Liver, biliary and pancreas

Effect of peritoneo-venous shunt on portal pressure

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SUMMARY The cause of variceal bleed after a peritoneo-venous shunt is not known. Portal haemodynamic consequences of a peritoneo-venous shunt are poorly understood. The most critical period after a peritoneo-venous shunt is the early postoperative period when rapid mobilisation of peritoneal fluid occurs. Serial changes in the portal pressure during the early postoperative period have not been recorded. In the present study preoperative wedged hepatic vein (WHV), right atrial (RA) and pulmonary capillary wedged (PCW) pressures, cardiac index (CI), and plasma volume (PV) were measured in five alcoholic cirrhotic patients with tense ascites for up to 20 hours postoperatively. The longterm effect was assessed by repeating the intrahepatic and/or wedged hepatic vein pressures in three of the surviving patients after 10 to 20 months. A significant increase in the circulatory dynamics and portal pressure was seen within two hours after shunt placement. Wedged hepatic vein pressure increased from 27.6 (8.2) mmHg to 37.2 (9.2) mmHg ($p < 0.01$), RA pressure increased from 6.8 (1.5) mmHg to 14.0 (4.3) mmHg ($p < 0.05$), PCW increased from 7.2 (3.5) mmHg to 19.3 (5.7) mmHg ($p < 0.01$), CI increased from 3.4 (0.27) lit/m²/min to 4.3 (0.85) lit/m²/min ($p < 0.05$). This was accompanied by a 34% increase in the plasma volume from 1838.5 (142.1) to 2471.4 (210) ml/m². These derangements were maintained up to 20 hours postoperatively. After 10 to 20 months, repeat measurements revealed a return to preoperative measurements. It is concluded that there is an acute increase in portal pressure after a peritoneo-venous shunt attributed to increased circulating plasma volume, resulting from rapid mobilisation of ascitic fluid after the shunt. A sudden increase in portal pressure might be an important provoking factor for variceal bleeding after peritoneo-venous shunt.

Rupture of oesophageal varices has been reported in 9 to 26% of patients receiving a peritoneo-venous shunt for refractory ascites.¹⁻⁶ Investigations were undertaken to determine the effects of this shunt on hepatic haemodynamics, because an increase⁷ and decrease^{8,9} in portal pressure has been reported. Measurements of wedged hepatic vein pressure were obtained preoperatively and serially during the first 20 hours to evaluate the acute influence of this procedure; repeat studies were obtained after 10 to 20 months to evaluate the longterm effects.

Methods

PATIENTS

Five patients with alcoholic cirrhosis and ascites received a peritoneovenous shunt after they had been treated with and were unresponsive to a 500 mg sodium diet, 250 mg spironolactone and 80-100 mg furosemide daily for four weeks. The preoperative plasma volume, measured by I¹³¹ labelled human serum albumin, was increased from a control mean value of 1500 (163.3) to 1838.5 (142.1) ml/m². Hepatic and cardiac haemodynamics were monitored pre- and postoperatively. Cournand and Swan-Ganz thermodilution catheters were introduced preoperatively in the hepatic vein¹⁰ and pulmonary

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Table Haemodynamic changes after peritoneo-venous shunt

	WHV pressure mmHg				Cardiac index lit/m ² /min				RA pressure mmHg			
	Pre	2 h	20 h	>10 Mo	Pre	2 h	20 h	>10 Mo	Pre	2 h	20 h	>10 Mo
GV	21	29	29	26.4	3	3.3	3.3	3.1	5	12	11	8
CM	30	36	36	29	3.2	3.7	3.5		6	10	11	
FR	34	48	40	28.5	3.7	4.6	4.8		7	21	15	
VH	17	30	26		3.3	5.5	4.4		7	12	12	
EF	36	44	42		3.5	4.5	4.2		9	15	13	
Mean	27.6	37.2	34.6	27.9	3.4	4.3	4.0	3.1	6.8	14.0	12.6	8
(SD)	8.2	9.2	6.9	1.3	0.27	0.85	0.62		1.5	4.3	1.6	
P		<0.01	<0.01			<0.05	<0.05			<0.05	<0.05	

	PA mean pressure mmHg				PCW pressure mmHg				SVR dynes/sec/cm ⁵			
	Pre	2 h	20 h	>10 Mo	Pre	2 h	20 h	>10 Mo	Pre	2 h	20 h	>10 Mo
GV	14.8	32	30	18	7.4	17	13	6	1485	1351	1157	1434
CM	11	18	15		2	10.5	10		1170	1016	978	
FR	16.5	32	26		9.7	23	18		797	676	560	
VH	10	31	23		6	25	16		964	627	746	
EF	14	23	22		11	21	14		1222	1080	1135	
Mean	13.0	27.2	23.2	18	7.2	19.3	14.2	6	1127.7	949.9	915.2	1434
(SD)	3.3	6.4	5.5		3.5	5.7	3.0		262.0	300.7	257.6	
P		<0.01	<0.01			<0.01	<0.01			<0.05	<0.01	

WHV – Wedged hepatic vein; RA – right atrial; PA – pulmonary artery; PCW – pulmonary capillary wedge; SVR – systemic vascular resistance.

artery¹¹ respectively and left in place for 20 hours postoperatively. The wedged hepatic vein (WHV), right atrial (RA), pulmonary artery (PA) and pulmonary capillary wedge (PCW) pressures, and cardiac output (CI) were obtained before surgery and postoperatively at hourly intervals for the first four hours then every four hours up to 20 hours. Intra-hepatic and/or wedged hepatic vein pressures were repeated in three of the surviving patients at 10 to 20 months after surgery. The plasma volume was determined preoperatively and two and 20 hours postoperatively^{12,13} in two patients.

Results

Before surgery, all the patients showed a raised WHV (27.6 (8.2) mmHg) pressure consistent with alcoholic cirrhosis. The CI (3.4 (0.27) lit/m²/min), mean RA (6.8 (1.5) mmHg), mean PA (13.0 (3.3) mmHg), and PCW (7.2 (3.2) mmHg) pressures were normal (Table). Postoperatively, each of the patients exhibited diuresis. None of the patients developed clinically manifest congestive heart failure or variceal haemorrhage.

Two hours postoperatively, the following haemodynamic changes were observed (Table and Figure): WHV pressure increased ($p<0.01$) from the preoperative value of 27.6 (8.2) mmHg to 37.2 (9.2)

mmHg, mean RA pressure increased ($p<0.05$) from 6.8 (1.5) mmHg to 14.0 (4.3) mmHg and the mean PA pressure increased ($p<0.01$) from 13.0 (3.3) mmHg to 27.2 (6.4) mmHg. This was accompanied by an increase in the PCW pressure from 7.2 (3.5) mmHg to 19.3 (5.7) ($p<0.01$). The CI increased significantly ($p<0.05$) from 3.4 (0.27) lit/m²/min preoperatively to 4.3 (0.85) lit/m²/min. There was a 34% increase in the plasma volume from 1838.5 (142.1) to 2471.4 (210.5) ml/m². Total systemic vascular resistance dropped significantly ($p<0.05$) from 1127.7 (262.0) dynes/sec/cm⁵ preoperatively to 949.9 (300.7) dynes/sec/cm⁵ postoperatively.

Twenty hours after the surgery, repeat measurement of the observed parameters revealed that the haemodynamic changes recorded two hours after the shunt insertion were still maintained. The WHV (34.6 (6.9) mmHg), mean RA (12.6 (1.6) mmHg), mean PA (23.2 (5.5) mmHg), and PCW (14.2 (3.0) mmHg) pressures remained significantly increased (Table and Figure). The CI (4.0 (0.62) lit/m²/min) and the plasma volume (2686.05 (68.6) ml/m²) showed further increase from the values recorded at two hours. The systemic vascular resistance remained low (915.5 (257.6) dynes/sec/cm⁵, $p<0.01$).

After 10 to 20 months (Table and Figure), the WHV pressure was 27.9 (1.3) mmHg, reflecting a

Figure Effect of peritoneo-venous shunt on cardiac output, portal pressure and plasma volume.

reversal to the preoperative value (27.6 (8.2) mmHg). Reevaluation of the systemic haemodynamics in one of these patients at 20 months, revealed no significant difference in the CI (3.1 lit/m²/min) and the mean RA (8 mmHg), PA (18 mmHg) and PCW (6 mmHg) pressures from their respective preshunt values (CI 3.0 lit/m²/min, RA 5 mmHg, PA 14.8 mmHg, PCW 7.4 mmHg).

Discussion

After the insertion of a peritoneovenous shunt there was a rapid mobilisation of the ascitic fluid into the circulating blood volume. This resulted in an increase in the plasma volume and cardiac output. The wedged hepatic vein pressure increased significantly after the insertion of the shunt and remained raised for 20 hours. The expansion of the plasma volume may have contributed to the rise in the portal pressure as a linear relationship exists¹⁴ between portal pressure and plasma volume in cirrhosis. An increase in plasma volume induced by infusion of albumin¹⁵ and dextran¹⁶ produces an increase in portal pressure in patients with cirrhosis. An acute increase in plasma volume is accommodated primarily in the venous system.¹⁷ In patients with cirrhosis, however, the splanchnic venous bed has a limited ability to accept and adjust to an increase in plasma volume.¹⁶ This appears to be because of an increase in hepatic vascular resistance in cirrhosis. The sustained expansion of plasma volume coupled with a limited accommodative capacity of the splanchnic vascular bed is probably the primary mechanism of the postoperative increase in portal pressure observed in the present study after the insertion of the peritoneo-venous shunt.

A sudden increase in portal pressure probably plays an important role in initiating variceal bleed, a complication seen in the early postoperative period after shunt insertion.⁹ Variceal bleed after albumin infusion^{18,19} or ascitic fluid reinfusion²⁰ seems to be closely related to a similar acute plasma volume expansion. Another factor which may precipitate this complication after the insertion of a peritoneo-venous shunt is the use of elastic abdominal binder in the early postoperative period. Its use has been advocated after a peritoneo-venous shunt¹² to increase the thoraco-abdominal pressure gradient and augment the ascitic fluid mobilisation. Our findings indicate that rapid mobilisation of a large amount of ascitic fluid carries the risk of a significant portal and systemic haemodynamic alterations. These findings corroborate the acute increase in portal pressure recorded by Markey and associates⁷ during an episode of variceal bleeding precipitated by peritoneo-venous shunt.

Longterm follow up of these patients revealed that once ascites had resolved and the source of expansion of plasma volume was absent, the cardiac output, the mean right atrial, the mean pulmonary capillary wedge and the portal pressures decreased and these parameters became comparable with the preoperative values.

Discrepancies in the change in portal pressure after a peritoneo-venous shunt insertion reported in the literature seem to be related to the varying postoperative period when the pressures are recorded. Pressures recorded after the ascites has resolved will not reflect the true haemodynamic consequences of such a shunt.

In view of the acute increase in portal pressure that follows a peritoneo-venous shunt, it is advisable that this procedure not be carried out on patients with a recent history of variceal bleed. Elastic abdominal binders, respiratory exercises and other manoeuvre that markedly increase the thoracoabdominal pressure gradient and augment ascitic fluid mobilisation should be used with prudence.

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