

# Management of Ascites

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## Abstract

This article is brief review of clinical management of ascites, especially in the cirrhotic population. It briefly covers appropriate investigations, initial management of ascites and further management of diuretic resistant ascites and complications. All materials have been appropriately referenced.

## SOURCE OF SUPPORT

Most of the references used in this article are sourced from UPTODATE.COM

## INTRODUCTION

Ascites: Greek ASKOS (bag)

Ascites is a common clinical finding, seen usually in cirrhotic patients, but its management is complex. It is one of the most common complication in cirrhotics, others are variceal bleeding, hepatorenal syndrome and spontaneous bacterial peritonitis etc. Natural history of ascites is variable, with 57% of cirrhotics developing this complications in 10 years time.<sup>[1]</sup>

Ascites formation involves various physiological, biochemical and pathological alterations over a period of time. Various theories have been proposed to explain the formation of ascites, with the older theories of ascites formation, the underfill theory <sup>[2]</sup> and the overflow theory <sup>[3]</sup>, appear to play a role at different stages of ascites formation <sup>[4]</sup>. The arterial vasodilation hypothesis, appears to be the most widely accepted theory which includes the previous accepted theories. <sup>[5]</sup>.

## CAUSES [.]

Based on Serum Ascitic Albumin Gradient

SAAG:  $\geq 1.1$ g/dL Cirrhosis Alcoholic hepatitis CHF  
Massive hepatic metastases Vascular occlusion Fatty liver  
disease of pregnancy Myxedema

SAAG:  $< 1.1$ g/dL Peritoneal carcinomatosis Peritoneal TB  
Pancreatitis Serositis Nephrotic syndrome Bowel obstruction  
/ infarction / perforation

Approximately 5 percent of patients with ascites have more than one cause, such as cirrhosis and TB peritonitis, heart failure, nephropathy or peritoneal carcinomatosis.

## DIAGNOSIS OF ASCITES

Thorough history and examination is crucial. Following factors in history are of more value: <sup>[8]</sup>

### Figure 1

#### USEFUL

	LR+	LR-	Sensitivity	Specificity
Increased girth	4.16	0.17	0.87	0.77
Weight gain	3.20	0.42	0.67	0.79
Hx of hepatitis	3.20	0.80	0.27	0.92
Ankle oedema	2.80	0.10	0.93	0.66
Hx of CCF	2.04	0.73	0.47	0.73

#### NOT USEFUL

Alcoholism	1.44	0.69	0.60	0.58
Hx of cancer	0.91	1.01	0.13	0.85

The most helpful physical finding in confirming the presence of ascites is flank dullness <sup>[9]</sup>. When flank dullness is detected, it is useful to elicit shifting dullness. Another useful physical finding to look for other causes is an elevated jugular venous pressure to detect heart failure or constrictive pericarditis.

## DIAGNOSTIC TESTS

A) Abdominal Ultrasound:

It is a very sensitive test and minimal amounts of fluid can

be detected. Also it is useful to assess the status of liver and spleen and direction of flow and the diameter of the portal vein. In difficult cases ultrasound guided aspiration of the ascitic fluid is useful.

### B) Diagnostic aspiration:

1. Microbiology: Gram stain and cell count, ascetic fluid culture in blood culture bottles filled at the bedside.
2. Biochemistry: Ascitic fluid albumin, serum albumin, total proteins( ascetic and serum), and serum ascites albumin gradient are useful.[<sub>10</sub>].

An ultrasound study demonstrated that a left lower quadrant tap site is superior to a midline site; the abdominal wall is relatively thinner in the left lower quadrant with greater depth of the fluid. [<sub>11</sub>].

Most bloody samples are due to a “traumatic tap” with blood clotting in the syringe if not injected into an anti-coagulated tube immediately. This can be confirmed by an immediate re-tap in a different site aspirating clear fluid. Where as hemoperitoneum has homogenous bloody fluid. Causes for hemoperitoneum include bloody cirrhotic ascites, malignancy – especially hepatocellular carcinoma, etc.

The serum-to-ascites albumin gradient (SAAG) accurately identifies the presence of portal hypertension.[<sub>7</sub>]. The SAAG is easily calculated by subtracting the ascitic fluid albumin value from the serum albumin value, which is obtained on the same day. The presence of a gradient 1.1 g/dL (11 g/L) indicates that the patient has portal hypertension with 97 percent accuracy [<sub>22</sub>]. A gradient <1.1 g/dL (<11 g/L) indicates that the patient does not have portal hypertension [<sub>22</sub>].

## INITIAL TREATMENT OF ASCITES

The treatment of cirrhotic ascites is aimed at the following general and specific measures.

### GENERAL MEASURES

1. Underlying cause of the hepatic disease
2. Removal of the ascitic fluid itself.
3. Patients must be encouraged to abstain from alcohol completely. In alcoholic cirrhosis, abstinence may lead to improvement of hepatic histology in some patients with marked fibrosis and inflammation [<sub>17</sub>], a reduction in the portal pressure and easier to treat ascites. In patients with

cirrhosis and chronic hepatitis C, the alcohol-related component of their liver disease may improve dramatically with abstinence.

4. Avoid the use of nonsteroidal antiinflammatory drugs. Through prostaglandin inhibition cause sodium retention, worsen renal function[<sub>17</sub>].

### SPECIFIC MEASURES

1. Limiting sodium intake to 88 meq (2g) per day (including all foods and medications) is the most important step. Thus is to counteract the central problem of sodium retention. [<sub>18</sub>]
2. Diet alone is useful only in a small number of patients; hence diuretics are very important for urinary sodium loss of more than 78meq/day (88 meq intake minus 10 meq of non-urinary losses).

Most patients with cirrhosis and fluid overload are treated with a combination of dietary sodium restriction and diuretics.

### CHOICE OF DIURETICS

A combination of potassium losing loop diuretic like furosemide with a potassium sparing diuretic like spironolactone is the best approach. Furosemide is a weaker diuretic in cirrhotic patients due to reduction of renal tubular excretion of furosemide in these patients [<sub>20</sub>].

A initial combination of 40mg Furosemide with 100mg Spironolactone, increased every 3-5 days to a maximum of 160mg Furosemide and 400mg Spironolactone is advised. [<sub>18</sub>]. The only setting in which we begin with spironolactone monotherapy is in patients with severe alcoholic hepatitis who have profound hypokalemia. Furosemide is added once the potassium normalizes.

### RATIONALE FOR ORAL DIURETICS INSTEAD OF INTRAVENOUS USE

Patients who only have ascites can mobilize edema fluid solely via the peritoneal capillaries unlike patients with fluid overload due to other conditions like heart failure. Hence a steady and gradual loss of fluid is better than brisk diuresis induced by intravenous diuretics, which might worsen glomerular filtration by reducing intravascular volume and may cause hepatorenal syndrome. If a quick removal of ascitic fluid is anticipated as in tense ascites with peripheral pedal oedema or respiratory splinting from a tense ascites, then a therapeutic paracentesis is a better approach.

The maximum rate at which this can occur is only 300 to

500 mL/day; more rapid fluid removal with diuretics can lead to plasma volume depletion and renal failure. Hence aim for 0.5kg/day weight loss ideally.

### ABDOMINAL PARACENTESIS

The risk of development of a large hematoma after abdominal paracentesis is only ~1% [12]. The risk of hemoperitoneum or iatrogenic infection is only about 1 per 1000 [11,12]. In one of the studies evaluating 1100 paracenteses, procedure was safe upto an INR as high as 8.7 and platelet count as low as 19,000 [14]. In another study of 4700 paracenteses the rates of severe hemorrhage and death were estimated to be 0.19% and 0.016%, respectively [15].

### ALBUMIN OR COLLOID REPLACEMENT

Removal of less than 5 liters of fluid does not appear to have hemodynamic or hormonal consequences; post-paracentesis colloid infusion does not appear to be necessary [19]. For larger paracenteses, albumin (10 g/L of fluid removed) can be administered which appears to be better than colloid. [28,29].

### COMPLICATION OF DIURETIC THERAPY HYPOKALEMIA

Hypokalemia should be avoided in patients with cirrhosis and ascites because potassium depletion can enhance renal ammonia production and possibly precipitate hepatic coma [21].

### HYPONATREMIA

Patients with cirrhosis and ascites usually have a marked reduction in systemic vascular resistance, mostly in the splanchnic circulation and in mean arterial pressure with an increase in cardiac output. [22]. This leads to increased ADH secretion and water retention.

Degree of hyponatremia parallels severity of liver disease and hyponatremia <120mmols is seen in <1-2% patients. Since symptomatic hyponatremia is unusual in cirrhosis, only modest water restriction that is tolerated by the patient is recommended in asymptomatic patients. Low serum sodium predicts a poor prognosis.[23]

Other treatments have been tried, but no definite benefit.

### DIURETIC RESISTANT ASCITES

The above approach is successful in up to 90% of the patients. In patients who appear to be diuretic-resistant, it is important to exclude lack of compliance with dietary sodium

restriction.

There is some evidence that a random urinary Na/K ratio may be nearly as good as a 24-hour collection [24]. If the random urine specimen reveal a urinary sodium greater than urinary potassium, this roughly equates to a daily urinary sodium loss of >78 meq/day. Patients with diuretic-resistant ascites have pre-hepatorenal syndrome and a very poor prognosis [25]. Survival in patients with diuretic-resistant ascites is 50 percent at six months and 25 percent at one year [26].

Options for further treatment include

1. Therapeutic paracentesis (large volume paracentesis-LVP)
2. Serial therapeutic paracentesis,
3. Liver transplantation,
4. Transjugular intrahepatic portosystemic stent-shunt (TIPS), and
5. Peritoneovenous shunt

Large-volume paracentesis (LVP) relieves respiratory splinting and early satiety that these patients experience. It also may be associated with collateral advantages, such as reductions in the hepatic venous pressure gradient, intravariceal pressure, and variceal wall tension [27].

Serial LVPs are very useful in diuretic resistant ascites. The problem with this approach is that repeated LVPs cause protein and complement depletion compared to diet/diuretic therapy, and may indirectly predispose to ascitic fluid infection [28].

Hence ensuring patient compliance with diet and diuretic is very important in selecting the appropriate patient for this procedure.

Further details on TIPPS, Liver transplantation and peritoneovenous shunting are beyond the scope of this article. Suggested reading –[30].

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