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Mykola Tutchenko
https://orcid.org/0000-0002-5050-6494
Department of Dentist Surgery, Bogomolets National Medical University, Kyiv, Ukraine

Diana Rudyk
https://orcid.org/0000-0001-9736-3614
Department of Dentist Surgery, Bogomolets National Medical University, Kyiv, Ukraine

Mykola Besedinskyi
https://orcid.org/0009-0009-8618-1458
Department of Dentist Surgery, Bogomolets National Medical University, Kyiv, Ukraine

Sergey Chub
https://orcid.org/0000-0001-6489-7345
Department of Dentist Surgery, Bogomolets National Medical University, Kyiv, Ukraine

ABSTRACT

SPONTANEOUS BACTERIAL PERITONITIS

Spontaneous bacterial peritonitis (SBP) is an infection of ascitic fluid, posing a severe complication in patients with portal hypertension (PH). Hospital mortality rates are exceedingly high, sometimes surpassing 85%. Only timely diagnosis and treatment can mitigate mortality and morbidity levels.

Purpose. Through literature analysis and review, demonstrate the necessity, appropriateness, and efficacy of SBP treatment in patients with decompensated portal hypertension. Additionally, highlight the potential development of ascites and subsequent infections, which may not always be linked to liver cirrhosis.

Materials and methods. This article conducts a literature review and assesses recommendations, findings from randomized controlled trials, meta-analyses, and other review articles published in databases such as PubMed, Scopus, Web of Science, and Google Scholar between 2000 and 2023.

Outcome. Diagnostic paracentesis is recommended for all ascites patients requiring emergency care or hospitalization to ascertain SBP presence. Accurate differentiation between SBP and secondary bacterial peritonitis is crucial, as treatment approaches differ for each condition.

Conclusions. Standard SBP treatment entails promptly administering broad-spectrum antibiotics, considering the potential for community-acquired or nosocomial SBP, and factoring in microbial antibiotic resistance. Given SBP's annual mortality rate surpassing 50%, liver transplantation is recommended for SBP survivors.

Keywords: spontaneous bacterial peritonitis, liver cirrhosis, portal hypertention, ascites fluid, antibiotic prophylaxis.

Corresponding author: *Besedinskyi Mykola, Department of Dentist Surgery, Bogomolets National Medical University, Kyiv, Ukraine*
e-mail: mr.besedinsky@gmail.com, tel.: +380672820892

РЕЗЮМЕ

Тутченко Микола Іванович
https://orcid.org/0000-0002-5050-6494
Кафедра хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, м. Київ, Україна

Рудик Діана Віталіївна
https://orcid.org/0000-0001-9736-3614
Кафедра хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, м. Київ, Україна

Беседінський Микола Сергійович
https://orcid.org/0009-0009-8618-1458
Кафедра хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, м. Київ, Україна

Чуб Сергій Леонідович
https://orcid.org/0000-0001-6489-7345
Кафедра хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, м. Київ, Україна

СПОНТАННИЙ БАКТЕРІАЛЬНИЙ ПЕРИТОНІТ

Спонтанний бактеріальний перитоніт (СБП) – це інфекція асцитичної рідини, яка є серйозним ускладненням у пацієнтів з портальною гіпертензією (ПГ). Госпітальна смертність дуже висока і може перевищувати 85%. Лише своєчасна діагностика та лікування можуть знизити рівень смертності і захворюваності.

Мета. На підставі аналізу та огляду літератури продемонструвати необхідність, доцільність та успішність лікування СБП у пацієнтів із декомпенсованою портальною гіпертензією, а також вказати на можливість розвитку асциту та подальшого інфікування, які не завжди пов'язані з цирозом печінки.

Матеріали та методи. У даній статті проведено огляд літератури та оцінку рекомендацій, результатів рандомізованих контрольованих досліджень, мета-аналізів та інших оглядових статей, що були опубліковані у базах даних PubMed, Scopus, Web of Science, Google Scholar в період з 2000 по 2023 рік.

Результати. Для всіх пацієнтів з асцитом, які потребують невідкладної допомоги або госпіталізації, рекомендується проведення діагностичного парацентезу з метою визначення наявності СБП. Важливо правильно відрізняти СБП від вторинного бактеріального перитоніту, оскільки ці патології вимагають різних підходів до лікування.

Висновки. Стандартним лікуванням СБП є негайне призначення антибіотиків широкого спектру з урахуванням можливості виникнення позалікарняного або нозокоміального СБП, а також з урахуванням антибіотикорезистентності мікрофлори. Оскільки загальна річна смертність від СБП перевищує 50%, трансплантація печінки показана для тих, хто пережив СБП.

Ключові слова: спонтанний бактеріальний перитоніт, цироз печінки, портальна гіпертензія, асцит, антибіотикопрофілактика.

Автор, відповідальний за листування: *Беседінський Микола Сергійович, кафедра хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, м. Київ, Україна*
e-mail: mr.besedinsky@gmail.com, tel.: +380672820892

Abbreviations

SBP - Spontaneous bacterial peritonitis
PH - portal hypertention
AF - ascitic fluid
PMN - polymorphonuclear cells
SAAG - serum–ascites albumin gradient
SBD - Selective bacterial decontamination

INTRODUCTION / ВСТУП

Clinical awareness, prompt diagnosis, and pathogenetic management are crucial in treating patients with SBP.

Portal hypertension results in a variety of disorders due to changes in liver parenchyma and hemodynamic alterations in portal blood flow. These complications include bleeding, ascites formation, and encephalopathy, necessitating a multifaceted approach to diagnosis and treatment [1]. SBP is a bacterial infection of ascitic fluid without an intra-abdominal infection source. Without treatment, mortality exceeds 90% [2]. SBP diagnosis occurs when the absolute neutrophil count (polymorphonuclear cells or PMN) in ascitic fluid (AF) reaches or exceeds 250 cells/mm3 [3, 4]. The outcome of ascitic fluid infection in cirrhosis patients is influenced by bacteria type and antimicrobial susceptibility [5]. Preventing SBP through antibiotic therapy is a crucial component for enhancing the survival of patients with decompensated PH.

Materials and methods. The purpose of this article is to provide support for clinicians and healthcare

professionals involved in assessing, researching, and managing patients with SBP. Our analysis focused on scientific literature sourced from the PubMed database, covering publications between 2000 and 2023. Key search terms included SBP, PH, antibiotics, ascites, paracentesis, microflora, treatment, and prevention.

Resultsand discussion. SBP emerges as the most prevalent and perilous infection among liver cirrhosis patients, necessitating immediate identification and treatment. It is characterized by the presence of >250 polymorphonuclear cells (PMN)/mm3 in ascites without intra-abdominal infection or malignancy [6]. Gram-negative aerobic organisms are responsible for the majority (75%) of SBP cases, with Klebsiella pneumoniae being implicated in 50% of these instances [7]. Notably, 93% of SBP cases exhibit a monomicrobial infection with aerobic gram-negative bacteria, with E.coli (70%) being the predominant microorganism, followed by Klebsiella [8].

According to current guidelines, third-generation cephalosporins represent the primary treatment for SBP, given the prevalence of Gram-negative Enterobacteriaceae [9]. However, hospitalization, escalating invasive procedures, and antibiotic prophylaxis in cirrhotic patients alter the bacterial landscape and resistance patterns of causative pathogens, contributing to an upsurge in SBP cases caused by Gram-positive and tigecycline-resistant bacteria [10].

Analysis of ascitic fluid is frequently omitted in patients with PH complicated by ascites who are hospitalized for various conditions, yet it remains essential for managing these patients effectively. When cirrhosis diagnosis is not clinically apparent, ascites resulting from portal hypertension can be distinguished from ascites caused by other factors through the serum–ascites albumin gradient (SAAG) [11].

SAAG serves as a vital clinical diagnostic tool for ascites evaluation, with an elevated SAAG (>1.1 gm/dL) indicating portal hypertension, discernible through portal hypertensive alterations in the upper gastrointestinal tract [12].

Early and prudent antibiotic administration can be life-saving, particularly for patients with end-stage liver disease characterized by a history of ascites, variceal hemorrhage, hepatic encephalopathy, hepatopulmonary syndrome, hepatorenal syndrome, SBP, or hepatocellular carcinoma [13].

However, in cases where clinical symptoms suggestive of SBP with an elevated neutrophil count in ascitic fluids are present, up to 60% of cultures may yield negative results [14].

Numerous studies have demonstrated the diagnostic and prognostic significance of either ascitic calprotectin or the calprotectin to total protein ratio in SBP among cirrhosis patients with ascites [15,16]. Calprotectin, a calcium-zinc-binding protein predominantly found in neutrophils, is proportionally present in body fluids alongside neutrophil influx [17]. Ascitic calprotectin levels correlate with polymorphonuclear lymphocytes, indicating its potential as a diagnostic marker for SBP. Additionally, fecal calprotectin levels, quantitatively associated with neutrophil migration to the intestine, serve as a marker of intestinal inflammation and a screening tool for SBP [18].

Diagnostic paracentesis should precede antibiotic administration [19]. Combining total ascitic fluid protein <1.5 g/dL with progressive liver failure (serum bilirubin level ≧3 mg/dL with a Child-Pugh score ≧9 points) or impaired renal function (serum creatinine level >1.2 mg/dL, blood urea nitrogen level >25 mg/dL, or serum sodium <130 mEq/L) increases the risk of SBP [20].

Other prevalent risk factors for the initial onset of SBP encompass acute variceal bleeding (with a 7.9% incidence of SBP even with antibiotic prophylaxis), acid-suppressive therapy, particularly proton pump inhibitors, as evidenced by three major meta-analyses, and asymptomatic bacteriuria [21, 22, 23]. Conversely, non-selective beta-blockers (such as propranolol) have shown a protective effect against initial SBP development [24].

The classical diagnosis of SBP relies on the count of polymorphonuclear leukocytes, a method that can be time-consuming. In light of this, the utilization of reagent strips, specifically for the colorimetric determination of leukocyte esterase activity, has been proposed as an alternative approach [25].

Regarding prognostic factors, a meta-analysis identified renal dysfunction as the primary prognostic indicator for cirrhosis patients with SBP, with a mortality rate of 67% compared to 11% in patients without renal dysfunction [26].

Nosocomial (49.5%) and staphylococcal infections (65.3%) were associated with higher mortality rates than community-acquired infections (23.8%) ($p = 0.0255$) and non-staphylococcal infections ($p < 0.001$) [27].

Although the overall mortality among patients with septic shock linked to SBP was 81.8%, a retrospective study illustrated that timely (<3 h) and appropriate antimicrobial therapy can substantially reduce mortality (OR = 0.54, $p = 0.02$) [28].

Considering SBP as the terminal clinical stage of liver cirrhosis, the annual mortality rate ranges from 53.9 to 78% [29]. In a comprehensive database study of cirrhosis patients, mortality rates at one, two, and three years after SBP-related hospitalization were 53.9%, 61.4%, and 66.5%, respectively [30].

The standard approach to treating SBP involves promptly administering broad-spectrum antibiotics along with albumin infusion, particularly in patients with impaired renal function [31].

If SBP is suspected, antibiotic therapy should be initiated immediately following analysis of ascitic fluid and culture results to minimize complications and mortality [32].

Bacterial resistance significantly increases the risk of mortality in SBP patients, with gram-positive cocci being the predominant bacteria in culture-positive SBP cases and various drug-resistant microorganisms emerging [34].

A prospective study suggested that the ineffectiveness of recommended empiric antibiotic regimens could negatively impact mortality [35]. Community-acquired and nosocomial SBP cases exhibit differences in the distribution of Enterobacter sp. and Enterococcus, with Enterococcus proportions notably higher in the nosocomial group [36].

Administering albumin (1.5 g/kg at diagnosis and 1 g/kg on day 3, maximum 100 g) is advisable for SBP patients, especially those with serum creatinine >1 mg/dL, blood urea nitrogen >30 mg/dL, or total bilirubin >4 mg/dL [37].

SBP patients treated with nonsteroidal beta-blockers, which affect systemic hemodynamics, may experience hepatorenal syndrome and reduced survival [38].

The use of mesenteric stromal cells shows promise for treating ascites infected with carbapenem-resistant bacteria [39].

Selective bacterial decontamination (SBD) is linked to decreased bacterial infection and mortality risk [40]. Antibiotic resistance to third-generation cephalosporins or quinolones can result in a poor prognosis and antibiotic treatment failure [41]. Antibiotic prophylaxis primarily functions by decontaminating the gut, thereby reducing the bacterial reservoir available for translocation [42].

Performing diagnostic paracentesis within <12 hours of hospitalization in cirrhosis patients with ascites may enhance short-term survival [43].

Three categories of high-risk patients who may derive benefits from SBP have been identified: those experiencing acute upper gastrointestinal bleeding, individuals with low total protein in ascites linked to advanced stages of liver failure or renal dysfunction and no prior SBP (primary prevention), and patients with a history of SBP (secondary prevention) [44].

A recent meta-analysis indicates significant uncertainty regarding the effectiveness of antibiotic prophylaxis and the optimal choice of antibiotics in patients with cirrhosis and ascites [45].

Acute gastrointestinal bleeding heightens the risk of SBP and other infections during or after the bleeding episode (within the first 7 days), with frequencies ranging from 16% (in compensated cirrhosis) to 66% (in decompensated cirrhosis) [46, 47]. Consequently, infections increase the chances of uncontrolled bleeding, rebleeding, and in-hospital mortality [48].

Selective bacterial decontamination (SBD) diminishes mortality risk and SBP-related infections. SBD should commence promptly in cases of gastrointestinal bleeding, with intravenous ceftriaxone 1 g/24 h being the preferred clinical antibiotic according to the recent Baveno VII consensus [49].

Clinical management of SBP in critically ill cirrhosis patients entails evacuating ascites fluid through therapeutic paracentesis, enhancing abdominal wall compliance, optimizing fluid administration, and optimizing systemic and regional perfusion [50].

The pathophysiological considerations surrounding intra-abdominal hypertension in individuals with decompensated liver cirrhosis and ascites suggest the safety and immediate beneficial impact of extensive therapeutic paracentesis. This intervention affects not only hemodynamic status but also kidney, respiratory, and liver function [51, 52].

Therefore, a multidisciplinary approach is essential for the diagnosis and treatment of SBP, upon which the timeliness and success of medical care provision depend.

CONCLUSIONS / ВИСНОВКИ

SBP represents a significant complication in patients with portal hypertension complicated by ascites. To mitigate mortality and morbidity in this patient cohort, clinical vigilance, swift diagnosis to rule out secondary bacterial peritonitis, and immediate treatment are imperative. Additionally, preventing SBP recurrence through antibiotic therapy while awaiting liver transplantation emerges as a critical clinical concern.

CONFLICT OF INTEREST / КОНФЛІКТ ІНТЕРЕСІВ

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS / ВКЛАД АВТОРІВ

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INFORMATION ABOUT THE AUTHORS / ВІДОМОСТІ ПРО АВТОРІВ

Тутченко Микола Іванович – доктор медичних наук, професор, завідувач кафедри хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, бульвар Тараса Шевченка, 13, Київ, 01601, Україна, https://orcid.org/0000-0002-5050-6494, e-mail: tutchenko@ukr.net, 0677883572
Рудик Діана Віталіївна – кандидат медичних наук, доцент кафедри хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, бульвар Тараса Шевченка, 13, Київ, 01601, Україна, https://orcid.org/0000-0001-9736-3614, e-mail: Dianavrudyk@ukr.net,
Беседінський Микола Сергійович - асистент кафедри хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, бульвар Тараса Шевченка, 13, Київ, 01601, Україна, https://orcid.org/0009-0009-8618-1458, e-mail: mr.besedinsky@gmail.com, 0672820892
Чуб Сергій Леонідович – аспірант кафедри хірургії стоматологічного факультету Національного медичного університету імені О.О.Богомольця, бульвар Тараса Шевченка, 13, Київ, 01601, Україна, https://orcid.org/0000-0001-6489-7345, e-mail: chub.mil.doc@gmail.com, 0930766868

Mykola Tutchenko, MD, professor, Head of Department of Dentist Surgery, Bogomolets National Medical University, Taras Shevchenko boulevard, 13, Kyiv, 01601, Ukraine; https://orcid.org/0000-0002-5050-6494, e-mail: tutchenko@ukr.net, 0677883572
Diana Rudyk, PhD, assistant professor, Department of Dentist Surgery, National Medical University, Taras Shevchenko boulevard, 13, Kyiv, 01601, Ukraine; https://orcid.org/0000-0001-9736-3614, e-mail: Dianavrudyk@ukr.net, 0638591178
Mykola Besedinskyi, assistant, Department of Dentist Surgery, Bogomolets National Medical University, Taras Shevchenko boulevard, 13, Kyiv, 01601, Ukraine; https://orcid.org/0009-0009-8618-1458, e-mail: mr.besedinsky@gmail.com, 0672820892
Sergey Chub, post graduate student, Department of Dentist Surgery Bogomolets National Medical University, Taras Shevchenko boulevard, 13, Kyiv, 01601, Ukraine; https://orcid.org/0000-0001-6489-7345, e-mail: chub.mil.doc@gmail.com, 0930766868