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ASCITES FOLLOWING PORTACAVAL SHUNTING

RELATIONSHIP BETWEEN WEDGED HEPATIC VENOUS PRESSURE AND ASCITES

RYUGO HIDEMURA

Second Department of Surgery, Nagoya University School of Medicine

AND

TELFER B. REYNOLDS

Department of Medicine, University of Southern California School of Medicine

ABSTRACT

When present, ascites usually disappears after portacaval shunt. In an occasional patient, however, ascites may appear after portacaval shunt even when not present before operation.

This work reviews the mechanisms of ascites formation following patent portacaval shunt. Hepatic hemodynamic studies were performed in 24 patients with alcoholic liver disease.

The results suggest that ascites is more likely to occur after end-to-side portacaval shunt in those patients who maintain a relatively high sinusoidal pressure and hepatic arterial blood flow.

INTRODUCTION

Ascites is one of the main symptoms in patients with cirrhosis of the liver. Though no one cause is alone responsible for the ascites, the pressure of the blood in the sinusoidal bed is of major importance. Increased sinusoidal pressure in cirrhosis is probably largely due to increased intrahepatic vascular resistance[1)2)3)].

In liver cirrhosis, portacaval shunt is often effective in relieving refractory ascites[4)5)]. Side-to-side shunt is more effective than end-to-side shunt, presumably because sinusoidal pressure is lowered to a greater degree[6)7)]. However, ascites sometimes makes its appearance immediately after a portacaval shunt or reappears when it had spontaneously subsided prior to the operation. Possible explanations for this anomalous development of ascites include (a) clotting of the shunt with increased transudation of fluid from the intestinal capillaries, (b) leakage from hilar hepatic lymphatics transected during the

秀 村 立 五

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shunt operation, (c) decrease in serum albumin incident to the stress of the operation.

In a few patients, postoperative ascites persists beyond the period of time that leakage from transected lymphatics would be expected, with an open shunt, and without a fall in serum albumin.

The purpose of this paper is to review the mechanisms of ascites formation following patent portacaval shunt.

MATERIALS AND METHODS

Ten patients were selected for study during a two-year period because they developed ascites after portacaval shunt. All had alcoholic liver disease with clinical symptoms of portal hypertension leading to elective, end-to-side portacaval shunt at Los Angeles County-University of Southern California Medical Center. Prior to surgery they had no detectable ascites while on a normal salt intake and their serum albumin level was above 3 g/100 ml. There was evidence, either direct or indirect, that the shunt remained patent.

For comparison, we used 14 patients, similar in all respects except for absence of postoperative ascites, who underwent routine postoperative hepatic vein catheterization during the same two year period. The number of patients with and without post-shunt ascites does not reflect accurately the incidence of this event since a special effort was made to study patients with ascites.

Hepatic vein catheterization was performed for measurement of wedged hepatic venous pressure (WHVP). WHVP was recorded on a Statham strain gauge in at least two locations on each patient, usually in the right lobe. Wedging of the catheter was confirmed by contrast media injection after recording pressure. Our zero reference point for wedged pressure measurement is the inferior vena caval pressure. Normal WHVP by our method is 0 to 4 mm Hg and there is no recognizable abrupt pressure drop as the catheter is withdrawn from the wedged position[8,9].

Hepatic blood flow was measured by analysis of the disappearance curve and hepatic extraction of indocyanine green following a single intravenous injection. The exactly weighed indocyanine green was given 0.5 mg per kg body weight. Following injection, blood collections were done from hepatic vein and femoral artery every three minutes for fifteen minutes[10].

RESULTS

WHVP was measured in all of the 24 patients after end-to-side portacaval shunting and in 10 of the 24 patients before shunting. Hepatic blood flow was measured in 13 of 24 patients using the indocyanine green single injection method. Patency of the shunt was ascertained by autopsy, splenoportography or catheterization, or was assumed because of a prolonged period without

TABLE 1. Cases with and without Postoperative Ascites

		WHVP (mmHg) Pre.	Post.	P.V.P. (cmH_2O) Pre.	Post.	H.B.F. (ml/min.) Pre.	Post.	Postoperative Ascites	Shunt Patency	Date of Ope.
1	B.H.		7.1	36	24			none	prob. open*	Apr. 1963
2	D.H.		10	35	20			none	open (c)	Jun. 1963
3	G.J.		6.7	38	23			none	prob. open*	1950
4	R.G.		15.4	33	23			none	open (c)	Jun. 1964
5	M.A.		4.1	28	17			none	open (c)	Jun. 1964
6	B.E.	17	3.9	38	22	1140		none	prob. open*	Mar. 1963
7	S.J.		8.6	35	14			none	open (a)	Apr. 1963
8	G.R.		14.2	45	25		1790	marked for 5 months	open (a)	Apr. 1965
9	O.Y.	17	10.8	30	26		500	moderate for 4 months	prob. open*	Apr. 1965
10	T.M.	20	13.8	46	24			marked for 5 months	open (a)	May 1966
11	E.C.		11.7	37	21			moderate for 3 months	prob. open*	Sep. 1966
12	C.J.	10	9.5	23	25			none	open (sp)	Jan. 1966
13	M.H.		15.3	33	18		1260	marked for 11 months	open (a)	Aug. 1966
14	P.R.		22.4	37	19			moderate for 6 months	prob. open*	Feb. 1964
15	C.C.	12.4	8.9	42	17	650	620	none	prob. open*	Jul. 1966
16	G.H.		5.7	56	26		443	none	prob. open*	Aug. 1964
17	M.S.	12.2	11	40	23	1260	474	none	prob. open*	Oct. 1966
18	S.R.	18.1	15.7	42	31	2700		mild for 1 month	prob. open**	Sep. 1965
19	G.F.	13.9	14.7	39	21	3200	950	mild for 1 month	prob. open**	May 1965
20	O.J.		5.9	41	16		603	none	open (a)	May 1965
21	N.M.		12.2	52	32		880	mild for 3 months	prob. open*	Aug. 1965
22	T.K.		6.6	30	18			none	prob. open**	Oct. 1964
23	V.A.	14	10	28	16		785	none	open (a)	Feb. 1967
24	B.A.	15	16	31	16	1600		moderate for 4 months	open (c)	Mar. 1965

(a): autopsy, (c): catheterization, (sp): splenoportogram, *: presumed open because of no GI bleeding since surgery,
**: presumed open because of no GI bleeding and development of recurrent encephalopathy since surgery.

evidence of variceal bleeding (Table 1). Three patients developed periodic encephalopathy postoperatively which provided additional evidence of an open shunt. The presence and duration of ascites was evaluated from the hospital records.

After end-to-side portacaval shunting, patients with ascites had a higher mean WHVP (14.6 mmHg) than patients without ascites (8.1 mmHg) (Fig. 1). The patients with postoperative ascites had high WHVP before and after shunting, and the patients without ascites after shunting had lower WHVP before and after shunting (Fig. 2).

There is a distinct difference between hepatic blood flow of patients with postoperative ascites and hepatic blood flow of patients without ascites after portacaval shunting (Table 2 and Fig. 3). Patients with postoperative ascites tended to have higher WHVP and a large amount of hepatic blood flow after shunt.

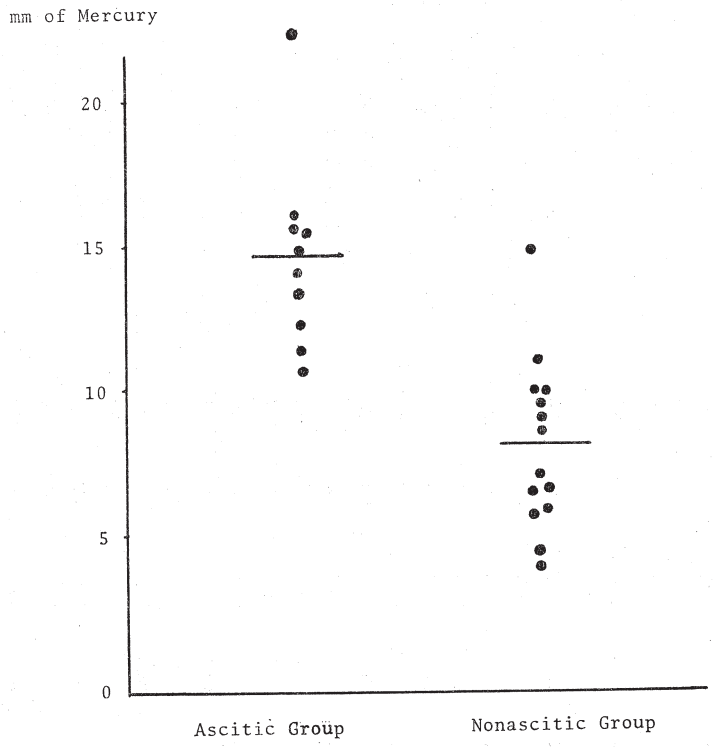

FIG. 1. Relationship between ascites and wedged hepatic venous pressure following portacaval shunting

DISCUSSION

After portacaval shunting, cirrhotic ascites usually disappears without

TABLE 2. Postoperative Hepatic Blood Flow

Ascitic Group		Nonascitic Group	
Case No.	H.B.F. (ml/min/kg)	Case No.	H.B.F. (ml/min/kg)
8	26	15	7.7
9	11.4	16	5.5
13	17.5	17	7.2
19	11	20	7.7
21	16	23	11.5

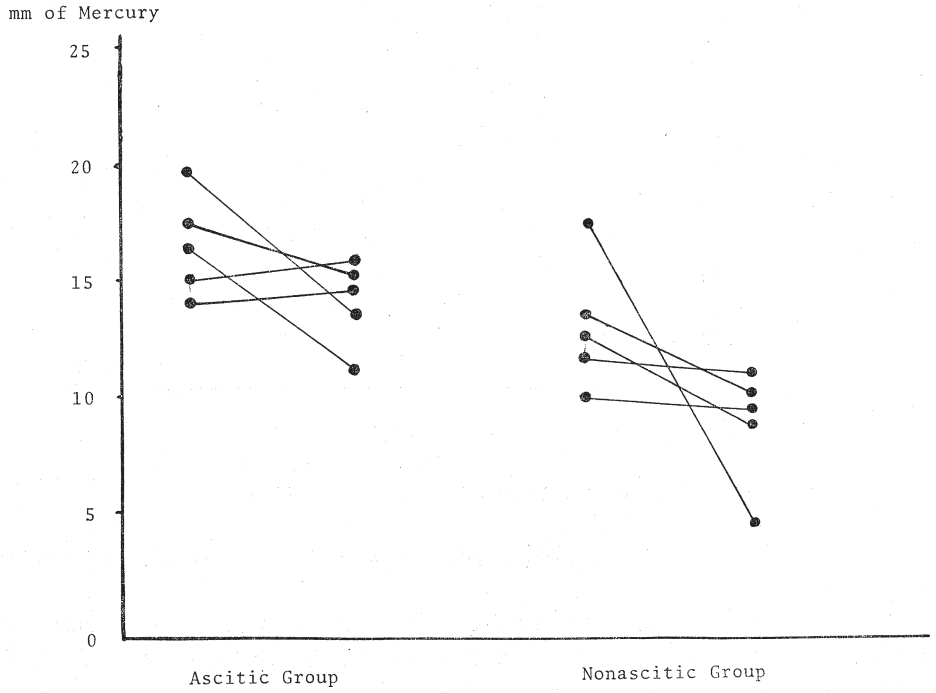

FIG. 2. Difference between preoperative and postoperative wedged hepatic venous pressure.

difficulty. In fact, portacaval shunt has been used for the treatment of intractable ascites[4)5)11)]. Development of ascites after a portacaval shunt when it was not present before the operation is surprising, therefore, and requires explanation. Ascites in this setting can be due to clotting of the shunt with increased transudation of fluid from the intestinal capillaries. Of three patients excluded from this study because of autopsy or splenoportographic evidence of thrombosed shunt, all had ascites postoperatively. However, the great majority of our patients with post-shunt ascites had a patent anastomosis at autopsy and one by catheterization. The remaining six patients have had prolonged observation without recurrence of gastrointestinal bleeding and two

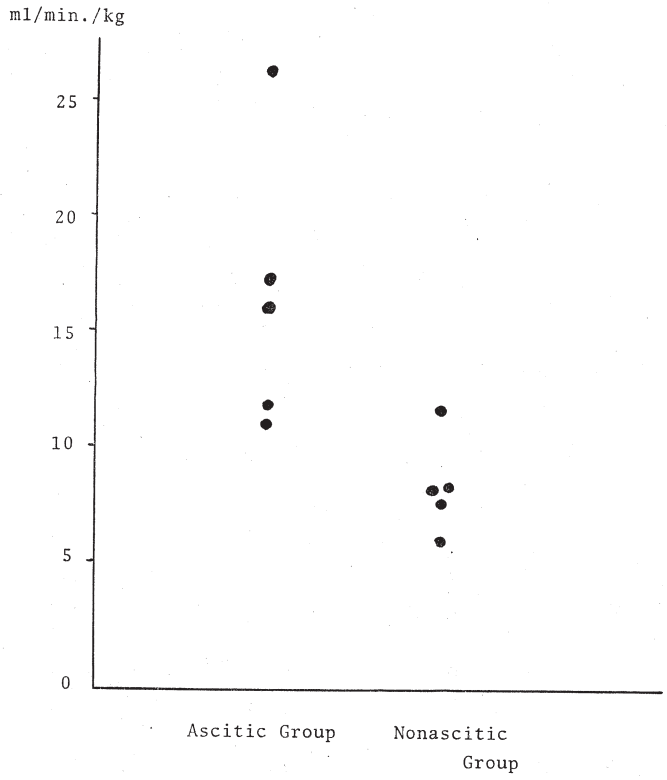

FIG. 3. Postoperative hepatic blood flow.

have developed recurrent encephalopathy so it is unlikely that their shunts have closed.

Ascites formation from the surface of the liver is probably directly related to the degree of pressure elevation in the hepatic sinusoids which in turn is a function of the volume of hepatic blood flow and the resistance in the vascular bed of the liver. Portacaval shunting reduces hepatic blood flow by removing the portal stream. Though there may be some compensatory increase in inflow through the hepatic artery, the usual result is a decrease in total hepatic blood flow and a fall in sinusoidal pressure[9]. The change in hepatic blood flow and sinusoidal pressure should be less in those patients with a greatly diminished portal inflow pre-operatively. Such patients may be more susceptible to the development of post-shunt ascites.

CONCLUSION

Our results suggest that ascites is more likely to occur after end-to-side portacaval shunt in those patients who maintain a relatively high sinusoidal pressure and hepatic arterial blood flow.

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