Ascites: Diagnosis and Management

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Abstract

Ascites, the collection of fluid in the peritoneal cavity, occurs with a variety of disease states. It is one of the earliest and most common complication of chronic liver disease. In cirrhosis, it is associated with circulatory dysfunction characterized by arterial vasodilatation, high cardiac output and stimulation of vasoactive systems.

As appropriate treatment depends on accurate diagnosis, paracentesis should be performed in every patient with new onset ascites to determine the cause and to detect potential complications. The treatment of ascites due to causes other than chronic liver disease is based on the underlying disease. In ascites associated with chronic liver disease, a combination of low sodium diet and the administration of diuretics remains the mainstay of therapy. Large volume paracentesis along with infusion of albumin is the preferred treatment for refractory ascites. The recently introduced technique of transjugular intrahepatic portosystemic shunt for the management of refractory ascites needs further evaluation.

Introduction

In clinical practice, the term ‘ascites’ refers to the detectable and pathologic collection of fluid in the peritoneal cavity. Usually it is a clinical finding and can be confirmed by a diagnostic paracentesis. Subclinical amount of fluid (i.e., less than 1.5 litre) can be detected using ultrasonography or computed tomography of the abdomen.

Aetiology

Chronic liver disease with portal hypertension, congestive cardiac failure, tuberculosis and malignancy are important causes of ascites. However, it can occur secondary to a number of pathological conditions. Various causes of ascites are shown in Table I

<table>
<thead>
<tr>
<th>Venous hypertension</th>
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<tr>
<td>Cirrhosis of liver</td>
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<tr>
<td>Congestive cardiac failure</td>
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<tr>
<td>Constrictive pericarditis</td>
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<tr>
<td>Hepatic venous outflow obstruction</td>
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<td>Acute portal vein thrombosis</td>
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<table>
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<tr>
<th>Hypoalbuminemia</th>
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<tr>
<td>Cirrhosis of liver</td>
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<tr>
<td>Nephrotic syndrome</td>
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<td>Malnutrition</td>
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<th>Infections</th>
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<tr>
<td>Tuberculosis</td>
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<td>Parasitic (strongyloidosis, entamoeba)</td>
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</table>

Malignancies

- Peritoneal carcinomatosis
- Lymphomas and leukaemias
- Primary mesothelioma

Miscellaneous

- Chylous ascites
- Systemic lupus erythematosus
- Ovarian disease
- Pancreatic ascites
- Pseudomyxoma peritonei

Pathogenesis

In a large number of patients, cirrhosis of liver is the cause of ascites. Several factors contribute to the development of ascites in chronic liver disease. Kidney plays a central role and is responsible for sodium and water retention, through complex mechanisms. The mechanism by which the diseased liver affects renal function is not fully understood. The ‘peripheral arterial vasodilatation hypothesis’ proposed in 1988 is based on the presence of characteristic circulatory abnormalities seen in cirrhotic patients. These patients show manifestations of increased cardiac output, arterial hypotension, decreased peripheral vascular resistance and splanchnic vasodilatation. Possible causes for vasodilatation include portosystemic shunting and/or impaired clearance of vasodilator substances like nitric oxide, endotoxins, prostacyclin, glucagon and adenosine. This peripheral and splanchnic vasodilatation is perceived as reduction in effective plasma volume. The effective hypovolemia brings into play the baroreceptor mediated activation of renin - angiotensin - aldosterone system and sympathetic nervous system which produce renal vasoconstriction and salt and...
water retention (Fig. 1)\textsuperscript{2,3}.

- decreased oncotic pressure of plasma due to impaired albumin production by the liver;
- portal hypertension which localizes the fluid within the peritoneal cavity; and
- an increased production of hepatic lymph due to post-sinusoidal obstruction by the hepatic nodules\textsuperscript{1}.

In ascites associated with other conditions, the pathogenesis depends on the cause. In congestive cardiac failure, elevation of right sided cardiac pressures results in the congestion of hepatic sinusoids and leakage of fluid from the surface of liver. In addition, reduction in effective blood volume leads to sodium and water retention by the kidney\textsuperscript{4}. In ascites associated with non-hepatic malignant disease, the pathogenesis depends on the type and location of tumour\textsuperscript{5}. In peritoneal carcinomatosis, the most common cause of malignant ascites, the leakage of protein rich fluid from the malignant cells causes exudation of extracellular fluid into the peritoneal cavity. Large liver tumours pressing on or growing into the portal or hepatic veins can cause portal hypertension and ascites. Infiltration of lymphatic channels by malignant disease especially lymphoma may lead to rupture of lymphatics and thereby produce chylous ascites. Chylous ascites can also occur after transection of lymphatics, such as after abdominal surgery\textsuperscript{6}. Filarasis is another uncommon but important cause of chylous ascites.

Pancreatic ascites results from rupture of the pancreatic duct or leakage of the pancreatic secretions from a pseudocyst. Irritation of the peritoneum by the pancreatic secretions can cause accumulation of protein rich exudate in the peritoneal cavity. Biliary ascites forms by similar mechanism. In infections such as tuberculosis, the mechanism is similar to that in carcinomatosis. There is leakage of protein rich fluid into the peritoneal cavity by the inflamed peritoneum. The mechanism of ascites in nephrotic syndrome and dialysis associated ascites is unclear but is probably related to volume expansion and abnormal peritoneal permeability. Hypoalbuminaemia also contributes to ascites in nephrotic syndrome.

**Diagnosis**

**History**

Ascitic fluid may accumulate rapidly or gradually depending upon the cause. Mild ascites may not
produce any symptoms. Moderate ascites may just produce an increase in abdominal girth and weight gain. Large amounts of fluid can produce abdominal discomfort, appearance of hernias particularly umbilical hernias and hinder the mobility of the patient. Elevation of diaphragm and restriction of its movements can produce breathlessness.

In many patients, a diagnosis of liver disease might have been established earlier, as ascites develops later when there is decompensation. However, ascites can be the first sign of liver disease. Thus, it is important to obtain a history of risk factors for liver disease like alcohol consumption, drug abuse, blood transfusions or hepatitis in the past. Sudden development of ascites in a previously stable patient of cirrhosis should raise the suspicion of hepatoma.

A history of heart failure and pericardial disease should make one suspect cardiac ascites. A history suggestive of malignancy elsewhere, e.g., breast, gastrointestinal tract, ovaries or lymphoma may suggest malignant ascites. In India, tuberculosis as a cause of ascites should be suspected if there is history of fever, constitutional symptoms and in the presence of known extra-abdominal tuberculosis. In patients with pancreatic ascites there is usually a history suggestive of chronic pancreatitis. The same patient may have more than one disease predisposing to ascites.

Physical examination

Ascites needs to be differentiated from abdominal distension due to other causes like gross obesity, gaseous distention, bowel obstruction, abdominal cysts or masses. The diagnosis may be obvious in patients with massive ascites, but when only a small or moderate amount of fluid is present, the accuracy of physical assessment is only about 50%, even by experienced gastroenterologists. Flank dullness which is present in about 90% of patients, is the most sensitive physical sign. Shifting dullness on percussion is more specific but less sensitive than flank dullness for detection of ascites. A fluid thrill or wave may be demonstrable in cases of tense ascites. Occasionally massive ovarian or hydatid cysts and pregnancy with hydranmios can masquerade as ascites as they can also be associated with fluid thrill. The puddle sign, reported to detect as little as 120 ml of fluid clinically requires the patient to be in knee-elbow position during examination. The utility of puddle sign and auscultatory percussion for detecting ascites has been assessed using ultrasound of abdomen as gold standard. It was observed that auscultatory percussion has a greater sensitivity (66% Vs 45%) but a lower specificity (48% Vs 68%) than the puddle sign.

Physical examination can provide clues to the cause of ascites in a given patient. Signs of chronic liver disease, e.g., palmar erythema, spider naevi, jaundice, etc. should be looked for. Presence of splenomegaly and large collateral veins may suggest portal hypertension. Patients with cardiac causes of ascites may show engorged jugular veins. Collaterals in the back may indicate an obstruction of the inferior vena cava. Presence of enlarged lymph nodes may suggest tuberculosis or lymphoma.

Investigations

Abdominal paracentesis and analysis of ascitic fluid

Abdominal paracentesis and a careful analysis of ascitic fluid is the single most important procedure and should be an early step in evaluating a patient with ascites. It should be performed in all patients with new onset ascites and whenever deterioration occurs in a patient with known ascites. Paracentesis can be performed easily and within minutes. The procedure has been found to be safe with about 1 percent risk of abdominal wall haematoma. This was despite the fact that two-thirds of the patients, most of whom had cirrhosis, had prolonged prothrombin time. Therefore, it is unnecessary to routinely administer fresh frozen plasma or platelets to cirrhotic patients who have a coagulopathy before performing paracentesis. Concerns regarding the introduction of bacterial peritonitis are also unfounded. As the midline caudal to the umbilicus is a relatively avascular area, this site is recommended for paracentesis. Either of the lower quadrants may be used if there is a surgical scar in the mid line. The bowel may adhere to the abdominal wall near surgical scars and a needle inserted close to a scar may enter the intestine.

Analysis of the ascitic fluid is useful in the differential diagnosis of ascites. The gross appearance of the fluid may be helpful in determining the pathologic process. In ascites due to portal hypertension or hypoalbuminaemia, the fluid is clear and straw coloured; turbid ascites may indicate infection. Chylous ascites typically has a milky appearance.
Blood stained fluid is usually due to malignancy but may occur with tuberculosis, pancreatitis, hepatic vein thrombosis, recent abdominal punctures or due to a traumatic tap. Dark brown fluid may indicate the presence of bile. The tests of ascitic fluid that are frequently performed are shown in Table II. Routine tests are performed on all the specimens. Optional tests are performed on specimens for patients with suspected infection of ascitic fluid, suspected tuberculosis or malignant ascites.

Table II: Tests of Ascitic Fluid

<table>
<thead>
<tr>
<th>Routine Tests</th>
<th>Optional tests</th>
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<tr>
<td>Total protein</td>
<td>Gram's stain &amp; culture</td>
</tr>
<tr>
<td>Albumin</td>
<td>AFB smear &amp; culture</td>
</tr>
<tr>
<td>Cell count</td>
<td>Cytology</td>
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<tr>
<td></td>
<td>Amylase</td>
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<tr>
<td></td>
<td>Lactate dehydrogenase (LDH)</td>
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<td></td>
<td>Glucose</td>
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</table>

The total protein concentration of ascitic fluid has traditionally been used to classify samples into the broad categories of exudate or transudate. Low protein ascites with total protein concentration of less than 2.5 g per decilitre is called transudative ascites and usually occurs with portal hypertension or hypoalbuminaemia. A higher protein ascites with total protein concentration of more than 2.5 g per decilitre is called exudative ascites and is usually associated with tuberculosis, malignancy, pancreatitis, myxoedema, etc. However, a total protein concentration of greater than 2.5 g per decilitre has recently been shown to have an accuracy of only 56 percent in detecting an exudate. The serum-ascites albumin gradient (SAAG) has been found to be superior to the ascites total protein concentration for the differential diagnosis of ascites. The gradient is calculated by substracting the ascitic fluid albumin level from the serum level obtained on the same day. A gradient of more than 1.1 g per decilitre indicates presence of portal hypertension with an accuracy of 97 percent. The SAAG correlates directly with portal pressure. An increasing hydrostatic pressure between the portal vasculature and ascitic fluid is balanced by corresponding difference in oncotic forces. In patients with mixed causes of ascites, the SAAG tended to resemble that in uncomplicated transudative ascites. A classification of the types of ascites according to the level of serum-ascites albumin gradient is shown in Table III.

Table III: Classification of types of ascites according to the level of serum-ascites albumin gradient (SAAG)

<table>
<thead>
<tr>
<th>High gradient (&gt; 1.1 g/dl)</th>
<th>Low gradient (&lt; 1.1 g/dl)</th>
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<tbody>
<tr>
<td>Cirrhosis</td>
<td>Peritoneal tuberculosis</td>
</tr>
<tr>
<td>Alcoholic hepatitis</td>
<td>Peritoneal carcinomatosis</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>Pancreatic ascites</td>
</tr>
<tr>
<td>Massive liver metastasis</td>
<td>Biliary ascites</td>
</tr>
<tr>
<td>Fulminant hepatic failure</td>
<td>Nephrotic syndrome</td>
</tr>
<tr>
<td>Budd-Chiari syndrome</td>
<td>Serositis</td>
</tr>
<tr>
<td>Portal vein thrombosis</td>
<td>Bowel obstruction or infarction</td>
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<tr>
<td>Veno-occlusive disease</td>
<td></td>
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<tr>
<td>Fatty liver of pregnancy</td>
<td></td>
</tr>
<tr>
<td>Myxoedema</td>
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<tr>
<td>“Mixed” ascites</td>
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</tbody>
</table>

Adapted from Runyon

Culture of the ascitic fluid for bacteria should be obtained routinely in patients with cirrhotic ascites, in whom spontaneous bacterial peritonitis (SBP) can occur. For optimal results, 10 ml of ascitic fluid should be inoculated at the bedside into a blood culture bottle. Gram staining is useful in detecting secondary peritonitis due to gut perforation but is only about 10 percent sensitive in detecting bacteria early in SBP. In tuberculous peritonitis, the smear for acid-fast bacilli (AFB) is rarely positive and culture is positive only in about 50% of cases. Ascitic fluid glucose can drop significantly in severe infections like secondary peritonitis or late stage of SBP. Low glucose can also be found in malignant ascites. Measurement of ascitic fluid amylase is useful when there is suspicion of pancreatic ascites. Chylos ascites may show Sudan staining fat globules on microscopic examination and an increased triglyceride content by chemical examination. Triglyceride levels are low in
pseudochylous ascites which can occur due to the presence of large number of degenerating malignant or inflammatory cells. Alkali will dissolve cellular proteins and thus reduce turbidity in pseudochylous fluid whereas extraction with ether will lead to clearing if turbidity is due to lipid18. Rarely, fluid may be mucinous in character suggesting pseudomyxoma peritonei.

Role of imaging

Radiologic studies are useful in detecting small amounts of ascitic fluid as well as helpful in assessing the aetiology of ascites19. Abdominal sonography may detect as little as 100 ml of intraperitoneal fluid20. Although sonography is more cost-effective than computed tomography (CT), CT detects even smaller amounts of ascitic fluid. The appearance of liver may suggest cirrhosis. Pancreatic pseudocyst, intra-abdominal tumours can be visualized. Doppler sonography can detect thrombosis of the portal or hepatic veins. In patients with tuberculous peritonitis, thickening of mesentery and bowel wall, matting of bowel loops and presence of mesenteric lymph nodes may provide a clue21. In patients with small amount of ascites, adhesions from previous surgery or where ascites is compartmentalised, sonography can be an invaluable guide for localising a safe and useful site for paracentesis. CT may provide information that may be difficult to obtain on ultrasonography. In patients with carcinomatosis or inflammatory peritonitis, a contrast enhanced CT scan may demonstrate enhancement of the peritoneal lining. Similar results with peritoneal abnormalities have recently been reported for magnetic resonance imaging using gadolinium22. In patients with pancreatic ascites alone or associated with liver cirrhosis, endoscopic retrograde pancreatography with fluoroscopy can demonstrate leakage of pancreatic juice from the pancreatic duct. In patients with cirrhosis and large hydrothorax, scintigraphy with Technetium sulfur colloid or radiolabelled albumin can be used to diagnose the intraperitoneal origin of the thoracic fluid.

Laparoscopy

With the availability of new imaging techniques, the need for laparoscopy in determining the cause of ascites has decreased. However, if the diagnosis remains unclear, laparoscopy with direct visualization of the peritoneum may be indicated. Typical peritoneal tubercles are found in most patients with tuberculous peritonitis and peritoneal biopsies detect the disease in 74% of cases23. This procedure is rarely needed to detect peritoneal carcinomatosis because of the sensitivity of the cytology5.

Complications of Ascites

Umbilical hernia

Some patients may develop or may show an increase in the size of already existent umbilical hernia. Most hernias recur after surgical repair unless the ascites is controlled.

Hydrothorax

Pleural effusion, particularly on the right side can develop in some patients with ascites. It occurs due to passage of fluid through small holes in the diaphragm. These effusions may be very large.

Spontaneous bacterial peritonitis (SBP)

SBP is defined as the spontaneous bacterial infection of the ascitic fluid without any apparent intra-abdominal source. Most cases are seen in patients with cirrhotic ascites. The risk for SBP is 15% within the first 3 years after the onset of ascites24. SBP is thought to occur as a result of prolonged bacteraemia due to impaired host-defense mechanisms and decreased bactericidal activity in the ascitic fluid. Bactericidal activity parallels the total protein concentration in the fluid. Prospective studies have shown that SBP is 10 times more likely to develop in patients with protein concentrations lower than 1 g/dl during hospitalisation than in those with concentration higher than 2 g/dl24.

Symptoms and signs of SBP can be very subtle and non-specific. The most common features are fever and abdominal pain, but patients may present with hypotension or hepatic encephalopathy. SBP should be suspected whenever sudden deterioration occurs in a patient with ascites. Definitive diagnosis requires bacterial culture of ascitic fluid. However, culture negative SBP is common. The most important finding in the ascitic fluid is an elevated neutrophil count. A count of 250 cells/mm³ or more is considered diagnostic24. Most of the episodes are due to single gram-negative enteric bacteria. Secondary bacterial peritonitis should be suspected if the infection is polymicrobial or if a patient with presumed SBP does not respond to antibiotic therapy. Patients suspected to have SBP should be treated empirically with
antibiotics without awaiting culture results, as they can deteriorate rapidly. Cefotaxime, a third generation cephalosporin, is the best studied antibiotic for treating SBP. A 5-7 day course of parenteral antibiotics seems to be sufficient. Aminoglycosides should be avoided because of increased risk of nephrotoxicity in patients with cirrhosis. Seventy percent of patients have another episode of SBP within 1 year and the 1-year survival rate is 38%\textsuperscript{26}.

Strategies have been developed to prevent hospital-acquired SBP among susceptible patients, by selective intestinal decontamination using oral antibiotics like norfloxacin. It is recommended that SBP prophylaxis with antibiotics should be restricted to cirrhotic patients at high risk, including bleeding cirrhotic patients, those with a past history of SBP, and those with low protein content in the ascitic fluid\textsuperscript{27}. However, prophylactic treatment with antibiotics may prevent recurrence but has not been proven to prolong survival\textsuperscript{28}.

**Treatment of Ascites**

**Ascites due to causes other than chronic liver disease**

Treatment of non-cirrhotic ascites is to be directed at the underlying cause. Appropriate chemotherapy is needed for infective causes. The management of chylous ascites will depend upon the underlying cause. However, a low fat diet with medium chain triglycerides substituted for the normal long chain triglycerides may help decrease the triglycerides content of the ascitic fluid. Treatment of pancreatic ascites is controversial. Some patients may respond to conservative measures like salt restriction, diuretics and IV hyperalimentation. Somatostatin infusion may help by reducing the pancreatic exocrine secretion\textsuperscript{29}. Occasionally surgical or endoscopic intervention may be needed.

The management of malignant ascites is an important clinical problem when ascites causes severe symptoms. Repeated therapeutic paracentesis is required. If malignant cells are present in the ascitic fluid and there are no intra-abdominal tumour masses, palliation may be achieved in some patients with chemosensitive malignancies by intraperitoneal injection of the appropriate cytotoxic drug. In patients without malignant cells in the ascitic fluid, a peritoneovenous shunt may be of value in the control of resistant ascites\textsuperscript{1}.

**Ascites due to liver disease**

Ascites in cirrhosis generally occurs within 10 years of diagnosis in about 50 % of patients. Patients may present with moderate ascites, tense ascites, refractory ascites, hyponatraemia, or with hepatorenal syndrome. A thorough search should be made for precipitating factors in patient with recent onset or worsening ascites. The general approach to management includes determining the baseline body weight, serum electrolytes, urea and creatinine and sodium concentration in a random urine sample.

*Sodium restriction, bed rest and use of diuretics are the mainstay of therapy.* The upright posture in patients with cirrhosis and ascites has been reported to be associated with marked activation of renin-angiotensin-aldosterone and sympathetic nervous system, reduction of glomerular filtration rate (GFR) and sodium excretion and a decreased response to loop diuretics\textsuperscript{30}. Thus, strict bed rest is often recommended because of improved renal clearance in supine position.

Sodium intake needs to be restricted to about 800-1000 mg (2g NaCl) in order to induce a negative sodium balance and permit diuresis. The factors favourable to respond to bed rest and salt restriction include recent onset ascites, a reversible liver disease, correctable precipitating factor, high urinary sodium excretion and normal renal function. In about 20% of cirrhotics with ascites, urinary sodium concentrations are relatively high. In these patients, restriction of sodium and bed rest alone may result in disappearance of ascites\textsuperscript{2}. Fluid restriction is not necessary unless hyponatraemia is present. In majority of patients, a negative sodium balance can only be achieved by increasing the urinary sodium excretion with diuretics.

For a patient with mild to moderate ascites, therapy can be undertaken as an outpatient and should be gradual and incremental. The goal of diuretic treatment is a loss of weight of not more than 1.0 kg/day if both ascites and edema are present and not more than 0.5 kg/day in patients with ascites alone\textsuperscript{3}. Spironolactone, an aldosterone antagonist is preferred as the initial diuretic. It decreases sodium reabsorption in the distal tubule. The recommended starting daily dose is 50 to 100 mg/day and a maximum of upto 400 mg/day may be given in one or more doses. Spironolactone may not provide
effective diuresis in all ascitic cirrhotic patients even in adequate doses. The most important factor determining the unresponsiveness to aldosterone antagonists is the presence of renal failure. Addition of a loop diuretic (furosemide) to spironolactone potentiates the effect of both drugs and reduces the risk of developing hyperkalaemia. A useful therapeutic approach may be to add 40 mg of furosemide for 100 mg of spironolactone. The maximum dose of diuretics recommended is a combination of spironolactone 400 mg/day with furosemide 160 mg/day. These patients should be monitored for weight reduction, electrolytes, urea and creatinine. If a patient fails to respond to these regimens, the physician should suspect presence of some of the responsible factors. These are, lack of compliance with salt restriction, use of prostaglandin inhibitors like NSAIDs, development of SBP, or progression of the underlying disease.

Thirty to fifty percent of patients develop complications with diuretic therapy. These include hyponatraemia, renal failure due to depletion of intravascular volume and hepatic encephalopathy. Other side-effects related to the use of spironolactone in cirrhotics are decreased libido, impotence and gynaecomastia in men, and menstrual irregularities in women. Muscle cramps may often be present.

Patients with severe or tense ascites who are refractory to diuretics should be treated with large volume paracentesis. Five or more litres of fluid should be removed to relieve dyspnoea, decrease early satiety, and prevent pressure related leakage from the site of paracentesis. Therapeutic paracentesis is haemodynamically beneficial in patients with tense ascites, contrary to the popular belief. Several randomized controlled clinical trials have demonstrated that large volume paracentesis with intravenous albumin infusion induces a lower incidence of complications than diuretic therapy. Paracentesis associated with intravenous albumin infusion (about 8 g of albumin per litre of ascitic fluid removed) is considered to be the treatment of choice in patients with tense ascites. Albumin infusion is required to prevent reduction in effective intravascular volume. A cheaper alternative is to use dextran or synthetic gelatins which are effective but may be followed by slight circulatory disturbances. A randomized controlled trial using the three substances as plasma expanders after large volume paracentesis has shown that dextran-70 or polygeline are less effective than albumin in prevention of post paracentesis circulatory dysfunction, particularly in patients in whom more than 5 litres of ascitic fluid is removed. When these patients are discharged from the hospital, they should be put on salt restriction and diuretics to prevent reaccumulation of the fluid.

Management of refractory ascites

In about 10% of patients, the ascites is refractory to treatment with first line measures mentioned above. A new definition and diagnostic criteria of refractory ascites has been proposed recently. Refractory ascites is now defined as the ascites that cannot be mobilized or early recurrence of which (i.e. after therapeutic paracentesis) cannot be satisfactorily prevented by medical therapy. It includes two different subtypes: diuretic resistant ascites and diuretic intractable ascites, i.e., development of diuretic induced complications that preclude the use of an effective diuretic dosage. Most of these patients with refractory ascites have severe disturbances of systemic haemodynamics and renal function. The development of refractory ascites usually indicates advanced underlying disease. The therapeutic options for this group of patients are discussed below.

Repeated large volume paracentesis at intervals of 2-3 weeks depending upon the severity of sodium retention and the amount of fluid removed each time. Many centres routinely perform total paracentesis on such patients. This can be performed on an outpatient basis. Ascites recirculation with removal, concentration and reinfusion of peritoneal fluid has been explored in USA and Japan. Technical problems and life threatening complications like infection and coagulopathy led to its unpopularity. The procedure has been performed at some centres as the standard therapy in diuretic refractory ascites.

Peritoneovenous shunt (PVS) or LeVeen shunt was introduced especially for the treatment of patients with refractory ascites. One limb of the shunt lies in the peritoneal cavity and the other in the superior vena cava close to the entrance of right atrium. A valve at the venous end prevents backflow of blood into the tubing. Flow is maintained by the peritoneovenous pressure gradient. This technique produces a marked increase in plasma volume and inhibits renin, aldosterone, noradrenaline and anti-diuretic hormone (ADH) concentrations leading to an increase in diuresis, natriuresis and free water clearance. In
addition, the procedure is also followed by an increase in renal blood flow and GFR. However, despite these positive effects of PVS, there are a large number of complications which may occur early in the postoperative period or at any time during follow up. Serious complications include sepsis, peritonitis, disseminated intravascular coagulation, and variceal haemorrhage which could occur due to rise in portal pressure. However, obstruction of the prosthesis is the most common complication and occurs in 40-60% of patients during first year of follow up.

In a recently conducted multicentric controlled clinical trial in cirrhotics with refractory ascites in which PVS was compared to therapeutic paracentesis, it was demonstrated that PVS is better than paracentesis in the long-term control of ascites. However, it also showed that PVS does not reduce the total hospital stay and does not increase survival. Therefore, paracentesis with intravenous albumin should be a better alternative to PVS in the management of refractory ascites.

Surgical portosystemic shunts in which the portal vein is used as an outflow tract (side to side portacaval and mesocaval shunt). They relieve portal hypertension and are effective in clearing ascites. However, because of high incidence of hepatic encephalopathy and of high surgical mortality in patients with advanced liver disease, they are seldom used nowadays.

Transjugular intrahepatic portosystemic shunt procedure (TIPS) which is a non-surgical portocaval anastomosis and behaves like a side-to-side portacaval shunt, has recently been proposed as an effective treatment for refractory ascites. In this procedure, a tract is created between branches of hepatic and portal veins, resulting in an intrahepatic portosystemic shunt with a concomitant reduction in portal pressure. The procedure is most commonly used for the treatment of recurrent oesophageal varical bleeding. Its main advantage over the traditional surgical shunts is a decrease in morbidity and mortality related with the procedure. Experience with TIPS procedure in treating ascites is still limited. There are few published reports and most are preliminary. Various studies have assessed the effects of TIPS in patients with refractory ascites. These studies have shown that the insertion of TIPS is associated with marked suppression of antinatriuretic systems and an improvement in renal function and in renal response to diuretics. However, TIPS may also impair hepatic function and induce severe encephalopathy. Moreover, this procedure has an additional problem of a high rate of shunt malfunction due to stenosis of the stent or the hepatic vein segment connecting with the prosthesis. Other technical complications that can occur with TIPS include haemobilia and biliary vascular fistula, liver haematoma, stent migration and intra-abdominal bleeding. A randomized trial comparing TIPS with paracentesis showed lower survival among patients treated with TIPS. At present, TIPS should be used for refractory ascites only as a part of randomized trials.

Liver transplantation can cure ascites by replacing the cirrhotic liver by a normal one. Overall, one year survival after liver transplantation exceeds 75%. Patients with refractory ascites who are otherwise good candidates for transplantation should be considered for liver transplantation.

References
11. Runyon BA, Montano AA, Akrivadias EA, et al. The serum-ascites albumin gradient is superior to the exudate-transudate


