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Spontaneous Bacterial Peritonitis in Cardiac Ascites: A Rare but Deadly Occurrence

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 85
Final Diagnosis: Spontaneous bacterial peritonitis
Symptoms: Abdomen distension • confusion • lethargy
Medication: —
Clinical Procedure: Paracentesis
Specialty: Gastroenterology and Hepatology

Objective: Unusual clinical course

Background: Spontaneous bacterial peritonitis is frequently described in cirrhotic patients who develop infected ascitic fluid. However, ascites can be cardiac in origin. The phenomenon of spontaneous bacterial peritonitis in cardiac ascites is an extremely rare but deadly occurrence.

Case Report: Here we present a unique case of a patient who was admitted for advanced cardiorenal syndrome in the setting of a viral colitis that likely promoted a bacterial translocation resulting in spontaneous bacterial peritonitis.

Conclusions: This case tends to shed light on a few quintessential points for clinicians to be aware of, including the potential intersection between the microbiota and metabolic effects of congestive heart failure and the necessity to lower the diagnostic threshold for spontaneous bacterial peritonitis cardiac ascites in patient's presenting for a congestive heart failure exacerbation.

MeSH Keywords: Ascites • Bacterial Translocation • Heart Failure • Microbiota

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/915944

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Background

Ascites, or the accumulation of fluid in the peritoneal cavity, is one of the most common complications of cirrhosis in the western part of the world [1]. Although less common, other etiologies include malignancy, heart failure, renal disease, and infectious causes [1]. Spontaneous bacterial peritonitis can develop when the ascitic fluid becomes infected in a patient with portal hypertension. Spontaneous bacterial peritonitis frequently arises in the background of ascites secondary to hepatic cirrhosis. In 1984, the first reported case of spontaneous bacterial peritonitis in association with cardiac ascites was published [2]. Since then, only 2 other case reports, from 1987 and 1986, have documented spontaneous bacterial peritonitis with cardiac ascites in hospitalized patients [3,4]. Here we present a unique case of spontaneous bacterial peritonitis in an elderly gentleman admitted for congestive heart failure in the setting of a viral colitis that likely promoted a bacterial translocation resulting in spontaneous bacterial peritonitis.

Case Report

An 85-year-old male presented, lethargic and confused, from a skilled nursing facility in which he was "found down", covered in non-bloody diarrhea. On arrival to the hospital, the patient was unable to recall events but as reported by the nursing staff at the nursing facility, he had been complaining of abdominal bloating. Th patient's medical history was significant for congestive heart failure, atrial fibrillation, hypertension, diabetes, and chronic kidney disease. There was no history of liver disease or alcohol or drug use.

On physical examination, he was found to be awake and oriented to self and place. A notable examination finding was a soft, non-tender, mildly distended abdomen, asterixis and 2+ lower extremity pitting edema. The patient was admitted with the preliminary diagnoses of a congestive heart failure exacerbation, metabolic encephalopathy, and an acute kidney injury. Due to concern for sepsis, he was empirically started on vancomycin and cefepime.

Initial laboratory tests revealed a brain natriuretic peptide (BNP) level of 3915 pg/mL, white blood cell count of 11 500/mm^3, hemoglobin of 11.5 g/dL, platelet count of 90×10^3/mm^3, serum bicarbonate of 25 mEq/L, blood urea nitrogen (BUN) of 89 mg/dL, creatinine of 3.51 mg/dL, total bilirubin of 2.3 mg/dL, direct bilirubin of 1.4 mg/dL, aspartate aminotransferase of 22 U/L, alanine aminotransferase of 9 U/L, and an international normalized ratio of 1.27. His initial cultures, urinalysis, and stool studies were negative for infection. A transthoracic echocardiogram was performed and was notable for an ejection fraction of 35–40% with global hypokinesis, a dilated inferior vena cava, and an elevated pulmonary artery (PA) pressure of 67 mmHg. A follow-up abdominal computed tomography (CT) scan showed a small right pleural effusion and a moderate amount of free intraperitoneal fluid.

Given these imaging findings, in conjunction with his symptoms of confusion, asterixis, and abdominal distension, a diagnostic paracentesis was performed. The aspirated fluid was dark yellow and opaque with a serum ascites albumin gradient (SAAG) calculated to be 1.9 g/dL, total fluid protein of 3.3 g/dL, polymorphonuclear (PMNs) of 1115 cells/mm^3, 49% PMN leukocytes, 2% lymphocytes, and 40% mononuclear leukocytes. With a SAAG of 1.9 g/dL and a total fluid protein count of 3.3 g/dL, we concluded that the peritoneal fluid represented cardiac ascites as there was no evidence of cirrhosis. Blood cultures taken were negative, which may reflect initiation of sepsis protocol on patient's admission reducing the yield of the culture. Diagnosis was made on the basis of previously reported cell counts of the fluid. Bacterial peritonitis was presumed due to a very high neutrophil count. The patient was subsequently treated with ceftriaxone 2 mg daily for 5 days and albumin (on day 1 and 3) with improvement in his encephalopathy.

Unfortunately, the patient exhibited a rapid acute and then chronic decline in his renal function. The patient and family declined hemodialysis resulting in further failure of his congestive heart failure treatment. Shortly afterwards, he became oliguric and expired from renal failure.

Discussion

Cardiac ascites resulting in spontaneous bacterial peritonitis is a rare, but deadly complication that warrants further investigation [2–4]. Due to the liver's extensive vascular supply, it is prone to hemodynamic disturbances which can result in passive hepatic congestion from right sided heart failure [5]. Recent studies have supported the diagnostic utility of serum BNP levels to distinguish ascites from cirrhosis or heart failure, in which BNP levels greater than 364 pg/mL had a 98% sensitivity in detecting congestive heart failure related ascites [5].

It has been generally well accepted that the low occurrence of spontaneous bacterial peritonitis in cardiac ascites may be related to the protein rich ascitic fluid that has both opsonic and bactericidal activity [6]. Interestingly, recent research exploring the relationship between the microbiome and heart failure has postulated that hemodynamic changes may be contributing to increased risks of spontaneous bacterial peritonitis [7]. The "gut hypothesis" suggests that intestinal hypoperfusion and congestion can lead to altered gut morphology, permeability, and the growth of microbiota that may ultimately stimulate bacterial translocation and endotoxin release [7,8]. Furthermore,

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the production of blood metabolites, such as indoxyl sulfate, has been shown to be a pro-inflammatory mediator that increases the risk of mortality in patients with chronic kidney disease [7]. As in our case, the patient had a rapid decline in renal function which could possibly be attributed to changes in his gut flora, metabolites, and other uremic toxins from his acute episode of congestive heart failure [7].

A study by Krack et al. further investigated the effects of intestinal hypoperfusion by measuring intragastric pCO2 and demonstrated that chronic endotoxin release from hypoperfused intestinal mucosa likely contributes to the progression of bacterial translocation in congestive heart failure patients [8]. This case tends to shed light on this new and emerging area of exploration. It is possible that our patient's diarrhea was initially due to a viral gastroenteritis that could have possible induced endotoxin release and gastrointestinal barrier changes in the setting of a congestive heart failure exacerbation.

Conclusions

Cardiac ascites is unusual, compromising only 5% of ascites patients [2] and has a high mortality rate [2,9]. This diagnosis could be easily overlooked or missed, especially in patients without history of liver disease or cirrhosis such as our patient. Since cardiac ascites may mimic cirrhotic ascites, it is essential to maintain high clinical suspicion and obtain appropriate ascitic fluid analysis to differentiate between the 2 conditions [2]. To our knowledge this is the fifth reported case of spontaneous bacterial peritonitis in cardiac ascites that was likely triggered by a viral colitis. Moving forward, we should reinforce the necessity of understanding the potential intersection between the microbiota and metabolic effects of congestive heart failure and lowering the diagnostic threshold for spontaneous bacterial peritonitis cardiac ascites in patients presenting with a congestive heart failure exacerbation.

Conflicts of interest

None.

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