

Contents lists available at ScienceDirect

IDCases

journal homepage: www.elsevier.com/locate/idcr



Case report

Infected ascites: Distinguishing secondary peritonitis from spontaneous bacterial peritonitis in a cirrhotic patient with classic symptoms



Marvin Louis Roy Lu*, Akanksha Agarwal, Josh Sloan, Aaron Kosmin

Albert Einstein Healthcare Network, United States

ARTICLE INFO

Article history: Received 27 January 2017 Received in revised form 10 February 2017 Accepted 25 February 2017

Keywords: Secondary bacterial peritonitis Cirrhosis Runyon's criteria

ABSTRACT

Introduction: Spontaneous bacterial peritonitis can be differentiated from secondary bacterial peritonitis by the absence of a surgically treatable intra-abdominal source of infection. However, oftentimes this is unapparent and other clinical clues need to be sought after to make the right diagnosis.

Case: A 64-year-old woman was admitted because of three days of worsening diffuse abdominal pain and distention. She was morbidly obese and had a history of non-alcoholic steatohepatitis (NASH) cirrhosis. She was febrile at 38.2 °C. Her abdomen was soft, diffusely tender and distended with a reducible umbilical hernia. Laboratory exam showed a white blood cell count 6700/mcl. Ascitic fluid analysis showed a yellow cloudy fluid with an absolute neutrophil count (ANC) of 720 cells/m³, a total protein of 1.1 g/dl and a lactate dehydrogenase of 242 IU\l. She was given ceftriaxone and albumin. The ascitic fluid culture grew pansensitive Viridans streptococcus. The following days she continued to have fever and abdominal pain and a repeat paracentesis was done which showed improvement in her ANC. Abdominal computed tomography scan was done which showed hernia inflammation with a rim-enhancing fluid collection. Surgery was consulted who did a primary repair of the umbilical hernia and over the next few days the patient improved and was discharged stable.

Conclusion: Persistence of signs and symptoms of peritonitis despite improvement in ascitic fluid analysis in cirrhotic patients treated for or early relapse of peritonitis with the same organism should prompt the physician to evaluate for secondary peritonitis and surgical management should be considered for potentially correctable sources.

© 2017 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Introduction

Spontaneous bacterial peritonitis (SBP) is a severe complication of cirrhotic patients with ascites. SBP is an acute ascites infection an ascitic fluid polymorphonuclear (PMN) cell count of ≥250 cells/mm³ both with or without a positive ascitic fluid bacterial culture. SBP can be differentiated from secondary bacterial peritonitis by the absence of a surgically treatable intra-abdominal source of infection. Other clues to differentiating these two entities include detecting polymicrobial infection by gram staining or culture and application of the Runyon's criteria [1,2], elevated blood markers of gut perforation such as elevated carcinoembryonic antigen (CEA) [3], imaging studies, and response to treatment. We present a case

E-mail address: lumarvin@einstein.edu (M.L.R. Lu).

of a patient with end stage liver disease with recurrent monomicrobial peritonitis that was successfully treated only after surgical repair of a presumed infected umbilical hernia.

Case

A 64-year-old woman was admitted because of three days of worsening abdominal pain and generalized weakness. She was morbidly obese and had a history of cirrhosis secondary to non-alcoholic steatohepatitis (NASH) complicated by refractory ascites and esophageal varices. She had a history of transjugular intrahepatic portosystemic shunt (TIPS) placement a few months before this admission and she is currently on spironolactone and furosemide. A month prior to this admission, she had been diagnosed with spontaneous bacterial peritonitis (SBP) at another hospital received ciprofloxacin for four days, which was later adjusted to cephalexin for 10 days because viridans streptococci was identified from the ascites fluid culture.

^{*} Corresponding author at: Albert Einstein Medical Center, 5501 Old York Road (Klein 403), Philadelphia, PA, United States.

She described her abdominal pain as diffuse, crampy and associated with abdominal distention and nausea unrelated to food intake. She took oxycodone which only offered temporary relief. She had no changes in her bowel movements. Other review of systems was unremarkable. She denied any alcohol or illicit drug use. On physical exam, her blood pressure was 135/58 mm Hg, heart rate of 110 beats/minute, respiratory rate of 20 breaths/minute, and temperature 38.2 °C. She was alert and oriented to time place and person and in no acute distress. She had anicteric sclera and no lymphadenopathy. Cardiopulmonary exam was unremarkable. Her abdomen was soft, distended with a positive fluid wave and a reducible umbilical hernia. It was diffusely tender and had normal bowel sounds. She had pitting edema on both her lower extremities and skin exam disclosed multiple spider angiomas in her chest and palmar erythema.

Laboratory exam showed a sodium of 125 mmol/L, creatinine 0.9 mg/dl, total bilirubin of 2.7 mg/dl, International Normalized Ratio (INR) 1.6, white blood cell (WBC) count 6700/mcl. Blood and urine cultures were done and a diagnostic paracentesis was performed. Ascites analysis was consistent with portal hypertension with spontaneous bacterial peritonitis (see Table 1). The patient was started on intravenous ceftriaxone 2g daily and albumin. The following day the patient still had persistent abdominal pain and developed a fever of 38.6°C and was tachycardic at 112 beats/min. Blood cultures has remained negative and ascitic fluid culture grew viridans group Streptococcus. again. On day 3 she was still febrile and a repeat paracentesis was done showing improvement of her absolute neutrophil count. Susceptibility studies showed that it was sensitive to ceftriaxone and so the antibiotics were continued. The following days she continued to develop fevers and her abdominal pain became more severe. Imaging studies were done to rule out secondary peritonitis. We escalated her antibiotics by adding metronidazole for anaerobic coverage. A repeat paracentesis was done which showed continued improvement of her absolute neutrophil count and antibiotics were de-escalated to ampicillin-sulbactam. She again developed more fevers, chills and worsening abdominal pain despite improvement in general fluid parameters. A computed tomography (CT) scan of the abdomen and pelvis with intravenous contrast was done which showed moderate inflammatory stranding in the surrounding subcutaneous fat of the umbilical hernia with an enhancing-rim fluid collection within the hernia. At this point we were able to obtain her previous abdominal CT scan from an outside hospital which showed the same umbilical hernia but with no inflammatory stranding. All blood cultures at this point has remained negative for bacterial growth. A 2D echocardiogram was also done which did not show any valvular vegetations.

We consulted surgery and they performed a primary repair of the umbilical hernia and the patient was given perioperative antibiotics vancomycin and piperacillin-tazobactam. The large hernia sac was drained, varicosities within the sac were ligated and the remaining defect was repaired. No bowel was found within the sac. The patient tolerated the procedure well with no post procedure complications. Over the next several days she no longer had fevers and her abdominal pain gradually improved. Repeat paracentesis was negative for bacterial growth and the patient was discharged improved on amoxicillin-clavulanate. She was seen one week later at the hepatology clinic and was doing well.

Discussion

Bacterial peritonitis is a common complication of liver cirrhosis. Its prevalence was 12% in 2001 [4] but is expected to be lower now with the widespread use of quinolones as SBP prophylaxis in highrisk patients. Less than 5% of bacterial peritonitis is due to an intraabdominal surgically treatable cause (secondary peritonitis) [2]. Signs and symptoms of spontaneous and secondary bacterial peritonitis are similar and do not help separate these patients [1]. Fever remains the predominant symptom occurring in 69% of these patients [5]. Secondary peritonitis can be divided into 2 categories: (a) Perforated viscus and (b) walled abscesses without perforation. There are several characteristics that distinguishes SBP from secondary perforation (Table 2).

Patients who have characteristics of secondary peritonitis with gut perforation should undergo emergent plain and upright abdominal radiographs, water-soluble contrast studies of the gut, and computed tomographic scanning [1,3]. Patients in whom secondary peritonitis with or without perforation is a likely diagnosis should receive anaerobic coverage in addition to a 3rd generation cephalosphorin. Surgery is indicated once a source is identified on imaging.

Our patient did meet Runyon's criteria. However, since it was a monobacterial infection, the initial treatment plan was to treat it as a spontaneous bacterial infection with intravenous ceftriaxone and albumin. A repeat paracentesis did show a decrease in her absolute PMN count but she continued to have signs of infection and so we treated it as a poor response to treatment and escalated the antibiotics. With imaging demonstrating a potentially infected hernia sac as a possible source of infection, and the initial ascites culture yielding the same isolate a month apart, our surgical team proceded with a primary repair and removal of the sac. Although the appearance of the fluid in the sac was serosanguinous and culture yielded negative results, the patient had been deteriorating on anitibotics and promptly and significantly improved postoperatively.

This was a case of a secondary peritonitis without perforation that was treated successfully with antibiotics and surgical intervention. Although the case did have certain characteristics of SBP, such as monomicrobial infection and improvement of absolute neutrophil count after 48 h of antibiotics, persistence of patient symptomatology prompted further evaluation to look for an alternate diagnosis. Runyon's criteria have an estimated sensitivity and specificity for predicting secondary bacterial peritonitis of 67 and 96%, respectively and our patient met the

Table 1 Ascitic fluid analysis.

	Day 1	Day 3	Day 7
Color	Yellow	Yellow	Yellow
Appearance	Cloudy	Cloudy	Slightly cloudy
Serum albumin to ascites gradient (SAAG) (g/dl)	2.1		
Absolute Neutrophil Count (ANC) (cells/m ³)	720	171	71
WBC (cells/m ³)	819	356	336
% PMN	88	48	21
% Lymphocytes	5	24	48
% Monocyte/Macrophage	7	28	29
Culture	viridians streptococci	Negative for bacterial growth	Negative for bacterial growth

Table 2

Characteristics of Secondary Peritonitis.

Ascitic Fluid Analysis

- Absolute PMN count >250 cells/mm³
- Multiple organisms on gram stain and culture
- *Runyon's Criteria (at least 2 of the following findings) (1)
- o Total protein >1 g/dL
- Glucose <50 mg/dL (2.8 mmol/L)
- LDH greater than the upper limit of normal for serum
- Carcinoembryonic antigen >5 ng/mL or alkaline phosphatase >240 units/L (3)

Poor response to treatment

- Repeat paracentesis after 48 h with: (a) absolute PMN count ≥ pre-treatment value; (b) persistence of bacteria on culture (1)
- Persistence of fever and signs of peritonitis

Abnormal structural findings on imaging procedure

- ⁴ 100% sensitivity and 45% specificity for detecting perforation; 50% sensitivity.
- € 92% sensitivity; 88% specificity.

criteria. That together with the lack of improvement prompted us to probe further. A repeat paracentesis is not necessary for all patients with infected ascites but should be considered in patients with one or more characteristics of secondary peritonitis as detailed above. Re-categorizing this case from SBP to secondary peritonitis allowed us to advocate for a likely curative surgical intervention.

Conclusion

Persistence of signs and symptoms of peritonitis despite improvement in ascitic fluid analysis in cirrhotic patients treated for or early relapse of peritonitis with the same organism should prompt the physician to evaluate for secondary peritonitis and surgical management should be considered for potentially correctable sources.

References

- [1] Akriviadis EA, Runyon BA. Utility of an algorithm in differentiating spontaneous from secondary bacterial peritonitis. Gastroenterology 1990;98:127.
- [2] Soriano G, Castellote J, Alvarez C, Girbau A, Gordillo J, Baliellas C, et al. Secondary bacterial peritonitis in cirrhosis: a retrospective study of clinical and analytical characteristics, diagnosis and management. J Hepatol 2010;52:39.
- [3] Wu SS, Lin OS, Chen YY, et al. Ascitic fluid carcinoembryonic antigen and alkaline phosphatase levels for the differentiation of primary from secondary bacterial peritonitis with intestinal perforation. J Hepatol 2001;34:215.
- [4] Borzio M, Salerno F, Piantoni L, Cazzaniga M, Angeli P, Bissoli F, et al. Bacterial infection in patients with advanced cirrhosis: a multicentre prospective study. Dig Liver Dis 2001;33:41–8.
- [5] McHutchison JG, Runyon BA. Spontaneous bacterial peritonitis. In: Surawicz CM, Owen RL, editors. Gastrointestinal and Hepatic Infections. Philadelphia: WB Saunders Company; 1994 p. 455.