Terrisporobacter glycolicus Bacteremia in a Patient With Cirrhosis

Monarch Shah, MD[1] ▪ Waleed Ahmed Mir, MD[1] ▪ Oluwatofunmi Olowoyo[2] ▪ Sanya Chandna, MD[1]

Abstract

Terrisporobacter is an anaerobic gram-positive bacterium causing infection in postoperative patients, particularly those with comorbidities, and is rarely linked to the disease process itself. Herein, we describe a case of an older man with cirrhosis found to have sepsis secondary to *terrisporobacter glycolicus*. The patient died from complications of cirrhosis leading to multiorgan failure. Although, more studies are needed to establish the pathogenicity of the bacteria, this case describes how bacteremia with this pathogen in a patient with chronic comorbidities can lead to a poor prognosis.

Key words: *Terrisporobacter glycolicus*, cirrhosis, sepsis, bacteremia

A 67-year-old man presented to the hospital with multiple episodes of vomiting, three of which involved hematemesis. His medical history was significant for cirrhosis secondary to nonalcoholic steatohepatitis, bleeding esophageal varices status post-banding, hypertension, hyperlipidemia, diabetes mellitus, and superior mesenteric artery thrombosis managed on warfarin 8 mg daily. The patient also had constipation for 2 days. On admission, his body temperature was 97.9°F, pulse was 118 beats per minute, blood pressure was 100/50 mm Hg, and respiratory rate was 18 breaths per minute.

Physical examination

The pertinent findings on physical examination were a distended abdomen with flank fullness, palmar erythema, and telangiectasias on the abdomen.

Laboratory testing

Laboratory values were significant for a hemoglobin level of 10.7 g/dL (normal range, 13-17 g/dL) and international normalized ratio of 2.18 (0.89-1.11). The rest of the blood counts and a comprehensive metabolic panel were within normal limits. The patient was taken for an emergent esophagogastroduodenoscopy (EGD), which was limited due to a large amount of food and liquid content in the stomach but showed large (>5 mm) nonbleeding esophageal varices in the middle and lower third of the esophagus **(Figures 1 and 2)**.

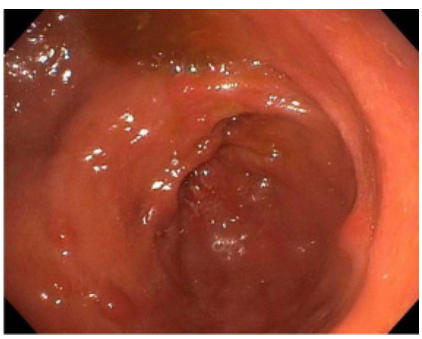

Figure 1. EGD showing nonbleeding varices in the middle-third region of the esophagus.

Intravenous (IV) octreotide infusion, IV pantoprazole 40 mg every 12 hours, and

AFFILIATIONS:
[1]Resident Physician, Department of Internal Medicine, Saint Peter's University Hospital, New Brunswick, NJ
[2]Medical Student, Department of Internal Medicine, Saint Peter's University Hospital, New Brunswick, NJ

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CORRESPONDENCE:
Monarch Shah, Saint Peter's University Hospital, 254 Easton Avenue, New Brunswick, NJ 08901 (monarch.shah08@gmail.com)

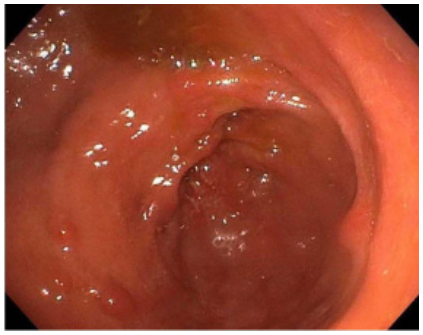

Figure 2. EGD showing nonbleeding varices in the lower-third region of the esophagus.

IV ceftriaxone 1 g daily were started. The patient remained hemodynamically stable and was scheduled to undergo repeat EGD in 48 hours. Due to persistent abdominal distention, an abdominal x-ray was performed, which revealed a small bowel obstruction. An abdominal computed tomography (CT) scan showed a high-grade small bowel obstruction (SBO) with dilation of the stomach and esophagus and a near complete collapse of the distal small bowel and colon **(Figures 3 and 4)**.

Treatment and management

A nasogastric tube was placed with an output of 3 L. Subsequently, the patient was hypotensive (blood pressure 92/52 mm Hg) and tachycardic (pulse 134 beats per minute) and was shifted to the intensive care unit (ICU) for a hypovolemic shock with possible sepsis. Concurrently, the patient developed hepatic encephalopathy. Antibiotic coverage was broadened to vancomycin and meropenem while awaiting the results of a blood culture. The nasogastric tube decompression was continued, and he was given rectal lactulose for hepatic encephalopathy. Blood cultures came back positive for *Terrisporobacter glycolicus* and *Lactobacillus casei*. Vancomycin was discontinued and meropenem was switched to piperacillin-tazobactam according to preliminary susceptibility results. Minimum inhibitory concentrations were not available at that time.

The patient initially responded to all the treatment given in the ICU, with improvement in SBO, mental status, leukocytosis, and tachycardia. He was

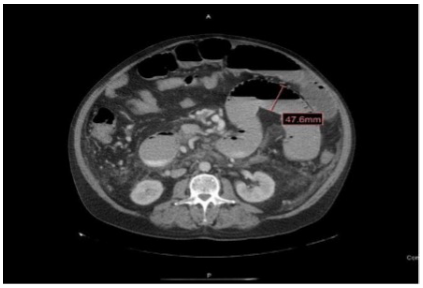

Figure 3. CT of the abdomen and pelvis showing a dilated stomach and SBO up to 47.6 mm in maximal caliber as well as multiple air-fluid levels.

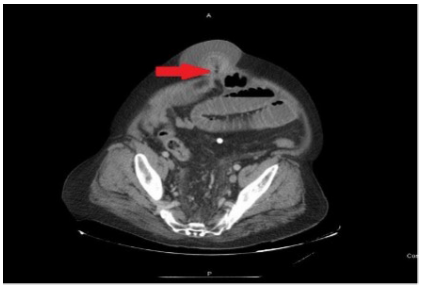

Figure 4. CT of the abdomen and pelvis showing a transition point in the periumbilical area (red arrow) with the more distal small bowel and colon completely collapsed.

transferred from the ICU to the medicine floor, but he was transferred back to the ICU after becoming hypoxic and hypotensive. Back in the ICU, he was found to have developed massive ascites and right-sided pleural effusion. He underwent paracentesis, with the fluid analysis showing white blood cell count of 191 cells/mm^3 and polymorphonuclear leucocyte count of 129 cells/mm^3, suggestive of no infection. Antibiotics were broadened to vancomycin, meropenem, and micafungin, and the patient was started on vasopressors for septic shock. Subsequently, he required intubation and mechanical ventilation secondary to worsening respiratory failure. The patient remained in shock despite the broadened antibiotic coverage and was on maximum doses of 3 pressors. Given the patient's worsening clinical condition and multiorgan failure, his family decided to withdraw all aggressive measures, as per the patient's previously expressed wishes, and the patient died.

Discussion

T glycolicus (formerly *Clostridium glycolcum*) was first described by Gaston and Stadtman in 1963 when they isolated a unique anaerobic gram-positive bacterium from a mud specimen obtained from a pond in Maryland.[1] The evidence so far has suggested that *T glycolicus* is identified in wound and blood cultures often in conjunction with other pathogens.[2] It is also seen in patients who develop postoperative surgical infections, and especially in patients with comorbidities[3]; in most cases, patients with *T glycolicus* deteriorate even after appropriate targeted treatment.[3] Although more commonly seen in patients with comorbidities, there has been one reported case directly linked to a disease process.[4] Therefore, it would be important to determine whether there is a specific timeline for identifying and initiating treatment, as this knowledge might improve outcomes. Our case report describes a patient with decompensated cirrhosis with *T glycolicus* bacteremia who deteriorated despite multiple treatments.

Although the mechanism of infection with gram-positive anaerobes is widely known, the pathogenicity needs to be considered when approaching complex cases such as ours. Gram-positive anaerobes have been known to colonize various areas in the body, such as skin, mucosal surfaces, gastrointestinal (GI) tract, genitourinary tract, and multiple organs, when coupled with any form of trauma, depressed immune system, or comorbidities, serve as a nidus for infection.[5] The processes known to enable anaerobes to evade the immune system include factors such as lectin, collagenase, phospholipase C.[5]

Clostridium is a group of anaerobic, endospore-forming gram-positive, rod-shaped bacteria with various phenotypic characteristics. Over 150 species of *Clostridium* have been described to date, and while most have typically been considered harmless soil saprophytes or inhabitants of the human or animal gut, an increasing number are being reported as causes of human disease.[6] Although *T glycolicus* has been isolated from hu-

man wounds, peritoneal fluid, and feces,[7] it was not clear until recently whether any pathogenic role could be ascribed to this microorganism. In 1987, the first and only published report of animal infection due to *T glycolicus* was described in a young addax with myonecrosis of the buttock and hind legs.[8] Recently, Elsayed and Zhang[2] reported the isolation of *T glycolicus* from blood cultures of a bone marrow transplant patient, implying clinical significance in an immunocompromised patient.

Recovery of this organism from clinical sources (eg, wounds, peritoneal fluid) has previously been documented in the literature.[2] However, Cai and colleagues[4] describe a case of a patient with acalculous cholecystitis whose cultures were positive for *T glycolicus*. Unlike our case, all cases mentioned above were subsequently resolved with treatment. Syuhadah and colleagues[9] described a recent case of an older patient with altered mental status, sacral ulcer, and subsequent deterioration. The deterioration of patients with comorbidities poses the question of whether *T glycolicus* leads to worsened outcomes in patients who have other chronic diseases.

Polymicrobial bacteremia is frequently observed in patients with *Clostridium* bloodstream infections, particularly in the setting of severe sepsis or septic shock, whereby *Clostridium* and *Enterococcus* species may act as copathogens. However, in our patient who presented with nonalcoholic-related cirrhosis, multiple dysregulated immune factors could have led to the increased pathogenicity of *T glycolicus*.[10] The pattern recognition receptors have proven to be most significant.[10] One proposed mechanism involves overstimulating these receptors by bacteria in the GI tract, commonly seen in cirrhotic patients.[10] As a result, the receptors are desensitized, leading to downregulation of immune response and susceptibility to infection.[10] In addition, patients with cirrhosis have been shown to possess a defect in the complement pathway secondary to altered mannose-binding lectin, which is essential in triggering opsonization, phagocytosis, and eventual elimination of organisms.[10] With cirrhosis affecting innate and adaptive immunity components, patients are at risk of worsening polymicrobial infection.

Conclusion

T glycolicus is an anaerobic gram-positive, rod-shaped bacterium that may be implicated as a cause of bacteremia and septic shock in immunocompromised patients. The clinical significance and pathogenic potential of *T glycolicus* await further study. However, clinicians must have a high index of suspicion in cases like ours. Compared with another case of *T glycolicus* in an immunocompetent patient with otitis media induced meningitis who recovered quickly,[11] this poses the question of whether the patient continued to deteriorate secondary to multiple risk factors or due to concomitant infection with *T glycolicus*. Thus, it can be hypothesized that a bloodstream infection with this organism in patients with comorbidities or immunocompromised patients may lead to a poorer prognosis.

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