Managing ascites in patients with chronic liver disease


Abstract
This article discusses the pathophysiology of ascites, a complication associated with chronic liver disease. The diagnosis and grading of ascites and assessment of patients with the condition are explored. In addition, the nursing and medical management of ascites is discussed, and recommendations for interdisciplinary working and education are suggested. Nursing knowledge of this complication is essential to ensure that patients with ascites are cared for effectively and that their comfort is maximised.

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Aims and intended learning outcomes
This article focuses on the assessment and management of ascites in patients with chronic liver disease. After reading this article and completing the time out activities you should be able to:
› Describe the pathophysiology of ascites.
› Discuss the diagnosis of the condition in patients with chronic liver disease.
› Explain the complications that may be associated with ascites.
› Outline the treatments and interventions used to manage the condition in patients with chronic liver disease.
› Describe the development and treatment of refractory ascites.

Introduction
Ascites is defined as the accumulation of fluid in the peritoneal cavity. It is not unique to liver disease and can be seen in patients with tuberculosis, peritoneal infection, pancreatic disease and malignancies (Rochling and Zetterman 2009). However, it is a frequent complication of liver dysfunction, and is observed in approximately 60% of patients with chronic liver disease (Ginés et al 1987). For the patient, the presence and experience of ascites causes significant discomfort and marks the transition from the liver being able to compensate for the damage, to a liver that can no longer perform its functions. This is known as decompensated liver disease. Once ascites is present, survival is markedly reduced, with median survival of approximately two years (D’Amico et al 2006).
Pathophysiology

The liver is supplied with 75% of its blood by the portal vein, with the remaining 25% of blood being delivered to the liver by the hepatic artery. The portal vein brings blood from the stomach, spleen, umbilicus, and small and large intestine (Bacon et al 2006), meaning the blood is rich in nutrients and toxins. The liver is comprised of millions of lobules, which are made up of hepatocytes and Kupffer’s cells. Each lobule has its own blood supply via a small branch of the portal vein and hepatic artery, along with a bile duct that make up the portal triad (McErlean 2011). About 1,500mL of blood is perfused through the liver each minute (Wong 2009), making it a highly vascularised organ.

In a healthy liver, endothelial cells that line the blood vessels are biologically active and produce several mediators. Endothelin and nitric oxide are both produced, inducing vasoconstriction and vasodilation, respectively. These mediators work together to regulate the diameter of and blood flow through blood vessels at a local level (Tousoulis et al 2012).

In liver disease, the flow of blood to the liver and the blood pressure in the blood vessels change, eventually leading to ascites. As the liver becomes damaged by the presence of disease, its architecture changes and it atrophies and appears shrunken because more scar tissue is formed and deposited. Increases in intrahepatic vasoconstriction agents, such as endothelin, also occur (Iwakiri and Groszmann 2007). The blood delivered to the liver, therefore, starts to meet resistance because of this change in architecture and intrahepatic vasoconstriction and pressure rises in the portal vein starting a process known as portal hypertension (Garcia-Tsao et al 2007). In addition, the regulation of mediators produced by the endothelial cells is altered and an imbalance occurs. Endogenous vasodilators such as nitric oxide are produced in excess in the presence of portal hypertension and result in vasodilation of the blood vessels in the splanchnic area, including the portal vein and its contributory vessels (Iwakiri and Groszmann 2007, Sargent 2009, Dooley et al 2011, Fullwood 2012).

This abdominal vasodilation results in blood being redirected away from major organs and an increased blood flow to the liver, worsening the pressure in the portal vein. The kidneys, which require a specific perfusion pressure to function effectively, receive a lower perfusion pressure than usual because the blood is redirected to the dilated abdominal vessels. This lower perfusion pressure is sensed and the kidneys activate the renin-angiotensin-aldosterone system to increase blood volume to improve their own blood supply. Renin production is stimulated, causing angiotensin to be released. This causes vasoconstriction of the arterial vessels leading to the kidney in an attempt to increase the pressure in the glomerulus. Angiotensin then activates aldosterone secretion and causes sodium to be reabsorbed in the loop of Henle, retaining water as a consequence. This increase in water absorption leads to an increase in blood volume (Sargent 2009, Dooley et al 2011). However, with the production of nitric oxide still in excess, vasodilation of the abdominal vessels continues as does the fall in blood pressure to the kidneys. Sodium and water, therefore, are continuously reabsorbed from the loop of Henle, making the blood volume increase, but the blood pressure remains inadequate (Rosner et al 2006) (Figure 1).

Eventually, the retained water in the engorged vessels starts to leak out of the intravascular space and into the lymphatic system. The amount of fluid exceeds the lymphatic drainage capacity, and fluid starts to leak from the lymphatic system into the peritoneal cavity (Sargent 2009).

**Grading, assessment and diagnosis**

The presence of fluid in the peritoneal cavity can cause abdominal distension. Depending on the severity of this distension, the ascites can be graded (European Association for the Study of the Liver (EASL) 2010) (Table 1).

**TIME OUT**

1. When examining a patient’s abdomen, palpation, inspection, percussion and auscultation can be used. In what order should these examinations be performed by a nurse to obtain the most accurate findings, and why?

**Complete time out activity**

Inspection of the abdomen should be performed initially. This involves looking at the abdomen carefully for signs of injury, such as bruises or wounds, and the presence of shiny, taut skin or prominent veins. The size, distension and symmetry of the abdomen should be noted along with the presence of bulging flanks (Rushing 2005). The abdomen should be auscultated to detect gurgling or rumbling reflecting gut peristalsis, and this must be performed before percussion and palpation because these examinations can cause such peristalsis to cease for a short period. Palpation is performed to detect any pain...
or tenderness, guarding or tensing of the abdomen followed by percussion.

Percussion can detect an accumulation of 1,000-1,500mL of fluid in the peritoneal cavity. Percussion is carried out with the patient in two positions. First, with the patient in the supine position, free fluid in the abdomen moves with gravity and will collect around the flanks. Percussion, therefore, reveals dullness in the flanks and tympanic sounds at the top of the abdomen where air has accumulated. Second, the patient is positioned on his or her side. Now the fluid moves with gravity to the front of the abdomen, allowing the air to be percussed at the top by the flank. This is known as the assessment of shifting dullness (Rushing 2005) (Figure 2), and can be done by a nurse with advanced assessment skills or a medical practitioner.

Once fluid has been detected and the ascites graded, it is important to confirm that the ascites is occurring as a result of liver dysfunction. As previously mentioned, there are other causes of ascites and it is important to rule these out before treatment options are considered.

A 30mL sample of ascitic fluid should be aseptically obtained by a medical practitioner, 10mL of which should be tested for white and red blood cell counts and albumin concentration. Table 2 shows the rationale for testing these parameters. The remaining 20mL of ascitic fluid is sent for culture and sensitivity testing. If infection is present, this will indicate which organism is responsible and the appropriate antibiotic therapy.

Psychological effects

The psychological needs of patients with ascites are important yet are often neglected because of the prioritisation of medical needs. The complications of and treatments for chronic liver disease can lead to lethargy, depression and reduced quality of life (Mucci et al 2013). The complications of end-stage liver disease, including ascites, have been shown to reduce mobility, limit physical activity and reduce patients’ health-related quality of life (Cox-North et al 2013). Nurses should be sensitive to the psychological needs of these patients and refer them to available support as appropriate. Referral to a counselling team may benefit these patients with ascites, particularly as they may experience loss of independence and deterioration in health (Sargent 2009). A multidisciplinary team approach to the management of patients with ascites is ideal to address mobility issues, optimal medical treatment, patient education and psychological support.
Complications

There are complications associated with ascites that affect both the morbidity and mortality of patients. Fluid accumulating in the peritoneal cavity reduces the space available for lung expansion and can lead to shortness of breath, particularly when in a supine position. The stomach can be compressed, leading to a reduced appetite, nausea and vomiting. When large volumes of fluid are present, the pressure can compress the bowel and reduce blood supply to abdominal organs. Reduced gut function and renal perfusion are possible consequences (Garcia-Tsao 2011). A list of further complications is shown in Table 3.

It is important that healthcare professionals are able to assess the presence of ascites and commence treatment in a timely manner to limit the risk of complications. It is also important that healthcare professionals are able to monitor patients with ascites for signs of any complications and to commence appropriate treatments as soon as possible.

Nursing care

While patients with grade 1 ascites may not require immediate treatment, measures should be taken to prevent the development of grade 2 or 3 ascites (Table 1). Patients with grade 2 or moderate ascites can be treated at home with follow up, unless other complications of cirrhosis are also present. Treatment is aimed at reducing sodium intake and increasing renal sodium excretion (Garcia-Tsao 2011). This reduces the formation of ascites by reducing the amount of sodium and water retained by the kidneys (Sargent 2009). Advising a reduced sodium diet may seem conflicting when hyponatraemia is a potential complication. Hyponatraemia may be dilutional in nature, caused by the excessive retention of salt and therefore water, which follows salt to form ascites. If less salt is consumed then less water will be reabsorbed, thus reducing ascites formation (Bacon et al 2006).

According to Garcia-Tsao (2011), salt should be restricted to 70-90mmol/day. This equates to a non-added salt diet rather than the more traditional recommendation of a low salt diet of 22-40mmol/day. The traditional regimen has been found to compromise protein and calorie intake in a patient group that is already experiencing malnutrition as a result of chronic liver disease (Garcia-Tsao 2011), and therefore it is no longer recommended.

Any patient prescribed a reduced salt diet will benefit from referral to a dietician for both a formal nutritional review and education (Moore and Aithal 2006). Relatives or those cooking for the patient should also attend any patient education sessions to aid compliance with this treatment regimen. The need for nursing education is evident as nurses should be able to promote this treatment and to educate patients about high salt content foods to be avoided. An awareness of medications that are high in salt is also required so that these may be avoided.

Alcohol abstinence is a vital component in the treatment of ascites caused by alcohol-related liver disease. Early abstinence may reverse portal hypertension and sodium retention (Runyon 2009). Referral to a specialist nurse is, therefore, beneficial and recommended in patients with alcohol or substance misuse issues (National Institute for Health and Care Excellence (NICE) 2011).

Complete time out activity

### Table 2

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Rationale</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>White cell count</td>
<td>Presence of &gt;250 white blood cells/mm³ indicates infection.</td>
<td>If the fluid is drained when infection is present, septicemia may occur.</td>
</tr>
<tr>
<td>Red cell count</td>
<td>Presence of red blood cells indicates bleeding within the peritoneal cavity.</td>
<td>Fluid in the abdomen can tamponade bleeding. If the fluid is drained, the bleeding point may haemorrhage further as the pressure pushing against it would decrease.</td>
</tr>
<tr>
<td>Albumin content</td>
<td>Comparing the serum albumin with the ascitic albumin content indicates the cause of ascites.</td>
<td>Serum ascites albumin gradient &gt;11g/L (&gt;1.1g/dL) indicates the ascites is present because of liver disease.</td>
</tr>
</tbody>
</table>

(Moore and Aithal 2006, Runyon 2013)

2) Aldosterone antagonists and loop diuretics are two groups of diuretics commonly used in the treatment of grade 2 ascites. Can you name at least one diuretic that falls in each category and outline their specific mode of action?
Diuretics are used in conjunction with a reduced salt diet to increase the excretion of sodium via the kidneys. Aldosterone antagonists are more effective than loop diuretics in the management of ascites because they treat the pathophysiology of the accumulation of ascites as well as preserving potassium levels. Patients with a first episode of ascites should receive an aldosterone antagonist such as spironolactone at doses of between 100mg and 400mg daily (Rosner et al 2006). The maximum recommended weight loss during diuretic therapy should be 0.5kg daily in patients without peripheral oedema and 1kg daily in patients with peripheral oedema (EASL 2010). In patients who do not respond to aldosterone antagonists, which means a reduction of body weight of less than 2kg per week, or if the patient develops hyperkalaemia or recurrent ascites, a loop diuretic such as furosemide should be added to the treatment regimen at a dose of 40-160mg daily (Runyon 2013). Patients should be monitored frequently for clinical and biochemical parameters, particularly during the first month of treatment. Side effects of diuretics include renal failure, hepatic encephalopathy, electrolyte disorders, gynaecomastia and muscle cramps (British National Formulary 2013). Monitoring should, therefore, include electrolyte balance, renal function and assessment for signs of hepatic encephalopathy.

Bed rest with legs elevated is advocated by Alexander et al (2006) as the most comfortable position and is likely to reduce peripheral oedema. The adoption of an upright position has been linked with activation of the renin-angiotensin-aldosterone system, a reduction in sodium excretion and a reduced response to diuretics (Bacon et al 2006). However, there is no evidence to support the relationship between bed rest and the effectiveness of diuretics; therefore, complications of bed rest must be considered (Moore and Aithal 2006). Bed rest may lead to muscular atrophy, constipation, increased risk of deep vein thrombosis and prolonged hospital stay.

Fluid restriction as a standard treatment is not advisable, and there are no studies proving its efficacy in the management of ascites (Runyon 2013). Fluid restriction may exacerbate the severity of central hypovolaemia, which may increase the secretion of antidiuretic hormone and result in a further deterioration in renal function. Fluid restriction may be recommended if dilutional hyponatraemia of less than 130mmol/L is present; however, it must always be used with caution (Garcia-Tsao 2011).

Large-volume paracentesis is the insertion of a catheter into the peritoneal cavity to drain the fluid and is the first-line therapy in patients with large or grade 3 ascites. It is usually performed by a medical practitioner and can be undertaken in an outpatient setting; however, ascites may re-accumulate quickly and require further drainage. There is no indication that coagulation or platelet count should be corrected before this procedure because the risk of bleeding is low (Pache and Bilodeau 2005).

Large-volume paracentesis should be performed using an aseptic technique by pulling the skin down, holding it firmly and inserting the paracentesis catheter – the Z-track technique.
for intramuscular injection (Rochling and Zetterman 2009). This technique reduces the risk of ascitic fluid leaking from the insertion site once the paracentesis catheter is removed (Figure 3). The nurse should be present to ensure the patient is comfortable and understands the procedure. Vital signs monitoring must be performed before the procedure to establish a baseline and then every 15-30 minutes to assess cardiovascular stability (Sargent 2009). A complication of large-volume paracentesis is post-paracentesis circulatory dysfunction, which manifests as a low blood pressure, a high heart rate (Salerno et al. 2010) and rapid re-accumulation of ascites (EASL 2010).

The fluid is then drained via the catheter continuously over four to six hours. The drain should not be clamped during this time because of the increased risk of infection (Sargent 2009). Fluid replacement is required to prevent cardiovascular complications. Studies have examined the most effective fluid to administer for paracentesis, and a meta-analysis of randomised controlled trials found that albumin was the most effective plasma expander in this patient population (Bernardi et al. 2012). Albumin should be administered at a ratio of 100mL 20% human albumin solution for every 2-3L of ascites drained (Moore and Aithal 2006, EASL 2010). In patients undergoing large-volume paracentesis of 5L or less of ascites, the risk of developing post-paracentesis circulatory dysfunction is low and, therefore, evidence suggests that volume replacement is not required (Moore and Aithal 2006, EASL 2010). In clinical practice, however, albumin is often administered for every 2-3L of ascites drained regardless of the total volume drained to minimise cardiovascular complications.

Once the paracentesis catheter is removed, a dry dressing can be applied over the insertion site if there is no fluid leaking. If the Z-track method of insertion is not used, a stoma collection bag is often required because of persistent leakage of ascitic fluid from the paracentesis site (Sargent 2009).

**Complete time out activity 3**

After large-volume paracentesis, patients should receive the minimum dose of diuretics to prevent the re-accumulation of ascites (Dooley et al. 2011). Patients need to have their blood results monitored for electrolyte abnormalities and renal impairment. A fluid balance chart will give an accurate account of the volume of fluid drained and replaced, and
daily weights will also show the volume of fluid lost. It is important that patients are weighed at the same time each day with the same clothes to obtain accurate readings. Pain scores should be obtained before draining ascites and reassessed throughout the procedure. In addition, patients should be encouraged to report any abdominal discomfort or bleeding around the paracentesis site.

**Complete time out activity 4**

**Contraindicated drugs**

Non-steroidal drugs are contraindicated in patients with ascites because of the increased risk of developing further sodium retention, hyponatraemia, and renal failure (Hampel et al 2001). Drugs that decrease arterial pressure or renal blood flow such as angiotensin-converting enzyme inhibitors and angiotensin-II receptor antagonists should be avoided because of the increased risk of renal impairment (Hampel et al 2001). The use of aminoglycoside antibiotics, for example gentamicin, is associated with an increased risk of renal failure (Garcia-Tsao 1998) and should be used with caution and in association with regular monitoring of creatinine levels in patients with ascites.

**Management of refractory ascites**

Refractory ascites refers to ascites that does not respond to medical management or re-accumulates immediately after treatment, or in patients for whom medical treatment cannot be given because of untoward side effects such as muscle cramps or severe electrolyte imbalance. Treatment options available for refractory ascites include large-volume paracentesis with administration of albumin, transjugular intrahepatic portosystemic shunt (TIPSS), implanted automated pump system insertion and liver transplantation.

**Transjugular intrahepatic portosystemic shunt**

A TIPSS is the insertion of a mesh stent between the portal vein and the hepatic vein. This assists in reducing the pressure in the portal vein and reduces the accumulation of ascites (Sargent 2009). The TIPSS has been found to be successful at controlling refractory ascites in 75% of cases (Riggio et al 2008). It has also been linked to a decrease in activation of the renin-angiotensin-aldosterone system and improved nutritional state because of reduced pressure on the stomach and gastrointestinal tract (Riggio et al 2008). However, although the TIPSS has been found to be more effective than large-volume paracentesis in preventing recurrence of ascites in randomised comparative trials, it is associated with a much higher risk of severe encephalopathy (Riggio et al 2008).

A stent reduces the pressure in the portal vein but also reduces the amount of blood passing through the liver for detoxification. Some blood will, therefore, return to the heart with raised levels of toxins from the gut, including ammonia. This is one of the hypothesised causes of hepatic encephalopathy (Houlston and O’Neal 2009) and raised levels in the blood can lead to neurological changes associated with hepatic encephalopathy. Despite the relative success of the TIPSS, it may not be suitable for all patients (Moore and Aithal 2006).

**Implanted automated pump system**

The automated pump system is surgically implanted subcutaneously, and it draws ascitic fluid from the abdominal cavity to the bladder where it is excreted through normal urination (Bellot et al 2013). Excess abdominal fluid is drawn automatically and continually into the bladder. The speed and quantity of fluid removal may be adjusted to the clinical and social requirements of the individual patient. This system helps to reduce the need for repeat paracentesis.

A study of 40 patients with refractory ascites who received an implanted pump showed that 90% of ascites was removed and the need for large-volume paracentesis was reduced. However, there were a large number of complications associated with both the insertion procedure and the system itself, for example infection and bladder catheter dislocation (Bellot et al 2013). There are larger studies underway looking at the efficacy of the implanted automated pump system in comparison to large-volume paracentesis.

**Liver transplantation**

Patients with refractory ascites should be considered for liver transplantation because their median survival is approximately six months (EASL 2010). At this advanced stage of liver disease, transplantation is the only option. Garcia-Tsao (2011) recommended early assessment, before further complications cause deterioration in the patient’s condition, which may limit his or her suitability for surgery. Because ascites marks the change from compensated liver disease to...
decompensated liver disease, transplant assessment may be suggested when this complication arises as a result of the reduced survival associated with it (D’Amico et al 2006).

Conclusion

Ascites is a common complication of chronic liver disease, marking the point where the liver is no longer able to function effectively because of the degree of damage present. The fluid in the peritoneal cavity causes significant discomfort to patients and increases their susceptibility to infection, and respiratory and renal dysfunction. Healthcare professionals should have an understanding of the treatments and interventions available for managing ascites. Interdisciplinary working is important in the management of these patients to ensure their physical and psychosocial needs are met.

There are treatments available for patients with refractory ascites; however, these can have adverse complications. Liver transplantation is the optimal treatment and should be considered for patients when ascites is first identified. NS

Complete time out activity

References


