Review Article

Primary Bacterial Peritonitis in Adults: Diagnostic Clues for Differentiation from Secondary Peritonitis and Current Evidence for Non-Surgical Medical Management

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Abstract

Spontaneous bacterial peritonitis is a severe infectious complication that predominantly affects patients with cirrhosis and ascites and remains a major cause of morbidity and mortality. Its pathophysiology is primarily driven by bacterial translocation from the intestinal lumen into the peritoneal cavity, a process facilitated by increased intestinal permeability, gut dysbiosis, immune dysfunction, and portal hypertension associated with advanced liver disease. These mechanisms explain not only the high incidence of spontaneous bacterial peritonitis but also the evolving microbiological profile, historically dominated by Gram-negative bacteria and increasingly

characterized by Gram-positive and multidrug-resistant organisms. The clinical presentation is often nonspecific, including abdominal pain, fever, worsening ascites, or altered mental status, which contributes to diagnostic delays. Diagnostic paracentesis is therefore essential, with an ascitic fluid absolute neutrophil count of at least 250 cells/mm³ representing the cornerstone of diagnosis. Additional biochemical and microbiological analyses, together with selective use of imaging studies, are critical for differentiating spontaneous bacterial peritonitis from secondary peritonitis, a condition associated with an identifiable intra-abdominal source and a fundamentally different therapeutic approach. Management is primarily non-surgical and relies on early initiation of empirical antibiotic therapy, followed by targeted treatment based on culture results, as well as the adjunctive use of albumin to reduce the risk of renal dysfunction and mortality. However, lack of clinical response, progression to sepsis, or radiological findings suggestive of secondary peritonitis require prompt reassessment and multidisciplinary intervention. Despite advances in diagnosis and treatment, significant challenges remain, particularly regarding antimicrobial resistance and the controversial role of secondary prophylaxis. These issues highlight the need for further research to refine preventive strategies and improve outcomes in this high-risk population.

Key words

Spontaneous bacterial peritonitis; Cirrhosis; Ascites; Bacterial translocation; Differential diagnosis; Antimicrobial therapy.

Introduction

Primary bacterial peritonitis is defined as an infection of the ascitic fluid that occurs in the absence of an identifiable intra-abdominal source of infection and is most commonly observed in patients with liver cirrhosis complicated by ascites [1, 2]. Its clinical relevance lies in the substantial burden of morbidity and mortality associated with the condition, as reported one-year mortality rates reach approximately 66 percent among affected individuals [3]. Despite this severity, the clinical presentation is frequently subtle or nonspecific. Many patients may remain asymptomatic or develop vague manifestations such as abdominal discomfort, fever, or altered mental status, which significantly complicates early recognition and timely diagnosis [1].

From an epidemiological perspective, primary bacterial peritonitis represents one of the most frequent infectious complications in patients with cirrhosis and ascites, carrying a high risk of recurrence and sustained mortality over time. Traditionally, the condition has been predominantly attributed to Gram-negative organisms; however, recent data indicate a growing prevalence of Gram-positive pathogens and multidrug-resistant bacteria, reflecting changes in antimicrobial exposure and healthcare-associated risk factors. Beyond its direct impact on patient outcomes, primary bacterial peritonitis imposes a considerable strain on healthcare systems, largely due to recurrent hospital admissions, prolonged inpatient stays, and the need for intensive medical management that includes broad-spectrum antibiotic therapy and close supportive monitoring [1, 2].

Within this context, the ability to accurately distinguish primary bacterial peritonitis from secondary peritonitis is of critical importance. Secondary peritonitis is characterized by the presence of an identifiable intra-abdominal source of infection, such as gastrointestinal perforation or abscess formation, and typically requires prompt surgical intervention for effective source control. In contrast, primary bacterial peritonitis is primarily managed through medical therapy, with antibiotics constituting the cornerstone of treatment. Failure to correctly differentiate between these entities may result in inappropriate therapeutic decisions, delayed

surgical management when indicated, or unnecessary operative interventions, all of which are associated with increased rates of complications and mortality [4, 5].

The aim of this article is to review primary bacterial peritonitis in adults, emphasizing the key clinical, laboratory, and imaging features that allow its differentiation from secondary peritonitis, and to summarize the current evidence supporting non-surgical medical management in appropriate patients.

Methodology

The present review on primary bacterial peritonitis in adults was conducted through a structured analysis of the available scientific literature with the aim of examining its clinical relevance, diagnostic approach, and current evidence supporting non-surgical medical management. Particular attention was given to the identification of clinical, laboratory, and imaging features that allow reliable differentiation from secondary peritonitis, as this distinction is essential for appropriate therapeutic decision-making.

A comprehensive literature search was performed using PubMed, Scopus, and Web of Science, selected for their wide coverage of high-quality publications in hepatology, internal medicine, infectious diseases, and acute care. Articles published between 2021 and 2026 in English or Spanish were included if they addressed key aspects such as epidemiology, pathophysiology, risk factors, diagnostic criteria, differential diagnosis, antimicrobial therapy, and clinical outcomes. Studies without peer review, with incomplete data, or containing duplicated information were excluded. The search strategy incorporated terms including *spontaneous bacterial peritonitis; cirrhosis; ascites; bacterial translocation; differential diagnosis; antimicrobial therapy.*

The selected publications, including original studies, reviews, and clinical guidelines, were critically analyzed using a qualitative and comparative approach. Artificial intelligence tools were used as supportive resources to facilitate thematic organization and synthesis of the evidence. This methodology allowed the integration of current knowledge into a coherent framework, highlighting diagnostic principles, evidence-based medical treatment, and factors influencing prognosis in adult patients with primary bacterial peritonitis.

Pathophysiology and predisposing conditions

Bacterial translocation represents a central mechanism in the pathogenesis of spontaneous bacterial peritonitis, whereby bacteria cross the intestinal barrier and gain access to the systemic circulation. This process is closely linked to increased intestinal permeability, a phenomenon commonly observed in patients with cirrhosis as a consequence of both structural and functional alterations of the intestinal mucosa [6, 7]. Pathogenic microorganisms further aggravate this disruption by degrading key tight junction proteins, including E-cadherin and occludin, which compromises epithelial integrity and results in a leaky intestinal barrier [8]. In parallel, dysbiosis, characterized by an imbalance of the gut microbiota, is frequently present in cirrhosis and contributes to both increased permeability and enhanced bacterial translocation, thereby facilitating the development of infection [9, 10].

These alterations occur within a broader context of immune dysfunction associated with cirrhosis, which markedly increases susceptibility to infections such as spontaneous bacterial peritonitis. This immune impairment is driven by dysregulated host defenses, including defective macrophage activation and altered cytokine production, leading to an inadequate response to translocated bacteria [11, 12]. Concomitantly, portal hypertension, a hallmark complication of advanced liver disease, further exacerbates bacterial translocation by intensifying intestinal congestion and permeability, thereby reinforcing

the cycle of microbial passage from the gut lumen into the systemic compartment [10, 13].

Within this pathophysiological framework, cirrhosis complicated by ascites emerges as the most significant risk factor for spontaneous bacterial peritonitis, as the presence of ascitic fluid provides a favorable environment for bacterial proliferation [1]. Additional conditions, such as nephrotic syndrome and states of immunosuppression, further compromise host defense mechanisms and increase vulnerability to infection [12]. From a microbiological perspective, spontaneous bacterial peritonitis is classically associated with Gram-negative organisms, particularly *Escherichia coli* and *Klebsiella pneumoniae*. Nonetheless, recent evidence indicates a growing prevalence of Gram-positive bacteria and multidrug-resistant organisms, reflecting shifts in microbial ecology and antibiotic exposure patterns. These changes are closely linked to alterations in gut microbiota composition, including reduced microbial diversity and expansion of pathogenic taxa, which have been associated with an increased risk of spontaneous bacterial peritonitis [1, 14].

Clinical presentation

Patients with primary bacterial peritonitis commonly present with nonspecific clinical manifestations, most frequently including abdominal pain, fever, and progressive worsening of ascites. In individuals with advanced liver disease, altered mental status is also a frequent finding and may represent one of the earliest clinical indicators of infection. From a diagnostic standpoint, a defining criterion for primary bacterial peritonitis is an ascitic fluid absolute neutrophil count of at least 250 cells per cubic millimeter. Diagnostic paracentesis therefore constitutes the procedure of choice and should be promptly performed when the condition is suspected, with ascitic fluid cultures obtained to identify the causative microorganism. Consistent with its pathophysiology, the microbiological profile of primary bacterial peritonitis is classically dominated by Gram-negative bacteria, although recent reports have documented a growing contribution of Gram-positive organisms and multidrug-resistant strains [1, 15].

Despite these typical features, atypical presentations are not uncommon and pose significant diagnostic challenges. A subset of patients may remain asymptomatic or present with minimal clinical findings, which can delay diagnosis and initiation of therapy and consequently increase the risk of complications [1]. Diagnostic complexity is further heightened in patients with concomitant liver lesions, as infectious processes may mimic malignant disease on imaging studies, complicating the distinction between inflammatory and neoplastic conditions [16]. In addition, there remains ongoing debate regarding the optimal use of prophylactic strategies for primary bacterial peritonitis, particularly in light of concerns related to antimicrobial resistance and the variable effectiveness of currently available prophylactic regimens [17].

When compared with primary bacterial peritonitis, secondary peritonitis is characterized by a distinct etiological and clinical profile. Secondary peritonitis arises from a direct intra-abdominal source of infection, such as a perforated viscus or bowel obstruction, which fundamentally differentiates it from the spontaneous nature of primary infection [18, 19]. Clinically, secondary peritonitis is more likely to present with severe systemic manifestations, including septic shock and organ failure, and is frequently associated with polymicrobial infections [20]. Various clinical tools, such as the qSOFA and SIRS scores, have been employed to predict adverse outcomes in this setting, although their diagnostic and prognostic performance remains variable [21].

Certain clinical and laboratory findings should raise suspicion of a secondary etiology in patients initially presumed to have primary bacterial peritonitis. An ascitic leukocyte count

exceeding 10,000 cells per cubic millimeter strongly suggests secondary peritonitis and warrants further investigation [22]. Similarly, persistent inflammation despite appropriate antimicrobial therapy, reflecting inadequate source control, represents a critical warning sign [18]. The isolation of enteric pathogens from ascitic fluid is also more indicative of secondary peritonitis and has been associated with poorer clinical outcomes, reinforcing the need for prompt diagnostic reassessment and escalation of care in such cases [20].

Diagnostic evaluation

Ascitic fluid analysis constitutes the cornerstone of the diagnostic evaluation in suspected spontaneous bacterial peritonitis. The absolute neutrophil count in ascitic fluid is the most critical parameter, with a threshold of at least 250 cells per cubic millimeter representing the diagnostic hallmark of the condition. This cutoff is essential for timely initiation of treatment, as delayed recognition and management are associated with significant morbidity and mortality [1]. In addition to cellular analysis, biochemical parameters provide important complementary information. The serum–ascites albumin gradient is routinely used to distinguish transudative from exudative ascites and to support identification of the underlying etiology. Other biochemical markers, including lactate dehydrogenase and glucose levels in ascitic fluid, can further aid in differentiating spontaneous bacterial peritonitis from secondary bacterial peritonitis, particularly when values are suggestive of an alternative intra-abdominal source of infection [23].

Beyond ascitic fluid assessment, blood tests and systemic inflammatory markers play a supportive role in the diagnostic process. The neutrophil percentage-to-albumin ratio has emerged as a novel and promising marker, demonstrating a sensitivity of 85.71 percent and a specificity of 66.67 percent at a cutoff value greater than 17. This parameter offers a non-invasive and cost-effective option that may assist in the early identification of spontaneous bacterial peritonitis. In parallel, elevated levels of C-reactive protein and increased total leukocyte count have been associated with a higher likelihood of culture-positive spontaneous bacterial peritonitis, reflecting the presence of systemic inflammation and infection [24].

Microbiological studies remain fundamental for confirming the diagnosis and guiding targeted therapy. Culturing ascitic fluid using blood culture bottles significantly enhances the detection rate of causative organisms and is therefore recommended in routine clinical practice. Blood cultures provide additional diagnostic value by identifying concurrent bacteremia, which may influence prognosis and therapeutic decisions [1, 25]. In recent years, ascitic fluid calprotectin has been identified as a highly accurate diagnostic biomarker, with reported sensitivity and specificity of 85.7 percent and 89.5 percent, respectively, at a cutoff value exceeding 21 micrograms per milliliter, supporting its potential role as an adjunctive diagnostic tool [26].

Imaging studies complement laboratory evaluation, primarily by facilitating procedures and excluding alternative diagnoses. Ultrasound plays a key role in guiding paracentesis, ensuring safe and effective sampling of ascitic fluid for diagnostic analysis [1]. Although computed tomography is not routinely required for the diagnosis of spontaneous bacterial peritonitis, it may be particularly valuable when secondary peritonitis is suspected, as it allows identification of intra-abdominal abscesses, perforations, or other structural abnormalities that may necessitate surgical intervention [25].

Differential diagnosis; primary vs secondary peritonitis

Primary peritonitis (spontaneous bacterial peritonitis)

Primary peritonitis most frequently affects patients with cirrhosis and typically presents with fever, abdominal pain, and altered mental status,

although symptoms may be subtle in advanced liver disease. The diagnosis is established by ascitic fluid analysis, with an absolute neutrophil count of at least 250 cells/mm³ representing the key diagnostic criterion. The neutrophil percentage-to-albumin ratio has emerged as a complementary marker, demonstrating high sensitivity and specificity for the diagnosis of spontaneous bacterial peritonitis [1, 24].

Secondary peritonitis

Secondary peritonitis is usually associated with more intense abdominal pain and clear signs of systemic infection. Laboratory findings supporting this diagnosis include an ascitic leukocyte count greater than 10,000 cells/mm³ and the absence of typical features of decompensated cirrhosis [22]. Additional biochemical abnormalities, such as elevated lactate dehydrogenase levels and low glucose concentrations in ascitic fluid, further suggest a secondary intra-abdominal source [23].

Ascitic fluid patterns: Monomicrobial vs Polymicrobial infection

Monomicrobial Infection: Monomicrobial growth is classically associated with spontaneous bacterial peritonitis and is most caused by Gram-negative organisms, particularly *Escherichia coli* [1]. Culture-negative spontaneous bacterial peritonitis is also frequent, which emphasizes the importance of integrating clinical and laboratory criteria when cultures are negative [24].

Polymicrobial Infection: Polymicrobial ascitic fluid cultures are more suggestive of secondary peritonitis and often reflect contamination by enteric pathogens. This pattern has been associated with poorer outcomes, especially among patients undergoing peritoneal dialysis [20].

Imaging features suggesting secondary peritonitis

Computed tomography plays a pivotal role in the evaluation of suspected secondary peritonitis by enabling identification of the underlying source of infection, such as gastrointestinal perforation or intra-abdominal abscess formation [25]. Radiological findings including free intraperitoneal air or localized fluid collections strongly favor a secondary etiology [22].

Practical diagnostic algorithms

Initial evaluation should include diagnostic paracentesis in all patients with ascites and suspected peritonitis to determine the absolute neutrophil count, obtain ascitic fluid cultures, and assess biochemical markers such as lactate dehydrogenase and glucose [1, 23]. When clinical or laboratory findings raise concern for secondary peritonitis, computed tomography should be performed to identify a potential intra-abdominal source. Diagnostic algorithms that integrate clinical assessment, laboratory data, and imaging findings support accurate differentiation between primary and secondary peritonitis and guide appropriate management decisions [25].

Medical (non-surgical) management of primary bacterial peritonitis

Empirical antibiotic therapy represents the cornerstone of initial management in spontaneous bacterial peritonitis and should be initiated promptly once the diagnosis is suspected. Third-generation cephalosporins, particularly ceftriaxone, have traditionally been considered first-line agents because of their proven efficacy against the most frequently implicated Gram-negative pathogens, including *Escherichia coli* and *Klebsiella pneumoniae* [1]. Nevertheless, in patients with recognized risk factors for multidrug-resistant organisms, such as recent antibiotic exposure, healthcare-associated infection, or nosocomial onset, the use of broader-spectrum agents may be warranted. In these scenarios, antibiotics such as carbapenems or piperacillin–tazobactam are often recommended to ensure adequate initial coverage [27].

Empirical regimens should be selected to provide effective coverage against both Gram-negative and Gram-positive organisms, although priority

is generally given to Gram-negative bacteria given their higher prevalence in spontaneous bacterial peritonitis [1]. The increasing incidence of Gram-positive pathogens and resistant strains has reinforced the importance of tailoring empirical therapy to local epidemiological data [27]. In particular, local resistance patterns and institutional microbiological surveillance should guide antibiotic selection, especially in nosocomial settings where resistant organisms are more commonly encountered. Patient-specific factors, including prior infections and comorbid conditions, must also be considered when choosing the initial antimicrobial strategy [28].

Once microbiological culture and susceptibility results become available, antibiotic therapy should be reassessed and, when appropriate, de-escalated to a narrower-spectrum agent. This strategy aims to reduce selective pressure for antimicrobial resistance while minimizing drug-related toxicity and aligns with established principles of antimicrobial stewardship [29]. Regarding treatment duration, a course of five to seven days is generally sufficient for uncomplicated spontaneous bacterial peritonitis, although the exact length of therapy may vary depending on clinical response and the presence of complications. Therapeutic success is typically reflected by clinical improvement, normalization of inflammatory markers such as the white blood cell count, and a reduction in ascitic fluid neutrophil count to below 250 cells per cubic millimeter [1].

In addition to antimicrobial therapy, adjunctive treatments play a crucial role in optimizing outcomes. Albumin infusion is recommended in selected patients to prevent renal dysfunction and reduce mortality, particularly among those with elevated serum bilirubin or creatinine levels. The commonly recommended dosing regimen consists of 1.5 grams per kilogram of body weight administered on the first day, followed by 1 gram per kilogram on the third day. Supportive care measures are equally important and include close monitoring of hemodynamic status, renal function, and electrolyte balance. Regular assessment of vital signs, renal parameters, and serum electrolytes is essential to guide fluid management and electrolyte replacement, thereby supporting overall clinical stability during the course of treatment [1].

Indications for surgical evaluation and treatment escalation

Failure to respond to appropriate medical therapy represents a key indication for surgical evaluation in patients with peritonitis. When clinical improvement is not observed despite adequate antimicrobial treatment, the underlying issue is often insufficient source control, which is a critical determinant of outcomes in intra-abdominal infections [18]. In this context, lack of response may be related to factors such as antimicrobial resistance, a phenomenon particularly prevalent in nosocomial peritonitis, where resistant organisms limit the effectiveness of empirical therapy and increase the need for surgical assessment [4].

Persistent or worsening sepsis despite optimized medical management further reinforces the indication for surgical intervention. Ongoing sepsis reflects uncontrolled infection and is associated with progression to multi-organ dysfunction, substantially increasing the risk of mortality [30]. The development of septic shock, often reflected by elevated Sequential Organ Failure Assessment scores, has been consistently linked to poorer outcomes and underscores the importance of timely surgical evaluation when medical therapy alone fails to achieve adequate control [18].

Radiological and clinical findings also play a decisive role in identifying patients who require surgical exploration. Imaging features suggestive of secondary peritonitis, such as the presence of free intraperitoneal air or localized fluid collections, strongly indicate an underlying intra-abdominal source and mandate prompt surgical intervention to prevent further complications.

Similarly, clinical signs including progressive abdominal distension and localized or diffuse tenderness may signal secondary peritonitis and should prompt reconsideration of the initial non-surgical approach [20].

Given the complexity of these clinical scenarios, multidisciplinary management is essential. Collaboration among surgeons, intensivists, and infectious disease specialists allows for a comprehensive approach that integrates timely source control, optimization of antimicrobial therapy, and advanced supportive care [4]. Such coordinated decision-making has been shown to enhance clinical outcomes by ensuring that both medical and surgical aspects of peritonitis management are addressed in a timely and effective manner [30].

Prognosis, prevention, and future directions

Prognosis in spontaneous bacterial peritonitis is closely related to the severity of underlying liver disease, the presence of organ failure, and the timeliness and adequacy of management. Delayed diagnosis and insufficient source control are consistently associated with increased mortality, underscoring the importance of early recognition and appropriate therapeutic intervention. Among prognostic tools, the Sequential Organ Failure Assessment score has emerged as a particularly important determinant of outcome, with higher scores correlating strongly with increased mortality risk. In critically ill patients, effective source control combined with appropriate antimicrobial therapy remains essential for improving survival and overall clinical outcomes [18, 31].

The risk of recurrence following an episode of spontaneous bacterial peritonitis represents a major clinical concern, and the role of secondary prophylaxis remains controversial. Available evidence suggests that secondary prophylaxis may paradoxically increase the risk of recurrence, with reported hazard ratios ranging from 1.63 to 1.68, corresponding to a 63 to 68 percent higher risk compared with no prophylactic therapy [32]. In addition, the prolonged use of prophylactic antibiotics has been linked to the emergence of resistant pathogens, including extended-spectrum beta-lactamase–producing organisms, which significantly complicates subsequent treatment and worsens prognosis [2]. This dilemma is widely recognized among hepatology specialists, many of whom advocate for updated and methodologically robust trials to reassess the efficacy and safety of current secondary prophylaxis strategies [17].

Preventive strategies in high-risk populations are therefore focused on careful patient selection and risk stratification. Individuals with advanced cirrhosis or low ascitic fluid protein levels are considered particularly vulnerable and may benefit from targeted preventive measures [2]. At the same time, the judicious use of proton pump inhibitors is recommended, as their administration has been associated with an increased risk of spontaneous bacterial peritonitis, although this association appears weak and varies according to study design and quality (Alhumaid et al., 2021). Albumin infusion has demonstrated benefits in reducing renal impairment and mortality in patients with spontaneous bacterial peritonitis, reinforcing its role not only as an adjunctive therapy but also as part of broader preventive strategies in selected patients [1].

Emerging evidence highlights the need for improved diagnostic and prognostic tools, including novel biomarkers such as the neutrophil percentage-to-albumin ratio, which has shown promising sensitivity and specificity for the diagnosis of spontaneous bacterial peritonitis [24]. At the same time, there is a clear demand for well-designed randomized controlled trials to address the limitations of existing prophylactic approaches and to explore alternative preventive strategies that balance efficacy with the risk of antimicrobial resistance [17]. Important research gaps persist, particularly

regarding the evolving microbiological landscape of spontaneous bacterial peritonitis, the increasing prevalence of multidrug-resistant organisms, and the development of new therapeutic options capable of improving outcomes in this high-risk population [1, 31].

Conclusions

Spontaneous bacterial peritonitis results from a complex interaction between increased intestinal permeability, dysbiosis, immune dysfunction, and portal hypertension in patients with cirrhosis. This interaction favors bacterial translocation and explains both its high incidence and its changing microbiological profile, with a progressive increase in Gram-positive and multidrug-resistant microorganisms.

Timely diagnosis remains the main prognostic determinant and requires a systematic approach based on early paracentesis, analysis of the absolute neutrophil count in ascitic fluid, biochemical and microbiological markers, and careful clinical and imaging evaluation to differentiate primary from secondary peritonitis, thus avoiding therapeutic delays or inappropriate interventions.

Non-surgical medical management, supported by appropriate empirical antibiotic therapy, culture-guided de-escalation, and selective use of albumin, is effective in most cases. However, lack of response, progression to sepsis, or findings suggestive of secondary peritonitis require immediate reassessment and a multidisciplinary approach, while prevention and prophylaxis remain controversial areas that require further clinical evidence**.**

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