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Emergency medicine updates: Spontaneous bacterial peritonitis

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ABSTRACT

Introduction: Spontaneous bacterial peritonitis (SBP) is a common infection in patients with cirrhosis and ascites and is associated with significant risk of mortality. Therefore, it is important for emergency medicine clinicians to be aware of the current evidence regarding the diagnosis and management of this condition.
Objective: This paper evaluates key evidence-based updates concerning SBP for the emergency clinician.
Discussion: SBP is commonly due to Gram-negative bacteria, but infections due to Gram-positive bacteria and multidrug resistant bacteria are increasing. The typical presentation of SBP includes abdominal pain, worsening ascites, fever, or altered mental status in a patient with known liver disease; however, some patients may be asymptomatic or present with only mild symptoms. Paracentesis is the diagnostic modality of choice and should be performed in any patient with ascites and concern for SBP or upper gastrointestinal bleeding, or in those being admitted for a complication of cirrhosis. Ultrasound should be used to optimize the procedure. An ascites absolute neutrophil count (ANC) ≥ 250 cells/mm^3 is diagnostic of SBP. Ascitic fluid should be placed in blood culture bottles to improve the culture yield. Leukocyte esterase reagent strips can be used for rapid diagnosis if available. While many patients will demonstrate coagulation panel abnormalities, routine transfusion is not recommended. Management traditionally includes a third-generation cephalosporin, but specific patient populations may require more broad-spectrum coverage with a carbapenem or piperacillin-tazobactam. Albumin infusion is associated with reduced risk of renal impairment and mortality.
Conclusions: An understanding of literature updates can improve the care of patients with suspected SBP.
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1. Introduction

End-stage liver disease resulting in cirrhosis and ascites is a major cause of death worldwide and is associated with a variety of complications [1-9]. One of the most common complications is bacterial infection due to the decreased humoral and cell-mediated immunity, abnormal gastrointestinal (GI) function and microbiome, reduced hepatic protein production, and bacterial translocation worsened by portal hypertension [4-10]. Infections account for 25–46% of admissions in those with decompensated cirrhosis, and infection increases mortality four-fold [4-6,10-13]. The most common infection in patients with cirrhosis is primary spontaneous bacterial peritonitis (SBP), which is an infection of the peritoneal ascitic fluid without an intra-abdominal focus of infection [4-7,10]. SBP accounts for over 30% of all infections in those with cirrhosis and is present in 5–30% of cirrhotic patients admitted with ascites [7,14-23]. Mortality associated with SBP is significant, with mortality rates per episode of SBP ranging from 15 to 40% [16-27]. Literature suggests a 40% survival rate at one year following an episode of SBP [10,28]. Older age, recurrent episodes of SBP, hepatorenal syndrome, hepatic encephalopathy, acute kidney injury (AKI), concurrent GI bleeding, and higher Model for End-Stage Liver Disease (MELD) scores are predictors of worse outcomes [4-7,29-31].

SBP is most commonly a monomicrobial infection. The most common microbes involved include Gram-negative bacteria such as *E. coli* (48–90% of cases) [32-44]. However, Gram-positive bacteria are becoming more common, as well as infections with multidrug resistant (MDR) bacteria, particularly among those with nosocomial infections [21,32-46]. Studies suggest SBP due to MDR bacteria or Klebsiella is associated with worse outcomes [47-49]. One study with over 1300 patients with cirrhosis found that half of infections were community acquired, while 25% were nosocomial and another 25% were healthcare associated [11,12].

While emergency clinicians regularly manage patients with cirrhosis, there have been several literature updates concerning primary SBP. The following questions will highlight some of these key updates, but this paper is not intended to serve as a review of SBP in its entirety.

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2. Discussion

2.1. What are the major risk factors for SBP?

There are several risk factors for development of SBP in those with known cirrhosis and ascites. Major risk factors include upper GI bleeding, low ascitic protein concentration (< 1.5 g/dL), history of prior SBP, serum total bilirubin >2.5 mg/dL, and malnutrition [4-8,50-56]. Many patients with these risk factors are placed on antibiotic prophylaxis such as norfloxacin [5-8]. SBP can occur in up to 25–65% of patients with ascites and GI bleeding [4-8,57]. A low ascitic protein concentration < 1.5 g/dL is also a significant risk factor for SBP, particular when combined with Child-Pugh score ≥ 9, serum bilirubin level ≥ 3 mg/dL, creatinine ≥1.2 mg/dL or blood urea nitrogen (BUN) level ≥ 25 mg/dL, or serum sodium ≤130 mEq/L [4-8,53,57-60]. Prior history of SBP also predicts greater risk, as the recurrence rate of SBP after an initial episode approaches 70% if prophylaxis is not provided [61]. Other risk factors include use of proton pump inhibitors (PPIs), refractory ascites, older age, and endoscopic management of esophageal varices [5-8]. PPIs may increase the risk of SBP by reducing gastric pH and impairing natural host defenses [62,63]. However, the literature is controversial, with some studies demonstrating increased infection and mortality with PPIs and others demonstrating no association [16,39,64-66].

2.2. When should SBP be suspected?

The classic presentation of SBP is fever, abdominal pain or tenderness, and altered mental status in a patient with ascites. However, 10–33% of cases are asymptomatic or demonstrate only mild symptoms [4-9,67,68]. SBP is rare in those with ascites caused by alternate etiologies (e.g., end-stage renal disease, heart failure, peritoneal carcinomatosis, pancreatic disease) in the absence of cirrhosis, though it may still rarely occur [4-6,69]. Clinical deterioration, including altered mental status, AKI, or worsening jaundice, may be suggestive of infection in patients with cirrhosis. Abnormal temperature is a common finding, including both fever and hypothermia. One study found that fever at the time of presentation was 90% specific but only 18% sensitive for the diagnosis [70]. This same study found fever within 24 h of presentation had a sensitivity of 35.5% with a specificity of 81.1% [70]. Importantly, mild hypothermia is normal in patients with advanced cirrhosis. Thus, a lower threshold for fever is often recommended (i.e., 37.8 °C) [4-9]. SBP may also present with marked hypothermia (specificity of 93.4%), which is a marker of more severe disease and associated with poor outcome [70].

Abdominal pain or tenderness is the most sensitive finding (94.1%) for SBP, but it is not specific (15.1%) [70]. However, pain and tenderness may be subtle due to the presence of ascites [4-7]. Most patients will demonstrate diffuse abdominal pain, but this will often be different than the pain due to stretching of the abdominal wall associated with worsening ascites. Approximately 50% of patients with SBP will present with some form of altered mental status, with a specificity of 95.3% [6,9,70,71]. However, mental status changes can be subtle and may best be detected by those who know the patient well (e.g., family, significant other). Diarrhea is also common in those with SBP due to alteration in gut flora [4-7]. Nausea and vomiting have a sensitivity approaching 30% and a specificity of 70% [70]. Severe illness can present with hypotension, paralytic ileus, and hypothermia, which are markers of critical illness and associated with increased mortality [4-7]. Examination may reveal abdominal tenderness, but abdominal rigidity does not typically develop even in those with critical illness. Vital sign abnormalities such as tachycardia may be present, and patients may demonstrate altered mental status. There may be stigmata of liver disease including jaundice, scleral icterus, spider angiomas, palmar erythema, and asterixis [4-9,70]. Unfortunately, clinician gestalt is not reliable in excluding the diagnosis of SBP. One study of ED patients with suspected SBP found that clinician gestalt for the diagnosis of SBP had a sensitivity of 76% and specificity of 34% [70]. Another study of 43 patients with SBP found that physician clinical suspicion had a sensitivity of 42% for SBP [72]. Of the 43 patients diagnosed with SBP, 25 had a pretest clinical suspicion of "none" or "low" by the emergency clinician [72]. Thus, clinical gestalt is not adequate to exclude the diagnosis of SBP without peritoneal fluid testing, and a low threshold for paracentesis is recommended, even in well-appearing patients with ascites [4-9,70,72].

2.3. Do patients with SBP routinely require blood product transfusion to address coagulation panel or platelet abnormalities?

The majority of patients with liver dysfunction have thrombocytopenia and abnormal coagulation panel testing. Literature suggests 70% of patients have an abnormal prothrombin time, and up to 84% have thrombocytopenia [73-77]. However, current evidence suggests that patients with cirrhosis have a balanced coagulopathy, with hypo- and hypercoagulable states [5,6,8,9,75]. This is primarily due to hepatic synthetic dysfunction with reduced production of procoagulant and anticoagulant factors. Studies suggest that patients have a low overall risk of bleeding with paracentesis and that the procedure is safe even in the setting of coagulopathy [5-9,75]. One study of 1100 patients receiving paracentesis found no bleeding complications requiring transfusion, even in patients with international normalized ratio (INR) > 8 and platelet counts of 19,000/microL [78]. A 1991 study found a 0.25% rate of bleeding after paracentesis [73]. A 2005 study found severe bleeding occurred in only 9 of 4729 paracenteses (0.19%), though neither INR nor platelet count were predictive of bleeding risk [79]. A separate study found a bleeding rate of 1% in those with platelet count <50,000/microL, while another study including 304 paracenteses performed on patients with platelet counts <50,000/microL found an overall event rate of 0.99% [80,81]. Another 2009 study of patients undergoing diagnostic paracenteses reported no bleeding complications among patients with an abnormal INR [82]. Based on current evidence, neither elevated INR nor thrombocytopenia is a contraindication to paracentesis, and guidelines state that routine assessment of prothrombin time and platelet count before paracentesis is unnecessary [5,6]. Transfusion to correct coagulation panel abnormalities is also not recommended by current guidelines, as it increases the risk of adverse events and is not associated with a reduced risk of bleeding or complications [5-9].

2.4. What diagnostic testing should be obtained?

The diagnostic modality of choice for SBP is ascitic fluid sampling via paracentesis, and an ascites absolute neutrophil count (ANC) ≥ 250 cells/mm^3 is diagnostic of SBP [4-9]. Paracentesis should be performed when SBP is suspected, with ascites fluid sent for cell count and culture [4-9]. Other testing, including serum laboratory assessment, may be helpful but should not be used to exclude the condition in isolation. Guidelines recommend performing paracentesis in patients with clinical suspicion for SBP or in a patient with cirrhosis and ascites who is being admitted for a complication of cirrhosis [5,6]. This includes those with worsening renal or liver function, encephalopathy, and GI bleeding [5,6,8]. Early diagnosis is critical, with one study suggesting that each hour of delay in obtaining ascitic fluid was associated with a 3.3% increase in mortality [83]. This same study found that paracentesis performed within 12 h was associated with lower in-hospital mortality and shorter length of stay when compared to paracentesis performed within 12–72 h (13% compared to 27%) [83]. Another study found a mortality rate of 8.9% for patients with ascites who did not undergo paracentesis versus 6.3% for those who did [84].

Of note, paracentesis is a relatively safe procedure with a low complication rate (iatrogenic infection 0.2%, hemorrhage 0.4%, bowel perforation 0.8%) [82,85,86]. A site that provides access to a deep pocket of fluid while avoiding vasculature is recommended. We recommend using ultrasound to confirm ascites and evaluate for the optimal site of drainage, starting the evaluation in the left lower and right lower

quadrants, lateral to the rectus sheath. Ultrasound can also be used to measure abdominal wall thickness and evaluate for the presence of any vessels running through the abdominal wall [87,88]. One study evaluating fluid depth and abdominal wall thickness found the left lower quadrant was superior to the infraumbilical midline site [89]. Once obtained, diagnosis of SBP only requires cell count to obtain the ANC, though culture and Gram stain should also be sent [5,6,9]. Other ascites fluid testing such as total protein and albumin, glucose, and lactate dehydrogenase (LDH) may be used to evaluate for other aspects of clinical importance, such as the patient with new onset ascites, but they are not necessary for diagnosis of SBP [5-9]. Approximately 1 mL of ascitic fluid should be injected into a purple top EDTA blood tube for cell count, with 2–3 mL sent for Gram stain in a red-top tube or sterile urine container [90,91]. Additional ascitic fluid should be inoculated into blood culture bottles, with at least 10 mL per bottle, which increases the sensitivity to over 90% [90]. In contrast, the sensitivity of cultures obtained in other types of containers is approximately 50% [91]. Blood cultures should be obtained around the same time as the ascitic fluid culture and prior to receiving antibiotics, to raise the likelihood of isolating the organism [61]. A different needle than the one used to obtain the ascitic fluid should be used to transfer the fluid to the blood culture bottle in order to reduce the risk of contamination.

While the most specific marker for SBP is an ascites ANC ≥ 500 cells/mm^3, a threshold of ≥250 cells/mm^3 is recommended for diagnosis of SBP [4-9,92,93]. Recent studies have evaluated the use of leukocyte esterase reagent strips for rapid diagnosis of SBP at the bedside [94]. There are several reagent strips available, including Aution Sticks, Combur, Multistix, and Periscreen reagent strips. These colorimetric strips typically provide readings ranging between 0 and 4. A 2021 meta-analysis found the Aution stick was the most sensitive but is the least studied, while the Multistix strip was the most specific (Table 1) [94]. Of note, the Periscreen reagent strip is not a urine testing strip but was developed as a high sensitivity screening test to evaluate peritoneal fluid for SBP in peritoneal dialysis patients. Reagent strip testing can be helpful in institutions where cell count is not rapidly available.

Serum laboratory abnormalities are common in patients with SBP, including anemia (due to GI bleeding, malnutrition, hypersplenism, alcohol use), thrombocytopenia (portal hypertension, hypersplenism), elevated total bilirubin and prothrombin time, hyponatremia (excess water intake), elevated aspartate and alanine aminotransferases, and elevated creatinine and blood urea nitrogen (BUN) [5,6,8,9,76,95]. However, these findings are neither sensitive nor specific for SBP [5,6,8,9,76,95]. C-reactive protein (CRP) and procalcitonin have been evaluated in those with SBP [76,96-99]. A 2022 meta-analysis found an area under the receiver operating curve (AUROC) for procalcitonin using a cut-off >2.0 ng/mL was 0.75 (95% CI 0.61–0.88), and CRP > 3.0 mg/L demonstrated an AUROC of 0.55 (95% CI 0.43–0.68) [98]. The combination of elevated procalcitonin with CRP demonstrated an AUROC of 0.76 (95% CI 0.61–0.90) for diagnosing all-cause SBP [98]. Another meta-analysis found procalcitonin had a pooled sensitivity of 79% (95% CI 64–89%) and specificity of 89% (95% CI 82–94%), while CRP had a sensitivity of 77% (95% CI 69–84%) and specificity of 85% (95% CI 76–90%) [99]. A separate 2022 meta-analysis evaluated neutrophil-to-lymphocyte ratio (NLR) for diagnosis of SBP and reported a sensitivity of 92.1% and a specificity of 72.6% [100]. However, the NLR thresholds ranged from 2.4 to 9.2, and there was significant heterogeneity in the included studies [100]. Blood cultures should be obtained, and positive blood cultures are associated with increased mortality [4-6,61,101].

Table 1
Reagent strips test characteristics [94].

Test	Sensitivity (95% CI)	Specificity (95% CI)
Aution Stick		
- Overall	96.2% (92.6–99.8%)	94.0% (90.4–97.6%)
- Visual	97.3% (94.5–1.00%)	93.5% (89.0–98.1%)
Combur 10		
- Overall	89.2% (84.6–93.8%)	92.2% (87.4–97.0%)
- Threshold ≥1	86.6% (81.5–91.7%)	87.1% (81.1–93.1%)
- Threshold ≥2	81.4% (68.9–93.9%)	97.0% (94.9–99.1%)
Multistix		
- Overall	80.6% (73.8–87.4%)	93.8% (96.2–98.5%)
- Visual	80.1% (72.9–87.2%)	97.6% (96.5–98.6%)
- Threshold ≥1	84.7% (77.1–92.3%)	96.4% (94.4–98.5%)
- Threshold ≥2	77.5% (59.8–95.2%)	97.6% (96.2–99.0%)
- Threshold ≥3	62.2% (49.2–75.3%)	99.2% (98.9–99.6%)
Periscreen		
- Overall	93.9% (90.0–97.9%)	67.2% (38.1–96.3%)

CI – confidence interval.

2.5. Antibiotics for SBP

Antibiotics are a cornerstone of treatment and should be administered immediately after paracentesis is performed if the patient has abdominal pain/tenderness, altered mental status, temperature > 37.8 °C (100° F), or if the ascitic fluid ANC is ≥250 cells/mm^3 [4-9]. Patients can rapidly progress to septic shock and multisystem organ failure, so rapid diagnosis and treatment with antibiotics are essential [4-9]. Mortality increases by 8–10% for every hour delay in antibiotics for those with cirrhosis in septic shock [11,13,102,103]. Antibiotics should not be withheld pending bacterial culture results [4-9].

Traditionally, a third-generation cephalosporin(e.g., ceftriaxone 2 g IV daily or cefotaxime 2 g IV every 8 h) is given for SBP and is effective in locations with local patterns of antimicrobial susceptibility [4-9,46]. However, cephalosporins have become less effective due to the increasing rates of nosocomial infections and MDR bacteria. Recent studies suggest MDR bacteria are present in 11–50% of patients with SBP [21,43–49,104–111]. One study found that a third-generation cephalosporin would have adequately covered 70% of community-acquired cases of SBP and 56% of nosocomial infections [109]. Current data demonstrate that the initial use of carbapenems may result in greater resolution of SBP and reduced mortality in patients with nosocomial infections or critically ill patients [21,110-114]. One study found carbapenem therapy in critically ill patients was associated with a reduction in mortality compared to third-generation cephalosporins (23% vs. 39%, odds ratio 0.84, 95% CI 0.75–0.94) [110], while another found carbapenem therapy reduced mortality and AKI in those with recurrent SBP [112]. Piperacillin-tazobactam may be utilized in place of a carbapenem [113,114]. Thus, patients with shock, critical illness, end-organ injury, recurrent SBP, or recent hospitalization and receipt of antibiotics should receive broad-spectrum coverage with piperacillin-tazobactam or a carbapenem (e.g., ertapenem, imipenem, meropenem) [4,5,7,21]. There is also an increasing prevalence of *Staphylococcus aureus* as the causative agent in those with nosocomial SBP, accounting for 19–48% of infections [115]. If risk factors for methicillin resistant *Staphylococcus aureus* (MRSA) are present or the patient has recent healthcare exposure, coverage for MRSA should be considered (e.g., vancomycin, linezolid) [4,5,7,21].

2.6. What is the role of albumin in SBP?

SBP commonly results in multiorgan failure, with the kidneys most commonly affected [4-9,116-119]. Literature suggests renal dysfunction occurs in 30–50% patients with SBP [116-119]. Acute kidney injury is the primary predictor of mortality in patients with SBP [4,5,7,118-122], with one study finding a 30% increase in mortality among patients with SBP and concomitant AKI [123]. A separate systematic review found in-hospital mortality for those with SBP and AKI was 67% versus 11% for those without AKI [119].

A meta-analysis found administration of albumin IV is associated with a 22.3% absolute reduction in risk of developing renal impairment (number-needed-to-treat [NNT] 5) and a 19.4% absolute reduction in risk of mortality (NNT 6) [124,125]. A meta-analysis published in 2022

found albumin infusion reduced all-cause mortality and renal impairment [126]. Guidelines recommend administering albumin in those with a bilirubin >4 mg/dL, creatinine >1 mg/dL, or BUN >30 mg/dL [5,6,127,128]. The regimen includes 1.5 g/kg of 20% albumin IV at the time of diagnosis, with 1 g/kg IV 48 h later.

If renal failure has developed, mortality significantly increases [4-9,127]. Renal failure in those with cirrhosis is associated with reduced effective arterial volume and activation of the renin-angiotensin system. Volume expansion with albumin is an integral component of therapy for these patients.

3. Conclusions

SBP is associated with significant risks of morbidity and mortality. There are a variety of causative organisms, including Gram-negative bacteria, Gram-positive bacteria, and MDR bacteria. The disease should be suspected in those with known liver disease and abdominal pain, worsening ascites, fever, and altered mental status. Paracentesis is recommended for diagnosis in any patient with ascites and concern for SBP, upper GI bleeding, or in those being admitted for a complication of their cirrhosis. Ultrasound can assist in locating the optimal site for paracentesis. Ascites ANC ≥ 250 cells/mm^3 is diagnostic. Further testing of ascitic fluid with leukocyte esterase reagent strips may be considered in settings in which peritoneal fluid testing is not available. Routine transfusion is not recommended to correct coagulation panel abnormalities. Management includes antibiotics depending on the patient and local sensitivities. Albumin infusion should also be considered for patients with SBP.

CRediT authorship contribution statement

Brit Long: Writing – review & editing, Writing – original draft, Visualization, Resources, Conceptualization. **Michael Gottlieb:** Writing – review & editing, Validation, Supervision, Resources, Conceptualization.

Declaration of Competing Interest

None.

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