Clostridium Difficile

Mallori Hooker, RN, MSN, NP-C, OCN®

Case Study: George, a 55-year-old retired businessman with a diagnosis of myelofibrosis, underwent an allogeneic stem cell transplantation from his human leukocyte antigen-matched brother in June 2006. He was admitted to the hospital for a possible flare of graft-versus-host disease (GVHD) of the gut. His medications included tacrolimus, budesonide, and bechlamethasone for immunosuppression and pantoprazole. A stool sample was positive for *Clostridium difficile* toxin A on October 31, 2006, and he was started on oral metronidazole.

A colonoscopy was completed on November 1, 2006, to determine the etiology of the diarrhea. The evaluation revealed ulceration with acute inflammation and pseudomembrane-like ulcer exudates in the left colon biopsy. George had rare apoptotic figures in the right colon that could have represented GVHD, but the findings were inconclusive. His diarrhea continued to be positive for *C. difficile,* so he was switched from metronidazole to oral vancomycin for one week. Repeat stool evaluation revealed *C. difficile* toxin A, so a higher dose of oral vancomycin was started. George continued to have positive tests, so oral metronidazole was restarted and vancomycin was continued. He had a negative *C. difficile* test on December 7, 2006, and completed the course of both medications. Reactivation occurred after another week, again with toxin A, and George was treated with IV metronidazole. The next day, he was started on oral vancomycin because the combination had the best results in past treatment. He was switched from IV to oral metronidazole once he could tolderate it without gastrointestinal symptoms. Vancomycin was continued. Another stool test three days later was positive for toxin B. A colonoscopy was done two weeks later after he presented with worsening diarrhea and abdominal cramping. The results were similar to the first, showing abundant acute inflammatory pseudomembrane-like exudates. Infectious disease physicians started him on much higher doses of oral vancomycin with a slow taper over two months. During the vancomycin taper, George remained on either oral or IV metronidazole. He then presented with vancomycin-resistant *Enterococcus bacteremia* at the end of that two-month period. His diarrhea resolved after five months of therapy.

C. difficile, named for the difficulty in culturing the species, originally was identified in 1978 as the main cause of antibiotic-associated diarrhea and pseudomembranous colitis (Schroeder, 2005). The infection was managed easily in the past; however, the onset of a new resistant strain has made treatment more challenging and appears to cause more clinical manifestations in patients.

The incidence of *C. difficile* has increased in the United States and Canada. In a study by McDonald, Owings and Jernigan (2006), cases of *C. difficile* increased significantly from 2000–2003 in the United States. The reason for the increase was unclear but could be related to increased use of an immunoassay test examining toxin A and B, increased use of alcohol-based waterless hand sanitizers, or the new, more resistant, strain (McDonald et al.). Researchers from Canada reviewed data from Quebec hospitals from 1991–2004 and concluded that the probability of recurrent *C. difficile* within 60 days of initial diagnosis increased significantly from 2003–2004 when compared to 1991–2002, especially in patients 18 years of age and older (Pepin et al., 2005). They also noticed a significant increase in the proportion of patients who experienced metronidazole failure from 2003–2004 (Pepin et al.).

What Is Clostridium Difficile?

C. difficile is an anaerobic gram-positive bacillus that accounts for approximately 15%–20% of antibiotic-related cases of diarrhea (Schroeder, 2005) (see Figure 1). This organism spreads by its ability to form spores. Fomites, such as bed rails, floors, and employees' hands have tested positive for *C. difficile,* where it can remain for as many as 40 days

Mallori Hooker, RN, MSN, NP-C, OCN®, is a bone marrow transplant nurse practitioner at Rocky Mountain Cancer Center in Denver, CO.

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