Title

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Authors

Lo, Jessica
Yuchno, David
Joo, Esther

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CLINICAL VIGNETTE

Spontaneous Bacterial Peritonitis Caused by Neisseria sicca: A Case Report and Literature Review

Jessica Lo, MD, David Yuchno, MD and Esther Joo, MD

Background

The genus *Neisseria* encompasses the well-recognized pathogens *N. meningitidis* and *N. gonorrhoeae* as well as a number of commensal species found in the human nasal and oral pharynx. These organisms include *N. sicca*, a gram-negative diplococcus first described by von Lingelsheim in 1906.[1] Though often regarded as non-pathogenic, *N. sicca* has been implicated as a causative agent in a range of infections including: endocarditis, pneumonia, meningitis, arthritis, bursitis, osteomyelitis, discitis, conjunctivitis, sinusitis and abscesses of the liver and Bartholin's gland.[2-13] In addition, there are three documented cases of peritoneal dialysis (PD)-associated peritonitis attributed to infection with *N. sicca*.[14-16]

We report a case of *N. sicca* spontaneous bacterial peritonitis (SBP) occurring in a non-cirrhotic patient with protein-losing enteropathy and multiple splanchnic vein thromboses previously treated with a transjugular intrahepatic portosystemic shunt (TIPS).

Case Report

The patient is a 44-year-old male with history of non-cirrhotic portal hypertension with ascites, hypoalbuminemia, protein-losing enteropathy of unknown etiology and splanchnic venous thromboses. Prior TIPS placement required multiple revisions for recurrent thrombosis despite anticoagulation therapy. He initially presented to the hospital for anasarca and worsening ascites. He denied recent fevers, nausea or vomiting, but reported mild, chronic abdominal pain due to abdominal distention. Pertinent home medications included bumetanide, spironolactone and warfarin. Social history was negative for alcohol or other substance use.

The patient was admitted to the hospital for titration of diuretics. On admission, he was afebrile and other vital signs were within normal limits. His physical exam was notable for soft but distended abdomen with mild lower abdominal tenderness to palpation and significant pitting edema in the lower extremities. His initial labs included: white blood cell (WBC) count 4.4 K/cumm, platelet count 183 K/cumm, sodium 127 mmol/L, creatinine 0.89 mg/dL, albumin <1 g/dL, total protein 4.4 g/dL, alkaline phosphatase (ALP) 462 U/L, aspartate transaminase (AST) 59 U/L, alanine transaminase (ALT) 47 U/L and total bilirubin 0.6 mg/dL. A paracentesis was performed on admission with removal of 4 liters of ascites fluid. Ascites fluid analyses showed WBC count 27/cumm with a differential of 8% neutrophils. Gram stain and culture of the ascitic fluid remained negative through the hospitalization. Blood cultures obtained on admission also remained negative for the duration of his stay. A contrast-enhanced computerized tomographic (CT) scan of the abdomen showed large volume of ascites and non-opacified TIPS, similar to a CT scan from 2 months prior.

On hospital day 7, while undergoing diuresis and awaiting interventional radiology evaluation of his occluded TIPS, the patient developed worsening abdominal pain. Repeat paracentesis and ascites fluid analysis revealed WBC count 1770/cumm with 92% neutrophils and intracellular cocci on cytology. The patient was assigned a diagnosis of SBP and started intravenous (IV) ceftriaxone 2 gram every 24 hours. Culture of the ascites fluid later grew *N. sicca*. Antibiotic susceptibility testing was not performed per lab protocol. He completed a 7-day course of IV ceftriaxone in the hospital and was discharged after symptoms improved, with plan for follow-up at a quaternary center for continued evaluation of his TIPS and other chronic conditions.

Discussion

SBP, also referred to as primary peritonitis, is defined as a peritoneal infection without a direct relationship to other intra-abdominal abnormalities. In adult patients, SBP is typically observed in the setting of cirrhosis with ascites, though it has also been identified in cases of acute and chronic viral hepatitis, congestive heart failure, metastatic malignancy, systemic lupus erythematosus and in patients without underlying disease.[17]

SBP is presumed to result from the translocation of bacteria from the gut lumen into the systemic circulation and ascitic fluid. As such, enteric organisms are the most frequently encountered pathogens, accounting for 69% of cases.[17] In order of prevalence, these include: *Escherichia coli*, *Klebsiella* species., *Proteus* species, *Enterococcus faecalis* and *Pseudomonas* species.[18]

Neisseria species have infrequently been identified as pathogens in cases of peritonitis, including SBP. Three reports of SBP due to *N. gonorrhoeae* have been documented. These cases

were all observed in women and were associated with a pre-existing diagnosis of cirrhosis in two of three instances.[19] *N. meningitidis* has also been implicated in cases of peritonitis. In 2004, Kelly and Robertson reported a total of 18 recorded cases of *N. meningitidis* peritonitis. These included two cases of PD-associated peritonitis and six cases occurring in patients with cirrhosis and ascites.[20] Following this report, two additional cases of SBP due to *N. meningitidis* have been published, one in a previously healthy 27-year-old male and the other in a 72 year-old woman with known cirrhosis.[21,22]

In addition to *N. gonorrhoeae* and *N. meningitidis*, commensal *Neisseria* species including *N. cinerea*, *N. elongata*, *N. macacae*, *N. mucosa* and *N. subflava* have been implicated in cases of peritonitis.[23-27] These organisms, however, have been identified as pathogens exclusively in the setting of PD-associated peritonitis. Prior to this report, only one instance of SBP due to a commensal *Neisseria* species has been documented. This occurred in a 66-year-old female with cirrhosis. *N. perflava* was isolated from blood and ascites fluid culture.[27]

N. sicca has been implicated in infections involving a number of organ systems including three cases of PD-associated peritonitis. The first of these cases was observed in a 5-year-old boy previously treated for *Staphylococcus aureus* peritonitis, and the second occurred in a 46-year-old man without a prior history of peritonitis.[14-15] Both of these patients were receiving immunosuppressive agents following renal transplant with subsequent graft failure. By contrast, Zhang, et al. have recently reported a case of *N. sicca* PD-associated peritonitis occurring in an immunocompetent 36-year-old man with a history notable only for diabetes and hypertension.[16]

Table 1 summarizes the known cases of peritonitis attributed to commensal *Neisseria* species.

To our knowledge, this report represents the first documented instance of SBP associated with *N. sicca* as well as the second case of SBP due to a commensal *Neisseria* species. As blood cultures remained negative throughout his hospital course, it is unclear how this patient's ascites fluid was inoculated with *N. sicca*. However, the diagnosis of SBP is strongly supported by the development of abdominal pain during the patient's hospital course, the presence of a neutrophil predominant pleocytosis in his ascites fluid and the absence of other intra-abdominal pathology accounting for his peritonitis on CT imaging. The identification of *N. sicca* as the causative organism in this case is supported by its isolation from ascites fluid culture and further corroborated by the observation of intracellular cocci on cytology.

Conclusions

N. sicca is one of several commensal *Neisseria* species found in the nasal and oral pharynx.[1] While typically regarded as non-pathogenic, *N. sicca* has been implicated as the causative organism in a range of infections, including PD-associated peritonitis.[14,15] Following its isolation from this patient's ascites fluid, *N. sicca* may now be regarded as a potential pathogen in cases of SBP.

Table 1. Cases of peritonitis caused by commensal species of *Neisseria*

Reference	**Patient information**	**Comorbidities**	**Organism**	**SBP, secondary, or PD-associated**
Haqqie [28] George*[29]	31 yo M	ESRD, lupus nephritis	N. cinerea	PD
Taegtmeyer [23]	38 yo M	ESRD, insulin-dependent diabetes, ischemic heart disease	N. cinerea	PD
Garcha [30]	37 yo F	ESRD, tuberous sclerosis, hypothyroidism, prior episodes of PD-associated peritonitis with *Staphylococcus spp*	N. cinerea	PD
Lin [31]	60 yo M	ESRD, hypertension	N. elongata	PD
Alsayed [24]	29 yo M	ESRD, chronic hepatitis C	N. elongata	PD
Iyama [25]	56 yo F	ESRD	N. macacae	PD
Macia [32]	30 yo F	ESRD, 2 failed renal transplants	N. mucosa	PD
Shetty [33]	17 yo M	ESRD	N. mucosa	PD
Awdisho [34]	28 yo M	ESRD, failed renal transplant, hypertension	N. mucosa	PD

Khan [35]	30 yo F	ESRD, migrated IUD	N. mucosa	PD
Ren [26]	55 yo F	ESRD, hypertension	N. mucosa	PD
McCue [27]	66 yo F	Postnecrotic cirrhosis	N. perflava	SBP
Vermeij [36]	45 yo F	ESRD	N. perflava	PD
Chen [37]	74 yo M	ESRD	N. subflava	PD
Neu [15]	5 yo M	ESRD, failed renal transplant (on low-dose prednisone only), recently treated *S. aureus* PD-associated peritonitis	N. sicca	PD
Konner [14]	46 yo M	ESRD, failed renal transplant (on low-dose cyclosporine and prednisone)	N. sicca	PD
Zhang [16]	36 yo M	ESRD, diabetes, hypertension	N. sicca	PD
Our case	**44 yo M**	**Non-cirrhotic portal hypertension, protein-losing enteropathy, splanchnic venous thromboses**	**N. sicca**	**SBP**

*same patient; determined to be re-infection with a different strain rather than relapse of infection with original strain

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