedly deprived of its portal blood flow, but (as shown in umbilical portography) there was a retrograde flow of hepatic blood in nearly all these cases.

Splenectomy effected an immediate reduction in the transumbilical portal pressure in 22 cases. When measured four to five days later *via* the cannulated umbilical vein, the pressure started to rise, but did not reach its presplenectomy level in 15 cases, and only in one case (no. 6) did it reach its presplenectomy level. Splenectomy had no effect on the transumbilical portal pressure in cases 2 and 7. In case 7 portal vein thrombosis was suspected on splenoportogram (Fig. 5a) and confirmed by umbilical vein portography (Fig. 5b) and dynamics (negative gradient with a very small transumbilical portal pressure); this explained the finding. In case 2 no similar data were demonstrable to provide an explanation.

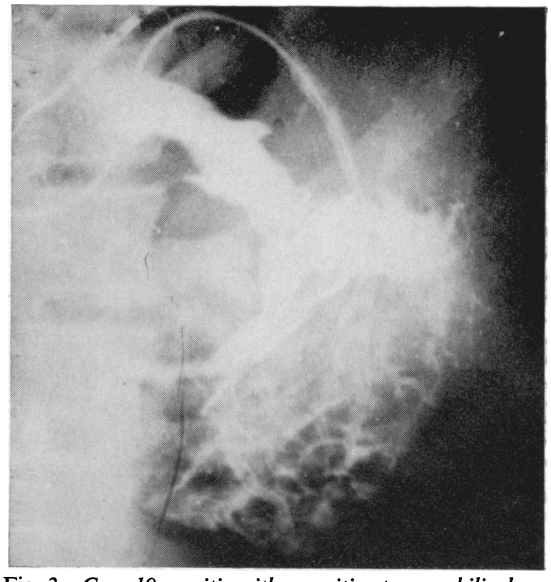

Fig. 3 *Case 19: ascitic with a positive transumbilical-transsplenic portal pressure gradient. Transumbilical portography shows (1) retrograde portal vein flow only as far as the junction of the superior mesenteric and splenic veins (the portal vein was visualised in splenoportography of the same case and showed stage II hepatic portal blood flow); (2) the characteristic periportal neovascular formations; and (3) indentations of the main portal vein which were found at operation to be due to pressure by enlarged lymph nodes in the porta hepatis.*

Discussion

Portal hypertension is a syndrome generated by contrasting haemodynamic patterns (Witte *et al.*, 1972; El-Gendi and Gemeuh, 1977). In this complex syndrome multiple changes occur in the vascular physiology of the splanchnic system (Warren *et al.*, 1967). Thus, a uniform approach to portal decompression will be unsatisfactory.

The transumbilical-transsplenic portal pressure gradient reflects the degree of portosystemic shunts

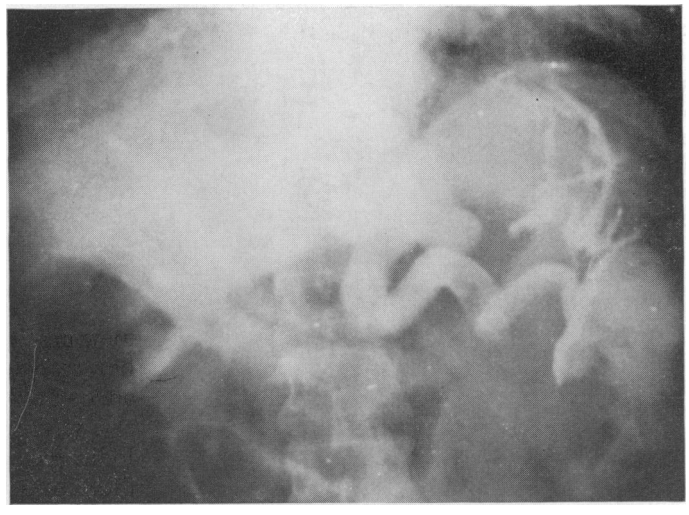

Fig. 4 *Case 11: bleeder with a negative transumbilical-transsplenic portal pressure gradient. Splenoportogram shows faint opacification of the portal vein (stage III hepatic portal vein flow) with preferential flow of the dye into the huge cephalic collaterals present.*

Table 3 *Haemodynamic findings in 28 cases of schistosomal hepatosplenomegaly and portal hypertension at different stages*

Case no.	*TUPP cm. saline*	*TSPP cm. saline*	*Gradient*	*TUPP after op.*	*TDF ml/min*	*TDOP cm. lymph.*	*Stage of liver flow*
Non-bleeders							
1	37	19	+18	24	7	11·5	I
2	33	24	+ 9	33	2	61	II
4	52	32	+20	37	8	22	I
5	34	30	+ 4	27	4·5	35	II
7	10	39	−29	10	8	37	IV
8	30	25	+ 5	28	2	9	II
9	37	30	+ 7	22	4·5	26	II
10	30	25	+ 5	25	5	45	I
12	30	33	− 3	20	3	28	III
14	18	13	+ 5	13	2·5	50	II
17	38	32	+ 6	33	6	67	II
18	32	28	+ 4	30	2·8	25	II
Mean	31·75	27·5			4·608		
SD	10·367	6·895			2·230		
SE	2·992	11·990			0·643		
Bleeders							
3	40	34	+ 6	26	1	20	II
6	50	37	+13	31	3	31	I
11	44	51	− 7	27	1	25	IV
13	38	30	+ 8	15	1	25	II
15	45	32	+13	30	3	32	II
16	42	54	−12	28	3·5	36	
Mean	43·166	39·666			2·083		
SD	4·215	10·250			1·200		
SE	1·721	2·045			0·489		
P	<0·005	<0·001			<0·01		
Ascitics							
19	35	32	+ 3	35	0·3	32	II
20	14	34	−20	9	4·2	22	IV
21	33	39	− 6	30	2	38	III
22	25	27	− 2	23	1·5	29	III
23	34	28	+ 6	23	1·2	28	II
24	35	30	+ 5	32	2	50	II
25	17	32	−15	12	3·2	25	IV
26	39	33	+ 6	21	0·9	42	II
27	22	34	−12	20	2·9	32	IV
28	37	29	+ 2	30	2·2	46	II
Mean	28·5	31·8			2·04		
SD	8·488	3·521			1·159		
SE	2·684	1·113			0·366		
P	>0·4	>0·05			<0·005		

P, compared with non-bleeders.
As estimated on splenoportaphy (Warren, 1967).

Table 4 *Haemodynamic finding in patients with positive TUPP-TSPP gradient: non-bleeders, bleeders, and ascitics and correlation with liver flow on splenoportography*

Case no.	*TUPP cm. saline*	*TSPP cm. saline*	*Gradient*	*TDF ml/min*	*Stage of liver flow*
Non-bleeders					
1	37	19	18	7	I
2	33	24	9	2	II
4	52	32	20	8	I
5	34	30	4	4½	II
8	30	25	5	2	II
9	37	30	7	4½	II
10	30	25	5	5	I
14	18	13	5	2½	II
17	38	32	6	6	II
18	32	38	+	2·8	II
Mean	34·1	25·8		4·43	
SD	8·504	6·064		2·117	
SE	2·689	1·919		0·669	
Bleeders					
3	40	34	6	1	II
6	50	37	13	3	I
13	38	30	8	1	II
15	45	32	13	3	II
Mean	43·25	33·25		2	
SD	5·377	2·986		1·154	
SE	2·688	1·493		0·577	
P	<0·05	<0·01		<0·02	
Ascitics					
19	35	32	3	0·3	II
23	34	28	6	1·2	II
24	35	30	5	2·0	II
26	39	33	6	0·9	II
28	31	29	2	2·2	II
Mean	34·8	30·4		1·32	
SD	2·863	2·073		0·785	
SE	1·280	0·927		0·351	
P	>0·9	>0·05		<0·005	

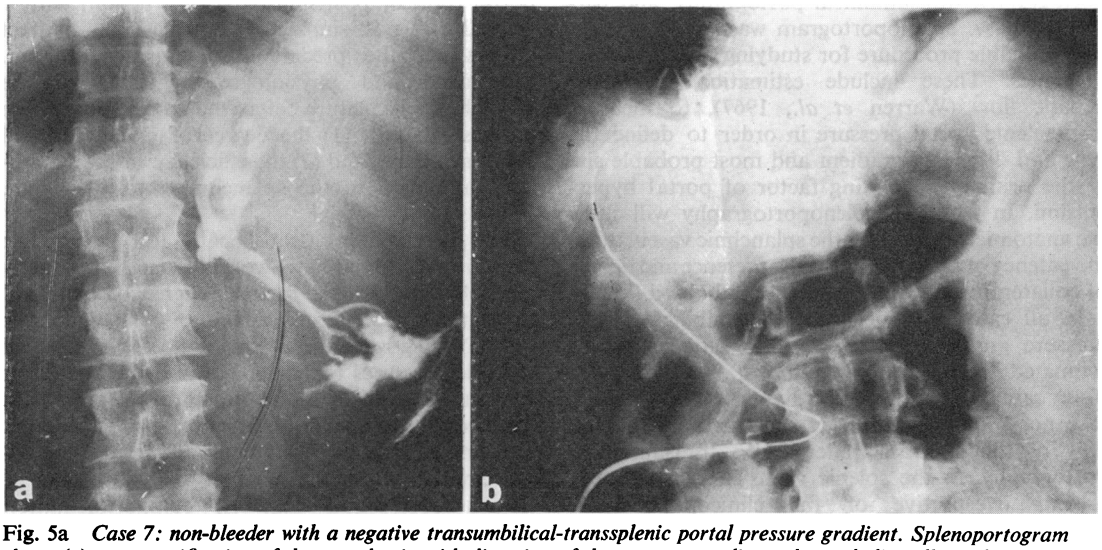

Fig. 5a *Case 7: non-bleeder with a negative transumbilical-transsplenic portal pressure gradient. Splenoportogram shows* (a) *non-opacification of the portal vein with diversion of the contrast media to the cephalic collaterals;* (b) *transumbilical portography showing the dye arrested at the upper end of the portal vein thrombus.*

and collateral veins. Besides, it points to the most probable site of the primary generating factor of portal hypertension. For instance, if the transumbilical portal pressure is higher than the transsplenic (+ve gradient), the primary generating factor of portal hypertension is most probably of hepatic origin (post or presinusoidal, organic and/or functional). While in cases with the transsplenic portal pressure higher than the transumbilical (−ve gradient), the primary generating factor of portal hypertension is most probably of splenic origin (hyperdynamic spleen) or portal vein (thrombosis). The gradient may be mild to moderate (between 1 and 10) or high (above 10). This is governed by the portosystemic shunts developed in each case individually: for instance, cases with mild-moderate gradient—that is, the nearer the two pressures in levels, the less the amount of blood shunted. In contradistinction, a high gradient is due to dispersion of the portal pressure and flow via opening up of portosystemic shunts and collaterals (El-Gendi and Gemeuh, 1977).

From this study we found that the pressures obtained in the bleeders were higher than those in non-bleeders or ascitics. In contrast, the thoracic duct lymph flow was highest in the non-bleeders and lowest in the ascitics. A similar finding was obtained when the cases (bleeders, non-bleeders, or ascitics) with positive gradient were compared. The number of cases with negative gradient were too few for useful statistical analysis.

To study the changes in the vascular physiology of splanchnic circulation we relied mainly on splenoportography, transumbilical portography, and barium swallow. Splenoportogram was found to be a very valuable procedure for studying portal haemodynamics. These include estimation of portal hepatic flow (Warren *et al.*, 1967), to measure transsplenic portal pressure in order to define the type and degree of gradient and most probable site of the primary generating factor of portal hypertension. In addition, splenoportography will show the anatomical changes in the splanchnic vasculature, the patency of the portal vein, the presence and degree of collateral veins, and the directional blood flow.

In all cases with negative transumbilical portal pressure gradient the liver portal blood flow, as estimated from splenoportograms, was grade III (four cases) and grade IV (five cases). All except case no. 7 of these same cases showed retrograde flow of the contrast material, on transumbilical portography, to the splenic vein. This change was present irrespective of the clinicopathological group (bleeders, non-bleeders, or ascitics) and the degree of gradient (high or low). This phenomenon could be due to an aetiological factor(s)—the primary generating factor of portal hypertension was of extrahepatic origin (hyperdynamic spleen, or portal vein thrombosis)—or a haemodynamic factor(s)—that is, it had a hepatic origin (post- or pre-sinusoidal) where the liver resistance to blood flow (arterial and/or portal) became very high and the portal vein showed retrograde flow and acted as a vent. Thus, this finding may be of value in the selection of the proper line of treatment. For instance, cases with a negative gradient will need splenectomy (for those who are hyperdynamic) or selective distal decompression.

In cases with a positive gradient, the hepatic venous flow as estimated from splenoportograms was usually stage I or II. In nearly all such cases the hepatic blush and intrahepatic vasculature were well demonstrated. On umbilical portography, retrograde flow of contrast media was shown in the main portal vein only in some cases. Perioesophageal collateral veins and filling of the left gastric vein were found in many of these cases, especially those complicated by haematemesis.

Thus one can say that the directional flow of the portal venous blood on splenoportography and transumbilical portography will be governed by many factors. These can be divided into direct and indirect factors. Direct factors are the underlying aetiopathological process—namely, (1) resistance to transsplenic blood flow: the presence or absence of intrasplenic arteriovenous shunt will play a great role in producing the hyperdynamic spleen with its consequences on the portal flow and pressure; (2) resistance to transhepatic blood flow: portal (pre- or post-sinusoidal obstruction) and/or arterial (in post-sinusoidal obstruction). Indirect factors are the predetermined, already present, anatomical and physiological patterns in the splanchnic vasculature before the start of the disease process—that is, (1) the degree of collaterals and shunt available; and (2) the efficiency of the lymphatic circulation in the splanchnic area (El-Gendi, 1978).

The direct factors (transhepatic and transsplenic resistances to blood flow) working together or singly aided by the indirect factors (degree of collateral circulation and the efficiency of the lymphatic system) will govern and explain not only the regional variable pressures obtained, but also the altered or sometimes reversed blood flows. In other words, these could provide an explanation for the different pressures obtained in the greater and lesser splanchnic circulation in the absence of valves and/or venous obstruction—that is, compartmentation in the splanchnic circulation in cases of schistosomal hepatosplenomegaly and portal hypertension.

Most of the cases with a positive gradient and liver

biopsy of pure schistosomal hepatic fibrosis, with stage I and II hepatic blood flow on splenoportography, showed highly satisfactory hepatograms on transumbilical portography, which demonstrated quite clearly the underlying schistosomal changes and degree of the liver pathology in each case. These findings were found to be useful in the selection of the most appropriate surgical treatment. For patients who are not bleeders or ascitics the only indications for splenectomy are the presence of hypersplenism, mechanical effects attributed to the grossly enlarged spleen that is usually present, and/or hyperdynamic spleen. For bleeders, if the pathology is mainly schistosomal (presinusoidal) with a positive gradient (stage I or II portal flow on splenoportography) a non-shunt (gastroesophageal decongestion with splenectomy) procedure is indicated, but in cases with an added postsinusoidal element (mixed), selective decompression (Warren *et al.*, 1967) may be indicated in some cases. For ascitics with positive gradient (stage I or II portal flow on splenoportography), the state of the lymphatic circulation and the type of ascitic fluid should be studied before selecting the proper line of treatment (El-Gendi, and Gemeuh, 1977).

Splenoportography is the classical method for estimating portal haemodynamics. But there are instances in which this procedure is too hazardous or cannot be done. In some other cases it cannot differentiate between organic occlusion of the portal vein and functional non-opacification. This situation was seen in most cases with the negative transumbilical-transsinusoidal portal pressure gradient met with in this study, where non-opacification of the portal vein was due to reversed portal vein blood flow and the contrast media was diverted into the collateral circulation. When properly done, transumbilical portography can solve this diagnostic problem; in addition, it visualises the intrahepatic vasculature and provides a direct portal pressure measurement to find out the gradient. In other words, it is a complementary diagnostic tool to splenoportography. Still, umbilical portography is more valuable to follow up the portal pressure after operation and it should be repeated on the fifth postoperative day to study the splanchnic vasculature after the operation. Preoperatively, splenoportography was found to be superior to barium swallow in demonstrating oesophageal varices. But, postoperatively barium swallow has its place in following up cases.

Conclusions

The anatomical and physiological changes in the hepatic and splanchnic vasculature can be safely investigated by barium swallow, splenoportography, and transumbilical portography.

Splenoportography, transsplenic portal pressure, transumbilical portography, transumbilical portal pressure and thoracic duct lymph flow were found to be the most useful parameters in the preoperative assessment of portal hypertension. They can help to establish the diagnosis of portal hypertension, the patency of the portal vein, the presence, degree, and site of collateral veins, and to estimate the intrahepatic portal blood flow.

Umbilical vein catheterisation provided haemodynamic and radiographic data which were of help in diagnosing and in differentiating extrahepatic from intrahepatic portal hypertension.

Rational therapy demands thorough estimation of portal haemodynamics, the possible changes in the splanchnic vasculature, and the clinicopathological stage. Only with these data at hand can selection of the appropriate treatment for portal hypertension be made for each case. When this is achieved better results can be expected and the possible changes after operation can be predicted.

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