ORIGINAL ARTICLES

Tense Ascites Redefined: Renal Consequences of Tense Ascites in Decompensated Cirrhotos

DILIP R KARNAD, PHILIP ABRAHAM, FRATIBHA D KARNAD, RAVI RAMAKANTAN

Departments of Medicine, Gastroenterology and Radiology, King Edward Memorial Hospital, Bombay 400 012

Abstract
Cirrhosis with tense ascites fail to achieve increased diuresis in the supine position. To assess the role of inferior vena cava compression in this phenomenon, we studied cirrhotes with mild to moderate (n=11) and tense (n=2) ascites, and patients with membranous inferior vena cava obstruction (n=2) before and after balloon dilatation, in the sitting, supine and 10° head down tilted positions for 2 hours each. Urinary output (p<0.005), creatinine clearance (p<0.025) and sodium excretion (p<0.025) increased in cirrhochs with mild to moderate ascites in the supine position, and further in the head down position. Similar changes occurred in patients with inferior vena cava membrane. In cirrhotoes with tense ascites, these parameters did not change significantly in the supine position, but increased in the head down position.

We conclude that failure to augment diuresis in the supine position in cirrhoses with tense ascites is not due to inferior vena cava obstruction alone but is probably also due to compression of the collaterals vessels. This clinical observation may serve as a criterion for diagnosing tense ascites. (Indian J Gastroenterol 1992; 11: 55-58)

Key Words: Natriuresis; diuresis; posture; vena cava, inferior.

Introduction
Patients with ascites and edema have been shown to have increased diuresis and natriuresis as well as improved renal function in the recumbent position.1 This posture causes a gravity-dependent shift of pooled blood from the lower limbs to the heart, which stimulates intrathoracic volume receptors, inducing diuresis.2 In a related study,3 we observed that patients with tense ascites did not develop these changes. Other workers4,5 have also identified a subset of cirrhotes who failed to excrete a water load, and also had inadequate diuresis on head-out water immersion, another method of achieving central hypervolemia without increasing total body volume. These 'non-excretors' too had tense ascites.4

It is likely that the pressure of tense ascites causes a 'functional' inferior vena cava (IVC) obstruction which prevents a central fluid shift. To test this hypothesis, we studied the effect of posture on renal function in cirrhotic patients with tense ascites as compared to that in patients with anatomical IVC obstruction and in cirrhotic patients with mild to moderate ascites. As a result of this study, we have attempted to redefine tense ascites on the basis of its physiologic consequences and prognostic significance.

Material and Methods

Cirrhosis
 Eleven patients with liver cirrhosis and mild to moderate ascites and edema (Group I) and two cirrhotic patients with tense ascites (Group II) were studied over a 3-year period. Data on some of these patients have been reported previously.1,2 Ascites was termed tense when it was large in volume, the skin was shiny, fluid thrill could be elicited, and enlarged abdominal viscera could not be palpated.4 Patients with systemic hypertension, respiratory or cardiac diseases, primary renal disease or peripheral neuropathy were excluded. The diagnosis of cirrhosis was established by liver biopsy in seven patients, and by the presence of esophageal varices on endoscopy in association with abnormal liver function tests and characteristic appearance on radionuclide scan, in the others. All patients had hypoproteinsinemia (<3.0g/dL).

Protocol: After obtaining informed written consent, diuretics were withdrawn in patients who were receiving these. They then received a diet that provided 24 mmol of sodium, 65 mmol of potassium, 60 g proteins and one liter of fluids per day. After allowing seven days for stabilization, tension of ascites was reconfirmed. Subjects were studied on two consecutive days. After an overnight fast, they ingested 100 mL of water hourly from
05.00 h onwards. At 07.00 h, they voided urine and then sat upright with their legs over the side of the bed for two hours. At 09.00 h, they voided urine and for the next two hours they assumed supine position on day 1 and a 10⁶ head down tilted position on day 2. At 11.00 h they voided urine again.

The 2-hour urine samples and blood samples collected at 08.00 h and 11.00 h were analyzed separately. Plasma and urinary creatinine were estimated by colorimetry, and electrolytes by flame photometry. Creatinine clearance and urinary sodium excretion were calculated for all patients.

**Inferior vena cava obstruction**

Two patients with supradiaphragmatic IVC obstruction due to a congenital membrane (Group III) were studied in a similar manner. Both had mild ascites, abnormal liver function tests and esophageal varices on endoscopy (Budd-Chiari syndrome).

After the initial study, a percutaneous balloon venography was performed, after which the gradient across the obstruction came down from 12.7 mmHg to 8.0 mmHg in one patient and 20.0 mmHg to 7.5 mmHg in the other. The patients were studied again 7 days later while on the diet detailed above, using the same protocol.

**Statistical methods**

Differences between various postures in the same group were compared using Wilcoxon's signed rank test for paired data; differences between groups in the same posture were compared by the Wilcoxon's rank sum test for unpaired data.

**Results**

At the start of the study, Group II had significantly lower (P<0.005) serum sodium levels (median 125 mmol/L) than Group I (median 157.5 mmol/L); serum sodium levels in Group II (median pre-dilatation 137.5 mmol/L, post-dilatation 140 mmol/L) were similar to those in Group I.

The 11 decompensated cirrhotics with mild to moderate ascites (Group I) had a significant increase in urine volume, creatinine clearance and urinary sodium excretion in the supine position, which rose further in the 10⁶ head down tilted position (Table).

The two cirrhotics with tense ascites (Group II) had significantly lower basal sitting posture urine volume and sodium excretion than Group I. In the supine position, there was no change in the urine volume, creatinine clearance or sodium excretion in Group II, but in the 10⁶ head down tilted posture, increase in all three variables,

### Table: Basal values in sitting position, changes observed on assuming supine position from sitting position (Δ1) and difference between 10⁶ head down tilted and supine positions (Δ2) in cirrhotics with mild to moderate ascites, cirrhotics with tense ascites, and patients with membranous obstruction of inferior vena cava before and after dilatation.

<table>
<thead>
<tr>
<th></th>
<th>Tense ascites</th>
<th>IVC obstruction</th>
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<tbody>
<tr>
<td></td>
<td>(n=2)</td>
<td>Before dilatation (n=2)</td>
</tr>
<tr>
<td><strong>Urine volume (mL/2 h)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sitting</td>
<td>60 (28-115)</td>
<td>20, 22.5</td>
</tr>
<tr>
<td>Δ1</td>
<td>21 (1-61)***</td>
<td>-3, -5.5†</td>
</tr>
<tr>
<td>Δ2</td>
<td>61 (8-141)***</td>
<td>18, 8</td>
</tr>
<tr>
<td><strong>Creatinine clearance (mL/min)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sitting</td>
<td>66 (30-109)</td>
<td>51.2, 38.1</td>
</tr>
<tr>
<td>Δ1</td>
<td>8.5 (1.4-10.9)**</td>
<td>-0.8, 4.6</td>
</tr>
<tr>
<td>Δ2</td>
<td>20.3 (2.1-52)*****</td>
<td>35.1, 5.3</td>
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<tr>
<td><strong>Urinary sodium excretion (mmol/min)</strong></td>
<td></td>
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<tr>
<td>Sitting</td>
<td>12.2 (1.2-38.7)</td>
<td>0.4, 0.9</td>
</tr>
<tr>
<td>Δ1</td>
<td>5.4 (1.4-14.1)**</td>
<td>0, 0.9#</td>
</tr>
<tr>
<td>Δ2</td>
<td>2.0 (1.1-13.2)*</td>
<td>1.0, -0.4</td>
</tr>
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Difference between postures in ascites: *p<0.05; **p<0.025, ***p<0.005
Difference between Δ1 and Δ2 in patients with tense ascites and IVC obstruction compared to mild/moderate ascites: #p<0.05; # #p<0.025
†Median (range)

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comparable in magnitude to that in Group I, was observed.

The two patients with membranous IVC obstruction (Group III) were studied twice, once with severe obstruction and again after balloon dilatation of the obstruction, which still left residual mild to moderate obstruction. This group had an increase in all three variables in the supine position, and a further rise in the 10° head down tilted posture, similar to that in Group I.

Discussion
In the supine position, cirrhosis with mild to moderate ascites and edema had a significant increase in urine volume, creatinine clearance and sodium excretion over basal (sitting) values. Blood is pooled in the leg veins in the upright position and shifts into the chest on assumption of the supine position. This leads to increased cardiac output and stimulates atrial volume receptors, resulting in decreased plasma renin activity, aldosterone, and antidiuretic hormone levels, and sympathetic tone, and increased circulating levels of atrial natriuretic factor. This produces diuresis, natriuresis and increased glomerular filtration rate (creatinine clearance).

The two patients with tense ascites studied showed no change in urine volume, sodium excretion or creatinine clearance in the supine position. Nichols et al. identified a group of cirrhosis patients with tense ascites who failed to develop adequate diuresis and natriuresis on head-out water immersion, which causes central hypervolemia without increasing total blood volume. They also failed to excrete a water load. Like our two patients, these non-excretors had lower basal glomerular filtration rates and serum sodium levels and, most importantly, had tense ascites. Do cirrhotic patients with tense ascites then differ from other cirrhotics in their response to central volume expansion? If so, why?

Tense ascites can exert pressure on the IVC along its retroperitoneal course. This could theoretically prevent a central shift of the pooled blood on assumption of supine position or on water immersion. If this was the only mechanism preventing diuresis, a similar outcome would be expected in patients with structural IVC obstruction. However, in two patients with membranous suprahepatic IVC obstruction who had compromised liver functions and mild transudative ascites and edema, assumption of the supine posture induced diuresis, natriuresis and increased creatinine clearance, which were similar before and after balloon dilatation of the obstruction and was comparable to that observed in the 11 cirrhotics with mild to moderate ascites and no IVC obstruction. This suggests that obstruction to IVC flow alone does not prevent postural shift of blood into the chest.

Both patients with IVC obstruction had massively dilated, tortuous abdominal veins with a cephalad flow; preserved venous return through these channels may have allowed the observed diuresis and natriuresis in spite of severe vena cava obstruction.

According to Laplace's law, tension in the walls of a cavity is proportional to the product of the intracavitary pressure and the radius of the cavity; both these are greatly increased in cirrhosis with tense ascites. Therefore, these patients probably have compression not only of the IVC but also of venous collaterals in the abdominal wall. This may explain the different responses to change in posture in our cirrhotics with tense ascites and those with membranous obstruction of the IVC.

In many decompensated cirrhoses, ascites may subside with hospitalization and bed rest. Increase in central blood volume in the recumbent posture is perhaps responsible for this. Our data suggest that patients with tense ascites may not respond to these measures. Pressure on the IVC and its collaterals may further reduce renal blood flow and worsen the already reduced glomerular filtration rate. Both our cirrhoses with tense ascites, and all of Nicholls' non-excretors, had lower glomerular filtration rates than other cirrhotics. This decrease in glomerular filtration of sodium, combined with hormonal changes, may make them retain water and sodium more avidly. These events may make treatment of ascites difficult, if not impossible, in patients with tense ascites.

A recent study has shown that large volume paracentesis in such patients results in increased cardiac output. This may probably be related to reduction of the pressure on the IVC and the collaterals. However, paracentesis or diuretic therapy further worsens renal blood flow and hypovolemia, and may cause azotemia. On the other hand, increasing ascites and IVC compression may further compromise renal function, resulting in hepatorenal syndrome. Both our patients developed hepatorenal syndrome and died within two months of the study. Only one of Nicholls' seven non-excretors with tense ascites was alive five months after the study; the rest developed hepatorenal syndrome and died.

In fact, given this high mortality rate, we did not consider it proper to withhold treatment for seven days as required by our protocol in patients with tense ascites. Hence after studying only two patients who were not already on therapy, this part of the study was terminated.

The small number of patients we studied makes a Type II error possible; however, this will only underestimate the validity of our conclusions.

This raises a vital question: what is tense ascites? Buhac defined tense ascites, on the basis of physical principles, as one where the ascitic fluid column in a manometer during paracentesis rises higher than the level...
of fluid dullness on percussion of the abdomen. In physiologic terms, ascites which is severe enough to compress the IVC and its collaterals should be called tense, because this significantly worsens the prognosis. This cannot be defined in terms of intra-abdominal pressures alone because compression of collaterals depends on the abdominal wall tension which in turn depends on abdominal girth and thickness of the abdominal wall (Laplace's law). Failure to excrete more than 25% of a water load may be one way of identifying this. Failure of the urine volume to rise by 25% or more in the supine position above the sitting value, as observed in our patients, may be another.

References


