Fecal Microbiota Transplant to Treat Recurrent *Clostridium difficile* Infections

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The prevalence of recurrent or refractory *Clostridium difficile* infection has been steadily increasing since 2000. Consequently, alternative treatments to the standard antibiotic therapies are now being considered. One alternative treatment is fecal microbiota transplant. Although fecal microbiota transplant is relatively new—and not appealing to most people—it has been around for many years and has great promise as an inexpensive, safe, and efficient treatment of refractory and recurrent *C difficile* infection. With a better understanding of the intricacies of the colonic microbiome and its role in colonic physiology and pathophysiology, critical care nurses will recognize that fecal microbiota transplant has the potential to become the standard of care for treatment of recurrent or refractory *C difficile* infection. The American College of Gastroenterology and the Infectious Diseases Society of America provide the latest treatment guidelines for care of patients with these clostridial infections. (*Critical Care Nurse*. 2015;35[2]:51-65)

***Clostridium difficile* infections (CDIs) are some of the most common health care–associated infections in hospitalized patients[1-8] and in patients residing in nursing homes.[9-11]** According to estimates, the diagnosis and treatment of CDIs cost more than $3.2 billion annually in the United States,[2,4,11,12] with approximately 333 000 cases and 15 000 to 20 000 deaths per year.[2,4,13-15] When the diarrhea associated with CDIs becomes severe and causes complications, the infections become life-threatening and are a marked cause of morbidity and death in hospitalized patients.[1-4] The incidence of severe and recurrent CDIs (RCDIs) has increased because of a new hypervirulent strain of *C difficile* that is less responsive to traditional medications. Patients with RCDI often are treated in an intensive care unit. Fecal microbiota transplant (FMT), also called fecal bacteriotherapy, is an adjunctive, cost-effective treatment for patients with RCDI.[2,12-19] Critical care nurses must understand the importance of a balanced gut microbiome[20,21] and how CDIs disrupt that balance.[22-25] Furthermore, the nurses must recognize the role of FMT in order to provide appropriate care, educate patients, and collaborate with health care professionals on the latest treatment options for patients with RCDI.

CE Continuing Nursing Education

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Identify patients at risk for developing *Clostridium difficile* infection (CDI)
2. Describe the role of the critical care nurse to ensure safety when caring for patients with recurrent CDI
3. Discuss the role of fecal microbiota transplant in the treatment of patients with recurrent CDI

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Dorothy, a 69-year-old woman who came to the emergency department, stated, "I have relentless diarrhea despite taking medication for 2 months and cannot deal with it any longer." She reported that she had "not urinated in over a day." Dorothy's medical history included a bout of pneumonia and bronchitis that had been treated 5 months earlier with an antibiotic. Table 1 describes Dorothy's time line of illness. Dorothy stated, "My bowels have not been the same since the pneumonia. I have been getting diarrhea on and off for months, but this time it is the worst ever." She had no relevant surgical history. She was taking metronidazole 500 mg orally 3 times a day for the "bowel issues."

Physical examination revealed a very pale appearance, flat neck veins, lethargy, and extreme fatigue. Other findings were oral temperature 100°F (37.8°C), heart rate

Table 1 Time line for Dorothy's course of illness

Month in 2013	Description
May	Dorothy went to her family medical doctor (FMD) because she had shortness of breath, fever, lethargy, and chills. Diagnosis: Pneumonia. Treatment: A "broad-spectrum antibiotic."
June	Two weeks after Dorothy finished taking her antibiotic and the pneumonia had resolved, she started to have diarrhea and returned to her FMD. Diagnosis: Antibiotic-related diarrhea. Treatment: Probiotics.
July	Dorothy went back to her FMD and reported having diarrhea for 5 days despite taking probiotics. Diagnosis: A stool specimen was obtained, and *Clostridium difficile* infection due to broad-spectrum antibiotic therapy was confirmed. Treatment: Metronidazole 500 mg orally 3 times a day for 10 days.
August	Dorothy went back to FMD and reported continued diarrhea, abdominal cramping, decreased appetite, and a 5-pound (2.25 kg) weight loss. Diagnosis: Recurrent infection with *C difficile.* Treatment: Vancomycin 125 mg orally 4 times a day for 10 days.
September	Dorothy reported back to her FMD, stating that she took the vancomycin, "felt a little better" but she still felt her abdomen was "distended, tender," and she was passing much more gas and had very soft stools. Diagnosis: Recurrent infection with *C difficile.* Treatment: Metronidazole 500 mg orally 3 times a day for 10 days and vancomycin 125 mg orally 4 times a day for 10 days.
November	Almost 3 months later, Dorothy was getting worse and her FMD encouraged her to go to the emergency department.

The Role of the Gut Flora

The gastrointestinal tract is complex and contains more than 500 species of microorganisms (intestinal microbiota), many of which are harmless in healthy individuals.[20-25]

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Although microorganisms vary greatly from one person to the next, each person has the same basic bacterial types,[20-25] which keep a harmonious balance in the gut to aid in protective, structural, and metabolic functions[4,20-25] (Table 2). The important role of the gut flora in colonization resistance or preventing potentially pathogenic organisms, such as *C difficile*, from establishing a colony within the gut has been recognized for a long time. Recently, the Human Microbiome Project[24,25] has emphasized the microbial components of the human genetic and metabolic landscape and how these components profoundly affect many diseases and conditions, from irritable bowel syndrome[20] to mental health,[26] immunity,[20,27] cystic fibrosis,[28] energy metabolism, and obesity.[20,29]

Definition and Transmission of CDI

Clostridium difficile, a gram-positive, rod-shaped, spore-forming bacterium spreads from person to person or surface to person via the fecal-oral route.[1-9,30,31] When spores

138/min with sinus tachycardia, respirations 18/min, and oxygen saturation 96% on room air as measured by pulse oximetry. Her blood pressure was 88/60 mm Hg; her usual blood pressure was "in the 130s." She had hyperactive bowel sounds on auscultation, slight abdominal distention with marked generalized tenderness on palpation, and dry mucous membranes. Dorothy rated her discomfort a 4 on the pain scale.

Laboratory values were white blood cell count 16 500/μL with a shift to the left (neutrophils 93%), serum albumin level low at 2.6 g/dL, serum potassium level 2.8 mEq/L, and serum creatinine level 1.5 mg/dL (to convert creatinine level to micromoles per liter, multiply by 88.4). Findings on a chest radiograph were normal, and she had no biomarkers for myocardial ischemia. An abdominal radiograph showed no obstruction, ileus, or perforation. Abdominal computed tomography displayed minimal thickening in the walls of the ascending and transverse colon. The hypovolemia and hypokalemia were treated with intravenous administration of fluid and electrolyte replacement.

On admission to the intensive care unit, Dorothy was placed in enteric contact isolation for treatment of suspected RCDI. A hospitalist ordered an enzyme immunoassay to detect *C difficile* toxins and also consulted a gastroenterologist. Vancomycin, which Dorothy had taken orally for 10 days about 2 months earlier when the diarrhea came back the second time, was administered intravenously. Treatment with metronidazole was discontinued because of possible neurotoxic effects and because the diarrhea had not abated. Shortly after she was admitted to the intensive care unit, a fecal management system was inserted. Rectal drainage on day 1 was 1100 mL. Rectal drainage of light brown liquid feces was 800 mL on day 2, 825 mL on day 3, 750 mL on day 4, 669 mL on day 5, and 730 mL on day 6. Despite the use of nitazoxanide and probiotics (*Lactobacillus rhamnosus* GG), no decrease in diarrhea occurred. On day 5, the gastroenterologist in collaboration with an infectious disease physician began discussing FMT. Dorothy's husband was assessed as a possible fecal donor. All pharmacological treatments for CDI were discontinued.

On day 7, in the endoscopy suite, the gastroenterologist administered the donor feces (400 mL) via colonic instillation. The rectal tube was discontinued before FMT. Dorothy was positioned on her left side and was anesthetized with propofol and a small amount of lidocaine. The gastroenterologist then infused small increments (30-40 mL) of feces throughout the colon, beginning at the cecum, via the biopsy port of the colonoscope. Dorothy was expected to retain the feces as long as she could (she was able to retain the material for about 3 hours) and was kept on her left side lying in bed for 2 hours after the transplant. The diarrhea resolved 10 hours after completion of the FMT. Dorothy was discharged on day 9; all laboratory values were within the reference range and abdominal tenderness was a 2 on a pain scale. At the 2-month follow-up at the gastroenterology office, she reported no further diarrhea or complications.

Table 2 Functions of the intestinal flora

Protective	Structural	Metabolic
Pathogen displacement	Barrier fortification	Regulation of differentiation and proliferation of intestinal epithelial cells
Nutrient competition	Induction of immunoglobulin A	Metabolization of dietary carcinogens
Receptor competition	Maintenance of mucosal integrity by tightening of apical junctions	Synthesis of vitamins such as vitamin K, vitamin B complex, and folate
"Colonization resistance" by production of antimicrobial factors such as bacteriocins and lactic acids to prevent exogenous or potentially pathogenic organisms from establishing a colony within the gut	Priming development of the mucosal immune system	Fermentation of nondigestive dietary residue and endogenous epithelially derived mucus
		Absorption of ions
		Salvage of energy

are ingested, they survive in the acidity of the stomach and enter the intestines, where they begin to germinate. Because of their durability and strength, spores germinate, and organisms rapidly outgrow the normal intestinal flora.[1,3,8,9,12-17] Although *C difficile* flourishes in anaerobic conditions such as the gut, it can survive on a variety of surfaces for more than 3 months.[1-7,31] The spores are difficult to eradicate from surfaces, creating an easy way to

transmit infection from one person to another.[1,30-33] In addition to transmission via the fecal-oral route, CDI may be a side effect of antimicrobial therapy.[1,4,24,31-35] Physiological alterations associated with antimicrobial therapy cause perturbations in the intestinal microbiota that allow colonization.[20,24,32,35]

Pathophysiology

Clostridium difficile produces 2 toxins that cause inflammation and disruption of the epithelial mucosal surface (see Figure), which lead to various degrees of diarrhea.[1-4,23-25,29] Once the toxins get into the cell, they interfere with cellular function, resulting in apoptosis or cell death.[4,8,23] Toxin A, an enterotoxin, causes increased intestinal permeability and fluid secretion. Toxin B, a cytotoxin, leads to intense colonic inflammation. The intestinal barrier function is lost, permeability increases, and granulocytes and fluids migrate into the intestines, resulting in diarrhea.[1-7,23,30-32] Newer, more virulent strains of *C difficile*, such as the North American PFGE type 1 (NAP1/027)[2,7,19,23,31,34] and PCR078, have highly mobile, mosaic genomes, a characteristic that enhances the drug resistance of the strains, making pharmacological treatment of CDIs caused by these strains more challenging than treatment of infections caused by less virulent strains of *C difficile*.[13,31]

Epidemiology

According to the Centers for Disease Control and Prevention,[6] the prevalence of CDIs has been markedly increasing since the early 2000s. Of more concern are the number of community-acquired cases and the number of cases resistant to metronidazole, the most common treatment for CDI.[14,18,35] About half of the infections occur in patients less than 65 years old[5]; however, patients more than 65 years old account for about 90% of the deaths due to CDI.[2,4,10,11] The highest incidence of CDI is among elderly hospitalized patients (25% of all cases)[2,4]; the remaining 75% of cases occur in patients in nursing homes[9,11,36] or in patients who recently visited doctors' offices and clinics.[1-10,19-21] Another increased rate has been noted in outpatients who are taking antibiotics (as Dorothy was) or proton pump inhibitors, children who are immunocompromised,[27,37] patients who have had gastric bypass,[1,8] and postpartum women.[1,8,13]

Signs and symptoms associated with CDI may appear shortly after antibiotic therapy is started or weeks or months later.

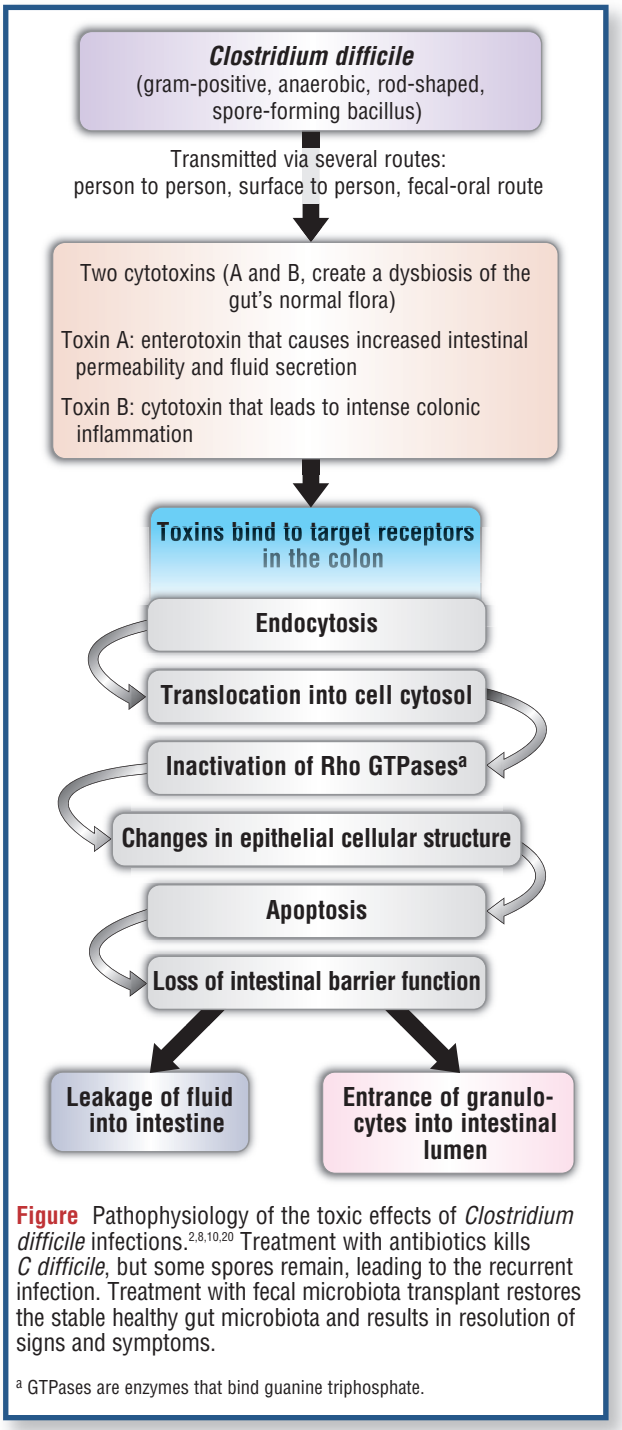

Figure Pathophysiology of the toxic effects of *Clostridium difficile* infections.[2,8,10,20] Treatment with antibiotics kills *C difficile*, but some spores remain, leading to the recurrent infection. Treatment with fecal microbiota transplant restores the stable healthy gut microbiota and results in resolution of signs and symptoms.

[a] GTPases are enzymes that bind guanine triphosphate.

Clinical Manifestations of CDI

The American College of Gastroenterology (ACG)[2,7] classifies CDIs as mild, mild-to-moderate, severe, severe and complicated, and recurrent (Table 3). Signs and symptoms associated with CDI may appear shortly after

Table 3 Scoring of severity of *Clostridium difficile* infection and recommended treatment from the American College of Gastroenterology[a]

Severity	Most common signs and symptoms and treatment
Mild	Diarrhea only, no other sign or symptom
Mild-to-moderate	Fever, chills, dehydration, poor skin turgor, dry mucous membranes, nausea, and anorexia Foul-smelling diarrhea and/or bloody diarrhea, abdominal distention, abdominal pain ranging from mild cramping to severe diffuse pain **Treatment:** metronidazole 500 mg by mouth 3 times a day for 10 days; if no improvement in 5-7 days or patient unable to take, then change to vancomycin at standard dosing of 125 mg by mouth 4 times a day for 10 days
Severe and complicated (fulminant pseudomembranous colitis)	Laboratory tests: serum albumin level < 3 g/dL plus 1 of the following: admission to intensive care unit, hypotension with or without use of vasopressors, white blood cell count > 15 000/μL, abdominal tenderness, serum lactate levels > 2.2 mmol/L, profuse diarrhea, pain in lower part of abdomen, leukocytosis, severe abdominal pain, high fever (> 38.5°C), chills, leukocytosis, abdominal distention Patients may have minimal diarrhea because of an ileus Patients may have severe *C difficile* infection without pseudomembranous colitis Fulminant pseudomembranous colitis may cause complications such as toxic megacolon, perforation of the colon, and death Recommended assessment: computed tomography of the abdomen **Treatment:** vancomycin by mouth 125 mg 4 times a day, metronidazole 500 mg intravenously 3 times a day, and, possibly, surgical consultation
Recurrent *C difficile* infection	Recurs within 8 weeks of completion of therapy **Treatment:** repeat metronidazole or vancomycin pulse regimen; consider fecal microbiota transplant after 3 recurrences

[a] Based on data from Surawicz et al,[2] Cohen et al,[4] and Hessen.[7]

antibiotic therapy is started or weeks or months later.[3] Although many patients with mild to moderate CDI are treated as outpatients, more patients, such as Dorothy, have severe CDI and require treatment in an acute care unit. Severe CDI is associated with abdominal distention and pain ranging from mild crampy feelings to severe diffuse pain, profuse diarrhea, leukocytosis, and hypoalbuminemia (< 3 g/dL). In severe and complicated CDI, at least one of the following is present or develops: hypotension, fever, leukocytosis, elevated serum lactate levels, any evidence of end-organ failure, and admission to an intensive care unit.[2,3,30,32]

Critical care nurses must recognize fulminant colitis, the most serious manifestation of CDI, which may lead to toxic megacolon, perforation of the colon, and death.[3] Typical indications of fulminant colitis are severe abdominal pain, diarrhea, high fever, chills, leukocytosis, and abdominal distention.[2,8,13] Some patients with fulminant colitis have minimal diarrhea because an ileus causes fluids to accumulate in the colon instead of being excreted.[9] Recurrent CDI, as in Dorothy's case, is defined by the ACG as a return of the signs and symptoms of CDI within 8 weeks of the completion of antibiotic therapy.[2,10] Recurrence rates after 1 bout of CDI are reported to be 20% to 25% whereas recurrence rates after 2 or more bouts of CDI have been reported to be as high as 50% to 60%.[7] Recognizing these classifications will help critical care nurses ensure timely therapy without overtreating or undertreating the patient.

Diagnostic Testing

Diagnostic testing for CDI includes laboratory and imaging studies. Laboratory tests include toxin enzyme immunoassays, toxigenic cultures, nucleic acid amplification tests , and the *C difficile* cytotoxin neutralization assay.[2-4,8] The enzyme immunoassays are used to detect *C difficile* toxins A and B; however, the sensitivity and specificity of detection vary greatly among the commercially available assays.[2,3] Toxigenic cultures have high sensitivities and specificities for detection of the organism but may take 2 to 5 days to produce results because of the incubation period required.[2,3] Although toxigenic cultures are highly sensitive and specific, they can produce false-positives if a formed stool is sent for testing.[2,3] The nucleic acid amplification tests are highly sensitive and specific but are only used in acute diseases because of concerns for false-positives. Finally, the *C difficile* cytotoxin neutralization assay produces results with 90%

Table 4 Microbiological testing for *Clostridium difficile*[a]

Test[b]	Sensitivity	Specificity	Availability	Application
Toxin enzyme immunoassays	Low	High	Wide	Must detect toxins A and B
Toxigenic culture	High	High	Limited	Limited diagnostic use; epidemiological tool
Nucleic acid amplification tests (polymerase chain reaction assays, isothermal amplification tests)	High	High	Wide	Used only in acute disease
C difficile cytotoxin neutralization assay	High	High	Limited	Limited diagnostic use; reference method
C difficile culture	Low	Moderate	Limited	No diagnostic use

[a] Based on data from Surawicz et al,[2] Kaseb and Novotne,[3] and Cohen et al.[4]
[b] How quickly results of these tests are available depends on the hospital's laboratory.

sensitivities and specificities quickly and requires little hands-on time from a technologist.[3,8] The ACG discourages repeat testing, stating that only a single sample needs to be assayed, and does not recommend testing for cure. Table 4 gives the sensitivities and specificities of the various tests.

Diagnostic colonoscopy and computed tomography of the abdomen and pelvis may be considered for complicated CDI.[2-4,9] The results of these procedures help detect the severity and extent of the CDI and its effects, including thickening of the colonic wall, ascites, toxic megacolon, ileus, and perforation.[2-9]

Traditional Pharmacological Interventions

According to the Infectious Diseases Society of America[2,4] and the ACG, traditional pharmacological treatment of CDI should be started only after testing for *C difficile* is completed. The exception is when a patient with suspected CDI is critically ill or has a rapidly worsening condition.[2,4] The first-line treatment for mild CDI is oral metronidazole 500 mg 3 times daily for 10 to 14 days.[2,4] Absorption of metronidazole occurs mostly in the upper part of the gastrointestinal tract, so this drug may be ineffective for infections due to some *C difficile* isolates.[2,4] If patients are unable to tolerate oral metronidazole, the drug can be administered intravenously at 500 mg every 6 hours. Metronidazole should not be used long-term because of the risk for neurotoxic effects.[2-4]

Traditional pharmacological treatment of CDI should be started only after testing for *C difficile* is completed.

For patients with moderate-to-severe signs and symptoms, such as Dorothy had, the drug of choice is oral vancomycin 125 to 500 mg 4 times daily for 10 days.[2-4] In patients unable to take medications orally, or patients in whom oral agents cannot reach a segment of the colon (Hartman pouch, ileostomy, colon diversion), vancomycin can be administered via enema, colostomy, ileostomy, or nasogastric tube.[2-4] Patients should be monitored for vancomycin resistance, which would be indicated by continuation of the diarrhea after vancomycin therapy was started. In such instances, fidaxomicin, a narrow-spectrum macrocyclic antibiotic, may be used at a dose of 200 mg orally twice a day. Compared with other CDI treatment options, administration of fidaxomicin is associated with lower rates of recurrence of the infection. Although fidaxomicin is quite expensive, if recurrences of CDI are prevented, the extra expense may be worthwhile.[4,22]

Fecal Microbiota Transplant

Approximately 20% of patients with CDI who are treated pharmacologically have a recurrence of the infection within 6 months after the antibiotic is discontinued.[2,3,7,8,16] Treatment options for RCDIs are limited. The ACG recommends FMT after a third RCDI[2,7,16] in order to reestablish the normal composition of the gut flora, restore the balance in metabolism, and stimulate both cellular and humoral immune responses in the gut mucosa.[3,4,7,13,14,38]

Although fecal transplants have been used since the 4th century in China, they are now becoming more widely accepted as a safe and effective method of treating RCDI.[4,13,38-46] Table 5 provides facts related to FMT. The first FMT documented in the English literature was in 1958. Eiseman et al[43] successfully used fecal enemas to treat 4 patients with pseudomembranous enterocolitis; all 4 had complete resolution of signs and symptoms.

Table 5 Facts on fecal microbiota transplant[a]

1. Fecal transplant was first documented in fourth-century China, was known as "yellow soup," and has been used regularly for decades in many countries as the first line of defense or the treatment of choice for infection with *Clostridium difficile*.
2. In all documentation, dating back to fourth-century China, not a single serious side effect has been reported.
3. In some parts of the world, a newborn infant routinely receives a small amount of the mother's stool orally, to jump start the baby's immune system.
4. Fecal transplant has been used in the United States sporadically since the 1950s and has recently gained popularity there. Experts estimate that the total number of treatments to date in the United States is around 500 patients.
5. In late spring of 2013, the Food and Drug Administration announced that it was classifying fecal matter as both an investigational new drug and a biologic.
6. In the summer of 2013, the Food and Drug Administration announced that qualified physicians could perform fecal microbiota transplant to treat recurrent infection by *C difficile*.
7. Many patients do not have a donor to assist them.
8. Many patients have never heard of this treatment.
9. Fecal transplant is a low-cost, low-risk, highly effective treatment.

[a] Data from Fecal Transplant Foundation.[13]

The first report[44] of using FMT for treatment of RCDI was published in 1983; a woman had prompt and complete resolution of gastrointestinal problems after FMT. In 2010, Garborg et al[40] reported the results of FMT in 70 patients with RCDI; the transplants were effective even in patients with CDI caused by the NAP1/BI/027 strain. By 2011, a total of 325 cases of RCDI had been treated with FMT worldwide, with a mean cure rate of 91%.[2,4] Most recently, in 2012, Kelly et al[37] reported the results of a retrospective, multicenter follow-up study of patients with RCDI who received FMT; the primary and secondary cure rates were 91% and 98%, respectively.

In FMT the stool (200-300 g) of a healthy donor is mixed with physiological saline or water to make a liquid slurry,[13,15,16] filtered to remove larger particulate matter, and then instilled in the upper or lower part of the gastrointestinal tract of a patient with RCDI.[38-47] Although early on the most common method of administration was retention enema,[17] more recently the donor fecal material has been administered via nasogastric tube,[47-49] nasoduodenal tube,[50,51] colonoscopy,[52] oral fecal capsules,[45] and self-administered enemas.[50,53-55] Instilling the fecal material via colonoscopy has many advantages.[52] First, with this method, the stool can be infused throughout the length of the colon. Second, the colonic mucosa can be directly visualized and any abnormal findings can be documented. Third, patients are sedated and generally tolerate FMT well.[1-3,19] Fourth, success rates range from 86% to 100% cure, whereas enema success rates are 81% to 100%. However, FMT via colonoscopy is associated with risks for perforation, infection, bleeding, and pain. Although the nasogastric method is least effective, with success rates of 73% to 83%, it is easier to perform than are the other methods, costs less, and poses lower risks to the patient.[49]

The preferred donor source is someone who is intimate with the patient; however, donors may also include family members or other unrelated healthy donors.[13,40,43] According to recent studies,[44,48] the material from donors whose fecal specimens are frozen (up to 6 months) until needed is just as effective as fecal specimens from patient-identified donors. The advantage of using a donor who is known to the patient, or known to the gastroenterologist, is that some providers will then allow the donor to opt out of screening for CDI to save costs and allow the patient to receive FMT more quickly.[13,35,38,41,53] Frozen stool can also be used quickly in emergent cases to save time. Fecal donors, like blood donors, are screened rigorously[2,10,13,15,16,40,47] (Table 6).

Meticulous hand washing with soap and running water is the most effective means of physically removing *C difficile* spores from the hands.

Although minimal risks associated with FMT have been documented so far, critical care nurses must recognize that the biggest risks are transmission of undetected infectious bacterial agents[2,4,7,13] and side effects such as headaches, sore throat, and various gastrointestinal

Table 6 Exclusion criteria for donors of fecal microbiota[a]

Criterion	Examples
Absolute	
Risk of infectious agents	Known human immunodeficiency virus (HIV), hepatitis B, or hepatitis C infection or known exposure to these within the past year High-risk sexual behaviors Use of illicit drugs, including intranasal cocaine Tattoo or body piercing within past 6 months Incarceration or history of incarceration Current communicable disease Risk factors for variant Creutzfeldt-Jacob disease Travel within 6 months to areas where diarrheal illnesses are endemic Malignant neoplasm
Gastrointestinal comorbid conditions	History of inflammatory bowel disease, irritable bowel syndrome, idiopathic chronic constipation, or chronic diarrhea History of gastrointestinal cancer or known polyposis
Factors affecting the gastrointestinal microbiota	Antibiotics within the preceding 3 months Major immunosuppressive medications Systemic antineoplastic agents
Additional considerations	Recent ingestion of anything to which the recipient is allergic (eg, shellfish, nuts)
Relative	History of major gastrointestinal surgery (eg, gastric bypass) Metabolic syndrome Systemic autoimmunity such as multiple sclerosis, connective tissue disease Atopic diseases such as asthma and eczema, eosinophilic disorders of the gastrointestinal tract Chronic pain syndromes such as chronic fatigue syndrome, fibromyalgia

[a] Many institutions use a donor questionnaire based on the Donor History Questionnaire prepared by the AABB Donor History Task Force to screen blood donors.

complaints. One case of norovirus gastroenteritis was recently reported after FMT despite asymptomatic donors and lack of sick contacts.[56] Uncertainty about the long-term safety of FMT is another factor to consider. In Dorothy's case, no side effects were reported.

Role of Critical Care Nurses

Although most patients with RCDI may be treated as outpatients, patients with severe infections, such as Dorothy, and patients who have fulminant CDI colitis often require treatment in the critical care environment. Critical care nurses have many responsibilities to ensure patients' safety, including recognizing when FMT may be a treatment option, identifying patients at risk for CDI, carrying out meticulous hand washing, promoting vigilant antibiotic stewardship, maintaining enteric contact isolation with attentive environmental cleaning, assessing and treating the patients' underlying illnesses, promoting comfort, maintaining skin integrity, and developing clinical practice guidelines to ensure safe patient care.[3,30,46] Teaching patients and their significant others is important in all areas when providing care to patients who may require an FMT.

Recognizing FMT As an Option for Patients With CDI

Critical care nurses must be aware of FMT as a treatment option for patients with RCDI. After stabilizing a patient's hemodynamic status, maintaining accurate stool records, and carrying out regular abdominal assessments, nurses must be able to answer questions of the patient and the patient's family members about the FMT procedure itself, know how to prepare the patient, and identify what to expect after the procedure.

Protocols for preparing patients differ depending on whether the route of administration is via the upper or the lower part of the gastrointestinal tract. For example, if FMT is performed via a nasogastric tube, a proton pump inhibitor is usually administered the night before the procedure, whereas, if the fecal material is transfused via a colonoscopy, a bowel preparation with polyethylene glycol orally may be administered the evening before the transplant.[30,37,40,42] Other times loperamide may be administered either before or after FMT. All pharmacological therapy for CDI is discontinued 24 to 48 hours before FMT.[2,5,19,20]

Identifying Patients at Risk for CDI

The role of critical care nurses starts with patient rounds and identification of patients at high risk for CDI. A thorough history from patients who have diarrhea is the beginning step. Questions should include history of antibiotic use, recent hospitalization or stay in a nursing home, onset of signs and symptoms, employment as a health care provider, and whether or not diarrhea is the primary sign or if a causative agent of the diarrhea has been identified.[49,57] Other factors to determine are whether the patient is immunocompromised, is elderly, or has a history of gastrointestinal disorders.[3,7,50-55,57] The answers to these questions will help guide the clinician in ordering diagnostic tests. If CDI is suspected, a fecal specimen should be obtained along with specimens for other laboratory studies.

Meticulous Hand Washing

Meticulous hand washing with soap and running water is the most effective means of physically removing *C difficile* spores from the hands.[57-59] Laboratory studies have shown that alcohol separates *C difficile* organisms from stool specimens but is ineffective for eradication. Alcohol-based hand rubs mostly cause displacement of the spores over the skin surfaces.[57] Health care providers must wash their hands for at least 15 to 20 seconds for maximal effectiveness.

Although much emphasis has been placed on ways to improve health care workers' compliance with hand hygiene,[58,59] little effort has been directed toward involving patients in the patients' own hand hygiene. Evidence suggests that patients' flora and the hospital environment are the primary source of many infections. Critical care nurses must educate and involve patients more directly in hand hygiene practices (eg, not putting their hands in their mouth or putting contaminated items in their mouth) and use patient-centered safety initiatives to provide recommendations for patient hand hygiene protocols.[59]

Vigilant Antibiotic Stewardship

One of the most important roles of critical care nurses is to be an advocate for the patient and to recognize that antibiotics are the primary risk factor for CDI. If antibiotics are the suspected cause of diarrhea, the drugs should be discontinued immediately[5-8] and consideration given to the underlying reason the patient was started on the therapy. Rapid diagnosis and treatment and collaboration with gastroenterologists are essential for the well-being of patients.

Nurses must be sure that any antibiotics are administered correctly, with no missed doses, and ensure that the drugs are discontinued as indicated. The Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America[4] recommend decreasing how often and for how long antibiotics are used and using the minimal number of antibiotics per patient. Nurses should be aware of these guidelines[4] to establish institutional programs to enhance antibiotic stewardship.

Maintaining Enteric Contact Isolation and Vigilant Environmental Cleansing

Because *C difficile* is so easily transmitted, critical care nurses must use personal protective equipment every time they enter the room of a patient with suspected or known CDI.[30,59] Having to don personal protective equipment every time nurses enter a patient's room may seem too burdensome; however, gowns and gloves are a necessity to prevent spreading *C difficile* to other patients. Wearing gowns limits potential contamination, especially when nurses come in contact with bodily fluids,[30,59] and inhibits cross-contamination to other patients and surfaces. After removing personal protective equipment, nurses must engage in thorough hand washing with soap and running water.

Clostridium difficile may thrive on frequently touched hospital surfaces like light switches, door knobs, call lights, television remote controls, and telephones.

Vigilant environmental cleaning is necessary because *C difficile* may thrive on frequently touched hospital surfaces, such as light switches, door knobs, call lights, television remote controls, and telephones. These surfaces should be cleaned with a hypochlorite-based disinfectant or a dilute (1:10) solution of bleach.[59]

Another key feature in preventing the spread of CDI is to have dedicated equipment for patients who are in isolation. Use of disposable blood pressure cuffs and thermometers can decrease rates of infection.[60] If equipment must be shared, it must be disinfected according to hospital policy immediately after use and before it is used for another patient. Patients with CDI should be placed in private rooms with their own bathrooms if possible. If they are placed in a semiprivate room, they must

have their own commodes.[60] Critical care nurses must be cognizant of the emotional stress associated with being in isolation.[6,59] A patient's access to the television, telephone, and call bell system should be maintained so the patient knows he or she is not totally disconnected. Patients should also be assessed for depression and sadness due to social isolation.

Assessing and Treating CDI Patients' Underlying Illnesses

CDI infections in patients who are hospitalized occur for many reasons. Critical care patients who have CDI require assessment and monitoring not only to determine the progress of their underlying illness but also to prevent complications of CDI, such as dehydration. Regular assessment of vital signs, hemodynamic status, intake, output, and daily weight is essential for rapid detection of dehydration.

Laboratory values such as serum levels of albumin, lactate, and electrolytes must be tracked. Serum lactate and albumin levels are assessed to differentiate the severity of the disease (see Table 3). Holistic assessment of all systems is also important to rapidly detect systemic manifestations.

Promoting Comfort and Maintaining Skin Integrity

Patients who have frequent loose stools, abdominal pain, and cramping are physically uncomfortable, especially if they are incontinent of stool. If a patient reports these findings, analgesics may be administered. Some patients may be extremely embarrassed if they are incontinent of feces; consequently, treating patients with dignity and respect is paramount to respectful compassionate nursing care.

In order to maintain skin integrity, patients should be cleaned promptly after each episode of incontinence and have skin creams or ointments applied to prevent breakdown. Nursing measures to prevent skin breakdown in patients with CDI are challenging because of the frequency, amounts, and characteristics of the stool. These patients require assessment by using a skin score and may require use of an indwelling bowel catheter system to divert the loose stool away from the skin to prevent excoriation and formation of pressure ulcers. Table 7 is a nursing care plan for patients with CDI.[57]

Establishing Clinical Practice Guidelines

Because the risks associated with FMT are low and the outcomes are positive, including a better quality of life and fewer hospitalizations, critical care nurses must accept FMT as a treatment modality with promising outcomes. Although standards of care and nursing clinical practice guidelines for FMT are being developed, critical care nurses will play a pivotal role establishing these guidelines and in implementing the latest standards of the ACG and the Infectious Diseases Society of America to ensure safe, effective patient care.[4]

Future Research

FMT has shown efficacy for RCDI, and research on the usefulness of this procedure for other conditions (eg, inflammatory bowel disease, irritable bowel syndrome, obesity, Parkinson disease, anxiety, schizophrenia, obsessive-compulsive disorders, autism) is under way. Future research depends on the outcomes of randomized control trials such as the Fecal Therapy to Eliminate Associated Long-standing Diarrhea trial[2,4,47] and findings from the Human Microbiome Project. As more patients and health care providers learn to overcome the unpleasant thought of FMT and more patients with RCDI are treated with FMT, critical care nurses will learn from the outcomes and develop future research.

FMT has the potential to enhance resistance to infection and reduce inflammatory diseases. Further targeted manipulation of microbial populations is a growing focus of investigation. The most important manipulation, depending on the microbiota composition and the recipient's genotype, could range from proinflammatory to anti-inflammatory effects.[4] Although the impact of FMT on a recipient's immune system is complex and unpredictable, ongoing discovery of commensal microbes and investigations of the effects of microbes on the host are important.

Summary

CDI continues to be a vexing health care problem for patients and clinicians alike. Because the prevalence of RCDIs is increasing, critical care nurses need to take further steps to be proactive in preventing CDI while at the same time remaining open-minded to alternative treatments such as FMT. Increasing evidence supports the role of FMT in the treatment of patients with RCDI. Critical care nurses must collaborate with hospitalists

Table 7 Nursing care plan for patients with diarrhea due to *Clostridium difficile* infection[a]

Nursing diagnosis	Nursing goals	Nursing interventions and rationales
Diarrhea related to infection by *C difficile* as evidenced by >3 loose stools per day	Patient will have no diarrhea	1. Assess the frequency of defecation (consistency and color), body temperature, abdomen (inspect, auscultate, palpate, and percuss), and history of antibiotic use. **Rationale:** Diarrhea may be due to *C difficile* infection. 2. Teach patient to keep a journal. **Rationale:** Information in the journal will help determine the treatment plan. 3. Seek to identify the cause of diarrhea. **Rationale:** The cause determines course of treatment. 4. Obtain stool specimens. **Rationale:** Results of laboratory tests can be used to rule out an infectious process. If *C difficile* infection is diagnosed, do NOT give medications that slow peristalsis. 5. Use standard precautions and possibly contact isolation. **Rationale:** Measures are needed to prevent spread of infection from patient to patient or to the health care worker. 6. Assess geriatric patients for the presence of impaction, ileus, and perforation. **Rationale:** Patients with an impaction have leakage of mucus or liquid stool around the impaction.
Risk for ineffective tissue perfusion related to decreased intravascular volume due to diarrhea associated with infection by *C difficile*	Patient's blood pressure and heart rate will remain within baseline values	1. Monitor vital signs. **Rationale:** Decreased blood pressure and increased heart rate are signs of dehydration. 2. Assess skin turgor. **Rationale:** Delayed skin turgor over sternum is a sign of dehydration. 3. Measure stool amounts. **Rationale:** Diarrhea is defined as passing more than 300 g of loose stool in 24 hours. The amount of diarrhea should be decreasing as treatment measures are successful. 4. Monitor for changes in urine output. **Rationale:** A decrease in urine output may signify dehydration and decreased renal perfusion. 5. Monitor for changes in mental status, restlessness, dysrhythmias, tachycardia, and cyanosis. **Rationale:** These findings may indicate dehydration.
	Patient will be free of signs of malnutrition	1. Monitor for signs of malnutrition (bruises, dry pale skin, muscle wasting, and cheilosis). **Rationale:** Brittle hair, dry and pale skin, and muscle wasting are signs of malnutrition. 2. Evaluate results of laboratory tests such as serum levels of albumin, total protein, prealbumin, transferrin, electrolytes, hemoglobin and hematocrit, and glucose. **Rationale:** Serum albumin level <3.5 g/dL is considered an indicator of risk of poor nutritional status. All other noted laboratory results will be low. 3. Track daily weights. **Rationale:** Tracking allows ready detection of weight trends and maintenance of a healthy body weight. 4. Collaborate with dietary staff. **Rationale:** Collaboration allows a multidisciplinary approach to patient care. 5. Note food intake and assess patient's ability to eat. **Rationale:** Stressors such as being in an intensive care unit and anticipating a stool transplant may cause a decrease in appetite. 6. Monitor patient for gastrointestinal bleeding by testing stools and gastric contents for guaiac. **Rationale:** Stressors as noted earlier may cause bleeding.
Anxiety related to change in health status and social isolation due to diarrhea	Patient will identify and verbalize symptoms of anxiety and report a decrease in anxiety	1. Assess the patient's level of anxiety and physiological reactions to anxiety (eg, tachycardia, tachypnea, nonverbal expressions of anxiety). **Rationale:** Anxiety may cause deleterious effects on patient recovery. 2. Encourage use of coping skills used successfully in the past. **Rationale:** Previously used coping skills for dealing with anxiety may help here. 3. Explain and teach the patients interventions that may reduce anxiety. **Rationale:** When possible remove sources of anxiety. 4. Decrease anxiety by using therapeutic touch. **Rationale:** Use therapeutic touch, and provide backrubs and massage. 5. Determine if patients feel socially isolated. **Rationale:** Patients in contact isolation often feel this way. Be sure the patient has the call bell and knows how to use the television and telephone.

[a] Based on information in Haugen and Galura.[57]

and gastroenterologists to provide patients with the most current treatment options. FMT is a promising inexpensive treatment for RCDI, with cure rates close to 100%. As FMT continues to develop, critical care nurses will play a unique role in helping patients with RCDIs find hope for healing. CCN

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None reported.

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To learn more about fecal transplant, read "*Clostridium difficile* Infection: Clinical Challenges and Management Strategies" by Walters and Zuckerbraun in *Critical Care Nurse*, August 2014;34:24-33. Available at **www.ccnonline.org**.

References

1. Knight CL, Surawicz CM. *Clostridium difficile* infection. *Med Clin North Am.* 2013;97(4):523-536, ix.
2. Surawicz CM, Brandt LJ, Binion DG, et al. Guidelines for diagnosis, treatment, and prevention of *Clostridium difficile* infections. *Am J Gastroenterol.* 2013;108(4):478-498.
3. Kaseb HO, Novotne T. The changing face of *Clostridium difficile* in critical care. *Nurs 2013 Crit Care.* 2013;8(3):26-34.
4. Cohen SH, Gerding DN, Johnson S, et al; Society for Healthcare Epidemiology of America; Infectious Diseases Society of America. Clinical practice guidelines for *Clostridium difficile* infection in adults: 2010 update by the Society for Health Epidemiology of America (SHEA) and the Infectious Diseases Society of America. *Infect Control Hosp Epidemiol.* 2010;31:431-455. doi:10:101086/651706.
5. Banning M. Ageing and *C difficile* infection—the immune and gastrointestinal impact. *Gastrointest Nurs.* 2011;9(4):42-47.
6. Centers for Disease Control and Prevention. CDC Vital Signs: stopping *C difficile* infections. http://www.cdc.gov/VitalSigns/Hai/StoppingCdifficile. Published March 2012. Accessed December 30, 2014.
7. Hessen MT. In the clinic: *Clostridium difficile* infection. *Ann Intern Med.* 2010; 153(7):ITC4-1-ITC4-15. doi:10.7326/0003-4819-153-7-201010050-01004.
8. Badger VO, Ledeboer NA, Graham MB, Edmiston CE. *Clostridium difficile*: epidemiology, pathogenesis, management, and prevention of a recalcitrant healthcare-associated pathogen. *JPEN J Parenter Enteral Nutr.* 2012; 36(6):645-662.
9. Simor AE. Diagnosis, management, and prevention of *Clostridium difficile* infection in long-term care facilities: a review. *J Am Geriatr Soc.* 2010; 58(8):1556-1564.
10. Gough E, Shaikh H, Manges AR. Systematic review of intestinal microbiota transplantation (fecal bacteriotherapy) for recurrent *Clostridium difficile* infection. *Clin Infect Dis.* 2011;53(10):994-1002.
11. Cober ED, Malani PN. *Clostridium difficile* infection in the "oldest" old: clinical outcomes in patients aged 80 and older. *J Am Geriatr Soc.* 2009; 57(4):659-662.
12. Department of Health. *Clostridium difficile*: guidance, data and analysis. https://www.gov.uk/government/collections/clostridium-difficile-guidance-data-and-analysis. Accessed February 2, 2015.
13. The Fecal Transplant Foundation. What is FMT? http://thefecaltransplantfoundation.org/what-is-fecal-transplant/. Accessed December 30, 2014.
14. Agito MD, Atreja A, Rizk MK. Fecal microbiota transplantation for recurrent *C difficile* infection: ready for prime time? *Cleve Clin J Med.* 2014;80(2):101-108.
15. Myers F. Beyond mainstream: making the case for fecal bacteriotherapy. *Nursing.* 2011;41(12) 50-53.
16. Guo B, Harstall C, Louie T, Veldhuyzen van Zanten S, Dieleman LA. Systematic review: faecal transplantation for the treatment of *Clostridium difficile*-associated disease. *Aliment Pharmacol Ther.* 2012;35(8):865-875.
17. Patel NC, Griesbach CL, DiBaise JK, Orenstein R. Fecal microbiota transplant for recurrent *Clostridium difficile* infection: Mayo Clinic in Arizona experience. *Mayo Clin Proc.* 2012;88(8):799-805.
18. Lofland D, Josephat S, Partin S. Fecal transplant for recurrent *Clostridium difficile* infection. *Clin Lab Sci.* 2013;26(3):131-135.
19. Khoruts A, Dicksved J, Jansson JK, Sadowsky MJ. Changes in the composition of the human fecal microbiome after bacteriotherapy for recurrent *Clostridium difficile*-associated diarrhea. *J Clin Gastroenterol.* 2010; 44(5):354-360.
20. Shih C. The gut flora. *J Lancaster Gen Hosp.* 2013;8(4):114-117.
21. Khanna S, Tosh PK. A clinician's primer on the role of the microbiome in human health and disease. *Mayo Clin Proc.* 2014;89(1):107-114.
22. Suwantarat N, Bobak DA. Fecal bacteriotherapy for recurrent *Clostridium difficile* infection: what's old is new again? *Curr Infect Dis Rep.* 2013; 15(2):101-103.
23. Warny M, Pepin J, Fang A, et al. Toxin production by an emerging strain of *Clostridium difficile* associated with outbreaks of severe disease in North America and Europe. *Lancet.* 2005;366(9194):1079-1084.
24. Taggart H, Bergstrom L. An overview of the microbiome and the effects of antibiotics. *J Nurse Pract.* 2014;10(7):445-450.
25. National Institutes of Health. Human microbiome project. http://commonfund.nih.gov/hmp/index. Accessed February 2, 2015.
26. Shaw W. Increased urinary excretion of a 3-(3-hydroxyphenyl)-3-hydroxypropionic acid (HPHPA), an abnormal phenylalanine metabolite of *Clostridia* spp in the gastrointestinal tract, in urine samples from patients with autism and schizophrenia. *Nutr Neurosci.* 2010;13(3):135-143.
27. Kelly CR, Ihunnah C, Fischer M, et al. Fecal microbiota transplant for treatment of *Clostridium difficile* infection in immunocompromised patients. *Am J Gastroenterol.* 2014;109(7):1065-1071.
28. Li L, Somerset S. The clinical significance of the gut microbiota in cystic fibrosis and the potential for dietary therapies. *Clin Nutr.* 2014;33(4): 571-580.
29. Ridaura VK, Faith JJ, Rey FE, et al. Gut microbiota from twins discordant for obesity modulate metabolism in mice. *Science.* 2013; 341(6150):1241214.
30. Keske LA, Letizia M. *Clostridium difficile* infection: essential information for nurses. *Medsurg Nurs.* 2010;19(6):329-333.
31. Sebaihia M, Wren BW, Mullany P, et al. The multidrug-resistant human pathogen *Clostridium difficile* has a highly mobile, mosaic genome. *Nature Genet.* 2006;38(7):779-786.
32. Grossman S, Mager D. *Clostridium difficile*: implications for nursing. *Medsurg Nurs.* 2010;19(3):155-158.
33. Vonberg R. Infection control measures to limit the spread of *Clostridium difficile*. *Clin Microb Infect.* 2008;14(2):20-26.
34. McFarland LV. Renewed interest in a difficult disease: *Clostridium difficile* infections—epidemiology and current treatment strategies. *Curr Opin Gastroenterol.* 2009;25(1):24-35.
35. Gardner E, Meghani N, Mancuso P, Thomson A. Recognizing metronidazole resistant *C difficile. Nurse Pract.* 2011;36(11):8-11.
36. Brandt LJ, Reddy SS. Fecal microbiota transplant for recurrent *Clostridium difficile* infection. *J Clin Gastroenterol.* 2011;45(suppl):S159-S167.
37. Kelly CR, de Leon, L, Jasutkar N. Fecal microbiota transplantation for relapsing *Clostridium difficile* infection in 26 patients: methodology and results. *J Clin Gastroenterol.* 2012;46(2):145-149.
38. Brandt LJ, Aroniadis OC, Mellow M, et al. Long-term follow-up of colonoscopic fecal microbiota transplant for recurrent *Clostridium difficile* infection. *Am J Gastroenterol.* 2012;107(7):1079-1087.
39. Bakken J. Fecal bacteriotherapy for recurrent *Clostridium difficile* infection. *Anaerobe.* 2009;15(6):285-289.
40. Garborg K, Waagsbø B, Stallemo A, Matre J, Sundøy A. Results of faecal donor instillation therapy for recurrent *Clostridium difficile*-associated diarrhea. *Scand J Infect Dis.* 2010;42(11-12):857-861.
41. Pamer EG. Fecal microbiota transplantation: effectiveness, complexities, and lingering concerns. *Mucosal Immunol.* 2014;7:210-214.
42. Mitchell I, Shropshire K, Ruel J. *Clostridium difficile* infection and fecal bacteriotherapy. *Gastroenterol Nurs.* 2013:36(1):42-50.
43. Eiseman B, Silen W, Bascom GS, Kauvar AJ. Fecal enema as an adjunct in the treatment of pseudomembranous enterocolitis. *Surgery.* 1958;44(5): 854-859.

44. Schwan A, Sjölin S, Trottestam U, Aronsson B. Relapsing *Clostridium difficile* enterocolitis cured by rectal infusion of homologous faeces. *Lancet.* 1983;2(8354):845.
45. Louie T, Cannon K, O'grady H, Wu K, Ward L. Fecal microbiome transplantation (FMT) via oral fecal microbial capsules for recurrent Clostridium difficile infection (rCDI). Abstract 89. Presented at: IDWeek 2013; October 2-6, 2013; San Francisco, CA. https://idsa.confex.com/idsa/2013/webprogram/Paper41627.html. Accessed December 30, 2014.
46. Brandt L. Faecal microbiota transplant. *Gastrointest Nurs.* 2012;10(2):6-7.
47. Owens C, Broussard E, Surawicz C. Fecal microbiota transplantation and donor standardization. *Trends Microbiol.* 2013;21(9):443-445.
48. Hamilton MJ, Weingarden AR, Sadowsky MJ, Khoruts A. Standardized frozen preparation for transplantation of fecal microbiota for recurrent *Clostridium difficile* infection. *Am J Gastroenterol.* 2012;107(5):761-767.
49. Aas J, Gessert CE, Bakken JS. Recurrent *Clostridium difficile* colitis: a series involving 18 patients treated with donor stool administered via a nasogastric tube. *Clin Infect Dis.* 2003;36(5):580-585.
50, Silverman MS, Davis I, Pillai DR. Success of self-administered home fecal transplantation for chronic *Clostridium difficile* infections. *Clin Gastroenterol Hepatol.* 2010;8(5):471-473.
51. van Nood E, Vrieze A, Nieuwdorp M, et al. Duodenal infusion of feces for recurrent *Clostridium difficile*. *N Engl J Med.* 2013;368:407-415.
51. Mattila E, Uusitalo-Seppälä R, Wuorela M, et al. Fecal transplantation, through colonoscopy, is effective therapy for recurrent *Clostridium difficile* infection. *Gastroenterology.* 2012;142(3):490-496.
53. Avery L, Hasan M. Fecal bacteriotherapy for Clostridium difficile infections—its time has come. *Clin Microbiol Newsl.* 2013;35(15):119-124.
54. Orenstein R, Griesbach CL, DiBaise JK. Moving fecal microbiota transplantation into the mainstream. *Nutr Clin Pract.* 2013;28(5):589-598.
55. Bakken JS, Borody T, Brandt LJ, et al; Fecal Microbiota Transplantation Workgroup. Treating *Clostridium difficile* infections with fecal microbiota transplantation. *Clin Gastroenterol Hepatol.* 2011;9(12):1044-1049.
56. Schwartz M, Gluck M, Koon S. Novovirus gastroenteritis after fecal microbiota transplant for treatment of *Clostridium difficile* infections despite asymptomatic donors and lack of sick contacts. *Am J Gastroenterol.* 2013;108:1367.
57. Haugen N, Galura S. *Ulrich & Canale's Nursing Care Planning Guides: Prioritization, Delegation, and Critical Thinking.* 7th ed. St Louis, MO: Elsevier; 2013.
58. MacLeod-Glover N, Sadowski C. Efficacy of cleaning products for *C difficile*: environmental strategies to reduce the spread of *Clostridium difficile*-associated diarrhea in geriatric rehabilitation. *Can Fam Physician.* 2010;56(5):417-423.
59. Landers T, Abusalem S, Coty MB, Bingham J. Patient-centered hand hygiene: the next step in infection prevention. *Am J Infect Control.* 2012; 40(4)(suppl):S11-S17.
60. Johnson J. Setting up an isolation and treatment unit for *C difficile*. *Nurs Times.* 2008;104(25):30-31.

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Fecal Microbiota Transplant to Treat Recurrent *Clostridium difficile* Infections

Facts

Clostridium difficile infections (CDIs) are some of the most common health care–associated infections in hospitalized patients. The incidence of severe and recurrent CDIs (RCDIs) has increased because of a new hypervirulent strain *C difficile* that is less responsive to traditional medications. Patients with RCDI often are treated in an intensive care unit (ICU). Fecal microbiota transplant (FMT) is an adjunctive, cost-effective treatment for patients with RCDI.

- *C difficile* spreads from person to person or surface to person via the fecal-oral route.
- Although *C difficile* flourishes in anaerobic conditions such as the gut, it can survive on a variety of surfaces for more than 3 months.
- CDI may be a side effect of antimicrobial therapy.
- About half of CDIs occur in patients less than 65 years old; however, patients more than 65 years old account for about 90% of the deaths due to CDI.
- In severe CDI, at least one of the following is present: hypotension, fever, leukocytosis, elevated serum lactate levels, any evidence of end-organ failure, and admission to an ICU.
- Typical indications of fulminant colitis, the most serious manifestation of CDI, are severe abdominal pain, diarrhea, high fever, chills, leukocytosis, and abdominal distention.
- Diagnostic testing for CDI includes laboratory and imaging studies. Laboratory tests include toxin enzyme immunoassays, toxigenic cultures, and nucleic acid amplification tests.
- The first-line treatment for mild CDI is oral metronidazole. For patients with moderate and severe signs and symptoms, the drug of choice is oral vancomycin.
- In patients unable to take medications orally, vancomycin can be administered via enema, colostomy, ileostomy, or nasogastric tube.
- In FMT the stool of a healthy donor is mixed with physiological saline or water and then instilled in the upper or lower part of the gastrointestinal tract of a patient with RCDI. The donor fecal material usually is administered via nasogastric tube, nasoduodenal tube, colonoscopy, oral fecal capsules, and self-administered enemas.
- The biggest risks associated with FMT are transmission of undetected infectious bacterial agents and side effects such as headaches, sore throat, and various gastrointestinal complaints.

Role of Critical Care Nurses

Patients with severe infections and patients who have fulminant CDI colitis often require treatment in the critical care environment. Critical care nurses have many responsibilities, including the following:

- Recognizing FMT as an option for patients with CDI
- Identifying patients at risk for CDI
- Meticulous hand washing
- Vigilant antibiotic stewardship
- Maintaining enteric contact isolation and vigilant environmental cleansing
- Treating CDI patients' underlying illnesses
- Promoting comfort and maintaining skin integrity
- Establish clinical practice guidelines
- Educating patients and their significant others

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