Spontaneous Bacterial Peritonitis

- Author: Steven M Bandy, MD, FACEP; Chief Editor: Rick Kulkarni, MD more...

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Background

Spontaneous bacterial peritonitis (SBP) is an acute bacterial infection of ascitic fluid. Generally, no source of the infecting agent is easily identifiable, but contamination of dialysate can cause the condition among those receiving peritoneal dialysis (PD).

Spontaneous bacterial peritonitis occurs in both children and adults and is a well-known and ominous complication in patients with cirrhosis.[1] Of patients with cirrhosis who have spontaneous bacterial peritonitis, 70% are Child-Pugh class C. In these patients, the development of spontaneous bacterial peritonitis is associated with a poor long-term prognosis.

Once thought to occur only in those individuals with alcoholic cirrhosis, spontaneous bacterial peritonitis is now known to affect patients with cirrhosis from any cause. In addition, spontaneous bacterial peritonitis can occur as a complication of any disease state that produces the clinical syndrome of ascites, such as heart failure and Budd-Chiari syndrome. Children with nephrosis or systemic lupus erythematosus who have ascites have a high risk of developing spontaneous bacterial peritonitis.

Pathophysiology

The mechanism for bacterial inoculation of ascites has been the subject of much debate since Harold Conn first recognized the disorder in the 1960s. Enteric organisms have traditionally been isolated from more than 90% of infected ascites fluid in spontaneous bacterial peritonitis, suggesting that the GI tract is the source of bacterial contamination.

The preponderance of enteric organisms, in combination with the presence of endotoxin in ascitic fluid and blood, once favored the argument that spontaneous bacterial peritonitis was due to direct transmural migration of bacteria from an intestinal or hollow organ lumen, a phenomenon called bacterial translocation. However, experimental evidence suggests that direct transmural migration of microorganisms might not be the cause.

An alternative proposed mechanism for bacterial inoculation of ascites is hematogenous transmission in combination with an impaired immune defense system. Nonetheless, the exact mechanism of bacterial displacement from the GI tract into ascites fluid remains controversial.

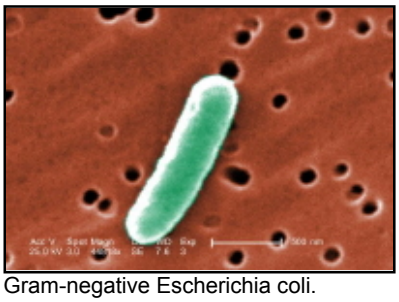
A host of factors contributes to peritoneal inflammation and bacterial growth in ascitic fluid. A key predisposing factor may be the intestinal bacterial overgrowth found in people with cirrhosis, mainly attributed to delayed intestinal transit time. Intestinal bacterial overgrowth, along with impaired phagocytic function, low serum and ascites complement levels, and decreased activity of the reticuloendothelial system, contributes to an increased number of microorganisms and decreased capacity to clear them from the bloodstream, resulting in their migration into and eventual proliferation within ascites fluid.

Interestingly, adults with spontaneous bacterial peritonitis typically have ascites, but most children with spontaneous bacterial peritonitis do not have ascites. The reason for and mechanism behind this is the source of ongoing investigation.

Etiology

Traditionally, three fourths of spontaneous bacterial peritonitis infections have been caused by aerobic gram-negative organisms (50% of these being *Escherichia coli*). The remainder has been due to aerobic gram-positive organisms

(19% streptococcal species). *E coli* is shown in the image below.



Gram-negative Escherichia coli.

However, some data suggest that the percentage of gram-positive infections may be increasing.[2, 3] One study cites a 34.2% incidence of streptococci, ranking in second position after Enterobacteriaceae.[3] *Viridans* group streptococci (VBS) accounted for 73.8% of these streptococcal isolates.

Anaerobic organisms are rare because of the high oxygen tension of ascitic fluid.

A single organism is noted in 92% of cases, and 8% of cases are polymicrobial.

Risk factors

Patients with cirrhosis who are in a decompensated state are at the highest risk of developing spontaneous bacterial peritonitis.[4] Low complement levels are associated with the development of spontaneous bacterial peritonitis. Patients at greatest risk for spontaneous bacterial peritonitis have decreased hepatic synthetic function with associated low total protein level or prolonged prothrombin time (PT).

Patients with low protein levels in ascitic fluid (< 1 g/dL) have a 10-fold higher risk of developing spontaneous bacterial peritonitis than those with a protein level greater than 1 g/dL.

Epidemiology

In patients with ascites, the frequency may be as high as 18%. This number has grown from 8% over the past 2 decades, most likely secondary to an increased awareness of spontaneous bacterial peritonitis and heightened threshold to perform diagnostic paracentesis.

No race predilection is known for spontaneous bacterial peritonitis. In patients with ascites, both sexes are affected equally.

Although the etiology and incidence of hepatic failure differ between children and adults, in those individuals with ascites, the incidence of spontaneous bacterial peritonitis is roughly equal. Two peak ages for spontaneous bacterial peritonitis are characteristic in children: the first in the neonatal period and the second at age 5 years.

Prognosis

The mortality rate in patients with spontaneous bacterial peritonitis ranges from 40-70% in adult patients with cirrhosis. Rates are lower in children with nephrosis. Patients with concurrent renal insufficiency have been shown to be at a higher risk of mortality from spontaneous bacterial peritonitis than those without concurrent renal insufficiency. Mortality from spontaneous bacterial peritonitis may be decreasing among all subgroups of patients because of advances in its diagnosis and treatment.

Contributor Information and Disclosures

Author

Steven M Bandy, MD, FACEP Adjunct Clinical Professor of Emergency Medicine, Virginia College of Osteopathic Medicine; Staff Physician, Department of Emergency Medicine, Johnston Memorial Hospital; Medical Director, Rejuvenage Medspa; Operational Medical Director, Virginia Operations, Wings Air Rescue Ambulance Service

Steven M Bandy, MD, FACEP is a member of the following medical societies: American Academy of Cosmetic

Spontaneous Bacterial Peritonitis Clinical Presentation

- Author: Steven M Bandy, MD, FACEP; Chief Editor: Rick Kulkarni, MD more...

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History

A broad range of signs and symptoms are seen in spontaneous bacterial peritonitis (SBP). A high index of suspicion must be maintained when caring for patients with ascites, particularly those with acute clinical deterioration. Completely asymptomatic cases have been reported in as many as 30% of patients.

Fever and chills occur in as many as 80% of patients. Abdominal pain or discomfort is found in as many as 70% of patients.

Other signs and symptoms may include the following:

- Worsening or unexplained encephalopathy
- Diarrhea
- Ascites that does not improve following administration of diuretic medication
- Worsening or new-onset renal failure
- Ileus

Physical Examination

Abdominal tenderness is found in more than 50% of patients with spontaneous bacterial peritonitis. Findings can range from mild tenderness to overt rebound and guarding. In some cases, the abdominal examination findings mimic an acute intra-abdominal catastrophe requiring emergency surgical evaluation. Physical examination may also disclose hypotension (5-14% of patients) or signs of hepatic failure such as jaundice and angiomata.

Contributor Information and Disclosures

Author

Steven M Bandy, MD, FACEP Adjunct Clinical Professor of Emergency Medicine, Virginia College of Osteopathic Medicine; Staff Physician, Department of Emergency Medicine, Johnston Memorial Hospital; Medical Director, Rejuvenage Medspa; Operational Medical Director, Virginia Operations, Wings Air Rescue Ambulance Service

Steven M Bandy, MD, FACEP is a member of the following medical societies: American Academy of Cosmetic Surgery, American College of Emergency Physicians, and American Society for Laser Medicine and Surgery

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Specialty Editor Board

Francisco Talavera, PharmD, PhD Adjunct Assistant Professor, University of Nebraska Medical Center College of Pharmacy; Editor-in-Chief, Medscape Drug Reference

Disclosure: eMedicine Salary Employment

Mark L Plaster, MD, JD Executive Editor, Emergency Physicians Monthly

Mark L Plaster, MD, JD is a member of the following medical societies: American Academy of Emergency Medicine and American College of Emergency Physicians

Spontaneous Bacterial Peritonitis Differential Diagnoses

- Author: Steven M Bandy, MD, FACEP; Chief Editor: Rick Kulkarni, MD more...

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Diagnostic Considerations

- Secondary bacterial peritonitis
- Perforated viscus
- Pyelonephritis

Clinical features do not distinguish secondary bacterial peritonitis from spontaneous bacterial peritonitis. However, patients with secondary bacterial peritonitis have a surgically treatable source of infection (eg, perforated duodenal ulcer, perinephric abscess).

In secondary bacterial peritonitis from free perforation of a viscus, the peritoneal fluid analysis characteristically shows an extremely elevated polymorphonuclear neutrophil count, multiple organisms (often including fungi and *Enterococcus*) on Gram stain and culture, and at least two of the following criteria[4] :

- Total protein greater than 1 g/dL
- Lactate dehydrogenase above the upper limit of normal for serum
- Glucose less than 50 mg/dL

Differentials

- Aneurysm, Abdominal
- Angioedema
- Appendicitis, Acute
- Mesenteric Ischemia
- Urinary Tract Infection, Female

[ ] References

Contributor Information and Disclosures

Author
Steven M Bandy, MD, FACEP Adjunct Clinical Professor of Emergency Medicine, Virginia College of Osteopathic Medicine; Staff Physician, Department of Emergency Medicine, Johnston Memorial Hospital; Medical Director, Rejuvenage Medspa; Operational Medical Director, Virginia Operations, Wings Air Rescue Ambulance Service

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Disclosure: Nothing to disclose.

Specialty Editor Board
Francisco Talavera, PharmD, PhD Adjunct Assistant Professor, University of Nebraska Medical Center College of Pharmacy; Editor-in-Chief, Medscape Drug Reference

Disclosure: eMedicine Salary Employment

Spontaneous Bacterial Peritonitis Workup

- Author: Steven M Bandy, MD, FACEP; Chief Editor: Rick Kulkarni, MD more...

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Approach Considerations

All patients suspected of having spontaneous bacterial peritonitis (SBP) must undergo peritoneal fluid analysis while in the emergency department. Diagnostic paracentesis should be performed in all patients who do not have an indwelling peritoneal catheter and are suspected of having spontaneous bacterial peritonitis. In peritoneal dialysis patients with a peritoneal catheter, fluid should be withdrawn with sterile technique. Ultrasonography may aid paracentesis if ascites is minimally detectable or questionable.

Blood and urine cultures should be obtained in all patients suspected of having spontaneous bacterial peritonitis. Blood culture results are positive for the offending agent in as many as 33% of patients with spontaneous bacterial peritonitis and may help guide antibiotic therapy. Urine culture may also prove useful, since asymptomatic bacteruria has been suggested to predispose to the development of spontaneous bacterial peritonitis.

Abdominal flat plate, abdominal upright, and chest radiographs are obtained if a perforated viscus is considered.

Peritoneal Fluid Analysis

Peritoneal fluid analysis must be performed in any patient in whom spontaneous bacterial peritonitis (SBP) is considered. In patients undergoing peritoneal dialysis (PD), this can be accomplished by obtaining a sample of the dialysate. In patients without a peritoneal catheter, diagnostic paracentesis must be performed.

The examination of ascitic fluid for SBP has routinely involved sending the fluid for cell count, differential, and culture. It has been accepted that the results of aerobic and anaerobic bacterial cultures, used in conjunction with the cell count, are beneficial in guiding therapy for those with SBP.[5]

Recent data, though, suggest that ascitic fluid cultures have generally been shown to be of low yield with respect to altering management of patients with ascites. In addition, positive culture and sensitivity results obtained from emergency department testing have not been shown to result in appropriate adjustment of antibiotic therapy by inpatient physicians. The reasons for this may include inpatient physicians' distrust of the culture results and the difficulty in determining what constitutes a true pathogen in ascitic fluid cultures.[6]

The sensitivity of microbiologic studies has been reported to increase significantly with the direct inoculation of routine blood culture bottles at the bedside with 10 mL of ascitic fluid.

Ascitic fluid neutrophil count

An ascitic fluid neutrophil count of more than 500 cells/µL is the single best predictor of spontaneous bacterial peritonitis, with a sensitivity of 86% and specificity of 98%. Lowering the ascitic fluid neutrophil count to more than 250 cells/µL results in an increased sensitivity of 93% but a lower specificity of 94%. (For simplicity, a threshold of 250 cells/µL is used for the remainder of this discussion.)

An exciting new development in the rapid diagnosis of spontaneous bacterial peritonitis is the proposed use of reagent strips that detect leukocyte esterase, which can be read at the bedside using a portable spectrophotometric device. In a pilot study that compared the reagent strips with the manual laboratory polymorphonuclear leukocyte count, the strips achieved a 100% sensitivity in diagnosis of spontaneous bacterial peritonitis.[7]

This diagnostic method holds promise in replacing manual cell counting, which is time-consuming and is often unavailable in many laboratories "after hours". Use of these reagent strips may result in a significant reduction of the

time from paracentesis to presumptive diagnosis and antibiotic treatment of spontaneous bacterial peritonitis.

In a small cohort, the average time saved from dipstick to laboratory results ranged from 2.73 hours (dipstick to validated result from automated counter) to 3 hours (dipstick to validated manual cell count of ascitic fluid). Although promising, this diagnostic method has not been investigated in a large-scale study.

Other ascitic fluid studies

Other studies of ascitic fluid to be considered include the following:

- Cytology
- Lactate level
- pH

An ascites lactate level of more than 25 mg/dL was found to be 100% sensitive and specific in predicting active spontaneous bacterial peritonitis in a retrospective analysis. In the same study, the combination of an ascites fluid pH below 7.35 and polymorphonuclear neutrophil count above 500 cells/µL was 100% sensitive and 96% specific for spontaneous bacterial peritonitis.

Combined ascitic fluid neutrophil count and culture

Combining the results of the ascitic fluid polymorphonuclear neutrophil (PMN) count and the ascitic fluid culture yields the following subgroups:

- Spontaneous bacterial peritonitis
- Culture-negative neutrocytic ascites (probable spontaneous bacterial peritonitis)
- Monomicrobial nonneutrocytic bacterascites

Spontaneous bacterial peritonitis is noted when the PMN count is 250 cells/µL or higher, in conjunction with a positive bacterial culture result. As mentioned previously, one organism is usually identified on the culture in most cases. Obviously, these patients should receive antibiotic therapy.

Culture-negative neutrocytic ascites (probable spontaneous bacterial peritonitis) is noted when the ascitic fluid culture results are negative, but the PMN count is 250 cells/µL or higher. This may happen in as many as 50% of patients with SBP and may not actually represent a distinctly different disease entity. It may be the result of poor culturing techniques or late-stage resolving infection. Nonetheless, these patients should be treated just as aggressively as those with positive culture results.

Monomicrobial nonneutrocytic bacterascites exists when a positive culture result coexists with a PMN count of 250 cells/µL or fewer. Although this may often be the result of contamination of bacterial cultures, one study found that 38% of these patients subsequently develop spontaneous bacterial peritonitis.[8] Therefore, monomicrobial nonneutrocytic bacterascites may represent an early form of spontaneous bacterial peritonitis.

All study patients described that eventually developed spontaneous bacterial peritonitis were symptomatic.[8] For this reason, any patient suspected clinically of having spontaneous bacterial peritonitis in this setting must be treated.

Contributor Information and Disclosures

Author
Steven M Bandy, MD, FACEP Adjunct Clinical Professor of Emergency Medicine, Virginia College of Osteopathic Medicine; Staff Physician, Department of Emergency Medicine, Johnston Memorial Hospital; Medical Director, Rejuvenage Medspa; Operational Medical Director, Virginia Operations, Wings Air Rescue Ambulance Service

Steven M Bandy, MD, FACEP is a member of the following medical societies: American Academy of Cosmetic Surgery, American College of Emergency Physicians, and American Society for Laser Medicine and Surgery

Disclosure: Nothing to disclose.

Specialty Editor Board
Francisco Talavera, PharmD, PhD Adjunct Assistant Professor, University of Nebraska Medical Center College of Pharmacy; Editor-in-Chief, Medscape Drug Reference

Spontaneous Bacterial Peritonitis Treatment & Management

- Author: Steven M Bandy, MD, FACEP; Chief Editor: Rick Kulkarni, MD more...

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Approach Considerations

A 2009 guideline from the American Association for the Study of Liver Diseases recommends that adult cirrhotic patients with ascitic fluid polymorphonuclear neutrophil (PMN) counts greater than 250 cells/ µL receive empiric antibiotic therapy (eg, an intravenous third-generation cephalosporin, preferably cefotaxime 2 g every 8 hours).[4] As an alternative to intravenous cefotaxime, inpatients with cirrhosis can be treated with oral ofloxacin (400 mg twice per day), if none of the following contraindications are present[4] :

- Prior exposure to quinolones
- Vomiting
- Shock
- Grade II (or higher) hepatic encephalopathy
- Serum creatinine greater than 3 mg/dL

Patients with a peritoneal fluid PMN count greater than 500 cells/µL should universally be admitted and treated for spontaneous bacterial peritonitis, regardless of peritoneal fluid Gram stain result. Antibiotics should be initiated as soon as possible. The regimen can be chosen empirically, unless microbiologic studies further guide treatment.

For patients with a peritoneal fluid PMN count below 250 cells/µL, management depends upon the results of ascitic fluid cultures. All symptomatic patients should be admitted. Patients whose culture results are positive should be treated for spontaneous bacterial peritonitis. A select subset of patients who are completely asymptomatic yet have positive culture results may be managed without treatment but must undergo a follow-up paracentesis within 24-48 hours.

All symptomatic patients with a peritoneal fluid PMN count of 250-500 cells/µL should be admitted and treated for spontaneous bacterial peritonitis.

Inpatient Care

For spontaneous bacterial peritonitis (SBP), a 10- to 14-day course of antibiotics is recommended. Although not required, a repeat peritoneal fluid analysis is recommended to verify declining PMN counts and sterilization of ascitic fluid.

If improvement in ascitic fluid or clinical condition does not occur within 48 hours, further evaluation is required to rule out bowel perforation or intra-abdominal abscess. Evaluation may include a combination of radiography, CT scanning, intraluminal contrast studies, or surgical exploration.

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Author

Steven M Bandy, MD, FACEP Adjunct Clinical Professor of Emergency Medicine, Virginia College of Osteopathic Medicine; Staff Physician, Department of Emergency Medicine, Johnston Memorial Hospital; Medical Director, Rejuvenage Medspa; Operational Medical Director, Virginia Operations, Wings Air Rescue Ambulance Service

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Spontaneous Bacterial Peritonitis Medication

- Author: Steven M Bandy, MD, FACEP; Chief Editor: Rick Kulkarni, MD more...

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Medication Summary

The goals of pharmacotherapy in patients with spontaneous bacterial peritonitis (SBP) are to reduce morbidity and prevent complications. Antibiotics are initially chosen empirically, as these patients may die from overwhelming infection if treatment is delayed until culture results become available.[4]

Antimicrobials

Class Summary

Traditionally, a combination of an aminoglycoside and ampicillin was used to treat spontaneous bacterial peritonitis (SBP). This regimen affords excellent empiric coverage of more than 90% of cases of spontaneous bacterial peritonitis caused by gram-negative aerobes or gram-positive cocci.

More recently, the third-generation cephalosporin cefotaxime has been demonstrated to be as efficacious as the ampicillin/aminoglycoside combination, and it does not carry the increased risk of nephrotoxicity in cirrhotic patients. Cefotaxime does not cover enterococci (up to 5% of cases).

View full drug information

Cefotaxime (Claforan)

A third-generation cephalosporin with broad gram-negative spectrum, cefotaxime has lower efficacy against gram-positive organisms and higher efficacy against resistant organisms. Thus, it provides excellent empiric coverage of SBP. By binding to 1 or more penicillin-binding proteins, cefotaxime arrests bacterial cell wall synthesis and inhibits bacterial growth.

View full drug information

Gentamicin

Gentamicin is an aminoglycoside antibiotic effective against Pseudomonas aeruginosa; E coli; and Proteus, Klebsiella, and Staphylococcus species. Dosing regimens are numerous; adjust dose based on creatinine clearance (CrCl) and changes in volume of distribution. Gentamicin may be given IV or IM.

View full drug information

Ampicillin

Ampicillin interferes with bacterial cell wall synthesis during active multiplication, causing bactericidal activity against susceptible organisms.

View full drug information

Norfloxacin (Noroxin)

Norfloxacin is used for prophylaxis in the outpatient setting (400 mg/d). It is a fluoroquinolone with activity against pseudomonads, streptococci, MRSA, S epidermidis, and most gram-negative organisms, but it has no activity

against anaerobes. It inhibits bacterial DNA synthesis and, consequently, growth.

View full drug information

Ciprofloxacin (Cipro)

Ciprofloxacin is used for prophylaxis in the outpatient setting (750 mg weekly). It is a fluoroquinolone that inhibits bacterial DNA synthesis and, consequently, growth, by inhibiting DNA gyrase and topoisomerases, which are required for replication, transcription, and translation of genetic material. Quinolones have broad activity against gram-positive and gram-negative aerobic organisms. This agent has no activity against anaerobes.

Sulfamethoxazole and trimethoprim (Bactrim DS, Septra DS)

This agent is used as prophylaxis in the outpatient setting (5 doses of double-strength trimethoprim-sulfamethoxazole per week (Monday through Friday). It inhibits bacterial growth by inhibiting the synthesis of dihydrofolic acid.

Contributor Information and Disclosures

Author
Steven M Bandy, MD, FACEP Adjunct Clinical Professor of Emergency Medicine, Virginia College of Osteopathic Medicine; Staff Physician, Department of Emergency Medicine, Johnston Memorial Hospital; Medical Director, Rejuvenage Medspa; Operational Medical Director, Virginia Operations, Wings Air Rescue Ambulance Service

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Disclosure: Nothing to disclose.

Specialty Editor Board
Francisco Talavera, PharmD, PhD Adjunct Assistant Professor, University of Nebraska Medical Center College of Pharmacy; Editor-in-Chief, Medscape Drug Reference

Disclosure: eMedicine Salary Employment

Mark L Plaster, MD, JD Executive Editor, Emergency Physicians Monthly

Mark L Plaster, MD, JD is a member of the following medical societies: American Academy of Emergency Medicine and American College of Emergency Physicians

Disclosure: M L Plaster Publishing Co LLC Ownership interest Management position

Chief Editor
Rick Kulkarni, MD

Rick Kulkarni, MD is a member of the following medical societies: Alpha Omega Alpha, American Academy of Emergency Medicine, American College of Emergency Physicians, American Medical Association, American Medical Informatics Association, Phi Beta Kappa, and Society for Academic Emergency Medicine

Disclosure: WebMD Salary Employment

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