MRSA Peritonitis Secondary to Perforation of Sigmoid Diverticulitis

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Abstract:
The occurrence of methicillin-resistant Staphylococcus aureus (MRSA) is well documented, but the pathology is usually associated with post surgical infections or long-term peritoneal dialysis. We report the case of a 50-year-old Caucasian man who presented with a one week history of left lower quadrant abdominal pain, poor appetite and nausea due to MRSA peritonitis secondary to perforated sigmoid diverticulitis. Despite a thorough search of the medical literature, we could not find that this problem has been previously described. We report this case to demonstrate the robust nature of MRSA, which has generally not been considered to be a normal colonizing bacterium of the sigmoid colon.

Introduction
Methicillin-resistant Staphylococcus aureus (MRSA) was first identified more than four decades ago. Penicillinase-producing strains of Staphylococcus aureus were universally present in hospitals as early as the 1950s, however, at that time, S. aureus in the community was considered to be largely penicillin-susceptible. MRSA was identified in hospital patients in the United Kingdom within one year of the introduction of semisynthetic penicillin in 1961. The prevalence of MRSA has progressively increased since the early 1980s, and by 2002, MRSA accounted for nearly 60 percent of S. aureus infections acquired in intensive-care units.

It is estimated that 25 percent to 30 percent of the population is colonized in the nose with S. aureus, and less than 2 percent are colonized with MRSA. A MRSA colonization is a strong risk factor for subsequent infection, although most colonized individuals do not develop any clinical disease. In 2005, there were an estimated 478,000 hospitalizations with a diagnosis of S. aureus infection in U.S. hospitals, and 278,000 of these hospitalizations were related to MRSA. It has been estimated that the number of people who developed an invasive MRSA infection in 2005 was 94,360. Approximately 18,650 persons died during a hospital stay related to serious MRSA infections that same year. Eighty-five percent of the infections were associated with exposures to health care delivery, 14 percent of all the infections occurred in persons without obvious exposures to health care and the remaining 1 percent could not be classified. MRSA infections are associated with higher mortality, increased length of stay and increased costs.

One study found that patients with surgical site infections colonized with MRSA had a hospital stay five days longer on average than patients colonized with methicillin-susceptible S. aureus. The study also found that patients with MRSA had a 1.19-fold increase in hospital costs.

As MRSA continues to be a health care burden worldwide, the species continues to evolve, becoming a dominant pathogen and resistant to even more antibiotics. MRSA isolates have a high resistance to many antibiotics. A recent study found high resistance to cefixime, doxycycline, oxacillin, gentamicin, trimethoprim/sulfamethoxazole, chloramphenicol, tobramycin, ofloxacin and ciprofloxacin. One isolate of the study was found to be vancomycin-resistant, demonstrating increasing emergence of vancomycin resistance.

MRSA infections are an important cause of skin infections, endocarditis, pneumonia and osteomyelitis. Although peritonitis is not a common presentation for MRSA, it has been well-documented in the literature. MRSA peritonitis is usually associated with post-surgical infections or long-term peritoneal dialysis. However, to the best of the authors’ knowledge, MRSA peritonitis has never been described in a patient with perforated sigmoid diverticulitis.
and no prior abdominal surgery. Thorough bibliographic database searches were completed in the following: PubMed (MEDLINE), ScienceDirect, BIOSIS Previews, ISI Web of Knowledge, Scitation, SpringerLink, Access Medicine, Wiley Online Library, EBSCOhost and Ingenta. The search strategy included the following MeSH headings and text words with truncation: periton*, perforation, perforated, diverticulitis, diverticular, diverticulosis, MRSA, Methicillin Resistant Staphylococcus Aureus, Staphylococ*, enteric, colon, colorectal, sigmoid, bowl.* We are reporting this case to demonstrate the robust nature of MRSA, which has generally not been considered a normal colonizing bacterium of the sigmoid colon.

**Case Presentation**

A 50-year-old Caucasian man presented to the clinic with a one-week history of left lower quadrant abdominal pain. The patient stated that he had been having an increasingly poor appetite and nausea. He had some relief with bowel movement and flatus; however, his last normal bowel movement was six days prior. He also stated he had fever up to 100°F. He also complained of pain in his right great toe to the point that it was difficult to bear weight. The latter was diagnosed as gout.

The patient had a history of asthma, chronic left hemidiaphragm paresis, gout and hyperlipidemia. He stated that he smoked a half pack of cigarettes per day and had for a number of years and admitted to drinking up to four alcoholic beverages per day. His list of medications included oxycodone, Flexeril, Pulmicort and Albuterol nebulizers, Foradil, Singulair, multivitamin, Indocin, Allopurinol, Crestor, Fenofibrate, Flonase, and omeprazole.

The patient was 72 inches tall, weighed 106 kg, and had a body mass index of 30.9 kg/m2. Tympanic temperature was 98.7°F, pulse 120 beats per minute, and his blood pressure was 134 mmHg systolic over 84 mmHg diastolic. Positive findings on physical examination included: scattered rhonchi, scattered course crackles that cleared with cough and an abdomen soft to palpation with voluntary guarding in the left lower quadrant.

Computed tomography (CT) of the abdomen and pelvis showed a short segment of sigmoid diverticulitis and fatty liver with no abscesses or obstructions. The CT also showed elevation of the left hemidiaphragm with some atelectasis. Pertinent laboratory findings included: a white blood cell count of 11,000, normal serum amylase and normal liver enzymes.

The patient began a ten-day regimen of oral Levaquin 500 mg daily and metronidazole 500 mg twice daily. He also began Miralax 17 grams with 8 ounces of fluid up to four times per day. The patient was advised to go on a clear-liquid diet until pain subsided and to avoid drinking alcohol. The patient was also advised to return promptly if symptoms worsened, fever recurred, pain increased, if he noticed blood in the stool or he was unable to defecate. A follow-up appointment was scheduled 10 days later with his primary care physician.

Three days later the patient presented to the clinic complaining of continuing fever and abdominal pain. At about 2 a.m., he had sudden onset of severe stabbing abdominal pain with fever and chills. The pain had worsened. Tympanic temperature was 104°F, pulse 120 beats per minute, and blood pressure 144 mmHg systolic over 88 mmHg diastolic. His white blood cell count was 18,700. The patient had a distended abdomen with tenderness to palpation and percussion across both lower quadrants greater on the right than left. Intravenous fluids and 2 mg of morphine sulfate were acutely given, and the patient was admitted to the hospital. The patient was started on IV Levaquin 500 mg daily and IV Flagyl 500 mg every eight hours.

The general surgeon was consulted due to suspected perforated diverticulitis. The patient was immediately taken to the operating room and found to have a generalized peritonitis secondary to a perforated mesosigmoid abscess as a result of diverticulitis. The anesthesiologist placed a thoracic epidural block at the T9-T10 level for pain control. The surgeon performed a Hartmann procedure, which is an excision of the perforated bowel with end sigmoid colostomy and closure of the distal sigmoid end or rectum. Cultures from the peritoneum were obtained, and subsequently were reported positive for Methicillin-resistant Staphylococcus aureus. After surgery, IV Levaquin was increased to 750 mg daily. The following day, Levaquin was discontinued and IV Invanz 1 gm daily was ordered.

While the patient seemed to improve initially, over the course of the next three days he became increasingly confused, diaphoretic, tachycardic and tachpneic. He developed pneumonia in the right middle and left lower lobes [Figure 1]. At this point IV Flagyl was discontinued and IV Zosyn 4.5 mg every six hours and IV Cefepine 1 gm every 12 hours were ordered. Because the patient also suffered from alcohol withdrawal, he was placed on the Clinical Institute Withdrawal Assessment (CIWA) protocol. On postoperative day three, there was significant drainage from the wound, which also looked erythematous. The patient developed wound dehiscence and he was immediately taken to the operating room for a laparotomy. There was marked intra-abdominal and abdominal wall edema along with free fluid. A culture from the peritoneal fluid grew MRSA. An initial abdominal wound vacuum device was placed, and the patient was re-admitted to the intensive care unit.
The patient was then started on IV vancomycin at 1 g every eight hours and IV Levaquin 750 mg daily. The following day the patient was again taken to the operating room for removal of the initial wound vacuum device and placement of the AbThera Open Abdomen Negative Pressure Therapy system. Marked intra-abdominal and abdominal wall edema as well as free fluid in the abdominal cavity were again noted. Another culture was obtained and again grew MRSA. After placement of the AbThera device, the patient was returned to the intensive care unit. Postoperatively, the patient continued to endure symptoms from bilateral pneumonia and alcohol withdrawal. A cortisol level was 16.06 μg/dL (2.3-11.9 μg/dL). Infectious disease was consulted at this time. The infectious disease physician recommended continuing vancomycin at 1 g IV every eight hours, starting Flagyl 500mg IV twice daily and Rocephin 2g IV daily, and discontinuing Zosyn and Levaquin.

Two days later, the patient was returned to the operating room for removal of the AbThera device. The intra-abdominal and abdominal wall edema had significantly decreased and there was no free fluid. Repeat peritoneal cultures were negative for MRSA. During this procedure, a small abscess in a remnant of the mesosigmoid was discovered and drained. Cultures of the abscess were positive for MRSA colonization. Due to the significantly decreased edema and absence of free fluid in the peritoneal cavity, a primary closure of the abdominal wall was performed.

The patient was on neuromuscular blockade therapy and a ventilator in the intensive care unit for five days postoperatively with the goals of keeping tension off the surgical incision site and to reduce abdominal wall stress. This decision was based on the World Society of Abdominal Compartment Syndrome’s non-operative treatment algorithm. On postoperative day 10, the patient was sputum culture positive for 1+ growth of Candida albicans. The patient did not receive any anti-fungal medications. On day 15, the patient was transferred out of the intensive care unit to the surgical floor. Four days later, the patient was discharged from the hospital in good condition following a 19 day hospitalization. On discharge, the patient was instructed to continue outpatient IV antibiotic therapy as managed through infectious disease, and was to follow up with the surgeon in the clinic. The patient is asymptomatic and hopes to have the colostomy reversed.

Conclusion

In summary, we treated a patient with MRSA peritonitis secondary to perforation of sigmoid diverticulitis using a Hartmann procedure. The patient had a complicated hospital course including development of bilateral pneumonia, alcohol withdrawal, and wound dehiscence. We believe that use of the AbThera device facilitated a dramatic decrease in intra-abdominal and abdominal wall edema and resolution of peritoneal cavity free fluid, allowing for reapproximation of the musculofascial layers of the abdomen. Although much is known about community-acquired MRSA infections, our literature search did not identify any previous case reports of MRSA peritonitis secondary to perforated sigmoid diverticulitis. We hope we have provided some clinical insight into the highly adaptable nature of MRSA to colonize areas of the body where MRSA is unlikely to exist.

REFERENCES


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