Clostridium sordellii: A cause of toxic shock syndrome after a breach in the GI tract

Sydney Pence[1], Rajshri Joshi[2], and Kasturi Shrestha[2]

[1]Ohio University
[2]Cleveland Clinic Akron General

March 17, 2023

Clostridium sordellii: **A cause of toxic shock syndrome after a breach in the GI tract**

Sydney Pence,[1] Rajshri Joshi,[2]Kasturi Shrestha[3]

1. Ohio University Heritage College of Osteopathic Medicine, 4180 Warrensville Center Rd, Warrensville Heights, OH 44122. Email: sp883713@ohio.edu

2. Internal Medicine, Cleveland Clinic Akron General, 1 Akron General Ave, Akron, OH 44307. Email: JOSHIR4@ccf.org

3. Infectious Disease, Cleveland Clinic Akron General, 1 Akron General Ave, Akron, OH 44307. Email: SHRESTK2@ccf.org

Corresponding Author Information:

Rajshri Joshi: 1 Akron General Ave, Ambulatory Care Center, Bldg. 301, Fl5, Akron Ohio 44307

Tel: 330-344-6015

Fax: 330-344-6820

Email: *joshir4@ccf.org*

There were no sources of financial support for this case report. Patient consent was obtained. We have no conflicts of interest to report, or acknowledgements to declare.

ABSTRACT

When found in humans, *Clostridium sordellii* is most often associated with infections of the uterus and perineum. This organism is highly virulent, causing tissue necrosis, leukemoid reactions, and death. This report describes an interesting case of *Clostridium sordellii* causing toxic shock syndrome post translocation through the GI tract.

KEYWORDS: Clostridium sordelli ; infectious disease medicine; shock, septic; leukemoid reaction

INTRODUCTION

Clostridium sordellii, first identified in 1922 by Alfredo Sordelli, is a beta hemolytic anaerobic gram-positive spore forming rod[1,2]. It is typically found in soil and the gut of many animals, including humans [1]. When found pathologically in humans, *C. sordellii* is almost exclusively reported with infections of the uterus and perineum; however, there have been rare cases of infection in other locations of the body reported

post-operatively [1,3] or with intravenous drug use[4,5]. In most of the cases of *Clostridial*bacteremia, patients were predisposed to infection because of their compromised immune system or underlying malignancy [6]. Suppressed immune system in many has also been a cause of delayed presentation of signs of infection thus making the organism invariably fatal.

Unfortunately, *C. sordelli* is highly virulent, causing death in nearly 70% of cases [1,7]. Its virulence is achieved with exotoxins, primarily the lethal and hemorrhagic toxins[1]. Infection with *C. sordelli* typically causes an acute onset leukemoid reaction accompanied by hypotension and tachycardia. Some reports have demonstrated this pathogen to cause a capillary leak syndrome, leading to hemoconcentration[1]. Even more severely, there have been reports of*C. sordelli* , almost exclusively involving the uterus or perineum, causing toxic shock syndrome [3,8].

Little guidance exists regarding the treatment of *C. sordelli;*although, some studies suggest that the infection is responsive to beta-lactams, clindamycin, tetracycline, and chloramphenicol[9].

This report presents a recent case that highlights the diagnosis and treatment of *Clostridium sordellii* causing toxic shock syndrome in the setting of a hemorrhagic necrotic renal mass and its fistulization with the adjacent splenic flexure of the colon. This is the first report of its kind.

CASE REPORT

A 69-year-old man presented to the emergency room with back pain and uncontrollable shaking. His past medical history was significant for metastatic renal cell carcinoma, a penicillin allergy, and surgical history of small bowel obstruction two weeks after undergoing transverse colostomy and one week post-renal biopsy. On physical exam, the patient was confused and found tachycardic, tachypneic, hypotensive, and febrile. Pertinent labs included lactate of 8mmol/L (0.5-2.2 mmol/L), initial hemoglobin of 10g/dL that fell to 7.7g/dl (11.5 - 15.5 g/dL), and a WBC count of 16,000/mL (4,500-11,000/mL). CT scan showed a left necrotic, hemorrhagic renal mass invading the adjacent splenic flexure of the colon. Invasion of the pancreatic tail and left adrenal gland with distal splenic vessels coursing through the mass could also not be excluded (Figure 1). The patient received one dose of aztreonam, vancomycin, metronidazole, and levofloxacin in the emergency department. Piperacillin/Tazobactam was not used given the patient's history of penicillin allergy.

After the patient was admitted to the intensive care unit for septic shock, he was treated with an antibiotic regimen of vancomycin, cefepime, and metronidazole. On day 2, the patient's WBC count increased to 49,000/mL (4,500-11,000/mL) and his procalcitonin was elevated to >100ng/mL (<0.25 ng/ml). Blood cultures identified *Clostridium sordellii* as the causative organism, and vancomycin was switched for clindamycin while meropenem was continued.

Over the next week, subsequent blood cultures showed no growth and the patient's clinical status stabilized. The antibiotic regimen was switched from meropenem to ertapenem for two weeks upon discharge, followed by oral clindamycin to continue for chronic suppression of the infection until resection of renal mass. Due to his cancer and dementia progression, his family chose to pursue hospice care soon after the hospital discharge.

DISCUSSION

This report describes a patient with back pain and uncontrollable shaking, ultimately diagnosed with a *C. sordelli* infection and successfully treated with a course of antibiotics. The majority of*C. sordelli* cases reported infect patients during childbirth or gynecologic procedures [1]; however, the patient described in this case presented in the setting of a hemorrhagic necrotic renal mass and its fistulization with the adjacent splenic flexure of the colon. It is suspected that the gastrointestinal tract was the source of this patient's *Clostridial* infection that likely reached the bloodstream post colonic fistulization with the necrotic, hemorrhagic renal mass.

The pathogenicity of *C. sordellii* has been mainly attributed to its hemorrhagic and lethal toxins that are known to cause local necrosis and edema [1]. These toxins share immunological cross reactivity with *C. difficile* toxin A and B, being a part of the large family of *Clostridial* glucosylating toxins. These toxins work at

the cellular level using similar molecular mechanisms involving glucosylation of Rho and/or Ras GTPAses. When infected, patients may first notice nonspecific symptoms that quickly evolve into massive tissue edema, effusions from the capillary leak, profound leukocytosis, hemoconcentration, refractory hypotension, and tachycardia. Typically, on initial presentation patients infected*with C. sordelli* are already experiencing symptoms of toxic shock, as this patient did, due to its rapidly progressive nature[1,10]. A recent article reported that leukemoid reactions, defined as a WBC count >50,000/ml, were highly suggestive of fatality. This article described 45 cases, which had a mortality rate of 69%. Of these patients, 80% had a leukemoid reaction, and the majority died within 2-6 days of infection[1].

As this patient's history does not follow the typical presentation, this report emphasizes the importance of recognizing the signs and symptoms of this infection and acting quickly due to its high mortality rate. *C. sordelli* must be considered in patients who present in septic shock following a recent surgery or procedure, given the fact that there is no rapid diagnostic test for this infection [1]. This creates a barrier to rapid diagnosis, which can cause a delay in treatment. Upon suspicion of this diagnosis, empiric antibiotic therapy should be started while awaiting blood cultures. While little information exists to support a standard treatment regimen, studies suggest *C. sordelli* is susceptible to beta-lactams, clindamycin, tetracycline, and chloramphenicol, and resistant to aminoglycosides and sulfonamides [9]. Although further investigation is warranted, use of anti-clostridial toxins as a form of treatment has been suggested [1,3], and may help guide treatment in such patients.

AUTHOR CONTRIBUTIONS

Author 1: Wrote and prepared manuscript for submission, edited manuscript.

Author 2: Helped prepare manuscript, edited manuscript.

Author 3: Edited manuscript, provided support to authors 1 and 2 as our attending

ACKNOWLEDGEMENTS

We have no acknowledgements to declare.

CONFLICTS OF INTEREST

We have no conflicts of interest to declare.

REFERENCES

1. Aldape MJ, Bryant AE, Stevens DL. Clostridium sordellii infection: epidemiology, clinical findings, and current perspectives on diagnosis and treatment. Clin Infect Dis. 2006 Dec 1;43(11):1436–46.

2. Smith LD, Holdeman LV. The pathogenic anaerobic bacteria. The pathogenic anaerobic bacteria [Internet]. 1969 [cited 2021 Nov 17]; Available from: https://www.cabdirect.org/cabdirect/abstract/19702201472

3. Sinave C, Le Templier G, Blouin D, Léveillé F, Deland E. Toxic shock syndrome due to Clostridium sordellii: a dramatic postpartum and postabortion disease. Clin Infect Dis. 2002 Dec 1;35(11):1441–3.

4. Brett MM, Hood J, Brazier JS, Duerden BI, Hahné SJM. Soft tissue infections caused by spore-forming bacteria in injecting drug users in the United Kingdom. Epidemiol Infect. 2005 Aug;133(4):575–82.

5. Bangsberg DR, Rosen JI, Aragón T, Campbell A, Weir L, Perdreau-Remington F. Clostridial myonecrosis cluster among injection drug users: a molecular epidemiology investigation. Arch Intern Med. 2002 Mar 11;162(5):517–22.

6. Myers G, Ngoi SS, Cennerazzo W, Harris L, DeCosse JJ. Clostridial septicemia in an urban hospital. Surg Gynecol Obstet. 1992 Apr;174(4):291–6.

7. Aldape MJ, Bryant AE, Ma Y, Stevens DL. The leukemoid reaction in Clostridium sordellii infection: neuraminidase induction of promyelocytic cell proliferation. J Infect Dis. 2007 Jun 15;195(12):1838–45.

8. Guzzetta M, Williamson A, Duong S. Clostridium Sordellii as an Uncommon Cause of Fatal Toxic Shock Syndrome in a Postpartum 33-Year-Old Asian Woman, and the Need for Antepartum Screening for This Clostridia Species in the General Female Population. Lab Med. 2016 Aug;47(3):251–4.

9. Nakamura S, Yamakawa K, Nishida S. Antibacterial Susceptibility of Clostridium sordellii Strains. Zentralblatt für Bakteriologie, Mikrobiologie und Hygiene Series A: Medical Microbiology, Infectious Diseases, Virology, Parasitology. 1986 May 1;261(3):345–9.

10. McGregor JA, Soper DE, Lovell G, Todd JK. Maternal deaths associated with Clostridium sordellii infection. Am J Obstet Gynecol. 1989 Oct;161(4):987–95.

FIGURE LEGENDS

Figure 1. Abdominal CT scan in a) coronal view and b) axial view. Arrows point to the area of the necrotic renal mass invading the colon.