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Contrasting Haemodynamic Patterns of Portal Hypertension in Hepatosplenic Schistosomiasis

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Summary

18 cases of hepatosplenic schistosomiasis taken at random were studied. The transumbilical portal pressure, the trans-splenic portal pressure, the occluded thoracic duct pressure, and thoracic duct lymph flow were measured simultaneously before and after splenectomy. The findings were correlated with the clinical data, liver pathology, and operative findings. The transumbilical portal pressure-trans splenic portal pressure gradient was found of significant importance. It was considered positive when the transumbilical portal pressure was higher than the trans-splenic portal pressure, in such cases the primary generating factor of portal hypertension is most probably of hepatic origin (post or presinusoidal obstruction; organic or functional). The gradient was considered negative when the transumbilical portal pressure was lower than the trans-splenic portal pressure; in such cases the primary generating factor of portal hypertension is most probably of splenic origin (hyperdynamic spleen) or in the portal vein (thrombosis). A significant correlation was found between the type of this gradient, the thoracic duct lymph flow and the clinico-pathological state. The thoracic duct flow was highest in the non-bleeders. Definite contrasting haemodynamic patterns were found between bleeders and non-bleeders. The bearing of these contrasting patterns on the selection of the operative procedure in the treatment of portal hypertension is suggested.

Introduction

Portal hypertension is a complex syndrome produced by different circulatory dynamics. Some authors believe in the foreward flow theory (1, 2). Others believe in the backward flow theory (3, 4), and some believe in the two theories (5). However, if portal hypertension is a syndrome generated by contrasting circulatory dynamics, a uniform approach to portal decompression may not be satisfactory (6). For instance, in the presence of increased portal blood flow, portal diversion operation may have deleterious metabolic and haemodynamic side effects. Likewise, splenectomy and gastro-oesphageal decongestion may fail to decompress the portal bed when the bulk of portal flow arises as in the normal situation from the superior mesenteric circulation. For these reasons we have studied different haemodynamic parameters in patients with hepatosplenic schistosomiasis taken at random and the pressures and flows obtained were correlated with the clinico-pathological state.

Material and methods

This study was done on 18 cases of hepatosplenic schistosomiasis mansoni. Diagnosis of schistosomiasis was based on the finding of schistosomal ova in the faeces and/or by rectal biopsy, history of the case, clinical examination for the presence of cirrhotic liver, splenomegaly and other evidences of schistosomiasis and portal hypertension. Each case was studied as follows:

1. Barium swallow and/or oesophagoscopy for oesophageal varices.
2. Splenoportography.
3. Under local anaesthesia the obliterated umbilical vein was cannulated to enter the portal vein. The portal pressure was estimated and transumbilical portography done before splenectomy.
4. The thoracic duct was exposed and cannulated in the neck just before opening the abdomen.

Immediately before laparotomy the following dynamic parameters were determined simultaneously i.e. trans-splenicportal pressure, transumbilical portal pressure, thoracic duct occluded pressure and thoracic duct flow per minute. All pressures were measured in cm. saline and flows in ml. lymph per minute. Through a left paramedian, the abdomen was then entered. The state of the liver was assessed and wedge liver biopsy taken. The size

of spleen, the presence of perisplenic adhesions, collaterals, and a thrill on the splenic hilum were looked for. The degree of retroperitoneal, perigastric, and perioesophageal collaterals were assessed particularly in cases with oesophageal varices. Splenectomy was done in all cases for the presence of hypersplenism, for the mechanical effects attributed to huge splenomegaly present in nearly all cases, and for its possible haemodynamic role in some cases. Non of these cases were ascitic. The transumbilical portal pressure and thoracic duct flow and pressure were re-estimated. In cases with a history of haematemesis, gastro-oesophageal decongestion was added as a treatment for the varices. Umbilical vein portography was repeated 4–5 days after operation. The transumbilical portal pressure was measured daily till the catheter was removed after the portography.

Results

The results obtained are shown in tables 1–6. Table (1) shows the clinico-pathological data obtained in the 18 cases (bleeders and non-bleeders) studied.

Table (2) demonstrates the haemodynamic patterns met with. It shows that portal pressures, transumbilical and transsplenic, were significantly higher in the bleeders than in the non-bleeders ($P < 0.005$ and < 0.001 respectively). In contrast, thoracic duct flow was higher in the non-bleeders than in the bleeders ($P < 0.01$).

Table (3) demonstrates that cases with a positive transumbilical portal pressure-trans splenic pulp pressure gradient bleeders and non-bleeders when compared to cases with negative gradient, bleeders and non-bleeders, were associated with non-significant difference in the thoracic duct flow ($P > 0.9$). Similarly the T.U.P.P. did not show a significant change between the two groups, but the T.S.P.P. showed a significant increase ($P < 0.01$).

The non-bleeders with a positive T.U.P.P.-T.S.P.P. gradient showed higher flows in the thoracic duct per minute (table 4), especially when compared with the bleeders associated with positive gradient ($P < 0.02$). Similarly, the T.U.P.P. and the T.S.P.P. showed significant changes when compared in the two groups, non-bleeders and bleeders, P was < 0.05 and < 0.01 respectively.

The number of cases associated with a negative T.U.P.P.-T.S.P.P. gradient were too small to draw statistically significant data (table 5).

Table (6) shows that cases of pure schistosomal hepatic fibrosis were usually accompanied with a significantly lower transumbilical portal pressure, ($P < 0.01$) and a non-significant drop ($P > 0.7$) in the thoracic duct flow per minute when compared to cases of schistosomal hepatic fibrosis with superadded cirrhosis (nutritional or post necrotic) called "Mixed".

The effect of splenectomy on the thoracic duct lymph flow was variable. It showed no change in two cases, an increase in four cases, and a decrease in eleven cases. These variable results may find an explanation in the fact that this was an immediate post-splenectomy estimation, hence the effects of different factors i.e.

Table 1 Clinico-pathological findings in the 18 cases of hepato-splenic Schistosomiasis and portal hypertension taken at random: 12 non-bleeders and 6 bleeders.

Group	Case No.	Liver		Collaterals	Spleen		
		Size*	Biopsy		Size*	Weight in gram	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	+

* = Finger below costal margin
M. = Mixed cirrhosis
Sc. = pure schistosomal fibrosis

Table 2 The T.U.P.P.-T.S.P.P. gradient and the thoracic duct dynamics in the non-bleeders and bleeders.

Group	Case No.	T.U.P.P. cm saline	T.S.P.P. cm. saline	Gradient	T.U.P.P. after operation	Thoracic duct	
						Flow ml./ minute	Pressure cm. lymph.
Non-bleeders	1	37	19	+ 18	24	7	11.5
	2	33	24	+ 9	33	2	61
	4	52	32	+ 20	27	8	22
	5	34	30	+ 4	27	4 1/2	35
	7	10	39	− 29	10	8	37
	8	30	25	+ 5	28	2	9
	9	37	30	+ 7	22	4 1/2	26
	10	30	25	+ 5	25	5	45
	12	30	33	− 3	20	3	28
	14	18	13	+ 5	13	2.5	50
	17	38	32	+ 6	33	6	67
	18	32	28	+ 4	30	2.8	25
Mean		31.75	27.5			4.608	
S.D.		10.367	6.895			2.230	
S.E.		2.992	1.990			0.643	
Bleeders	3	40	34	+ 6	26	1	20
	6	50	37	+ 13	31	3	31
	11	44	51	− 7	27	1	25
	13	38	30	+ 8	15	1	25
	15	45	32	+ 13	30	3	32
	16	42	54	− 12	28	3.5	36
Mean		43.166	39.666			2.083	
S.D.		4.215	10.25			1.200	
S.E.		1.721	2.045			0.489	
P.		0.005	0.001			0.01	

T.U.P.P. = Transumbilical portal pressure.
T.S.P.P. = Trans-splenic portal pressure.

anaesthesia, operative trauma, fluid losses, etc. On the other hand, splenectomy effected a reduction in the transumbilical portal pressure in 16 cases and no drop in two cases. This drop in the transumbilical portal pressure is immediate after splenectomy. When measured 4–5 days later, through the cannulated umbilical vein, the transumbilical portal pressure started to rise again, but it did not reach the pre-splenectomy level in 15 cases. In one case (No. 6) the T.U.P.P. reached its pre-splenectomy level on the 5th post-operative day. Splenectomy showed no effect on the transumbilical portal pressure in two cases (case No. 2 and 7).

In case number 7, portal vein thrombosis was suspected on splenoportography and confirmed by umbilical vein portography, this explains the finding. In case number 2 no similar finding was demonstrable, even the liver biopsy was not available and one can not offer an explanation. The radiographic changes will be the subject of another communication.

Discussion

The increased thoracic duct lymph flow is a hallmark of hepatic cirrhosis whether or not ascites is present (7). Two different haemodynamic patterns in portal hypertension are

Table 3 Thoracic duct flow in cases with a positive T.U.P.P.-T.S.P.P. gradient as compared to cases with a negative gradient.

Gradient	Case No.	T.U.P.P. cm. saline	T.S.P.P. cm. saline	Degree of gradient	T.D.F. ml. per minute
Positive	1	37	19	18	7
	2	33	24	9	2
	3	40	34	6	1
	4	52	32	20	8
	5	34	30	4	4.5
	6	50	37	13	3
	8	30	25	5	2
	9	37	30	7	4.5
	10	30	25	5	5
	13	38	30	8	1
	14	18	13	5	2.5
	15	45	32	13	3
	17	38	32	6	6
	18	32	28	4	2.8
	Mean	36.714	27.928		3.735
	S.D.	8.668	6.305		2.169
	S.E.	2.317	1.685		0.580
Negative	7	10.	39	29	8
	11	44	51	7	1
	12	30	33	3	3
	16	42	54	12	3.5
	Mean	31.5	44.25		3.875
	S.D.	15.609	9.912		2.954
	S.E.	7.804	4.956		1.477
	P >				0.9

Table 4 Thoracic duct flow in non-bleeders with a positive T.U.P.P.-T.S.P.P. gradient as compared to bleeders with a positive gradient.

Gradient	Case No.	T.U.P.P. cm. saline	T.S.P.P. cm. saline	Degree of gradient	T.D.F. ml. per minute
Non-Bleeders	1	37	19	18	7
	2	33	24	9	2
	4	52	32	20	8
	5	34	30	4	4.5
	8	30	25	5	2
	9	37	30	7	4.5
	10	30	25	5	5
	14	18	13	5	2.5
	17	38	32	6	6
	18	32	28	4	2.8
	Mean	34.1	25.8		4.43
	S.D.	8.504	6.069		2.117
	S.E.	2.689	1.919		0.669
Bleeders	3	40	34	6	1
	6	50	37	13	3
	13	38	30	8	1
	15	45	32	13	3
	Mean	43.25	33.25		2
	S.D.	5.377	2.986		1.154
	S.E.	2.688	1.493		0.577
	P <	0.05	0.01		0.02

Table 5 Thoracic duct flow in non-bleeders with a negative T.U.P.P.-T.S.P.P. gradient as compared to bleeders with a negative gradient.

Gradient	Case No.	T.U.P.P. cm. saline	T.S.P.P. cm. saline	Degree of gradient	T.D.F. ml. per minute
Non-bleeders	7	10	39	29	8
	12	30	33	3	3
	Mean	20	36		5.5
	S.D.	14.142	4.242		3.535
	S.E.	10.001	3		2.5
Bleeders	11	44	51	7	1
	16	42	54	12	3.5
	Mean	43	52.5		2.25
	S.D.	1.414	2.211		1.767
	S.E.	1.000	1.500		1.250
	P	> 0.1	< 0.05		> 0.3

distinguished: post-sinusoidal portal hypertension in which wedge hepatic vein pressure is very high and presinusoidal portal hypertension, as found in prehepatic cases of portal hypertension in which wedge hepatic vein pressure is within normal limits (8).

In hepatosplenic schistosomiasis normal wedge hepatic vein pressure was found in around 70 % of cases and slightly elevated pressures in 30 % of cases (9). Simultaneous measurement of wedge hepatic vein pressure and intrasplenic pressure disclosed another important feature of hepatosplenic schistosomiasis. Splenic pressure is almost always very high so that a pressure gradient exists between the splenic and wedge hepatic vein pressure (10).

Table 6 Thoracic duct Flow and T.U.P.P.-T.S.P.P. gradient in pure and Mixed Schistosomal cases.

Group	Case No.	T.U.P.P. cm. saline	T.S.P.P. cm. saline	T.U.P.P.-T.S.P.P. gradient	T.D.F. ml/min.
Pure cases	7	10	39	−29	8
	8	30	25	+5	2
	10	30	25	+5	5
	11	44	51	−7	1
	12	30	33	−3	3
	14	18	13	+5	2 1/2
	15	45	32	+13	3
	16	42	54	−12	3 1/2
	Mean	31.125	34		3.5
	S.D.	12.529	13.742		2.154
	S.E.	4.430	4.859		0.761
Mixed Cases	3	40	34	+6	1
	4	52	32	+20	8
	5	34	30	+4	4 1/2
	6	50	37	+13	3
	9	37	30	+7	4 1/2
	Mean	42.6	32.6		4.2
	S.D.	8.000	2.550		2.564
	S.E.	3.571	1.383		1.146
	P.	0.01	0.6		0.7

It is usually considered that the portal venous pressure is comparable throughout the portal system, and that its measurement at any site between the digestive tract and the liver is similar. During this study we found that the portal pressure level varied in the same patient with the site of measurement, whether via the splenic pulp, omental vein (though not included in the results since it was not measured in all cases) or the cannulated umbilical vein. These measurements were repeated in each case more than twice to be sure of the obtained results, since it was difficult to accept that there is compartmentalization of circulatory dynamics in the portal system in the absence of venous obstruction or valves. This point had been previously investigated on experimental animals where a significant localized venous hypertension relative to the general portal pressure was achieved by arteriovenous fistulae made in the portal circulation (11). More recently embellishment to this point was suggested (12). The present study showed that a pressure gradient does exist between the transumbilical portal pressure measurement and the trans-splenic portal pressure measurement i.e. the two pressures are different in the same patient. Our belief is that the transumbilical portal pressure-trans-splenic portal pressure gradient could reflect the degree of collaterals and portosystemic shunts, besides it points to the generating factor of portal hypertension. For instance, if the transumbilical portal-vein pressure is higher than the trans-splenic portal pressure, the primary generating factor of portal hypertension is most probably post-sinusoidal or presinusoidal hepatic obstruction (anatomical or functional). In such cases the gradient between the two pressures will be positive (T.U.P.P.-T.S.P.P. = + ve gradient) i.e. the trans-splenic portal pressure was lower than the transumbilical portal pressure, inspite of the absence of valves in the portal venous system and/or venous obstruction. This could be explained by the shunting of blood through the natural portosystemic shunts, i.e. that difference in pressures is due to dispersion of the portal pressure and flow via opening up of shunts and collaterals as a compensatory response to the increased portal pressure and/or flow.

In cases with trans-splenic portal pressure higher than the transumbilical portal vein pressure, the primary generating factor of portal hypertension is most probably in the spleen (hyperdynamic spleen) or in the portal vein outside the liver (thrombosis), the gradient in such cases was considered negative.

Therefore, the T.U.P.P.-T.S.P.P. gradient might help in pointing to the most probable primary generating factor of portal hypertension, and thus the choice of the most suitable surgical procedure.

Table 3 demonstrates that cases with a positive gradient (bleeders and non-bleeders) when compared to cases with a negative gradient (bleeders and non-bleeders) showed a non significant change in thoracic duct lymph flow ($P > 0.9$). In other words,when all cases with the positive gradients lumped with the lymph flows were compared with all the cases with the negative gradients

lumped with lymph flows, the thoracic duct lymph flow was nearly the same in each group.

In contrast, when all the cases with the positive gradients were analysed into bleeders and non-bleeders, the results obtained are shown in table (2). There was a significant increase in the transumbilical and the trans-plenic portal pressures in the bleeders than in non-bleeders ($P < 0.005$ and < 0.001, respectively) while, the thoracic duct lymph flow per minute was significantly higher in the non-bleeders ($P < 0.01$). Thus the thoracic duct lymph flows could be correlated from a clinical stand point.

The thoracic duct lymph flow could be considered as a vent for the relief of the increased portal pressure by absorbing the interstitial tissue fluid from the splanchnic capillary bed helping in portal decompression. This point might be of diagnostic help to predict patients susceptible to variceal bleeding. Furthermore, we believe that the more efficient the lymph is drained via the thoracic duct from the splanchnic area, the less is the liability to bleeding varices, the lower the pressures obtained from the portal area, and the less the liability of ascites formation (13, 14).

On the basis of wedge hepatic vein pressure, free hepatic vein pressure may be higher or lower than the transumbilical portal pressure, splenic pulp pressure, clinical and pathological data, it was possible to distinguish three degrees of liver damage in hepatosplenic form of schistosomiasis mansoni, namely, mild, moderate and severe (15).

In this study (Tables 6), we found that in cases with pure schistosomal hepatic fibrosis, the trans-splenic portal pressure, but in cases of schistosomal hepatic fibrosis with superadded cirrhosis, the transumbilical portal pressure was higher than the trans-splenic portal pressure in all cases. This finding is not comparable to that of others who found a linear relation between the wedge hepatic vein pressure-intra splenic gradient, size of the spleen, and liver pathology (15). We did not find such a correlation between size of the spleen, the trans-splenic portal pressure and the transumbilical portal pressure in neither pure schistosomal hepatic fibrosis nor in mixed cases.

Conclusions

- The use of the cannulated umbilical vein, instead of the wedge hepatic vein pressure measurement, provides an easy, safe, and reproducible route for direct portal pressure measurement.
- The transumbilical portal pressure-trans-splenic portal pressure gradient may help in pointing to the most probable primary generating factor of portal hypertension and hence the choice of the surgical procedure.
- Compartmentalization of the circulatory dynamics in the portal system in patients with hepatosplenic schistosomiasis and portal hypertension was found.
- The thoracic duct lymph flows can be correlated from a clinical stand point, it was higher in non-bleeders as compared to bleeders.

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