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# *Clostridium difficile* Infection and Fecal Bacteriotherapy

## ABSTRACT

*Clostridium difficile*, also called “*C. diff*,” is a gram-positive bacillus associated with nosocomial infections involving diarrhea, most often seen in developing countries. The severity of *C. diff*-associated diarrhea varies tremendously from mild and self-limiting to fulminant and life-threatening. *C. diff* has become an extremely important pathogen in community health but can be minimized with attention to proper hygiene. This article presents a case study regarding the treatment and management options of *C. diff* infection using a recent update of clinical guidelines for patient management.

**C***lostridium difficile*, also called “*C. diff*,” is a gram-positive bacillus that has been found to be the culprit in nosocomial infections involving diarrhea in several developed countries (Vaishnavi, 2009), including the United States. This discussion will focus on its implications related to pseudomembranous colitis (PMC) and antibiotic-associated diarrhea.

## Background

According to Vaishnavi (2009), *C. diff* is the cause of almost all cases of PMC and is responsible for 15%–25% of diarrhea that can be associated with antibiotic use. The severity of *C. diff*-associated diarrhea (CDAD) varies tremendously from mild and self-limiting to fulminant PMC that could be life-threatening (Bakken, 2009). *C. diff* infection (CDI) has become a major issue in healthcare recently because of a significant

increase in incidence and severity in CDAD. From 1999 to 2004, mortality rates related to CDI and CDAD significantly increased from 5.7 million deaths per 1 million persons to a whopping 23.7 million (Drekonja et al., 2011).

*C. diff* normally lives within the gut and was first discovered in the fecal flora of healthy newborns (Vaishnavi, 2009). Today it can be found in the stool of 50% of all healthy neonates (Vaishnavi, 2009). In the gastrointestinal (GI) tract of a healthy individual, it is a component of the normal flora, but in the presence of immunosuppression or changes in the normal environment within the gut, it seizes its opportunity to flourish (Vaishnavi, 2009).

Treatment guidelines have been updated because of increased virulence and incidence (Garborg, Waagsbo, Stallemo, Matre, & Sundoy, 2010). Since the release of the Society for Healthcare Epidemiology of America position paper on *C. diff* in 1995, there have been several updates related to the epidemiology and treatment of CDI (Cohen et al., 2010). *C. diff* has become an extremely important pathogen in community health, especially with the emergence of a more virulent strain (Cohen et al., 2010). There have also been new data, which are discussed later in the article, on metronidazole as a treatment option for CDI (Cohen et al., 2010). The recently updated guideline seeks to address this new information regarding the epidemiology, diagnosis, treatment, infection control, and environmental management related to CDI (Cohen et al., 2010).

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## Risk Factors Related to CDAD/CDI

There are several factors that may put one at an increased risk for CDI. As mentioned before, *C. diff* flourishes in the presence of extreme changes within the GI tract. Most of these changes occur from human intervention. These risk factors include most often antibiotic (antimicrobial), immunosuppressant, proton pump inhibitor, or chemotherapy use (Vaishnavi, 2009).

Antimicrobial use, which includes antibiotics, antifungals, and antivirals, is the biggest and most notable risk factor of all (Vaishnavi, 2009). In most cases, a recent history of antibiotic therapy can be found and nosocomial transmission is normally common (Drekonja et al., 2011). According to Vaishnavi (2009), CDAD prevalence is directly linked to the emergence of broad spectrum antibiotics in the 1960s and 1970s.

There are several factors that increase the risk of contracting CDI that are not related to medication use. These risk factors are directly host related. The chance of CDAD increases with older age due to the ample use of antibiotics in the elderly and the presence of other comorbidities (Vaishnavi, 2009). Populations who were previously considered to be at low risk for CDI are now becoming a concern. Data now suggest that peripartum women and individuals within the community, including those not previously exposed to healthcare, are now at an increased risk for contracting CDI (Cohen et al., 2010).

There are also risks associated with the actual pathogen, such as the presence of large amounts of *C. diff* molecules within a population, adhesiveness of the strain, production of cytotoxins by the actual strain, and the presence of other organisms that influence toxin production and activity (Vaishnavi, 2009). The patient's milieu also plays a significant role in CDI transmission. Hospital units, nursing homes, and day-care facilities are highly infectious and serve as huge reservoirs for *C. diff* because of improper hand hygiene and inadequate sterilization of equipment (Vaishnavi, 2009).

Healthcare providers know that thorough hand hygiene is a key component in reduction of transmission of most nosocomial infections. Improper hand hygiene is a direct method of transmission of *C. diff*, as well as contaminated stethoscopes, commodes, bathtubs, and other various types of medical equipment (Vaishnavi, 2009). This reinforces the need for proper sterilization of medical equipment and the availability of the proper disinfectants for staff use to control the transmittal rate of nosocomial infections. Vaishnavi (2009) reported that there are approximately 3 million cases of hospital-acquired diarrhea a year within the United States and the annual cost is projected to supersede \$1.1 billion each year.

## Case Study

A patient was seen by her primary care provider (PCP) after 2 weeks of having intermittent diarrhea. The patient's stool culture was negative for *C. diff* at that time. The patient then returned to her PCP's office 1 week later due to persistent diarrhea, and a computed tomographic (CT) scan was completed. The results of the CT scan showed colonic dilation and diffuse colonic wall thickening, consistent with colitis. The patient's symptoms persisted, she was admitted into the hospital with dehydration and was then diagnosed with a mild to moderate case of CDI.

**Chief complaint:** Abdominal pain with diarrhea.

**History of the present illness:** A 44-year-old African American woman presented to the emergency department, with complaint of intermittent abdominal cramps with watery diarrhea that persisted for 3–4 weeks. She rated her pain as 8/10 and denied radiation. Pain was localized to the epigastric region. The patient complained of 6–8 watery diarrhea episodes per day. Initially she complained of 10–12 watery stools, but started loperamide (Imodium) 4–6 times per day at the onset of symptoms. She was seen by her PCP 1 week ago, with complaint of diarrhea. Finding from her digital rectal examination was negative for fecal occult blood and comprehensive metabolic panel, and complete blood cell (CBC) count with differential was within normal limits. A CT scan of the abdomen was performed for persistent abdominal pain, but results are unknown at this time. Eating and drinking fluids made her diarrhea worse, so she had been limiting her intake. The patient denied nausea, vomiting, fever, chills, hematochezia, or melena. She denied recent travel outside of the country. She was treated for a respiratory infection with levofloxacin (Levaquin) 6 weeks ago following treatment failure with azithromycin (Zithromax). The patient stated, "I get those a lot."

**Differential diagnosis:**

- Antibiotic-associated diarrhea
- Benign diarrhea
- *C. diff* colitis
- Celiac disease
- Crohn disease
- Gastroenteritis
- Irritable bowel syndrome
- Ischemic colitis
- Norwalk disease
- Viral hepatitis

**Prior medical history:** Chronic obstructive pulmonary disease (COPD), uterine fibroids, frequent upper respiratory infection.

**Medications:** Advair diskus 250/50 twice daily, Albuterol inhaler 1–2 puffs Q 4 hours as needed, loperamide 2 mg orally Q 4 hours as needed.

**Allergies:** Denies food, drug, or seasonal allergies.

**Prior surgical history:** Cholecystectomy 2001, total abdominal hysterectomy 2005.

**Transfusions:** Not applicable.

**Immunizations:** Up to date; influenza 10/18/11.

**Preventative healthcare:** Last physical examination 2011. Denies dental care or vision examinations. Last pap smear 2005. Last pelvic examination 8/2011. Last mammogram 8/2011.

**Family history:** Denies diabetes, COPD, hypertension, heart disease, cancer, colitis, or irritable bowel syndrome.

**Personal/social history:** Single, currently unemployed, lives with two children. Denies alcohol or illicit drug abuse, +40 pack/year tobacco abuse history; currently smoking 1 pack/day. Liquid diet secondary to diarrhea. States she has lost 22 lb since the onset of symptoms. Denies eating raw or undercooked meat/eggs.

### Review of Systems

**General:** + fever. Denies chills or malaise. Able to perform daily duties without difficulty.

**Skin:** Complains of dry skin. Denies cracks or open lesions in skin.

**Nose:** Denies nasal drainage, sneezing, and nasal congestion.

**Mouth/oropharynx:** Denies sore throat, hoarseness, difficulty eating and swallowing, voice changes, sores in mouth, sores, and lesions on tongue. Denies difficulty chewing or swallowing.

**Neck:** Denies any neck pain, stiffness, tenderness, or enlarged lymph nodes.

**Cardiovascular:** Denies palpitations, chest pain/discomfort, or syncope.

**Chest/lungs:** Recent COPD exacerbation. Denies dyspnea, cough, congestion, orthopnea, dyspnea, or pain. Normally uses albuterol inhaler 1–2 times daily approximately 3 times weekly; usually unable to participate in strenuous activity without inhaler use. No home oxygen supplementation.

**Gastrointestinal:** Complains of epigastric abdominal pain, cramping, anorexia, bloating, and watery diarrhea. Denies nausea, vomiting, heartburn, and indigestion. Denies the use of nonsteroidal anti-inflammatory drugs, antacids, or proton pump inhibitors for abdominal pain. Computed tomographic scan of abdomen and pelvis shows colonic dilation and diffuse colonic wall thickening.

**Genitourinary:** Voiding 3–4 times per day. Denies any voiding issues.

### Physical Examination

**Vital Signs:**

- Blood pressure 100/54 mmHg sitting and 102/60 mmHg standing

- Heart rate 120 beats/minute sitting and standing
- Temperature 99.5°F orally
- Respiratory rate 24 per minute
- Saturation of peripheral oxygen (SPO<sub>2</sub>) 99% 2-L nasal cannula
- 5 feet, 4 inches tall
- 220 lb
- Basal metabolic index 33.5.

**General:** Visibly uncomfortable obese African American woman sitting upright on hospital bed with hands over abdomen. Alert and oriented 3×, mood and affect congruent.

**Skin:** Skin intact, warm, dry, color within normal limits for ethnicity. Normal turgor.

**Eyes:** Eyes symmetric and aligned; no tearing or drainage; conjunctivae pink and moist; pupils equally responsive and reactive to light and accommodation; extraocular movements intact.

**Nose:** Mucosa pink, moist, no drainage.

**Mouth:** Oral buccal mucosa pink and moist with no lesions.

**Neck:** Neck supple. No thyromegaly, nodules, or tenderness noted. No lymphadenopathy.

**Chest/lungs:** Chest symmetrical, normal contour, anterior to posterior diameter less than lateral. Respiratory effort symmetric, even and nonlabored. Lungs clear to auscultation without adventitious sounds bilaterally.

**Cardiovascular:** No jugular venous distention. S1, S2 strong; no extra sounds or murmurs heard; no carotid bruits bilaterally. +3 peripheral pulses palpated symmetrically in all extremities. Capillary refill less than 2 seconds. No peripheral edema.

**Abdomen:** Abdomen symmetrical and rounded. Hyperactive bowel sounds 4 times quadrants. Abdomen tympanic to percussion in all four quads; abdomen soft and tender with light and deep palpation in all four quadrants. No organomegaly. Abdominal reflexes present.

**Rectal:** Normal rectal tone; + tenderness; no masses or lesions noted. Stool positive for trace red blood.

**Neurological:** Cranial nerves I–XII grossly intact. Smooth rapid rhythmic alternating movements. Deep tendon reflexes + 2 in all extremities.

### Laboratory Tests Completed

- Comprehensive metabolic panel (electrolytes/Chem 7, alanine transaminase, aspartate aminotransferase, blood urea nitrogen, creatinine)
- CBC count with differential
- Urinalysis
- Stool cultures for ova and parasites
- Stool for *C. diff* toxins

**Pertinent laboratory test findings:** Urine analysis negative, white blood cell count 16.9 q/dl, hemoglobin 14.8, hematocrit 44.2, platelets 165, sodium ( $\text{Na}^+$ ) 148, potassium ( $\text{K}^+$ ) 3.1, chloride ( $\text{Cl}^-$ ) 98, carbon dioxide ( $\text{CO}_2$ ) 20, blood urea nitrogen 39, creatinine 1.3, random glucose 100, aspartate aminotransferase 34, alanine transaminase 27. Stool positive for *C. diff*.

**Assessment:**

- *C. diff* colitis
- Dehydration
- Leukocytosis
- Hypokalemia
- Tobacco abuse
- COPD
- Tachycardia secondary to dehydration

**Plan:**

1. Check stool for *C. diff*. 3× samples.
2. Stool culture, ova, and parasites.
3. Discontinue loperamide until infectious processes are ruled out.
4. Admit patient to medical–surgical unit. Start D5.45% normal saline (NS) at 150 ml/hour. Repeat basic metabolic panel (BMP) in morning. Check electrocardiogram.
5. Repeat CBC count with differential in 24 hours.
6. Blood cultures if temperature is higher than 101.0°F.
7. Potassium chloride (KCl) 10 meq intravenous push piggy back 4× doses q 1 hour. Repeat potassium ( $\text{K}^+$ ) 2 hours after the last dose.
8. Deep vein thrombosis prophylaxis; heparin 5,000 units SubQ twice daily.
9. Gastrointestinal prophylaxis; famotidine (Pepcid) 20 mg IVP q day.
10. Smoking cessation education.

## Discussion of Patient Outcome

Once the diagnosis of CDI was established, the patient was transferred to an isolation room and started on metronidazole 500 mg orally every 8 hours and vancomycin 250 mg orally every 6 hours as the first-line treatment of choice (Cocanour, 2010). According to Cocanour (2010), vancomycin is also the drug of choice in severe CDI cases, and the dose is increased to 500 mg orally every 6 hours when the condition is complicated by ileus, shock, or megacolon. The goal of this patient's therapy was to restore hydration, treat the infections, and prevent the development of any further complications of CDI. Treatment of CDI is discussed more thoroughly in the treatment section of the CDI information later.

## Conclusion of the Case Study

The patient in this case developed CDI after being treated with levofloxacin for an exacerbation of

COPD. It is presumed that she was started on this broad-spectrum respiratory fluoroquinolone due to failed treatment with azithromycin and the cost-effectiveness of levofloxacin as a second-line therapy. She has a significant history of COPD exacerbations that occur yearly during the fall and winter seasons, and usually requires antibiotic therapy every winter because of her tobacco abuse and risk for *Haemophilus influenza* infection (McPhee & Papadakis, 2011). Some of the consequences and complications that this patient experienced from CDI included weight loss, dehydration, tachycardia, and volume loss secondary to anorexia and diarrhea.

The patient's hydration status was corrected with intravenous fluids for 1 day. She received a total of 4 L of D5.45% NS and 40 meq of intravenous potassium, and her electrolytes returned to normal limits. She was continued on metronidazole and vancomycin, as an inpatient, as mentioned previously.

Patients with CDI in the outpatient arena can be treated with oral metronidazole 500 mg every 8 hours (Vaishnavi, 2009). Contact isolation precautions should be continued in the home setting and should include recommendations for the use of a separate bathroom, if possible, and proper hand and environmental hygiene to avoid the spread of the infectious agent. Once the patient has been on a 10- to 14-day course of antibiotics, their stool should be rechecked for CDI (Vaishnavi, 2009). Patients with CDI should be educated on early signs and symptoms of dehydration such as dry mouth, dry mucous membranes, and dry flakey skin and hair (McPhee & Papadakis, 2011). Patients need to be advised to increase their fluid intake by 1–2 L per day with water or Gatorade to help maintain their electrolyte balance.

Smoking is known to be a hazardous habit and results in a variety of associated complications such as hypertension, COPD, numerous cancers, cardiovascular disease, and much more. It is vital that advanced practice registered nurses (APRNs) take a proactive role in health promotion that addresses the underlying cause of illnesses as opposed to just treating the illness and future complications. Patients who smoke should be assessed for readiness to quit and counseled on smoking cessation at every office visit (Armitage, 2008). This patient did receive literature and resources on smoking cessation and a nicotine patch, while she was admitted to the hospital. She was instructed to follow up with her PCP on discharge for further smoking-cessation management.

Family and provider assistance are essential. Advanced practice registered nurses can actively promote health and reduce risks by individualizing care with the patient and family input, while continuously reevaluating and making adjustments when indicated.



Comorbidities, family history, and patient and family preferences should be considered before implementing a plan (Armitage, 2008).

### What Is Known About *Clostridium difficile*?

#### *Diagnosing CDI*

There is no one way to diagnose CDI. A diagnosis can be made clinically, endoscopically, radiologically, or through identification of *C. diff* and related toxins by assays (Vaishnavi, 2009). Pseudomembranous colitis, which is usually associated with CDI as a late manifestation, can be detected endoscopically as multiple yellow-white plaques in the mucosa of the upper GI tract (Vaishnavi, 2009). Using endoscopy as a diagnostic tool can be extremely beneficial in the late phase of CDI because of the speed and accuracy of the results, as well as the avoidance of major abdominal surgery (Vaishnavi, 2009).

Enzyme immunoassays that test for *C. diff* toxins A and B are rapid tests; however, they are not as sensitive as cell cytotoxin assays so are thought to be suboptimal for diagnosing CDI (Cohen et al., 2010). Enzyme immunoassays typically range from 50% to 90% in sensitivity and 70% to 95% in specificity and approximately 100–1000 pg of the toxin (or toxins) tested for must be present to produce a positive result (Vaishnavi, 2009).

Once the enzyme immunoassay has been performed, the results should be confirmed with a cell cytotoxic assay or toxigenic culture due to a lack in sensitivity (National Guideline Clearinghouse, 2010). To avoid the trouble of ordering multiple tests and to make the process easier, a polymerase chain reaction test, which is rapid and manifests both sensitivity and specificity, can be done; however, the Society for Healthcare Epidemiology and the Infectious Diseases Society of America (2010) expert panel has not yet recommended polymerase chain reaction testing for diagnosing CDI because of the need of more data related to its usefulness in establishing a diagnosis of CDI. If one wishes to order the most sensitive and indispensable epidemiological study, a stool culture would be the test of choice (Society for Healthcare Epidemiology and the Infectious Diseases Society of America, 2010).

When making a clinical diagnosis of CDI, the patient normally presents with a chief complaint of persistent watery, green, malodorous, or bloody diarrhea with abdominal cramps, which are all presumptive symptoms of PMC (Vaishnavi, 2009). The patient's CBC count may reflect leukocytosis and leukocytes may be present in the stool sample; however, neither of the two characteristics may be seen with benign diarrhea (Vaishnavi, 2009). Pseudomembranous colitis can be diagnosed most quickly using endoscopy,

which prevents the need for abdominal surgery when diagnosed early in the course of the disease (Vaishnavi, 2009). As mentioned with the patient in the case study, a CT scan may reveal a diffusely thickened and edematous colonic wall with pericolic inflammation, which may be indicative of PMC and the need for antibiotic therapy or other treatment modalities can be decided (Vaishnavi, 2009).

#### Treatment Options of CDAD/CDI

**Oral antibiotics.** According to the National Guideline Clearinghouse (2010), metronidazole 500 mg 3 times daily for 10–14 days should be used to treat the initial occurrence of mild to moderate CDI; however, vancomycin in suspension form is the drug of choice for an initial episode of severe CDI at a dosage of 125 mg given orally 4 times per day for a duration of 10–14 days. Vancomycin is usually preferred by the patient in capsule form, which may also promote compliance (instead of suspension) due to its relatively bitter taste (Vaishnavi, 2009). On the contrary, oral suspensions are most efficient in maintaining higher concentrations within the GI tract (Vaishnavi, 2009). Because of its poor absorption and bioavailability within the GI tract, vancomycin has been named the drug of choice in severely ill patients suffering from PMC (Vaishnavi, 2009).

Most patients treated with vancomycin respond to treatment with the resolution of diarrhea within 5 days of initiation (Vaishnavi, 2009). The drug of choice, treatment regimen, and route given vary on the basis of disease severity and the presence of any other comorbidities or complications. In a patient unable to take or tolerate oral forms of vancomycin, the medication can be given via nasogastric (NG) tube, long intestinal tube, enema, colostomy, or ileostomy. If any of these routes are impossible, it is given intravenously (Vaishnavi, 2009). Intravenous vancomycin is not the route of choice, because of poor availability in the GI tract, but it is given with the faith that some portion of the dosage will reach the mucosa of the GI tract (Vaishnavi, 2009).

Metronidazole is normally indicated in mild to moderate disease and is the alternative medication in patients or situations where vancomycin is contraindicated (Vaishnavi, 2009). It is sometimes chosen over vancomycin because of its cost-effectiveness in relation to vancomycin (Vaishnavi, 2009). Vaishnavi (2009) reports a success rate of 95% in patients with mild to moderate disease treated with metronidazole. Although there is very little evidence supporting the superiority of vancomycin over metronidazole (Drekonja et al., 2011), metronidazole has several shortcomings in the treatment of CDI.

Metronidazole is almost completely absorbed in the upper GI tract, which lessens its bioavailability in the

colon to nearly nonexistent, and some *C. diff* isolates can be resistant to the drug (Vaishnavi, 2009). For these reasons, vancomycin is the drug of choice in the presence of severe disease or severely ill patients (Vaishnavi, 2009). Metronidazole is also not recommended for long-term use or beyond the first recurrence, because of the increased risk for neurotoxicity (National Guideline Clearinghouse, 2010). Providers should make treatment decisions on the basis of evidence-based practice and in congruence with current treatment guidelines to avoid toxin resistance.

In the 1990s, a group of researchers published a study that confirmed that clinical treatment success was comparable with the use of either metronidazole or vancomycin, but metronidazole was favored because of cost-effectiveness and the emergence of vancomycin-resistant enterococci (Drekonja et al., 2011). In addition, a Cochrane Review performed in 2007 revealed that there was not an antibiotic superior to metronidazole in the treatment of CDI; however, the use of vancomycin is still increasing (Drekonja et al., 2011). According to Drekonja et al. (2011), this trend may be due to the recent data that prove that neither vancomycin nor metronidazole is more efficacious than the other in the treatment of mild to moderate CDI, and both medications have played a role in the emergence of vancomycin-resistant enterococci.

It has also been found that intravenous vancomycin may be easily obtained and more cost-effective than vancomycin capsules when compounded into an oral suspension, which may influence the provider's choice to use vancomycin (Drekonja et al., 2011). The increase in the incidence and severity of CDI may also contribute to the increase in vancomycin use, or it could be related to the providers' experience with achieving success with vancomycin over metronidazole (Drekonja et al., 2011).

Fidaxomicin belongs to the macrolide class, but it is the first macrocycle (a ribonucleic acid polymerase inhibitor) (Grant, 2011). Like vancomycin, because of its absorption in the gut, it is able to potently combat CDI within the GI tract (Grant, 2011). In a clinical trial, the response to fidaxomicin did not supercede that of vancomycin at the treatment cessation; however, its ability to prevent recurrence of CDI/CDAD was greater than that of vancomycin (Grant, 2011). These high recurrence rates with traditional treatment regimens imply the need for new treatment options.

Fidaxomicin is just one newly proposed option. Recent studies showed that the rate of relapse with fidaxomicin was 12.7%–14.6% lower than that of vancomycin. Simply speaking, this amounts to one less reoccurrence for every seven to eight patients who experience recurrence with vancomycin (Grant, 2011). The downfall of using fidaxomicin is directly related to

cost-effectiveness. A 10-day course of fidaxomicin twice daily costs approximately \$2,800 compared with \$1,061 for 125 mg of vancomycin given orally 4 times a day over a 10-day period (Grant, 2011).

**Fecal bacteriotherapy.** Fecal bacteriotherapy is a process that restores the colon homeostasis by instilling normal bacterial flora from a healthy person, or donor, into the GI tract of the affected patient (Cocanour, 2011). The procedure is performed by placing a liquid suspension of stool from a healthy donor into the GI tract of a patient with CDI (Bakken, 2009). Although it is not that commonly heard of, it is not a new procedure. In fact, the first successful fecal transplant was performed in 1958 (Brandt, Borody, & Campbell, 2011). Veterinarians have used a similar procedure termed transfaunation for many years. The first transplantation of enteric bacteria was performed by Fabricius of Acquapendente, who died in 1619 (Brandt et al., 2011).

The donor-screening process usually starts with the patient's spouse/significant other or a household family member (Bakken, 2009). Individuals who have an intimate relationship with the patient are considered the most suitable stool donors for the procedure (Bakken, 2009). If the patient lives alone, a donor sample of stool from any prospective healthy person is then considered (Bakken, 2009). People who are actively being treated for intrinsic bowel disease, chronic infections, or malignancies, or who are on immunosuppressive therapy should not be considered because they are high-risk patients (Bakken, 2009). Another critical consideration for potential donors is the receipt of systemic antibiotics within the last 6–12 weeks before donating the stool sample (Bakken, 2009). Recent antibiotic use in a donor is contraindicated (Bakken, 2009).

Potential donors are also screened for exposures to contagious agents 30 days prior to the instillation procedure (Bakken, 2009). The donor is rigorously screened serologically for potential contagious infectious diseases such as Hepatitis A, B, and C, HIV-1 and HIV-2, and syphilis (Bakken, 2009). Each sample should be tested for CDI, ova, and parasites, and examined for enteric bacterial pathogens on selective growth media (Bakken, 2009). Donors who test positive for any of the diseases described previously are excluded and informed of the invading pathogen (Bakken, 2009).

Once a donor is selected and sufficiently screened, the instillation process can begin. The procedure is usually performed on an outpatient basis (Bakken, 2009). Care is coordinated between the clinical laboratory, radiology department, and the GI group that will perform the procedure (Bakken, 2009). The donor stool subsequently gets broken down into a liquid

suspension (Bakken, 2009). The stool sample can be placed either distally or proximally into the GI tract (Bakken, 2009).

Most patients receive their fecal bacteriotherapy through a retention enema placed in the rectum or via a colonoscopy; however, there is an increased risk of bowel perforation associated with this method of instillation (Bakken, 2009). A large amount of stool is also required when using this method, which may result in stool leaking backward out of the rectum after instillation (Bakken, 2009). This can lead to repeat procedures over several days for the procedure to be successful (Bakken, 2009). Fecal enemas have also been used to treat inflammatory bowel disease, chronic constipation, and pouchitis (Bakken, 2009).

Fecal transplants can also be accomplished by placing a NG tube into the patient's upper GI tract (Bakken, 2009). This method has relatively low risk, is cost-effective, and usually requires only a single fecal sample to be successful (Bakken, 2009). The complications include a small risk of perforation of the upper GI tract when placing the NG tube and the risk of aspiration of the feces into the lung (Bakken, 2009). Radiography is needed to confirm proper placement of the NG tube before using this method to help avoid any complications (Bakken, 2009).

The recipient of the fecal transplant needs to be pretreated with 250 mg of oral vancomycin thrice daily for 4 days or 500 mg twice daily for 7 days prior to the procedure in order to reduce the possibility of vegetative *C. diff* colonies (Bakken, 2009). The pretreatment should be discontinued the night before the procedure (Bakken, 2009). If patients are receiving the stool transplant via NG tube, they will need to receive omeprazole 20 mg orally the night before and the morning of the procedure. This decreases gastric acid production and creates a therapeutic environment for vegetative bacteria needed within the transplant. For patients receiving their stool sample via enema, a single pretreatment oral lavage with a purgative is recommended (Bakken, 2009).

Preparation of the donor stool sample varies depending on which method of installation is being used. In general, the stool sample is collected as close to the procedure as possible. In the case of transplantation via rectum, the stool should be collected 24 hours prior to the procedure (Bakken, 2009). The stool is diluted with either preservative-free NS or 4% milk that can be added to the stool sample before using a household blender to liquefy the stool into a liquid slurry consistency (Bakken, 2009). The stool is then filtered through a standard coffee-filter or gauze barrier to remove particulate matter before the instillation procedure takes place. Once the stool slurry is finished, it may be used immediately or frozen for later use (Bakken, 2009).

When patients are treated using a NG tube route, they are usually able to go home the night of the procedure. Typically their abdominal pain is resolved, and their stool frequency and consistency normalizes within 24–48 hours of the fecal transplant (Bakken, 2009). However, as previously mentioned, patients who are treated with the more common method of rectal enemas usually require repeat procedures for the procedure to be successful, which can be conducted over a 3- to 5-day period (Bakken, 2009).

Fecal bacteriotherapy is considered safe and effective in the treatment of CDI. There have been no infectious complications or adverse physical effects reported in the literature thus far from fecal transplant therapy (Bakken, 2009). The treatment modality is reported to have a success rate of 81%–100%, regardless of the treatment technique that was used (Bakken, 2009). Fecal bacteriotherapy is cost-effective, breaks the cycle of repeated antibiotic use, prevents emergence of resistant strains, avoids the risk of allergic reaction, reduces cost by eliminating the need of antibiotics, is considered a “low-tech” procedure, and can be performed on an outpatient basis (Bakken, 2009; Johnson, 2009).

### Health Promotion and Disease Prevention

Once the patient's diarrhea began, she originally thought she had some sort of stomach virus and felt it would resolve on its own in a few days. After the diarrhea persisted past a few days, she visited her PCP, who performed some testing. She was diagnosed with noninfectious diarrhea and told that her prognosis was good. She then chose to start herself on loperamide for the diarrhea.

This patient has limited access to healthcare and was on a fixed income. She was unemployed and had an insurance plan that she paid for out of pocket. Therefore, she tried to battle her diarrhea at home by using loperamide, increasing her oral fluids, and modifying her diet for as long as she could in an effort to avoid further medical expenses. The fact that she is a single mother of two young children and does not have any one to watch her children when/if she is hospitalized is just another issue that the patient was faced with. She had a significant other, but he did not contribute to the household or the children.

Before becoming hospitalized, the patient had already acquired a significant amount of medical debt. She received a colonoscopy for a positive fecal occult blood sample that was negative, as well as numerous laboratory tests and stool samples. She also had a CT scan of the abdomen although she never received its results. She was becoming distrustful of the healthcare system. During the interview for the case study, the patient stated, “I didn't want to come in here, and they

may not be able to tell me what's wrong with me, and I have to spend all of my money for nothing."

Upon being diagnosed with CDI, the patient searched the Internet to find out exactly what CDI is and how it is detected and treated. The diagnosis and treatment made the patient feel angered and enraged with her PCP because she felt that it should have been detected earlier. This patient is on a limited income and has limited access to healthcare, so a thorough laboratory workup should have been initiated before ordering expensive tests such as a CT scan of the abdomen.

*C. diff*-associated diarrhea can be a self-limiting condition, but it also has the potential to create a tremendous amount of stress, imbalance, and life-threatening complications in one's life. Trying to maintain and manage a normal life while plagued with CDAD can be a very draining experience. The affected person may have to make a toileting schedule to ensure time for defecation. Dehydration develops and causes a cascade of other symptoms.

The patient's personal life may be affected because of the avoidance of public activity or the shame he or she may feel. The cost related to hospitalization, treatment, diagnostic testing, and time missed at work can lead to financial burden. *C. diff*-associated diarrhea can completely disrupt one's life. According to Bakken (2009), patients tormented by the effects of CDI and CDAD for long periods of time are uniformly more receptive to the use of fecal bacteriotherapy and tend to overlook the unattractiveness of the procedure.

## Implications for Use of Fecal Bacteriotherapy in Practice

The most critical issue with CDI and its current treatment options is the failure of treatment in relation to reoccurrence. The frequency of recurrent CDAD, whether it is caused by relapse or reinfection, occurs in 5%–35% of cases (Bakken, 2009). Recurrent CDI (RCDI) is most likely to occur in older adults (those older than 65 years), in the presence of low serum albumin concentrations, a positive history for recent abdominal surgery, and a recent history of a prolonged hospitalization on an intensive care unit (Bakken, 2009). Although the guidelines currently support treating RCDI with a second course of metronidazole or vancomycin, the development of a chronic cycle of relapse and treatment with improvement occurring for short periods in between tends to occur (Bakken, 2009). Even with the implementation of several new alternative pharmacological therapies and treatment strategies, there is still a pattern of relapse present (Bakken, 2009).

Fecal bacteriotherapy can be the solution to ending this vicious cycle and improving the quality of life for

patients suffering from the intensity of RCDI. There is neither an abundance of clinical trials available on this subject nor a huge amount of experience with the procedure; however, those trials that have been completed within the United States and Europe reported successful results (Bakken, 2009). Bakken (2009) mentioned a trial completed by a group of researchers in 2003 that reported success in 94% ( $n = 16$ ) of the patients treated with fecal bacteriotherapy included in the trial.

Fecal instillation is definitely a squeamish subject. There is a potential risk of transmission of contagious diseases with this procedure; however, this is the reason for a diligent screening process (Bakken, 2009). Because of screening, there has not been any report of crosscontamination (Bakken, 2009). The simplicity and the minute amount of technology used to perform the actual procedure make it easy and affordable. Fecal bacteriotherapy can be implemented in most health-care institutions (Bakken, 2009). Compared to the price of antibiotic therapy in the treatment of RCDI, hospital bills, and cost incurred because of time off of work, fecal bacteriotherapy is considered to be very inexpensive (Bakken, 2009).

As previously mentioned, research has shown that patients tormented by CDAD or RCDI are receptive to fecal bacteriotherapy (Bakken, 2009). The chance of restoration of a normal life is more glorious than the lifestyle imposed on them by RCDI. In a study, Garborg et al. (2010) presented the idea of fecal bacteriotherapy and the risks and complications related to RCDI to patients in the early stage of recurrent CDAD and found that patients were less likely to any objections to the procedure.

## The Role of the APRN

As a provider, especially within the primary care arena, the APRN is responsible for providing preventative, educational, and medical services to every patient. As defined by the Consensus Model for APRN Regulation, the APRN is responsible and held accountable for health promotion services, as well as the assessment, diagnosis, and management of their patients' issues (APRN Joint Dialogue Group Report, 2008).

Patients should be educated on the potential risks related to frequent antibiotic use, including the risk for CDI/CDAD. They should also be educated on the importance of avoiding the risky behaviors that may lead to the need for antibiotic therapy. For instance, patients with asthma, COPD, or any other ailment related to the respiratory system should be educated on the risk of bronchitis or frequent upper respiratory infections related to tobacco use. These patients should be presented with smoking cessation materials and options to assist them in quitting. Smoking cessation



may reduce their risk for infection, need for antibiotic therapy, and risk for CDI.

## Summary

In the presence of the current spike in the incidence of CDI/CDAD and the emergence of the NAPI/BI/027 strain of *C. diff*, it is important to be aware of the need for a timely diagnosis and a thorough and efficient management plan to decrease morbidity and mortality. It is also essential that the APRN is aware of the available treatment modalities in the presence of CDI. The information in this article presents the basic information needed to identify the at-risk population, as well as diagnose and initiate a therapeutic treatment regimen for the patient with CDI. This article also supports the need for more clinical trials and data to support the use of fecal bacteriotherapy. It is a hope that this information will spark an interest in fecal bacteriotherapy among APRNs as well as other clinicians.

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