Ascitic fluid removal: does it cause renal or hemodynamic decompensation?[1]

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Twenty-seven patients with cirrhotic ascites lost variable amounts of weight (0–19.7 kg) as a result of ascitic fluid removal during peritoneoscopy. No attempt was made to correct these fluid losses, yet no evidence of untoward hemodynamic, renal, or electrolyte abnormalities was found. The apparent safety of ascites removal, even in large amounts, lends support to the overflow theory of the pathophysiology of ascites formation and also suggests that its role in the clinical management of tense ascites should be reevaluated.

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Previously it was taught that removal of ascitic fluid in cirrhotic patients might have deleterious effects by virtue of rapid reformation of ascites at the expense of intravascular volume.[1,2] On the other hand, before the importance of salt restriction was recognized and before diuretics were readily available for the treatment of ascites, removal of large volumes was done frequently.[1] Moreover, when a patient with ascites requires surgery, large volumes of ascitic fluid are frequently removed without untoward consequences. Because of our observations[3] of the relative safety of performing peritoneoscopy in patients with ascites (when variable amounts of fluid must be removed in order to obtain adequate visualization), we reviewed our peritoneoscopy experience to see whether deleterious effects on renal function or clinically measurable correlates of intravascular blood volume could be detected. Specifically we investigated whether there is a relationship between the

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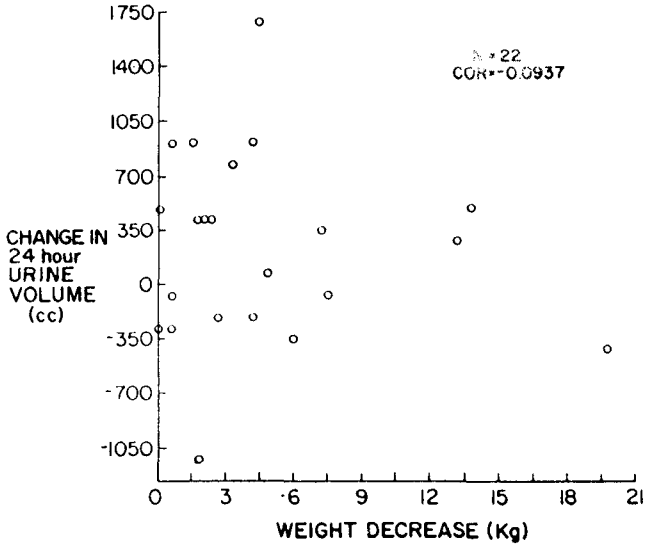

Fig. 1. There is no apparent tendency for urine volume to fall as increasing amounts of acute weight loss occur, reflecting ascitic fluid loss.

volume of ascites removed and the hemodynamic and renal responses.

Methods

The charts of all patients who underwent peritoneoscopy between April 1974 and May 1981 were reviewed retrospectively. Cases of ascites due to cirrhosis were selected for analysis. Correlations between weight loss due to ascitic fluid removal and changes in blood pressure, pulse, urine volume, blood urea nitrogen (BUN), creatinine, and serum electrolytes were recorded.

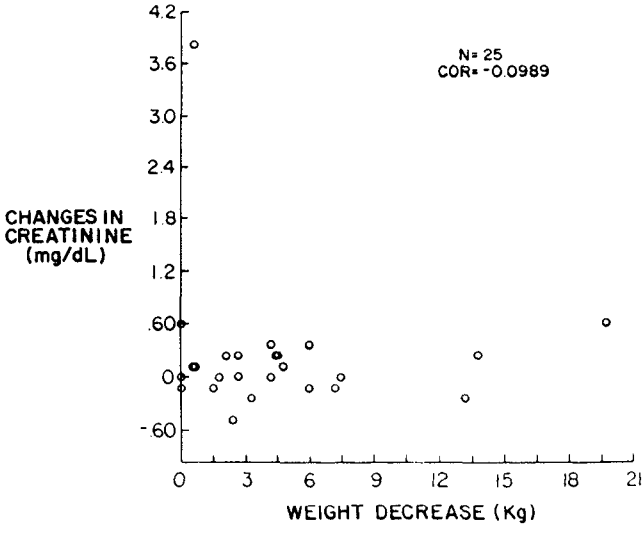

Fig. 2. Serum creatinine does not show any tendency to rise as increasing amounts of acute weight loss occur, reflecting ascitic fluid loss.

Only cases in which paired values (preperitoneoscopy and postperitoneoscopy) were available are included for analysis. In all cases the value closest to day zero (the day of peritoneoscopy) was considered the preperitoneoscopy value, and the "worst case" change occurring within the first five days was taken as the post-peritoneoscopy value. The type and amount of intravenous solutions given, urine volumes, and medications, including diuretics, were also recorded. The three physicians performed the peritoneoscopies in similar ways. When ascites was removed, no attempt was made to replace volume loss with intravenous fluid administration although an intravenous line was established should intravenous fluids have become necessary. Hypotension was defined as a systolic blood pressure less than 100 mm Hg. Tachycardia was defined as a pulse rate greater than 100/min. Laboratory tests were done in the Clinical Laboratory of the Cleveland Clinic Hospital.

Results

Between April 1974 and May 1981, 450 peritoneoscopies were performed. Sixty-two patients had ascites, and in 32 the ascites was secondary to cirrhosis. The 27 patients for whom paired data are available are the subject of this analysis. Peritoneoscopy weight loss varied from 0 to 19.7 kg, with a mean of 4.4 kg, indicating an average loss of 4.4 liters of ascitic fluid. Dietary sodium was restricted to ≤2 g/day in 26 of 27 patients for whom complete data are available. In 17 patients dietary sodium intake was ≤1 g/day.

Neither hypotension nor tachycardia was seen in any patient following peritoneoscopy (N = 27). Renal test results remained substantially unchanged. Urine volume on the day following peritoneoscopy compared to the day before peritoneoscopy varied widely (*Fig. 1*) and was not correlated with weight loss at peritoneoscopy. The mean urine volume decrease of 231 cc/day is of no clinical significance. In no patient whose preperitoneoscopy urine volume was in excess of 500 cc/24 hr did the urine volume fall below 500 cc/24 hr. Levels in 2 patients whose urine volumes were <500 cc/day remained below this level, although one patient had an increase of 35 cc/24 hr and the other a decrease of 70 cc/24 hr. Similarly, changes in serum creatinine were minor and not related to the weight loss that resulted from ascitic fluid removal (*Fig. 2*). The only patient who experienced a substantial rise

in creatinine postperitoneoscopy had unrecognized progressive oliguric renal failure before the procedure. In this patient, in fact, no ascites fluid was removed. No trend suggests that those who lost more ascites were more likely to have a significant change in serum creatinine concentration. Similar data exist for BUN changes.

Electrolyte abnormalities did not develop as a consequence of paracentesis. The majority of patients had a slight decrease in sodium concentration, which averaged 1.75 mEq/L. The degree of change in serum sodium concentration was unrelated to the amount of ascitic fluid removed (N = 24).

Discussion

Observations in the 1950s[4] that renal sodium abnormalities were customarily present in patients with cirrhotic ascites resulted in the treatment of ascites by paracentesis being replaced by dietary salt restriction and natriuretic agents.[5] Further observation that ascites fluid begins reforming within hours of paracentesis led to concern that the ascites was reforming at the expense of intravascular volume.[6] Such ascitic fluid reformation might result in intravascular volume depletion. According to the classic theory, ascites forms because of an imbalance in Starling's forces (caused primarily by portal hypertension and abetted by diminished colloid osmotic pressure). It implies that removal of ascitic fluid should result in rapid reaccumulation of the ascites, even at the expense of intravascular volume.[7] Indeed, experimental data have shown the ascitic fluid space to be in communication with the intravascular space,[8] and Gabuzda and others have repeatedly warned about the potential hazards of large volume ascitic fluid removal in man.[7,9] However, intravascular volume depletion as a result of large volume ascitic fluid removal has not been observed experimentally.

Our data suggest that the predictions of Gabuzda and others are incorrect. Our patients tolerated varying amounts of ascitic fluid removal without any apparent untoward hemodynamic, renal, or electrolyte consequences. Larger weight reductions resulting from ascitic fluid loss had no different effects than smaller weight reductions on any clinical or laboratory feature measured. Had we studied a larger group of patients, we might have found a subset more sensitive to ascitic fluid removal; similarly, had we removed more fluid in each individual, deleterious effects might have been noted. Since we did not measure intravascular volume or renal blood flow directly minor perturbations of intravascular volume may have resulted from ascitic fluid removal. However, at the clinical level, these changes, if present, were too small to be apparent. We have presented only patients for whom paired data are available (*Figs. 1* and *2*). A review of patients for whom paired data are not available similarly reveals no apparent change in clinical status.

Other studies also cast doubt on the deleterious hemodynamic effects of large volume ascites removal. For example, removal of from 2 to 5 liters of ascitic fluid is accompanied by an increase in cardiac output over baseline.[10,11] Shear and Gabuzda showed that their patients did not suffer any untoward reactions from large volume ascites removal.[6] Removal of ascitic fluid from dogs with experimentally induced cirrhosis resulted in no hemodynamic changes.[12] Moreover, there is evidence in both experimental animals and in man that when dietary sodium is severely curtailed (≤10 mEq/day), reformation of ascites does not occur.[5,11] The apparent contradiction between studies that demonstrate rapid ascites reformation and those that suggest that no significant fall in intravascular volume occurs, may be reconciled. Perhaps reformation of ascites occurs at the expense of edema fluid rather than intravascular fluid volume. Also, ascites may reform rapidly without a fall in intravascular volume if free access to dietary sodium is allowed.[12] Many of our patients had edema, but this retrospective analysis did not allow us to know how often it was present.

Our findings also suggest that Starling's forces alone are not the principal ones involved in ascites formation. Instead, they support the "overflow" theory, which holds that the principal physiologic defect in ascites formation is an abnormal signal received by the kidney to conserve sodium.[13] According to this hypothesis, when positive sodium balance persists long enough, ascites will form by "overflowing" from the intravascular pool. If this theory is correct, no deleterious hemodynamic or renal effects would result from removal of this overflow pool.

Our data do not suggest that repetitive large volume paracentesis should be considered appropriate therapy for cirrhotic ascites. Although paracentesis, combined with dietary salt restriction, might be effective, additional evidence is needed. The potentially deleterious effects of protein re-

moval in the ascites is another reason to avoid large volume paracentesis. However, the true metabolic effects of removal of this protein are unknown and deserve future study. It seems evident that one-time removal of several liters of ascites, particularly if the patient also has peripheral edema, is safe and well-tolerated and may afford the patient with tense ascites substantial comfort.

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