INTRAVENOUS INFUSION OF DIALYZED, AUTOGENOUS, ASCITIC FLUID IN THE MANAGEMENT OF CIRRHOTIC ASCITES

A Preliminary Report of Favorable Results in Six Patients

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IN recent years the traditional treatment of repeated paracenteses in the management of cirrhotic ascites has waned in favor of attempts to correct the underlying physiologic defects. The relative importance of abnormal renal tubular absorption of sodium, portal hypertension, abnormal osmoreceptor responses, secondary aldosteronism, and reduced protein oncotic pressure in the formation of cirrhotic ascites has been shown.[1-6] Sodium retention appears to be basic to the formation of ascites and, in many patients, strict salt restriction is sufficient to control ascites.[7-9] However, in patients with imminent or actual hemorrhage from esophageal varices, prolonged dietary measures are impractical and potentially dangerous. A reduced surgical mortality in patients free of ascites has been the impetus for seeking a safe rapid means of eliminating or controlling ascites.[10]

Repeated paracenteses reduce total body sodium, but also have the disadvantage of depleting plasma proteins. Infusion of ascitic fluid intravenously has been advocated in the past, but this amounts to a transfer of sodium and water from one extracellular space to another, and the volumes to be infused are prohibitive.[11-13]

Intravenous infusion of concentrated ascitic fluid without water and sodium and with conservation of proteins would be most desirable. This report describes a method of concentrating ascitic fluid and rendering it sodium poor. Such concentration is possible by ultrafiltration and dialysis. This technic has proved practical in nine dialyses of ascitic fluid from six cirrhotic patients. No serious reactions occurred, and ascites formation was arrested after single treatments in five patients, and after four treatments in one patient. Only each patient's own processed fluid was given to him intravenously.

Selection of Patients

All six patients, three men and three women, had postnecrotic cirrhosis as confirmed by liver biopsy studies. Five patients had large volumes of ascites for longer than three months; of these, three patients had had one or more paracenteses with rapid reaccumulation of ascites; the sixth patient had active hepatitis with

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hepatosplenomegaly, thrombosis of the portal vein, confirmed by splenoportography, and actively forming ascites. On a high-protein, high-caloric diet with sodium intake limited to 10 mEq. daily, each patient excreted less than 10 mEq. per liter of sodium in the urine, and gained from one to three pounds in body weight daily.

Technic

The procedure was uniform for each of the six cirrhotic patients. All measurements and analyses, such as of body weight, serum electrolytes, serum proteins, urinary volume and electrolytes, were made immediately before and after paracentesis and before and after infusion of the autogenous ascitic fluid. Ascitic fluid was analyzed for electrolytes, glucose, and osmolality. Samples were used for culture, paper electrophoresis, and cytologic study.

Paracentesis. Trocar paracentesis was performed in the operating room under aseptic conditions. Ascitic fluid was drained into 10-liter sterile plastic bags containing between 20 and 40 mg. of heparin-sodium to prevent clotting of fibrin. The bags were equipped with inflow and outflow tubes.

Dialysis and ultrafiltration. The inflow and outflow tubes on the plastic bag were attached to a twin-coil disposable artificial kidney* having a dialyzing surface of 18,000 sq. cm. Ultrafiltration pressures were manually controlled by a screw clamp on the outflow tube; pressures ranged from 280 to 300 mm. of Hg in the coils. An automatic device† controlled pressure to prevent rupture of the cellophane membranes in the coils.

The 50-l. dialyzing solution of the artificial kidney consisted of 15 per cent dextrose in water. The dextrose, as shown by Lenggenhager,[14] enters the ascitic fluid as sodium leaves it, and prevents the precipitation of protein. Duration of dialysis ranged between 4 and 10 hours, depending upon the volume of ascitic fluid to be processed. One change of the dialyzing solution reduced dialysis time in obtaining ascitic fluid sodium concentrations of less than 10 mEq. per liter. Tetracycline hydrochloride, 2 gm., was added to the rinsing fluid. The usual filters of the artificial kidney help to eliminate particulate matter. Immediately after dialysis and ultrafiltration, the concentrated, salt-poor fluid was stored in sterile 250-ml. containers at 5 C. for overnight.

Infusion. The day after dialysis and ultrafiltration, the treated ascitic fluid was intravenously infused in the patient. The fluid was viscous and was administered through 18-gauge needles. Plastic blood filters were connected in the tubing to collect all residual particulate material not eliminated by dialysis. Infusion time averaged 250 ml. per hour. During infusion and every four hours thereafter, blood pressure, pulse, respiration, and temperature were measured.

**Manufactured by Travenol Laboratory, Inc., Morton Grove, Illinois.*

†Mercoid Control, manufactured by Mercoid Corporation, Chicago, Illinois.

Results

Alterations in ascitic fluid. *Tables 1* and *2* summarize the effects of dialysis and ultrafiltration on composition of ascitic fluid. The average reduction in sodium content to 5.8 mEq. per liter, and concentration of protein to five times that of the original ascitic fluid have resulted in a solution with a salt-poor albumin con-

Table 1.—*Quantitative alterations in ascitic fluid after dialysis and ultrafiltration*

(Average data; nine samples from six patients)

Determination	Before dialysis	After dialysis
Total volume, ml.	7000	750
Total protein, gm./100 ml.	1.6	8.3
Albumin, gm./100 ml.	(0.9)	(4.8)
Globulin, gm./100 ml.	(0.7)	(3.5)
Alpha-1	(0.05)	(0.30)
Alpha-2	(0.06)	(0.31)
Beta	(0.19)	(0.74)
Gamma	(0.52)	(2.12)
Sodium, mEq./l.	130.0	5.8
Potassium, mEq./l.	3.9	0.3
Chloride, mEq./l.	103.0	4.3
Glucose, mg./100 ml.	151.0	10044.0

Table 2.—*Quantitative sodium, water, and albumin exchange after dialysis of ascitic fluid*

(Nine samples from six patients)

Sample	Total sodium removed, mEq.	Total water removed, ml.	Total albumin returned, gm.
1	1071	7500	24.7
2	1081	7430	96.9
3	1536	10300	93.2
4	747	5350	28.6
5	1051	7800	35.7
6	739	5000	30.0
7	272	1700	23.8
8	750	5200	59.0
9	827	5400	17.1

tent approaching that supplied by the Red Cross, or that commercially available. Cultures were sterile before and after dialysis, except for one sample that grew *Staphylococcus albus* before, but not after, dialysis, and was thought to be a contaminant. Cytologic study showed the absence of neoplastic cells, except for one patient with cirrhosis and carcinomatosis whose pleural fluid contained neoplastic cells.

Reactions to infusion. Three patients had mild chills, and fever as high as 102 F., transient in nature, without further reaction during infusion. No significant alterations in serum electrolytes or low-sodium syndrome occurred. Diuresis and natruresis, mild and transient, occurred in all patients.

Clinical responses. Stabilization of ascites occurred in five patients, as shown by stable body weight and clinical examination. Portacaval shunts were performed one week later in two of these patients. The sixth patient regained 45 pounds of ascites the month after his first tap and infusion, 20 pounds in the month after the second infusion, 8 pounds 10 days after the third infusion; ascites has remained stable since the fourth infusion. He had been considered to have "intractable ascites" and required monthly paracentesis despite strict medical management. The ascites in the other five patients has remained stable for as long as four months of follow-up study. With improvement in hepatocellular function on an intensive medical program, increasing sodium excretion in the urine has permitted easing the restriction of sodium intake without recurrence of ascites.

Discussion

Infusion of ascitic fluid, autogenous and homologous, fresh, refrigerated, or lyophilized, had considerable use in the late 1930's, primarily as a substitute for blood in the treatment of shock, nephrosis, and hypoproteinemic states.[15-17] Few serious reactions were observed except with contaminated fluid or homologous fluid; anaphylaxis, fever, urticaria, and chills were reported. In certain clinics, suitably cross-matched ascitic fluid was used in homologous transfusion, while in others no attempt at matching was made, largely with impunity.[15] The ready availability of blood and blood derivatives made infusion of ascitic fluid unnecessary for these purposes.

In 1951, Emmrich and Fliege[18] reported good response to repeated infusion of untreated ascitic fluid in two patients, with fair results in a third patient. It is possible that the benefit obtained was largely due to the associated medical treatment, and to time, rather than to a direct effect upon retention of sodium and water.

The procedure of ultrafiltration and dialysis of ascitic fluid, and infusion intravenously, appears to be practical in removing excessive total body sodium and water without protein loss. Prolonged clinical benefit probably depends more upon improvement in hepatocellular function than in the transient physicochemical alterations that result from infusion of concentrated salt-poor ascitic fluid.

However, if surgical intervention is imperative, rapid preoperative *control* of ascites, is important if surgical mortality is to be lessened.

Summary

1. Controlled reduction of water and sodium content of ascitic fluid without protein loss can be achieved by ultrafiltration and dialysis in a twin-coil disposable artificial kidney.

2. Intravenous infusion of this autogenous, concentrated ascitic fluid appears to be a safe method of reducing excessive total body sodium without protein loss.

3. This technic has been useful in stabilizing ascites in six patients with postnecrotic cirrhosis.

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