Clinical experience with the Rhône-Poulenc ascites reinfusion apparatus

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Summary

Ascites reinfusion is not free from complications. The Rhône-Poulenc ascites reinfusion apparatus could be of value in those patients with cirrhosis and ascites who cannot be managed by diuretic therapy.

ONLY six patients with cirrhosis and ascites have been treated with the Rhône-Poulenc ascites reinfusion apparatus. They were admitted 24 hr before the initiation of the treatment in an intensive care unit for patients with liver diseases. On the previous day, and during the procedure, the following were observed hourly: respiratory rate; heart rate; blood pressure; central venous pressure; portal pressure by catheterization of a para-umbilical vein; urine volume and body temperature. The concentration in urine, plasma and ultrafiltrate of urea, creatinine, sodium and potassium as well as osmolality were determined every six hours during the procedure. The volume of ultrafiltrate was also recorded.

All the patients had a normal ECG, chest X-ray and heart on physical examination and none had infection of the ascitic fluid.

Results

The results of the treatment were excellent in the first three cases (Table 1) and these patients were discharged from hospital without ascites. However, the last three patients presented serious complications. One patient died of acute pulmonary oedema. The second patient died from a massive gastrointestinal haemorrhage due to rupture of an oesophageal varix. The third patient died from an acute myocardial infarction. Two of the patients who died had been responding to standard diuretic therapy before starting the procedure.

There were no differences, between the patients

who survived and those who died, in relation to age, duration of the perfusion or amount of ultrafiltrate produced per hr. The total amount of ultrafiltrate produced during the treatment was lower in those patients who had serious complications (Table 1).

The infusion of concentrated ascitic fluid was followed by an increase in urine volume and natriuresis in all patients (Fig. 1). However, there were no significant changes in glomerular filtration rate as measured by the endogenous creatinine clearance.

The most striking features found were the haemodynamic changes induced by the treatment. There was a significant increase in heart rate and central venous pressure in all but one patient (Fig. 2). The changes in blood pressure were only minimal and the respiratory rate only increased significantly in those two patients who had cardiac complications.

Portal pressure increased in two patients, but in the other two it decreased during the procedure (Fig. 3). In the patient who had bled from oesophageal varices, it was surprising that the portal pressure had decreased during the infusion.

FIG. 1. Urine volume (a) and urinary sodium excretion (b) before and during the infusion of concentrated ascitic fluid.

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TABLE 1. Data and evolution of the patients

Age	Response to diuretics	Ultrafiltration			Evolution
		Duration (hr)	Volume (ml)	Ultrafiltrate (ml/hr)	
64	Yes	28	7550	269.6	Good
51	Yes	16	6675	417.2	Good
49	No	62	17,400	280.6	Good
54	Yes	20	6560	378	(+) Left ventricular failure
41	Yes	14	5050	360.7	(+) Gastro-intestinal haemorrhage
65	No	12	5100	425	(+) Myocardial infarction

+, Patients who died.

FIG. 2. Maximal values of heart rate and respiratory rate before and during the infusion of concentrated ascitic fluid. The closed dots represent the values of the patients who had left-sided ventricular failure (LVF) and myocardial infarction (MI). (a) Heart rate; (b) central venous pressure.

FIG. 3. Maximal values of portal pressure before and during the infusion of concentrated ascitic fluid. The closed dots represent the values of the patient who had a gastro-intestinal haemorrhage (GIH).