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Prevention and Treatment of *Clostridium difficile* Enterocolitis

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Keywords

• *Clostridium difficile* • Enterocolitis • Infection

Key points

- *Clostridium difficile* infection is a major cause of nosocomial infection and is increasing in incidence and severity.
- Metronidazole and vancomycin are considered first-line medical therapies.
- Surgical management is subtotal colectomy with preservation of the rectum and end ileostomy.
- Diverting loop ileostomy with colonic lavage and installation of antibiotics via the ileostomy may be appropriate for selected patients.

INTRODUCTION

Clostridium difficile infection (CDI) is a symptomatic disease caused by the spore-forming and toxin-producing anaerobic bacterium *C difficile*. CDI is the most common cause of pseudomembranous colitis and remains a major cause of nosocomial infection and antibiotic-associated diarrhea. Surgical patient populations, especially patients undergoing colorectal procedures and those with inflammatory bowel disease, are susceptible. Rates of CDI are increasing in incidence in the United States, and associated morbidity and mortality are high. Antibiotic stewardship, hand hygiene, and environmental control are critical to help prevent spread of this transmissible disease. Most cases will respond to medical management, but early operative consultation is recommended. The mainstay of surgical management is subtotal colectomy with preservation of the rectum and end ileostomy. Some patients may be appropriate for a newer

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approach using diverting loop ileostomy with colonic lavage and installation of antibiotics via the efferent limb of the ileostomy.

HISTORY

The first reported case of pseudomembranous colitis was published in 1893 by JM.T. Finney at The Johns Hopkins Hospital [1]. A 22-year-old woman developed bloody diarrhea on the tenth postoperative day after uncomplicated peptic ulcer surgery and died on postoperative day 15. The autopsy report described pseudomembranous changes of the colon: "…appearing gray in contrast with the haemorrhagic mucous membrane about it, and a coating of what is apparently fibrin (and blood) can be scraped from the surface." Although the *C difficile* organism would not be identified until more than 40 years later, this case has been cited as the first report of a *C difficile*–associated diseaselike process [2,3]. Pseudomembranous colitis became a commonly recognized complication of antibiotic use in the early 1950s. Surgeons reported rates as high as 14% to 27% among postoperative patients [4]. An early study used routine endoscopy and reported high rates of pseudomembranous colitis (21%) among patients receiving the antibiotic clindamycin [5]. Further efforts were directed at identifying the cause of "clindamycin colitis." By the late 1970s, *C difficile* was isolated from the stool of patients with pseudomembranous colitis [6,7].

SIGNIFICANCE AND EPIDEMIOLOGY

CDI is common and remains the most frequently reported health care–associated pathogen in the United States [8]. CDI is an independent predictor of increased length of stay in the intensive care unit, increased hospital length of stay, and higher total hospital charges [3]. A major burden for both patients and hospitals, CDI has demonstrated increasing incidence and greater severity of disease since the year 2000 [9–11]. Currently, at least 500,000 cases are reported each year in the United States, and mortality may be as high as 5% to 10% [11,12]. The economic burden of CDI on the US health care system is estimated at more than $1.5 to $2 billion per year [9,11].

Although most CDI cases are acquired in the hospital, a growing number of individuals without traditional risk factors have acquired the infection in the community [13]. Community-acquired CDI may account for more than one-third of *C difficile*–associated diarrhea. Specifically, nursing home–acquired CDI represents nearly one-quarter of all cases in the United States with a 19% recurrence and 8% 30-day mortality [14]. Although community-acquired cases are usually associated with lower mortality than hospital-acquired cases, rates of hospitalization, morbidity, and mortality remain high.

PATHOGENESIS

C difficile is present within the normal microbial population of the gut in as many as 5% to 15% of healthy adults and 40% to 60% of neonates [15,16]. Asymptomatic colonization is not a risk factor for symptomatic CDI and may in fact protect against development of the disease [17]. Healthy carriers are thought to be