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CURRENT STRATEGIES IN TREATING AND PREVENTING SPONTANEOUS BACTERIAL PERITONITIS IN ADULTS

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ABSTRACT Spontaneous bacterial peritonitis (SBP) is a life-threatening infection in patients with cirrhosis and ascites. Early recognition and prompt treatment with empirical antibiotics, albumin administration, and supportive care are essential to improve outcomes. This review discusses the pathogenesis, clinical manifestations, diagnosis, treatment, and prevention strategies for SBP, emphasizing the importance of timely intervention. Preventive measures, including secondary prophylaxis with antibiotics, are recommended for high-risk patients to reduce recurrence rates. Enhanced diagnostic techniques and tailored treatment approaches are critical for managing SBP effectively.

KEYWORDS : Spontaneous bacterial peritonitis; Cirrhosis; Empirical antibiotics; Albumin.

INTRODUCTION

Spontaneous bacterial peritonitis (SBP) is a severe and common complication in patients with advanced cirrhosis, characterized by the infection of ascitic fluid without an evident intra-abdominal surgically treatable source. It is most frequently observed in patients with high Model for End-stage Liver Disease (MELD) scores. SBP primarily results from bacterial translocation across the intestinal wall into the ascitic fluid, facilitated by factors such as increased intestinal permeability and bacterial overgrowth. Early recognition and intervention are crucial, as delayed treatment can lead to septic shock and multi-organ failure. Current strategies for managing and preventing SBP involve empirical antibiotic therapy, prophylactic antibiotics in high-risk patients, and careful monitoring of cirrhotic patients with ascites. This review aims to summarize the current approaches to treating and preventing SBP, highlighting the importance of timely diagnosis and effective management to improve patient outcomes (1,2).

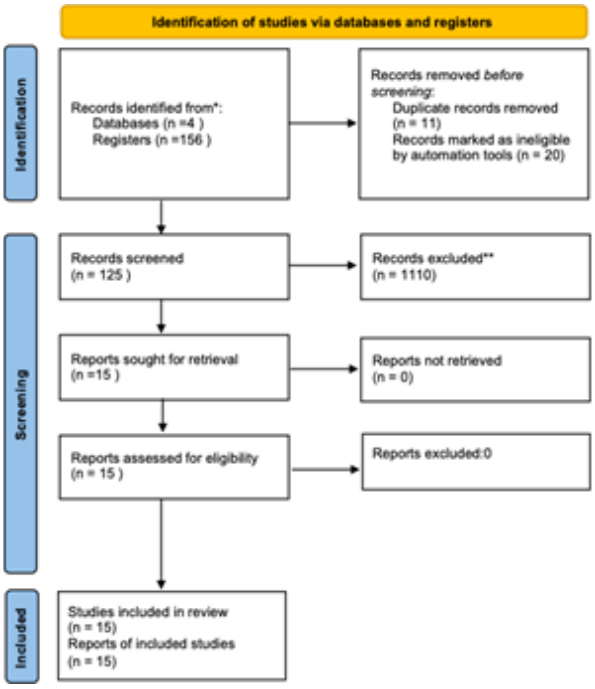

Figure 1. PRISMA.

Methods

The methodology for this narrative review involved a comprehensive search of four databases: PubMed, Scopus, Web of Science, and Cochrane Library. Keywords used in the search included "spontaneous bacterial peritonitis," "SBP," "cirrhosis," "antibiotic therapy," and "prophylaxis." Studies and reviews published in English that focused on the treatment and prevention of SBP in adults were included. Articles were screened for relevance based on their titles and abstracts, followed by a full-text review. After applying inclusion and exclusion criteria, 15 references were selected to provide a detailed and focused overview of current strategies in managing and preventing SBP.

Epidemiology

Spontaneous bacterial peritonitis (SBP) is a prevalent and severe complication in patients with cirrhosis, affecting approximately 10-30% of hospitalized patients with ascites. The incidence of SBP is higher in patients with advanced liver disease, with a Model for End-stage Liver Disease (MELD) score greater than 15. The annual risk of developing SBP in cirrhotic patients with ascites ranges from 7% to 25%. Despite advances in management, the in-hospital mortality rate for SBP remains significant, varying between 20% and 40%. Early detection and prompt treatment are critical to improving survival outcomes in patients with SBP (3,4).

Pathophysiology

The pathogenesis of spontaneous bacterial peritonitis (SBP) is multifaceted, primarily involving bacterial translocation from the intestinal lumen to the ascitic fluid. This process begins with disturbances in gut flora, commonly due to factors such as cirrhosis-related small intestinal dysmotility, increased intestinal permeability, and bacterial overgrowth. Patients with cirrhosis often exhibit altered gut microbiota and impaired mucosal barriers, facilitating the passage of bacteria into the mesenteric lymph nodes and subsequently into the bloodstream and ascitic fluid (5).

Bacterial translocation involves the migration of bacteria or bacterial products across the intestinal epithelium, entering the mesenteric lymph nodes and potentially leading to bacteremia. Escherichia coli, Klebsiella pneumoniae, and Streptococcus species are the most commonly isolated organisms in SBP, reflecting the gut origin of the infection. Once in the ascitic fluid, bacteria must overcome local immune defenses, which are often compromised in cirrhotic patients (6).

Several immune system impairments contribute to the development of SBP in cirrhotic patients. These include reduced complement levels, impaired neutrophil function, and decreased opsonic activity in the ascitic fluid. Complement deficiency is particularly significant, as complement proteins play a crucial role in the opsonization and clearance of bacteria. Ascitic fluid in cirrhotic patients typically has low protein content, further reducing its antibacterial capacity (6,7).

Inflammatory cytokines such as tumor necrosis factor-alpha

(TNF-α) and interleukins (IL-6, IL-1β) are elevated in SBP, promoting an inflammatory response that, while aimed at controlling infection, can lead to further tissue damage and systemic inflammatory response syndrome (SIRS). This inflammatory milieu exacerbates the permeability of the intestinal barrier, perpetuating bacterial translocation and infection (8)

Additionally, the use of proton pump inhibitors (PPIs) in cirrhotic patients has been associated with an increased risk of SBP, potentially due to alterations in gastric pH and subsequent changes in gut microbiota. Advanced liver disease itself, characterized by portal hypertension and hypoalbuminemia, creates an environment conducive to the persistence of bacteria in the ascitic fluid (8,9).

Clinical Manifestations

The clinical manifestations of spontaneous bacterial peritonitis (SBP) are often subtle and nonspecific, posing a challenge for early diagnosis. However, prompt recognition of these signs and symptoms is crucial for timely intervention and improved patient outcomes. Fever is the most common symptom, occurring in approximately 69% of patients. Patients with advanced cirrhosis may present with only a slight elevation in temperature, making it essential to treat any temperature above 37.8°C (100°F) with suspicion. Abdominal pain and tenderness, present in about 59% of cases, are also hallmark features. The pain is usually diffuse rather than localized, differing from the acute, localized pain seen in secondary peritonitis. Due to the presence of ascites, patients typically do not develop the rigid abdomen characteristic of classic peritonitis, but rebound tenderness may be noted in advanced cases (9,10).

Altered mental status, such as confusion or hepatic encephalopathy, is another significant indicator, occurring in about 54% of patients. This symptom can be subtle, with changes often noted by family members or caregivers before healthcare providers. The presence of hepatic encephalopathy in a cirrhotic patient with ascites should prompt consideration of SBP. Additional symptoms include diarrhea (32%), paralytic ileus (30%), hypotension (21%), and hypothermia (17%). Laboratory findings commonly reveal leukocytosis, metabolic acidosis, and azotemia. The development of hypotension and hypothermia are particularly concerning, as they indicate severe infection and a poor prognosis (10,11).

Ascitic fluid analysis is critical for diagnosis. An elevated ascitic fluid absolute polymorphonuclear leukocyte (PMN) count of ≥250 cells/mm^3 is a key diagnostic criterion. A positive ascitic fluid culture further supports the diagnosis, although cultures may be negative if antibiotics were administered before paracentesis. It is important to note that approximately 13% of patients may be asymptomatic at the time of diagnosis, identified only through routine paracentesis performed upon hospital admission or due to clinical deterioration (11).

Diagnosis

The diagnosis of spontaneous bacterial peritonitis (SBP) involves a combination of clinical assessment, laboratory analysis, and imaging studies. Early and accurate diagnosis is crucial due to the high morbidity and mortality associated with untreated SBP (11).

Clinically, SBP should be suspected in any patient with cirrhosis and ascites who presents with symptoms such as fever, abdominal pain or tenderness, altered mental status, or signs of systemic inflammatory response syndrome (SIRS). Given that some patients may be asymptomatic, routine diagnostic paracentesis is recommended for all hospitalized cirrhotic patients with ascites, especially upon admission or if there is any clinical deterioration (11,12).

Laboratory analysis of ascitic fluid is central to the diagnosis. The most critical diagnostic criterion is an elevated absolute polymorphonuclear leukocyte (PMN) count of ≥250 cells/mm^3 in the ascitic fluid. This threshold is sufficient to initiate empirical antibiotic therapy, even in the absence of a positive culture. Ascitic fluid cultures, although specific, may have low sensitivity, particularly if antibiotics have been administered before paracentesis. Therefore, cultures should be obtained using bedside inoculation of blood culture bottles to enhance yield (12).

Additional ascitic fluid tests include total protein concentration, glucose levels, lactate dehydrogenase (LDH), and Gram stain, which can help differentiate SBP from secondary bacterial peritonitis. Serum blood tests may show leukocytosis, elevated C-reactive protein (CRP), and procalcitonin levels, indicating infection and inflammation. Imaging studies, such as ultrasound or computed tomography (CT), are not diagnostic for SBP but can help rule out secondary causes of peritonitis, such as bowel perforation or abscess formation (12,13).

Treatment

The treatment of spontaneous bacterial peritonitis (SBP) is a medical emergency that requires prompt initiation of empirical antibiotic therapy and supportive care to reduce morbidity and mortality. The primary goal is to eradicate the infection, manage symptoms, and prevent complications (13).

Empirical Antibiotic Therapy

Empirical antibiotic therapy should be initiated immediately upon suspicion of SBP, without waiting for culture results. The choice of antibiotics depends on local resistance patterns, but third-generation cephalosporins such as cefotaxime (2 g every 8 hours) are commonly recommended due to their efficacy and low toxicity. Alternatives include ceftriaxone or piperacillin-tazobactam. For patients with a high risk of infection with multidrug-resistant organisms, such as those with recent antibiotic use or nosocomial infections, carbapenems or a combination of ceftazidime and vancomycin may be considered (13).

Duration Of Therapy

The standard duration of antibiotic therapy is 5 to 10 days, with clinical improvement usually seen within 48 hours. Therapy should be tailored based on culture results and sensitivity patterns when available (13,14).

Albumin Administration

In addition to antibiotics, intravenous albumin administration is recommended, especially for patients with renal dysfunction or elevated bilirubin levels. Albumin helps to prevent renal impairment, a common complication of SBP. The typical regimen involves 1.5 g/kg of albumin on the day of diagnosis, followed by 1 g/kg on day three (14).

Supportive Care

Supportive care is essential and includes careful monitoring of vital signs, fluid balance, and renal function. Aggressive management of hypotension and hypovolemia is crucial, often necessitating intravenous fluids and vasopressors (14).

Prophylaxis

Secondary prophylaxis with antibiotics is recommended for patients who have recovered from an episode of SBP to prevent recurrence. Oral norfloxacin (400 mg daily) or trimethoprim-sulfamethoxazole (one double-strength tablet daily) are commonly used. Primary prophylaxis may be indicated in high-risk patients, such as those with low protein

concentration in ascitic fluid (<1.5 g/dL) and advanced liver disease (14,15).

Monitoring And Follow-up

Patients should be closely monitored for signs of treatment failure, complications, and adverse effects of antibiotics. Follow-up paracentesis may be performed in non-responders to confirm resolution of infection or identify alternative diagnoses (15).

CONCLUSION

Early diagnosis and prompt treatment of spontaneous bacterial peritonitis (SBP) are crucial to improving patient outcomes. Implementing empirical antibiotic therapy, supportive care, and prophylactic measures significantly reduces morbidity and mortality associated with this severe complication in cirrhotic patients.

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