BMJ Best Practice

Spontaneous bacterial peritonitis

Straight to the point of care

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Summary

Spontaneous bacterial peritonitis (SBP) is one of the most frequently encountered bacterial infections in patients with cirrhosis, and most commonly seen in patients with end-stage liver disease.

Key symptoms are abdominal pain, fever, vomiting, altered mental status, and gastrointestinal (GI) bleeding. However, patients are commonly minimally symptomatic, and may even be asymptomatic.

Ascitic fluid laboratory tests should include cell count and culture.

SBP is diagnosed by an ascitic fluid absolute neutrophil count >250 cells/mm^3, in the absence of an intra-abdominal surgically treatable source of infection. Positive blood cultures confirm the diagnosis.

Treatment is directed primarily at early administration of appropriate empirical antibiotic regimens. The practitioner must be aware of local resistance patterns, with particular reference to increased third-generation cephalosporin and fluoroquinolone resistance.

Patients with sepsis, history of fluoroquinolone prophylaxis, nosocomial-acquired SBP, or a history of previous infections with resistant organisms are likely to require broader initial empirical coverage.

Albumin is indicated in the treatment of patients with SBP; particularly for those with kidney dysfunction.

Continuous antibiotic prophylaxis is indicated in patients with a previous episode of SBP, upper GI bleeding, or in patients with an ascitic fluid protein concentration <15g/L (<1.5 g/dL) plus evidence of severe liver failure (Child-Pugh score >9 points with serum bilirubin >51.31 micromol/L [>3 mg/dL]) and/or renal dysfunction (serum creatinine >106 micromol/L [>1.2 mg/dL], urea >8.92 mmol/L [>25 mg/dL], or serum sodium <130 mmol/L [<130 mEq/L]).

Definition

Spontaneous bacterial peritonitis (SBP) is an infection of ascitic fluid that cannot be attributed to any intra-abdominal, ongoing inflammatory, or surgically correctable condition. It is one of the most frequently encountered bacterial infections in patients with cirrhosis.

Epidemiology

Studies have demonstrated a SBP prevalence of 12% in patients with ascites admitted for decompensated cirrhosis, 18% in those admitted for hepatic encephalopathy, and 10% to 14% in those admitted with acute gastrointestinal haemorrhage.[4] [5] [6] [7] Among asymptomatic patients receiving outpatient paracentesis, there is an approximately 2% prevalence.[8] [9] [10] There are no data on sex or race prevalence of SBP beyond that which would be associated with ascites itself.

Although SBP may occur in the patient with ascites caused by malignancy, kidney failure, or congestive heart failure, it is a much less common occurrence than in patients with ascites due to end-stage liver disease.

Increased infections due to gram-positive cocci have been reported. Studies suggest that these changes are associated with long-term hospitalisation of patients with end-stage liver disease and the use of prophylactic antibiotics with superior activity against gram-negative organisms after an initial episode of SBP.[11] [12] However, gram-negative bacteria remain the most common pathogens in SBP.

Studies from different countries indicate that SBP pathogens isolated from ascitic fluid are increasingly resistant to antimicrobial therapy. One study found antibiotic resistance in SBP in North America to be 17.8%, with methicillin-resistant *Staphylococcus aureus* the most common resistant organism.[13] Resistance rates to cephalosporins and fluoroquinolones may be as high as 40%; 30% prevalence of extended spectrum beta-lactamases (ESBL) resistant *Escherichia coli* has been reported.[14] [15]

Aetiology

The aetiology of SBP is infection of the ascitic fluid. More than 92% of all cases of SBP are monomicrobial.[16] The presence of polymicrobial infection significantly increases the risk for secondary peritonitis.

Gram-negative bacteria remain the most common pathogens in SBP. However, there has been an increase in infections due to gram-positive cocci. Studies have suggested that these changes are associated with long-term hospitalisation of patients with end-stage liver disease and the use of prophylactic antibiotics after an initial episode of SBP. Prophylactic antibiotics generally cover gram-negative organisms better than gram-positive organisms.[11] [12] [17] There has also been a case report of carbapenem-resistant *Klebsiella pneumoniae* , which is of particular concern due to the potential for widespread transmission of resistance due to its mobile genetic elements.[18]

The most common pathogens are:[19] [20] [21]

- *Escherichia coli* (reported in 39% to 61% of cases)
- *Staphylococcus aureus* (3% to 12%)
- *Streptococcus pneumoniae* (2% to 11%)
- *Enterococcus faecalis* (4% to 17%)
- *Klebsiella pneumoniae* (4% to 20%)
- *Pseudomonas aeruginosa* (3% to 9%).

Less common pathogens are:

- *Proteus* species
- *Acinetobacter* species
- *Citrobacter freundii*

- *Bacteroides fragilis*
- *Aeromonas hydrophila*
- *Listeria monocytogenes* [22]
- *Vibrio vulnificus* .

Rare organisms noted in case reports include:

- *Haemophilus influenzae* , non-typeable[23] [24]
- *Haemophilus parainfluenzae* [25]
- *Neisseria meningitidis* [26]
- *Salmonella typhimurium* [27]
- *Salmonella paratyphi A* [28]
- *Leclercia adecarboxylata* [29]
- *Leminorella grimontii* [30]
- *Aerococcus urinae* [31]
- *Gemella morbillorum* [32]
- *Actinomyces* species[33]
- *Streptococcus salivarius* [34]
- *Ochrobactrum anthropi* [35]
- *Arcanobacterium haemolyticum* [36]
- *Cryptococcus neoformans* (even in HIV-negative patients)[37] [38]
- *Coccidioides immitis* [39]
- *Candida* species[40]
- *Brucella* species[41]
- *Enterococcus hirae* [42]
- *Enterococcus gallinarum* [43]
- *Enterococcus casseliflavus* [43]
- *Bordetella bronchiseptica* [44]
- *Plesiomonas shigelloides* [45]
- Expanded dengue syndrome[46]
- *Edwardsielle tarda* [47]

Streptococcus viridans commonly grows as a contaminant in peritoneal fluid cultures.[48] However, it also has been identified as a pathogen in other studies.[49] [50]

Pathophysiology

SBP is believed to develop primarily through haematogenous spread of bacteria with subsequent colonisation of the ascitic fluid. The source of the bacteria can be classified into intestinal (more commonly) and non-intestinal (less commonly).

With intestinal sources, bacterial translocation from the intestinal flora occurs by movement to the mesenteric lymph nodes and from there to the bloodstream. The pathophysiology of cirrhosis predisposes to this colonisation and impairs the ability to resist subsequent infection. The bacterial translocation is believed to involve numerous mechanisms that are found in patients with advanced cirrhosis:[51]

- Depression of the reticulo-endothelial system function of the liver
- Intestinal bacterial overgrowth, likely to be caused by intestinal hypomotility
- Venous stasis, resulting from portal hypertension, which causes increased intestinal permeability to enteric bacteria.

SBP is sometimes caused by organisms that are not part of the intestinal flora. In such cases, the source of bacteria is believed to be an extraintestinal infection or procedure, such as:

- A respiratory infection
- A urinary tract infection
- An invasive procedure (e.g., endoscopic sclerotherapy for oesophageal varices, which is associated with a 5% to 30% rate of bacteraemia; central venous catheterisation; urinary catheterisation; paracentesis; transjugular intrahepatic portosystemic shunt placement).[51] [52][53]

After haematogenous spread of the bacteria to the ascitic fluid, complement in the fluid can serve to protect from infection. However, many patients with cirrhosis have low ascites protein concentration, which correlates with decreased opsonic activity and predisposes to infection.[54]

Classification

International Ascites Club[1]

- Spontaneous bacterial peritonitis (SBP)
  - Defined by an absolute neutrophil count (ANC) >250 cells/mm³.
  - Because of the difficulties in culturing the pathogen, the criteria do not require a positive culture, although some manuscript authors have used this as part of their diagnosis of SBP.
- Culture-negative neutrocytic ascites (CNNA)
  - Defined by an ANC >250 cells/mm³, with no culture growth, this is considered a variant of SBP.
  - Studies have demonstrated similar short- and long-term mortality in patients with CNNA and SBP.[2] [3]
- Bacterascites
  - The patient must fulfil all of the following criteria: positive ascitic fluid culture; ANC <250 cells/mm³; and no evidence of systemic or local infection.

Case history

Case history #1

A 53-year-old man with a history of hepatitis C presents with a complaint of abdominal distention, fever, vomiting, and blood in his stool. Paracentesis has improved symptoms on the numerous occasions that he has previously presented with abdominal distension.

Case history #2

A 46-year-old woman with a history of long-standing alcoholism and previous episodes of hepatic encephalopathy presents with altered mental status and worsening abdominal distention.

Approach

Diagnosis is made, first by eliciting the presence of ascites, then by looking for signs and symptoms consistent with peritoneal irritation or signs of systemic infection, and finally by confirmation with peritoneal fluid testing.

History and physical examination

Patients with end-stage liver disease presenting with hepatic encephalopathy, decompensated cirrhosis, increase in ascites volume and/or frequency, or gastrointestinal (GI) bleeding are at particularly high risk for SBP. Patients who have recently had a therapeutic endoscopy are also at risk. Ascites due to malignancy, renal insufficiency, or congestive heart failure also carry a risk, albeit one that is less well-described than the risk in patients with end-stage liver disease.[78]

The typical presentation of SBP includes abdominal pain, fever, increasing ascites, ileus and/or altered mental status in a patient with known liver disease; however, one third of patients also may be asymptomatic or present with only mild symptoms.[1] [61][79]

The wide range of possible physical examination findings include symptoms of peritonitis (e.g., vomiting, diarrhoea, ileus, abdominal tenderness), systemic inflammation (e.g., hypothermia, hyperthermia, tachycardia, tachypnoea), shock, hepatic encephalopathy, renal failure, and GI bleeding.[80]

Peritoneal fluid testing is the only way to confirm or rule out SBP; signs, symptoms, and clinical gestalt are unreliable.[81] [82]

Detection of ascites

There are several manoeuvres for the detection of ascites, including examining for flank dullness, shifting dullness, fluid wave, and auscultatory percussion.

Flank dullness is elicited by percussion of the abdominal wall starting at the periumbilical region and going outwards to the dependent areas of the flanks. If ascites is present, there is a change from tympany to dullness.

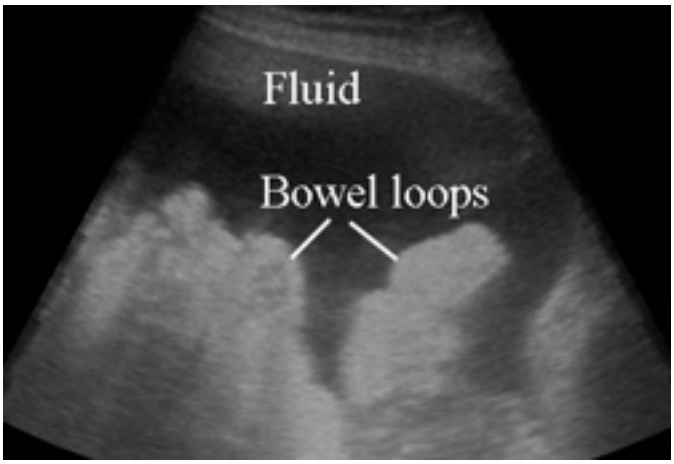
To detect shifting dullness, the abdomen should be percussed from the umbilicus laterally and the level noted at which tympany turns to dullness. Then the patient should be positioned in the right lateral decubitus position. The abdomen is percussed again, starting on the left side and going toward the right. If ascites is present, the level at which tympany turns to dullness will have shifted.

Assistance is required to detect a fluid wave. The patient should be in the supine position, and the ulnar side of the assistant's hand and forearm is placed lengthways in the midline of the anterior abdominal wall. The examiner's hands are then placed on either side of the abdomen. When one hand strikes the abdomen, a fluid wave will be felt by the other hand in a patient with ascites.

Auscultatory percussion is conducted with the patient standing. Auscultation is started just above the symphysis pubis while percussing from the costal margin down to the pelvis. Normally there is a sharp transition from quiet to loud at the pelvic border. In a patient with ascites, the transition occurs higher up.

The sensitivities and specificities of these signs for ascites vary widely. Percussion of the abdominal wall is the most sensitive of all the manoeuvres for ascites, with a sensitivity of 84%.[83]

Ultrasound is the definitive test for the detection of ascites. Up to 25% of patients thought to have ascites by physical examination techniques who go on to have an abdominal ultrasound are found to have no or minimal ascites.[84] Sonography can determine adequacy of fluid for paracentesis and can help localise the procedure.



Abdominal ultrasound showing large amount of ascites with bowel loops

From the personal collection of Brian Chinnock, MD; used with permission

Initial investigations

Initial laboratory tests should include:[61] [85]

- FBC, which may show an elevated white cell count; anaemia may be a clue to a GI bleed.
- Creatinine, as hepatorenal syndrome may occur concomitantly.
- Liver function tests, to establish baseline labs and monitor the health of the liver.
- PT/INR, which should be performed if there is GI or other bleeding.
- Blood cultures, which may assist in identifying the pathogenic organism, as the yield from peritoneal fluid culture is poor. The Infectious Diseases Society of America (IDSA) recommends 2-3 sets of blood cultures for identification of concomitant bacteraemia.

Diagnostic paracentesis

Owing to the high prevalence of SBP in hospitalised patients with cirrhosis and ascites, diagnostic paracentesis should be performed on all patients with these two conditions, even in the absence of symptoms suggestive of infection.[61] [64] Patients with known ascites who present with GI bleed or hepatic encephalopathy should also generally be evaluated for SBP. Diagnostic paracentesis has been shown to be safe in patients with significant coagulopathy or thrombocytopenia; fresh frozen plasma or platelet transfusion is not indicated before diagnostic paracentesis in patients with coagulopathy.

Diagnostic paracentesis should be performed as early as possible.[61] Early paracentesis of hospitalised patients with ascites was associated with lower all-cause mortality, SBP mortality, and 30-day re-admission rate in a large inpatient database study.[90]

Ascitic fluid laboratory analysis

The key tests on peritoneal fluid for the analysis of SBP are a cell count and culture.[61] A minimum of 10 mL (and up to 50 mL if available) of peritoneal fluid should be cultured aseptically at the bedside in aerobic and anaerobic blood culture bottles before giving antibiotics.[61] [85] Additional laboratory testing

should include fluid analysis for protein, lactate dehydrogenase (LDH), and pH.[85] The gross appearance of the fluid can also be examined by laboratory staff.

Cell count

- A peritoneal fluid absolute neutrophil count (ANC) >250 cells/mm³ is the accepted criterion for the diagnosis of SBP.[61] [64]
- Although an ANC >500 cells/mm³ is more specific for the diagnosis, the danger of missing SBP in a patient with an ANC count between 250-500 cells/mm³ is unacceptably high.[1] Therefore, a patient who is felt to be at high risk for SBP should be considered for treatment.
- Automated cell counters have been found to be equivalent to manual cell counts in the examination of ascitic fluid.[91] [92] [93]

Culture

- Culture of ascitic fluid, even in patients with obvious SBP, has a low yield because of the low concentration of bacteria compared with infections in other organic fluids (e.g., urine).
- Inoculating ascitic fluid directly into blood culture bottles at the bedside has demonstrated significantly increased yield and should be the standard method of collection.[61] [94] However, cultures are still negative in approximately 50% of patients with an ascites ANC >250 cells/mm³.[1] [19]
- Polymicrobial growth may be suggestive of secondary peritonitis.

Fluid appearance

- Subjective descriptions of ascitic fluid by laboratory technicians as abnormal with the descriptors 'hazy', 'cloudy', or 'bloody' have a sensitivity of between 72% and 98% for the detection of SBP.[82] [95]
- Clinical impression, including an assessment of ascitic fluid appearance, should not be used to exclude the diagnosis.[82]

Other tests that may be performed on ascitic fluid include glucose, acid fast bacterium (AFB) stain and culture, fungal culture, and microscopy for ova and parasites, depending on the clinical context.[61] [85] [96] The measurement of carcinoembryonic antigen and alkaline phosphatase can be performed to help differentiate SBP from secondary peritonitis.[97]

Measurement of the serum-ascites albumin gradient (SAAG) and ascitic total protein concentration should be considered for a first episode of ascites, with SAAG measurement recommended if a cause of ascites different from cirrhosis is suspected.[61] [64] Ascitic fluid lactoferrin can also be measured. Along with helping to identify SBP in a cirrhotic patient with ascities, an elevated lactoferrin in a cirrhotic patient without SBP can indicate a developing hepatic carcinoma.[98]

Highly-sensitive leukocyte esterase reagent strip testing (Periscreen), a test created to examine peritoneal dialysis fluid for infection, has been studied in ascitic fluid to rule out SBP and may be of use if laboratory peritoneal fluid testing is not available. In a multi-centre study that assessed 84 ascitic fluid samples from 9 outpatients (17 ascitic fluid samples) and 31 inpatients (67 ascitic fluid samples) diagnosed with SBP, the leukocyte esterase reagent strip test had a sensitivity of 92% and specificity of 57%.[99] An emergency department-based study demonstrated a sensitivity of 95%.[100]

Bedside (standard urine) leukocyte esterase reagent strip testing of ascitic fluid has been studied in the evaluation of SBP. The reagent strip is dipped into ascitic fluid, and after 60-120 seconds the result is analysed according to the colourimetric scale for that reagent strip. Most studies used a strip colour that

gives a positive result as corresponding to between 15 (1+) and 125 leukocytes/mL (3+). One meta-analysis found sensitivities ranging from 45% to 100% and specificities ranging from 81% to 100%.[101]

Low sensitivity demonstrates that bedside (standard urine) leukocyte esterase reagent strip testing is not suitable for rapidly ruling out SBP. At this time they are not widely used, nor recommended in current EASL or AASLD guidelines. However, they may play a role in facilitating prompt administration of antibiotic therapy, particularly in settings without available ascitic fluid microscopy testing.

CT scan abdomen

If perforation is suspected within the abdomen, CT imaging should strongly be considered.[102]

CT should also be considered in patients with findings suggestive of secondary peritonitis (such as bile-stained fluid, polymicrobial growth on ascites fluid culture, no clinical improvement despite appropriate antibiotics for 48 hours, and no history of liver disease or malignancy to explain the ascites) as it may demonstrate free air.[103]

Clinical decision score

The chronic liver failure-sequential organ failure assessment (CLIF-SOFA) can help to determine the severity of illness in patients presenting with SBP. It is similar to the SOFA score, the predictive scoring system that assesses severity of illness in patients with sepsis. CLIF-SOFA has been shown to have better predictive value for in-hospital mortality in cirrhotic patients with infection compared to Sepsis-3 criteria or qSOFA.[104] Patients with CLIF-SOFA scores ≥7 have >20% mortality and so might benefit from broader empirical antibiotic therapy.[105]

DIAGNOSIS

History and exam

Key diagnostic factors

presence of risk factors (common)

- Patients with end-stage liver disease presenting with hepatic encephalopathy, decompensated cirrhosis, increase in ascites volume and/or frequency, or gastrointestinal bleeding are at particularly high risk.
- Patients who have recently had a therapeutic endoscopy are also at risk.
- Malignant ascites also carries a risk, albeit one that is less well-described than the risk in patients with end-stage liver disease.[78]

abdominal pain or tenderness (common)

- Common presenting complaint or finding, occurring in 50% to 94% of patients.[16] [81]

signs of ascites (common)

- Clinical manoeuvres for the detection of ascites include examining for flank dullness, shifting dullness, fluid wave, and auscultatory percussion.
- The sensitivities and specificities of these signs for ascites vary widely. Percussion of the abdominal wall is the most sensitive of the manoeuvres for ascites, with a sensitivity of 84%.[83]

fever (common)

- Fever is detected in 35% to 68% of patients.[81] [106]

nausea/vomiting (common)

- Caused by the intestinal hypomotility and bacterial overgrowth associated with cirrhosis and SBP.

diarrhoea (common)

- Caused by the intestinal hypomotility and bacterial overgrowth associated with cirrhosis and SBP.

altered mental status (common)

- In patients admitted to the hospital with hepatic encephalopathy, there was an 18% prevalence of SBP in 1 series.[5]

gastrointestinal bleed (common)

- In patients with ascites hospitalised for acute gastrointestinal bleeding, there is a 10% to 14% prevalence of SBP.[6] [7]

Other diagnostic factors

hypothermia (common)

- Signs of sepsis may be present.

hypotension (common)

- Signs of sepsis may be present.

tachycardia (common)

- Signs of sepsis may be present.

Risk factors

Strong

decompensated hepatic state (usually cirrhosis)

- In patients with advancing cirrhosis (increasingly frequent episodes of tense ascites, gastrointestinal bleeding, hepatic encephalopathy), there can be worsening bacterial intestinal overgrowth with increased haematogenous spread, as well as decreased ascitic protein content and opsonic activity to fight off infection.

low ascitic protein/complement

- A randomised, placebo-controlled trial found that patients with a total ascitic protein concentration <15 g/L (<1.5 g/dL) were at increased risk for development of SBP compared with those with a higher protein concentration.[55] However, subsequent cohort studies have failed to replicate this finding.[56] [57]

gastrointestinal bleeding

- In patients with ascites hospitalised for acute gastrointestinal bleeding, there is a 10% to 14% prevalence of SBP.[6] [7] This is believed to be due to increased accessibility of enteric bacteria to the bloodstream during the haemorrhagic episode.

endoscopic sclerotherapy for oesophageal varices

- Causes bacteraemia in 5% to 30% of patients, which increases the risk of haematogenous spread to the ascitic fluid.[51] [52] [53] Endoscopic band ligation has not been shown to confer an increased risk.

Weak

ascites due to malignancy, renal insufficiency, or congestive heart failure

- There are no studies that describe whether patients with ascites due to end-stage liver disease are at higher risk for SBP than those with ascites not due to liver disease. However, there is some suggestion that mechanisms in cirrhosis that cause increased susceptibility to infection may not be present in patients without cirrhosis.[58]

extra-intestinal infection

- Respiratory and urinary tract infections may seed to the ascitic fluid; in these cases, the organisms causing the SBP may not be part of the normal intestinal flora.

invasive procedures

- Invasive procedures, such as central venous catheterisation, urinary catheterisation, paracentesis, and transjugular intrahepatic portosystemic shunt placement, have been associated with SBP.

use of proton-pump inhibitors (PPIs)

- PPIs facilitate enteric colonisation, overgrowth, and translocation into the peritoneum, which might increase the risk for SBP. Meta-analyses demonstrate PPI use as an independent predictor of increased SBP risk in cirrhotic patients.[59] One recent meta-analysis looking at over 10,000 patients demonstrated a weak but statistically significant association between SBP and PPI use.[60] The decision to prescribe a PPI for a patient with cirrhosis should be made carefully.

Investigations

1st test to order

Test	Result
FBC • Leukocytosis common with SBP, but may be absent. Worsening anaemia may suggest gastrointestinal bleeding.	**leukocytosis, anaemia**
serum creatinine • Hepatorenal syndrome may occur in patients with decompensated cirrhosis.	**may be elevated**
LFT • Used to establish baseline labs and monitor the health of the liver. In the patient with end-stage liver disease, bilirubin testing can be used to calculate a Model for End-Stage Liver Disease (MELD) score, MELD-Na, or Child-Pugh score to determine mortality rate and may assist in decision-making for SBP prophylaxis.	**In end-stage disease, liver enzymes and bilirubin often elevated; albumin decreased**
prothrombin time/INR • An elevated PT/INR is not a contraindication for diagnostic or therapeutic paracentesis.[107] Useful if the patient has GI haemorrhage or other bleeding complication. Is a component of Child-Pugh and MELD scoring systems to determine mortality rate.	**elevated**
blood cultures • As yield of peritoneal fluid culture is poor, blood cultures may assist in identifying the pathogenic organism. The Infectious Diseases Society of America (IDSA) recommends 2-3 sets of blood cultures for identification of concomitant bacteraemia.[85]	**growth of causative organism**
ascitic fluid appearance • Subjective descriptions of ascitic fluid by laboratory technicians as 'hazy', 'cloudy', or 'bloody' have a sensitivity of between 72% and 98% for the detection of SBP.[82] [95] • Clinical impression, including an assessment of ascitic fluid appearance, should not be used to exclude the diagnosis.[82] •	**'hazy', 'cloudy', 'bloody'**
ascitic fluid absolute neutrophil count (ANC) • ANC is diagnostic for SBP. If haemorrhagic ascites is present, subtract 1 neutrophil for every 250 RBCs. • Although an ANC >500 cells/mm³ is more specific for the diagnosis of SBP, the danger of missing the diagnosis of SBP in a patient with an ANC count of 250-500 cells/mm³ is unacceptably high.[1] • Automated cell counters have been found to be equivalent to manual cell counts in the examination of ascitic fluid.[91] [92] [93]	**>250 cells/mm³**
ascitic fluid culture • Must be performed by bedside inoculation of 10 mL fluid into blood culture bottles. • Even with bedside inoculation, culture is negative in 50% of patients with SBP.[1] [19] • Polymicrobial growth is suggestive of secondary peritonitis.	**growth of causative organism**

Test	Result
ascitic fluid protein, glucose, lactate dehydrogenase (LDH), pH • Normal ascites should have low protein and LDH, and a glucose >50 mg/dL, and normal pH. A study comparing ascitic protein, glucose, and LDH in 6 patients with gastrointestinal perforation into their ascitic fluid (secondary peritonitis) and 32 patients with SBP found that all 6 of the patients with secondary peritonitis met at least two of the criteria for secondary peritonitis as follows: protein >10 g/L (>1 g/dL); glucose <2.8 mmol/L (<50 mg/dL); LDH >225 units/L. Only two of the patients with SBP fulfilled two of these criteria.[85] [108] [109]	**protein >10 g/L (>1 g/dL); glucose <2.8 mmol/L (<50 mg/dL); LDH >225 units/L raises likelihood of secondary peritonitis; ascitic fluid pH often decreased in SBP**

Other tests to consider

Test	Result
serum-ascites albumin gradient (SAAG) • Calculated by subtracting the ascitic fluid albumin from the serum albumin in simultaneously obtained samples.[61] Indicated for new-onset ascites.	**>11 g/L (>1.1 g/dL) highly suggestive of portal hypertension, usually caused by liver disease; ≤11 g/L (≤1.1 g/dL) suggests other causes of ascites**
ascitic fluid carcinoembryonic antigen (CEA) • Not routinely used, but can be useful in that an elevated level indicates secondary peritonitis. Therefore, if level is normal (<5 micrograms/L [<5 nanograms/mL]), it raises the likelihood of secondary peritonitis.[97]	**<5 micrograms/L (<5 nanograms/mL)**
ascitic fluid alkaline phosphatase • Not routinely used, but can be useful in that an elevated level indicates secondary peritonitis. Therefore, if level is normal (<240 units/L) it raises the likelihood of secondary peritonitis.[97]	**<240 units/L**
ascitic fluid AFB stain and culture, fungal culture, microscopy for ova/parasites • Can help diagnose the cause of peritonitis.[85]	**positive = abnormal**
ascitic fluid lactoferrin • Can help identify SBP in a cirrhotic patient with ascites. Sensitivity is 96% and specificity is 97% for the detection of SBP.[110] • Not routinely performed, but if a qualitative bedside assay can be developed, it might significantly reduce the time to diagnosis.[98]	**level elevated in SBP; an elevated lactoferrin in a cirrhotic patient without SBP can indicate a developing hepatic carcinoma**
CT scan abdomen • May be considered in patients with findings suggestive of secondary peritonitis, such as bile-stained fluid, polymicrobial growth on ascites fluid culture, no clinical improvement despite appropriate antibiotics for 48 hours, and no history of liver disease or malignancy to explain the ascites. May demonstrate free air.[102] [103]	**demonstrates diffuse ascites; excludes pneumoperitoneum in patients with secondary peritonitis**

Emerging tests

Test	Result
highly-sensitive leukocyte esterase reagent strip testing of ascitic fluid (Periscreen) • Rapidly rules out SBP. • In a multi-centre inpatient/outpatient, and accident and emergency department studies, a negative colorimetric reading had a sensitivity of 92% to 95% for the detection of SBP.[99] [100]	**reading of 'negative' on colorimetric strip at 3 minutes considered to rule out SBP**
bedside (standard urine) leukocyte esterase reagent strip testing of ascitic fluid • Can be done at the bedside within 2 minutes. • The reagent strip is dipped into ascitic fluid, and after 60-120 seconds the result is analysed according to the colorimetric scale for that reagent strip. Most studies used a strip colour that gives a positive result as corresponding to between 15 (1+) and 125 leukocytes/mL (3+). • One meta-analysis found sensitivities ranging from 45% to 100% and specificities ranging from 81% to 100%.[101] • Low sensitivity demonstrates that bedside (standard urine) leukocyte esterase reagent strip testing is not suitable for rapidly ruling out SBP. However, the high specificity suggests that it has a role in the rapid diagnosis of SBP, facilitating prompt administration of antibiotic therapy.	**elevated leukocytes measured by comparison with a colour strip**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Secondary peritonitis	• Much rarer than SBP as a cause of infected ascitic fluid should be suspected when localised abdominal symptoms or signs, presence of multiple organisms on ascitic culture, very high ascitic neutrophil count and/or high ascitic protein concentration, or in those patients with an inadequate response to therapy.[64] Secondary peritonitis may cause more rigidity and the patients are usually, overall, appear much more ill. Sepsis is common in these patients. Have a higher suspicion if history of intestinal perforation, abdominal surgery, or small bowel or if there is no history of liver disease or malignancy. • Typically not the large-volume distention seen with ascites caused by liver disease or malignancy and therefore associated with SBP.	• Polymicrobial growth on ascitic fluid culture, which is particularly suggestive of secondary peritonitis if there is an anaerobic or fungal organism. • Ascitic fluid is more likely to have increased protein and lactate dehydrogenase with reduced glucose.[108] • Ascitic fluid is more likely to have increased carcinoembryonic antigen and alkaline phosphatase.[97] • There is less likely to be a decreased absolute neutrophil count on repeat paracentesis.[111] CT abdomen should be considered to confirm diagnosis and cause in high-risk patients.[64]
Tuberculous peritonitis	• There may be extra-abdominal signs and symptoms of tuberculosis (pleural, pulmonary, CNS, bony, genitourinary). Abdominal symptoms may be similar to those of SBP.	• The definitive test is peritoneal biopsy with examination for granulomas. • Acid-fast staining of ascitic fluid is not a good differentiator, because it is negative in up to 92% of patients with peritoneal tuberculosis.[112] • CT scan may show enlarged abdominal lymph nodes. • Adenosine deaminase level >39 units/L is highly suggestive of peritoneal tuberculosis.[113]
Intraperitoneal haemorrhage into ascitic fluid	• Signs of haemorrhagic shock may be present. A history of a recent large-volume paracentesis may be a clue to haemorrhage. Abdominal	• The presence of grossly bloody ascitic fluid on paracentesis, especially if prior paracentesis did not demonstrate haemorrhagic

Condition	Differentiating signs / symptoms	Differentiating tests
	pain and distention may be similar to SBP.	ascites, is suggestive of intraperitoneal haemorrhage.
Pancreatic ascites	• There may be a history of previous pancreatitis. Abdominal symptoms and signs may be difficult to differentiate from SBP.	• Peritoneal fluid absolute neutrophil count likely to be normal. • Amylase is typically elevated (>1000 units/L), and the ratio of ascitic fluid amylase to serum amylase is approximately 6.[114] • In a case series of 8 patients with pancreatic ascites, ascitic fluid amylase values ranged from 280 to 5730 units/L.[115] • The serum albumin-ascites albumin gradient (SAAG) is usually <11 g/L (<1.1 g/dL), whereas in SBP (which typically occurs in the patient with portal hypertension), SAAG is >11 g/L (>1.1 g/dL). • CT scan may demonstrate a pancreatic pseudocyst.
Choleperitoneum (rupture of gallbladder into peritoneum)	• It should be suspected with bile staining of ascitic fluid (dark orange or brown colour).	• If bile staining of ascitic fluid consider measuring ascitic fluid bilirubin concentration. If both ascites bilirubin >102.6 micromol/L (>6 mg/dL) and ascites : serum bilirubin ratio >1.0 this is very suggestive of choleperitoneum. If ascitic fluid amylase obtained, normal amylase would suggest upper gastrointestinal perforation rather than gallbladder perforation.

Approach

Treatment for SBP is directed primarily at early administration of appropriate empirical antibiotics.[61] [64] Ascitic fluid should ideally be obtained by paracentesis prior to antibiotic administration but antibiotics should be started before culture results are known to avoid delay.[61] [64]

Aggressive resuscitation is essential if sepsis is present, with fluid resuscitation and pressor support to maintain a mean arterial pressure >65 mmHg.[116] Empirical broad-spectrum antibiotic therapy is required as soon as possible after recognition.[61] [64] Assess for signs of sepsis, antibiotics should ideally be started within 1 hour once sepsis is suspected. See Sepsis in adults .

Antibiotic selection relies on the following factors:

- Community-acquired infection versus nosocomial infection
- Presence of risk factors for multi-drug-resistant (MDR) species
    - Recent ascitic fluid, urine, or blood culture demonstrating MDR
    - Patient not improving on appropriate therapy
    - Patient taking SBP prophylaxis
- Local bacterial resistance patterns
- Clinical signs of severe infection

Community-acquired infection with low risk for resistant species

First-line empirical antibiotic therapy for community-acquired SBP is an intravenous third-generation cephalosporin (e.g., cefotaxime, ceftriaxone).[61] Alternative options include an intravenous fluoroquinolone (e.g., ciprofloxacin) or ampicillin/sulbactam.[117] Treatment should continue for 5-7 days.[61] [64] [118] [119] [120] If the patient shows clinical improvement over 48 hours, it is reasonable to consider switching to an oral antibiotic.[117]

Systemic fluoroquinolone antibiotics, such as ciprofloxacin, may cause serious, disabling, and potentially long-lasting or irreversible adverse events. This includes, but is not limited to: tendinopathy/tendon rupture; peripheral neuropathy; arthropathy/arthralgia; aortic aneurysm and dissection; heart valve regurgitation; dysglycaemia; and central nervous system effects including seizures, depression, psychosis, and suicidal thoughts and behaviour.[121]

- Prescribing restrictions apply to the use of fluoroquinolones, and these restrictions may vary between countries. In general, fluoroquinolones should be restricted for use in serious, life-threatening bacterial infections only. Some regulatory agencies may also recommend that they must only be used in situations where other antibiotics, that are commonly recommended for the infection, are inappropriate (e.g., resistance, contraindications, treatment failure, unavailability).
- Consult your local guidelines and drug information source for more information on suitability, contraindications, and precautions.

Despite increasing cephalosporin and fluoroquinolone resistance, a recent randomised, controlled trial comparing cefotaxime, ceftriaxone, and ciprofloxacin demonstrated similar resolution rates and mortality, and at rates similar to prior studies.[122]

Patients at high risk for MDR including nosocomial infection

Nosocomial SBP is associated with higher mortality than community-acquired SBP.[123] Patients with nosocomial infection or with other high risk factor for MDR should be started on empirical broad-spectrum intravenous antibiotics that cover the most likely MDR organism.[61] Overall, increased prevalence of infection from gram-positive cocci, such as MRSA and *Enterococcus faecalis* , and extended spectrum beta-lactamase (ESBL)-producing gram-negative bacilli, along with the emergence of carbapenem-resistant *Klebsiella pneumoniae* puts these patients at higher risk.[124]

Options include a carbapenem (e.g., imipenem/cilastatin, meropenem) or piperacillin/tazobactam.[61] [64] Due to the concern of cephalosporin resistance in this population, and the higher mortality, primary treatment with carbapenems is recommended by the EASL.[64] [125] [126] Vancomycin can be added when better coverage of gram-positive cocci is needed (e.g., for patients with sepsis or a history of fluoroquinolone prophylaxis, or in areas with a high prevalence of gram-positive MDR organisms).[64] [127] Daptomycin is recommended for patients with previous vancomycin-resistant enterococcus (VRE) infection or a VRE-positive surveillance swab.[61] The choice of of broad-spectrum antibiotics should be tailored to the local prevalence and type of MDR organisms, and antibiotic coverage should be narrowed as soon as culture results are available.[61] There are no large randomised, controlled trials comparing efficacy of antibiotic regimens in nosocomial/high risk MDR patients.

Patients who are responding and clinically improving after 48 hours may be considered for a switch to oral antibiotics.[50] [117] [128] [129] Antibiotics should be continued to give a total duration of treatment of 5-7 days.[61]

Patients with high severity of infection

While standard therapy has excellent efficacy in SBP patients, the risk of an MDR pathogen being undertreated in a patient who presents critically ill (e.g., septic) is unacceptably high and antibiotic therapy should be broadened accordingly. This includes patients with nosocomial infection, recent hospitalisation, and patients who are admitted to the intensive care unit.[61] In addition, patients with CLIF-SOFA scores ≥7 are at higher risk of short-term mortality and should be treated more aggressively.[105]

Albumin

Intravenous albumin treatment has been shown to reduce mortality and decrease kidney dysfunction in patients with SBP.[130]

Subgroup analysis of studies examining albumin use for SBP show the greatest mortality and renal dysfunction prevention benefits occur in patients with serum bilirubin >68.42 micromol/L (>4 mg/dL) or serum creatinine >88.4 micromol/L (>1 mg/dL) and serum urea >10.7 mmol/L (>30 mg/dL).[131] Because of this, the AASLD recommends albumin in all patients with SBP, but notes that patients with acute kidney injury and/or jaundice at time of diagnosis of SBP are more likely to benefit.[61] Albumin decreases renal insufficiency, probably by increasing the circulatory volume and by binding proinflammatory molecules.[106] [132]

Large-volume paracentesis (LVP)

LVP can improve abdominal discomfort in patients with tense ascites. However, there is little evidence on the safety of LVP in SBP and further research is warranted.[133]

Studies in patients with uncomplicated SBP (no sepsis, hepatic encephalopathy, GI bleeding, or significant renal dysfunction) have demonstrated that LVP with albumin replacement can be safe.[134] [135] There are no studies that have examined whether LVP is safe in patients with complicated SBP.

Repeat paracentesis and broadened antibiotic coverage in treatment-resistant patients

Patients who have not demonstrated significant clinical improvement, or who are lacking a confirmed antibiotic-susceptible organism from their initial ascitic fluid culture, should undergo repeated diagnostic paracentesis after 48 hours of treatment.[61] [136] Treatment failure is believed to occur if the absolute neutrophil count has decreased by <25% on 48-hour repeat paracentesis.[61]

Change in antibiotic therapy can be done according to blood or ascitic fluid culture results. If no growth has occurred, the addition of, or change to, vancomycin to cover MRSA and group D enterococci should be considered. Also, antibiotics that cover resistant Enterobacteriaceae (such as *E coli*) should be considered.

Failure to demonstrate significant improvement should also increase concern for secondary peritonitis, and imaging tests or surgical consultation may be needed.

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute		(summary)
community-acquired infection with low risk for resistant species		
	1st	**empirical intravenous antibiotics**
	adjunct	**albumin**
	adjunct	**large-volume paracentesis (LVP)**
nosocomial infection, septic shock, high risk for MDR organisms		
	1st	**empirical intravenous antibiotics**
	adjunct	**vancomycin or daptomycin**
	adjunct	**albumin**
	adjunct	**broaden empirical regimen and assess further or switch to oral regimen**
	adjunct	**large-volume paracentesis (LVP)**

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

community-acquired infection with low risk for resistant species

1st **empirical intravenous antibiotics**

Primary options

» cefotaxime: 2 g intravenously every 12 hours

OR

» ceftriaxone: 1-2 g intravenously every 12-24 hours

Secondary options

» ciprofloxacin: 400 mg intravenously every 12 hours

OR

» ampicillin/sulbactam: 1.5 to 3 g intravenously every 6 hours
Dose consists of 1 g ampicillin plus 0.5 g sulbactam (1.5 g), or 2 g ampicillin plus 1 g sulbactam (3 g).

» First-line empirical antibiotic therapy for community-acquired SBP is an intravenous third-generation cephalosporin (e.g., cefotaxime, ceftriaxone).[61] Alternative options include a fluoroquinolone (e.g., ciprofloxacin) or ampicillin/sulbactam.[64] [117][118] [119] [120] Do not use fluoroquinolones if patient is already on fluoroquinolone prophylaxis or in areas where there is a high prevalence of fluoroquinolone-resistant bacteria.[80]

» If continued improvement over 48 hours, it is reasonable to consider switching to an oral antibiotic.[117]

» Systemic fluoroquinolone antibiotics, such as ciprofloxacin, may cause serious, disabling, and potentially long-lasting or irreversible adverse events. This includes, but is not limited to: tendinopathy/tendon rupture; peripheral neuropathy; arthropathy/arthralgia; aortic aneurysm and dissection; heart valve regurgitation; dysglycaemia; and central

Acute

nervous system effects including seizures, depression, psychosis, and suicidal thoughts and behaviour.[121] Prescribing restrictions apply to the use of fluoroquinolones, and these restrictions may vary between countries. In general, fluoroquinolones should be restricted for use in serious, life-threatening bacterial infections only. Some regulatory agencies may also recommend that they must only be used in situations where other antibiotics, that are commonly recommended for the infection, are inappropriate (e.g., resistance, contraindications, treatment failure, unavailability). Consult your local guidelines and drug information source for more information on suitability, contraindications, and precautions.

» Emerging patterns of resistance must be examined closely at each institution to determine if more broad-spectrum empirical coverage is warranted from the outset.

» Treatment course: 5-7 days.

adjunct **albumin**

Treatment recommended for SOME patients in selected patient group

» Intravenous albumin treatment has been shown to reduce mortality and decrease kidney dysfunction in patients with SBP.[130] Albumin decreases renal insufficiency, probably by increasing the circulatory volume and by binding pro-inflammatory molecules.[106] [132]

» Subgroup analysis of studies examining albumin use for SBP show the greatest mortality and renal dysfunction prevention benefits occur in patients with serum bilirubin >68.42 micromol/L (>4 mg/dL) or serum creatinine >88.4 micromol/L (>1 mg/dL) and serum urea >10.7 mmol/L (>30 mg/dL).[131] Because of this, the AASLD recommends albumin in all patients with SBP, but notes that patients with acute kidney injury and/or jaundice at time of diagnosis of SBP are more likely to benefit.[61]

adjunct **large-volume paracentesis (LVP)**

Treatment recommended for SOME patients in selected patient group

» LVP can improve abdominal discomfort in patients with tense ascites. However, there is little evidence on the safety of LVP in SBP and further research is warranted.[133]

» Studies in patients with uncomplicated SBP (no sepsis, hepatic encephalopathy,

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gastrointestinal bleeding, or significant renal dysfunction) have demonstrated that LVP with albumin replacement can be safe.[134] [135]

» There are no studies that have examined whether LVP is safe in patients with complicated SBP.

nosocomial infection, septic shock, high risk for MDR organisms

1st **empirical intravenous antibiotics**

Primary options

» piperacillin/tazobactam: 3.375 g intravenously every 6 hours
Dose consists of 3 g piperacillin plus 0.375 g tazobactam.

OR

» imipenem/cilastatin: 0.5 to 1 g intravenously every 6 hours, or 1 g every 8 hours
Dose refers to imipenem component.

OR

» meropenem: 1-2 g intravenously every 8 hours

» Patients should be started on empirical broad-spectrum intravenous antibiotics that cover the most likely MDR organism.[61]

» Antibiotic options include a carbapenem (e.g., imipenem/cilastatin, meropenem) or piperacillin/tazobactam.[61] [64]

» Due to the concern of cephalosporin resistance in this population, and the higher mortality, primary treatment with a carbapenem regimen is recommended by the EASL.[64] [125] [126]

» The choice of of broad-spectrum antibiotics should be tailored to the local prevalence and type of multidrug resistant organisms, and antibiotic coverage should be narrowed as soon as culture results are available.[61]

» The risk of an MDR pathogen being undertreated in a patient who presents critically ill (e.g., septic) is unacceptably high and antibiotic therapy should be broadened accordingly. This includes patients with nosocomial infection, recent hospitalisation, and patients who are admitted to the intensive

Acute

care unit.[61] In addition, patients with CLIF-SOFA scores ≥7 are at higher risk of short-term mortality and should also be treated more aggressively.[105]

» Patients who are responding and clinically improving after 48 hours may be considered for a switch to oral antibiotics.[50] [117][128] [129]

» Treatment course: 5-7 days.

adjunct **vancomycin or daptomycin**

Treatment recommended for SOME patients in selected patient group

Primary options

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours
A loading dose of 25-30 mg/kg intravenously is recommended in critically ill patients.

OR

» daptomycin: 4-6 mg/kg intravenously every 24 hours

» Vancomycin can be added when better coverage of gram-positive cocci is needed (e.g., patients with sepsis or a history of fluoroquinolone prophylaxis, or in areas with a high prevalence of gram-positive multidrug resistant organisms).[64][127] Daptomycin is recommended for patients with previous vancomycin-resistant enterococcus (VRE) infection or a VRE-positive surveillance swab.[61]

» The choice of antibiotic should be tailored to local MDR prevalence and narrowed once culture results are available.[61]

adjunct **albumin**

Treatment recommended for SOME patients in selected patient group

» Intravenous albumin treatment has been shown to reduce mortality and decrease kidney dysfunction in patients with SBP.[132]

» Subgroup analysis of studies examining albumin use for SBP show the greatest mortality and renal dysfunction prevention benefits occur in patients with serum bilirubin >68.42 micromol/L (>4 mg/dL) or serum creatinine >88.4 micromol/L (>1 mg/dL) and serum urea >10.7 mmol/L (>30 mg/dL).[131] Because of this, the AASLD recommends albumin in all

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patients with SBP, but notes that patients with acute kidney injury and/or jaundice at time of diagnosis of SBP are more likely to benefit.[61] Albumin decreases renal insufficiency, probably by increasing the circulatory volume and by binding pro-inflammatory molecules.[106] [132]

adjunct **broaden empirical regimen and assess further or switch to oral regimen**

Treatment recommended for SOME patients in selected patient group

» Consider broadening the antibiotic coverage and assess further (including repeat diagnostic paracentesis) if the patient does not demonstrate significant improvement after 48 hours. Change in antibiotic therapy can be made according to the blood or ascitic fluid culture results. If no growth has occurred, consider addition of, or change to, vancomycin to cover MRSA and group D enterococci, and consider antibiotics that cover resistant Enterobacteriaceae if the patient is not already on antibiotics that cover these organisms. Failure to demonstrate significant improvement should also increase concern for secondary peritonitis, and imaging tests or surgical consultation may be needed.

» If the patient responds to treatment after 48 hours, consider switching to a suitable oral antibiotic regimen.[117]

adjunct **large-volume paracentesis (LVP)**

Treatment recommended for SOME patients in selected patient group

» LVP can improve abdominal discomfort in patients with tense ascites. However, there is little evidence on the safety of LVP in SBP and further research is warranted.[133]

» Studies in patients with uncomplicated SBP (no sepsis, hepatic encephalopathy, gastrointestinal bleeding, or significant renal dysfunction) have demonstrated that LVP with albumin replacement can be safe.[134] [135]

» There are no studies that have examined whether LVP is safe in patients with complicated SBP.

Emerging

Eravacycline

Eravacycline, a tetracycline derivative antibiotic, has been approved in the US and Europe for the treatment of complicated intra-abdominal infections in adults. It has shown activity against multi-drug-resistant (MDR) species, including extended spectrum beta-lactamase (ESBL)-producing species, and has been used to treat SBP.[137]

New beta-lactam/beta-lactamase inhibitors

Numerous beta-lactam/beta-lactamase inhibitor combinations have been developed to treat infections from MDR pathogens, particularly carbapenem-resistant Enterobacteriaceae (CRE). As these drugs were developed for the treatment of the most difficult-to-treat CRE and ESBL-producing organisms, they should be used only in patients with culture-confirmed diagnosis or who are very ill at high likelihood for infection with resistant organisms. Options include meropenem/vaborbactam, imipenem/cilastatin/relebactam, and ceftazidime/avibactam. Each of these combinations has been approved for use in the US and Europe. Aztreonam/avibactam has also been approved in Europe, but not currently the US, for the treatment of complicated intra-abdominal infections.

Granulocyte-macrophage colony-stimulating factor (GM-CSF)

In a randomised controlled trial in which difficult to treat SBP (defined as nosocomial infection with inadequate response to antibiotics within 48 hours) patients were randomised to receive meropenem (a carbapenem antibiotic) plus placebo or meropenem plus GM-CSF. The meropenem plus GM-CSF group had better resolution rates (30% vs. 60%, respectively).[138]

Primary prevention

Antibiotics for primary prophylaxis, the prevention of a first episode of SBP, should be used judiciously, taking into account adverse effects and risk of promoting resistance.[61] The potential benefit of antibiotic prophylaxis must be balanced against the increased likelihood of risks from long-term antibiotics, and decisions should be individualised according to patient characteristics, current evidence, and drug availability.[61] [62] [63]

Primary prophylaxis for SBP should be considered in patients at highest risk of infection which includes patients with cirrhosis and acute upper gastrointestinal bleeding, and patients found to have low total protein content in ascitic fluid plus evidence of liver or kidney impairment.[61] [64] The most recent 2021 AASLD guidelines do note that several of the studies looking at giving antibiotics for primary prophylaxis of SBP have been considered to be of variable quality and considered insufficient to make a consensus recommendation for primary prophylaxis, other than in patients with advanced cirrhosis and at high risk of infection, such as in the clinical scenarios above. A low concentration of ascitic protein (<15 g/L [<1.5 g/dL]) has been demonstrated as a risk factor for the development of SBP and systematic reviews have found that oral antibiotic prophylaxis in this patient population reduces the rate of first-episode SBP and other bacterial infections, and results in reduced mortality.[65] [66] The greater the degree of liver and kidney dysfunction, also the greater the benefit of prophylaxis. In patients with low concentration of ascitic protein and either severe liver dysfunction (Child-Turcotte-Pugh score ≥9, with serum bilirubin ≥51.31 micromol/L [≥3 mg/dL]) or kidney dysfunction (serum creatinine level ≥106 micromol/L [≥1.2 mg/dL], urea ≥8.92 mmol/L [≥25 mg/dL], or serum sodium level ≤130 mmol/L [≤130 mEq/L]) , prophylaxis with norfloxacin was associated with a decreased 6-month SBP rate and hepatorenal syndrome rate.[67]

While most studies have been done with norfloxacin, which has been discontinued in some countries (including the US), prophylaxis with ciprofloxacin, trimethoprim/sulfamethoxazole, or rifaximin have also shown benefit. Rifaximin, a poorly absorbed oral antibiotic with broad-spectrum activity against both gram-positive and gram-negative intestinal bacteria, has been studied as a means of primary prevention of SBP. In meta-analyses, rifaximin appeared to reduce the risk of first-episode SBP in people with cirrhosis.[68]

[69] [70] [71] In terms of which antibiotic regimen is more efficacious, the AASLD does not recommend any antibiotic, but two more recent meta-analyses (one that has been published since those guidelines) have suggested rifaximin as potentially being more efficacious.[71] [72]

Beta-blockers

Evidence for the use of non-selective beta-blockers for SBP prophylaxis is conflicting.[61] One meta-analysis of three randomised controlled trials (one on primary prevention; two on secondary prevention) found that propranolol and nadolol may prevent new episodes of SBP in patients with cirrhosis and ascites.[73] A subsequent randomised controlled trial in patients with compensated cirrhosis showed that use of a non-selective beta-blocker was associated with a reduced incidence of decompensated cirrhosis or death, suggesting that their use in early cirrhosis may be beneficial.[74]

However, continued use of a non-selective beta-blocker in patients with cirrhosis and established SBP was associated with reduced (transplant-free) survival, increased hospital stay, and higher rates of hepatorenal syndrome and acute kidney injury.[75] Later studies demonstrated that this was likely limited to patients with reduced mean arterial pressure.[76] [77] Therefore, the AASLD advises to not continue the drug in hypotensive patients, while it can be resumed when the mean arterial pressure normalises.[61]

Secondary prevention

Antibiotics for secondary prophylaxis against SBP should be considered in patients following an episode of SBP.[61] [117][130] [158] The 1-year cumulative recurrence rate is around 70% in those that survive SBP.[117] Treatment should continue until ascites resolves, the patient becomes critically ill, or liver transplantation takes place.[51] See Primary Prevention for more information on prophylaxis in patients with no history of SBP.

One systematic review and network meta-analysis (where different antibiotic prophylaxes were treated as different interventions) of antibiotic prophylaxis for the prevention of SBP in people with cirrhosis found no evidence of difference in mortality or serious adverse events in any of the direct comparisons or network meta-analysis.[159] There was no evidence of difference based on whether the prophylaxis was primary or secondary. Overall quality of evidence was low or very low.[159]

Local bacterial resistance patterns should be considered when selecting the most appropriate antibiotic.

Rifaximin

One meta-analysis of studies of rifaximin for primary and secondary prevention of SBP suggested a protective effect; in subgroup analysis, rifaximin reduced the risk of SBP by 74% compared with systemic antibiotics for secondary prophylaxis.[68]

Beta-blockers

Evidence for the use of non-selective beta-blockers for SBP prophylaxis is conflicting.[61] One meta-analysis of three randomised controlled trials (one on primary prevention; two on secondary prevention) found that propranolol and nadolol may prevent new episodes of SBP in patients with cirrhosis and ascites.[73] A subsequent randomised controlled trial in patients with compensated cirrhosis showed that use of a non-selective beta-blocker was associated with a reduced incidence of decompensated cirrhosis or death, suggesting that their use in early cirrhosis may be beneficial.[74]

However, continued use of a non-selective beta-blocker in patients with cirrhosis and established SBP was associated with reduced (transplant-free) survival, increased hospital stay, and higher rates of hepatorenal syndrome and acute kidney injury.[75] Consideration should be given to stopping non-selective beta-blockers if SBP develops. Further randomised controlled studies using hard end points are required to establish the benefits of beta-blockers in patients with refractory ascites, and the American Association for the Study of Liver Diseases advises caution if their use is considered for patients with hypotension, hyponatraemia, or acute kidney injury.[61]

Patient discussions

Patients should be advised not to take part in any heavy activity for 24 hours after paracentesis. They should also be advised to call their doctor or return to the emergency department if any of the following are present after paracentesis:

- Signs of infection, such as increasing redness, swelling, or drainage of pus from the puncture site
- Worsening abdominal pain
- Fever
- Severe vomiting
- Bleeding from the site that does not stop after 1 hour of applying direct pressure to the area
- Continued drainage of more than a small amount of fluid from the puncture site for >24 hours.

Following discharge from the hospital after an episode of SBP, patients should be advised to take their medications (most importantly antibiotics) as directed. They should also be reminded to call their doctor or go to the emergency department immediately if any of the following occur:

- Worsening of abdominal pain
- Fever
- Severe vomiting
- New blood in stool or blood when vomiting.

Monitoring

Monitoring

Repeat paracenteses may be necessary to ensure resolution of SBP in patients with continued symptoms.

FOLLOW UP

Complications

Complications	Timeframe	Likelihood
sepsis/septic shock	**short term**	**high**
While there are no data on the frequency of sepsis or septic shock in SBP, sepsis in cirrhosis is estimated to occur in at least 30% to 50% of hospital admissions, and patients with cirrhosis are much more likely than other patients to develop a nosocomial infection.[148]		
tense ascites	**short term**	**medium**
Worsening ascites with subsequent abdominal distention and pain may be the presenting symptom of SBP. Large-volume paracentesis with albumin replacement in the patient without haemodynamic compromise is safe and efficacious in this scenario.[134] [135]		
bleeding after paracentesis	**short term**	**low**
Bleeding after a paracentesis may occur as an intraperitoneal haemorrhage, an abdominal wall haematoma, or, more rarely, external bleeding. Reasons for bleeding include injury to the inferior epigastric artery caused by poor selection of puncture site for the paracentesis catheter; puncture of a recanalised umbilical vein or intra-abdominal varices, which may occur more commonly using a midline puncture site; and rupture of mesenteric varices, which is postulated to occur as a result of the sudden reduction in intraperitoneal pressure that can occur during a large-volume paracentesis (this sudden pressure reduction results in an increased pressure gradient across the wall of the mesenteric varices, which may cause a life-threatening haemoperitoneum).[149] [150][151] However, studies have found a low rate of bleeding associated with the procedure.[107] [152] [153] [154]		
bowel perforation after paracentesis	**short term**	**low**
Puncture of the bowel wall with the paracentesis catheter, with subsequent peritonitis or abdominal wall abscess, is a known complication. Ultrasound guidance to help find pockets of fluid that are free from bowel loops may decrease this complication. One study of 242 diagnostic paracenteses reported one case of bowel wall perforation.[155]		
leakage from paracentesis puncture site	**short term**	**low**
Approximately 1% to 5% may develop a persistent leak at the site of the paracentesis.[83] [154] This may be prevented in 3 ways: by using a smaller gauge paracentesis needle, by not making the pre-needle incision too wide or too deep, or by using a 'Z-tract' technique of needle insertion. With this technique, the needle is inserted and advanced a short distance. The direction of needle insertion is then changed by about 90° to 120° and advanced another short distance. Finally, the needle direction is changed again to its initial direction. It is hoped that the Z-shaped tract formed by the needle will make it more difficult for ascitic fluids to form a persistent tract. A persistent leak can be treated by applying a purse-string suture while the patient is lying with the affected side up.[156] Applying 2-octyl cyanoacrylate has also been described.[157]		
abnormal kidney function	**variable**	**high**
In one study examining 252 episodes of SBP, there were 83 (33%) episodes of abnormal kidney function.[146] Another 2023 study found that in 55.96% of patients with SBP it was associated with abnormal kidney function.[147] This and other studies have demonstrated abnormal kidney function to be the strongest independent predictor of mortality in SBP patients.[144]		

Prognosis

One-year SBP recurrence rates as high as 69% have been reported.[139] Randomised controlled trials comparing antibiotic regimens have described an in-hospital mortality rate of 10% to 28%.[50] [118] [128] [140] Infection-related mortality rates as low as 0% have been described in patients with uncomplicated SBP at the time of treatment.[141] [142] The chronic liver failure-sequential organ failure assessment (CLIF-SOFA) can be used to help determine the severity of illness in patients presenting with SBP. Patients with CLIF-SOFA scores ≥7 have >20% mortality and so might benefit from broader empirical antibiotic therapy.[105]

Survival rates after an episode of SBP are 30% to 50% at 1 year and 25% to 30% at 2 years. Because survival rates after liver transplantation are higher than this, patients should be considered for evaluation for transplantation.[1]

In one systematic review of studies examining prognostic factors in patients with SBP, kidney and liver impairment were shown to be the main prognostic factors of cirrhosis mortality in patients with SBP, with Model for End-Stage Liver Disease (MELD) score and the Charlson index being good markers of survival.[143] The in-hospital mortality rate in patients with SBP and kidney dysfunction was found to be 67%, compared with 11% in patients with SBP and normal kidney function.[144] Other prognostic factors under investigation include ascitic polymorphonuclear leukocyte percentage (PMN-%), which has shown promise in assessing risk of death and future SBP.[145]

Diagnostic guidelines

United Kingdom

Cirrhosis in over 16s: assessment and management (https://www.nice.org.uk/guidance/ng50)

Published by: National Institute for Health and Care Excellence **Last published:** 2023

Europe

EASL clinical practice guidelines for the management of patients with decompensated cirrhosis (https://easl.eu/publications/clinical-practice-guidelines)

Published by: European Association for the Study of the Liver **Last published:** 2018

North America

Guide to utilization of the microbiology laboratory for diagnosis of infectious diseases: 2024 update by the Infectious Diseases Society of America (IDSA) and the American Society for Microbiology (ASM) (https://www.idsociety.org/practice-guideline/practice-guidelines)

Published by: Infectious Diseases Society of America and the American Society for Microbiology **Last published:** 2024

Diagnosis, evaluation, and management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome (https://www.aasld.org/publications/practice-guidelines)

Published by: American Association for the Study of Liver Diseases **Last published:** 2021

Treatment guidelines

United Kingdom

Cirrhosis in over 16s: assessment and management (https://www.nice.org.uk/guidance/ng50)

Published by: National Institute for Health and Care Excellence **Last published:** 2023

Guidelines on the management of ascites in cirrhosis (https://www.bsg.org.uk/clinical-resource/guidelines-on-the-management-of-ascites-in-cirrhosis)

Published by: British Society of Gastroenterology **Last published:** 2020

Europe

Use of albumin infusion for cirrhosis-related complications: an international position statement (https://www.jhep-reports.eu/article/S2589-5559(23)00116-7/fulltext)

Published by: European Association for the Study of the Liver **Last published:** 2023

EASL clinical practice guidelines for the management of patients with decompensated cirrhosis (https://easl.eu/publications/clinical-practice-guidelines)

Published by: European Association for the Study of the Liver **Last published:** 2018

North America

Diagnosis, evaluation, and management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome (https://www.aasld.org/publications/practice-guidelines)

Published by: American Association for the Study of Liver Diseases **Last published:** 2021

Key articles

- Biggins SW, Angeli P, Garcia-Tsao G, et al. Diagnosis, evaluation, and management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome: 2021 practice guidance by the American Association for the Study of Liver Diseases. Hepatology. 2021 Aug;74(2):1014-48. Full text (https://www.doi.org/10.1002/hep.31884) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/33942342?tool=bestpractice.bmj.com)

- European Association for the Study of the Liver. EASL clinical practice guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018 Aug;69(2):406-60. Full text (https://www.doi.org/10.1016/j.jhep.2018.03.024) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/29653741?tool=bestpractice.bmj.com)

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Images

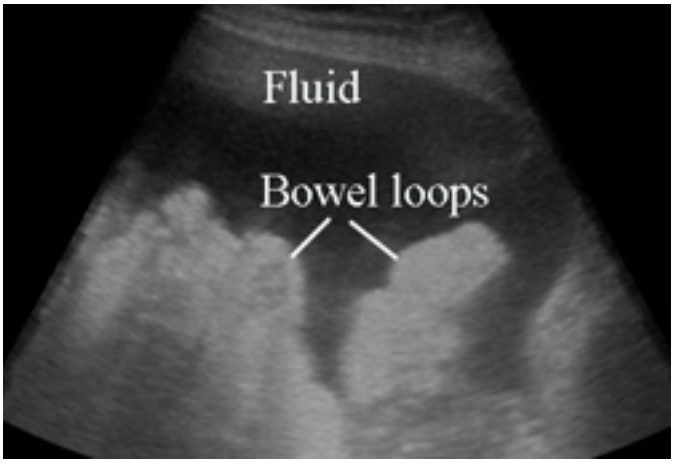

Figure 1: Abdominal ultrasound showing large amount of ascites with bowel loops

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Please note that recommended formulations and doses may differ between drug databases drug names and brands, drug formularies, or locations. A local drug formulary should always be consulted for full prescribing information.

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Regardless of the language in which the content is displayed, numerals are displayed according to the original English-language numerical separator standard. For example 4 digit numbers shall not include a comma nor a decimal point; numbers of 5 or more digits shall include commas; and numbers stated to be less than 1 shall be depicted using decimal points. See Figure 1 below for an explanatory table.

BMJ accepts no responsibility for misinterpretation of numbers which comply with this stated numerical separator standard.

This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

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