SHEA Position Paper

Clostridium difficile in Long-Term–Care Facilities for the Elderly

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ABSTRACT

Antimicrobial agents are among the most frequently prescribed medications in long-term–care facilities (LTCFs). Therefore, it is not surprising that *Clostridium difficile* colonization and *C. difficile*–associated diarrhea (CDAD) occur commonly in elderly LTCF residents. *C. difficile* has been identified as the most common cause of non-epidemic acute diarrheal illness in nursing homes, and outbreaks of CDAD in LTCFs have also been recognized. This position paper reviews the epidemiology and clinical features of CDAD in elderly residents of LTCFs and, using available evidence, provides recommendations for the management of *C. difficile* in this setting (*Infect Control Hosp Epidemiol* 2002;23:696-703).

Clostridium difficile is the major cause of nosocomial infectious diarrhea, with reported rates ranging from 1 to 10 cases per 1,000 discharges and 17 to 60 cases per 100,000 bed-days.[1-4] The organism may cause severe, even life-threatening disease, and has the potential to cause outbreaks in hospitals and other healthcare settings. Most individuals with symptoms attributable to *C. difficile* are older than 60 years,[5] and increasing age has been identified as a risk factor for *C. difficile* acquisition and for *C. difficile*–associated diarrhea (CDAD).[3,5-10] Community-acquired CDAD also appears to occur more frequently in the elderly.[11] In one study, approximately 10% of those older than 65 years were found to be colonized with *C. difficile* on admission to the hospital.[12] Aronsson et al. found that *C. difficile*–associated colitis occurred more often in older adults (older than 60 years) than in younger adults, and this difference did not appear to be due to greater exposure to antibiotics.[7] The increased risk of acquiring *C. difficile* infection in the elderly may be due to age-related changes in fecal flora, immune senescence, or the presence of other underlying diseases.

With an increased risk of *C. difficile* acquisition in older adults, it is not surprising that this infection also occurs commonly in nursing homes and other long-term–care facilities (LTCFs). However, much remains uncertain about the appropriate management of *C. difficile* in these settings. A previous Society for Healthcare Epidemiology of America position paper summarized knowledge about CDAD in hospitals.[13] This position paper reviews the epidemiology, clinical features, and diagnosis of *C. difficile* infection in LTCFs for the elderly and makes recommendations for appropriate investigation, management, prevention, and control of CDAD in this setting. In this paper, *C. difficile* infection refers to the presence of symptoms attributable to *C. difficile* (eg, CDAD), and colonization or carriage refers to the detection of *C. difficile* or one of its toxins in the absence of symptoms.

EPIDEMIOLOGY IN LTCFS

Antibiotic-associated diarrhea is common among LTCF residents, with a reported incidence as high as 1,600 per 1,000 resident-years.[14] In one study, 70% of chronic-care ward patients and 26% of nursing home residents (overall 33% of the LTCF residents) were found to have acquired *C. difficile* within 2 weeks of antimicrobial therapy, although only a minority experienced diarrhea.[14] *C. difficile* has been identified as the most common infectious cause of acute diarrheal illness in nursing homes.[15,16]

The prevalence of *C. difficile* colonization in LTCF residents in the absence of a recognized outbreak has ranged from 4% to 20%.[15,17-23] A significant proportion of residents may already be carrying *C. difficile* on admission to the LTCF, and an additional 10% to 20% may acquire the organism during their stay.[24,25] In a prospective study, the rate of acquisition of *C. difficile* during 1 year of follow-up in an LTCF was found to be 0.52 per 1,000 resident-days.[15] However, most of the residents with *C. difficile* remained asymptomatic during the year of study.

Outbreaks of CDAD have been reported in geriatric hospital units, rehabilitation hospitals, and freestanding

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skilled nursing facilities.[24,26-29] During outbreaks, up to 30% of LTCF residents have been found to harbor *C. difficile* or its toxin. Transmission of the organism in LTCFs is likely facilitated within a closed environment with a high rate of exposure to antibiotics. Why *C. difficile* is endemic or epidemic in some LTCFs but not in others is uncertain. Differences in the debility of the residents and the patterns of antimicrobial use and regional differences in strain virulence and infection control practices could influence the incidence of *C. difficile* colonization and infection in LTCFs.

Several risk factors for acquiring *C. difficile* in nursing homes have been identified. Not surprisingly, antibiotic use, especially the use of cephalosporins and trimethoprim–sulfamethoxazole, has been associated with *C. difficile* acquisition.[15,23,30] In one study, *C. difficile* acquisition was associated with the presence of a nasogastric or gastrostomy feeding tube and fecal incontinence,[15] whereas the use of a histamine-2 antagonist appeared to be a significant risk factor in another study.[23] Most LTCF residents colonized with *C. difficile* will spontaneously clear their feces by 2 months, although prolonged colonization for 3 or more months has been noted in up to 19% of residents.[15]

When *C. difficile* is present in LTCF residents, multiple strains of the organism are often found in the facility.[15,25] However, clonal outbreaks[28] and exogenous acquisition of new strains[25,31] have also been documented. Transmission likely occurs by direct spread from the hands of personnel, fomites, or the nursing home environment.[32,33] Healthcare providers probably contribute to transmission of *C. difficile* because of transient hand carriage.[1,34,35] Reduced rates of CDAD associated with glove use in a hospital setting provide further support for the importance of hand carriage.[36] Contamination of the inanimate environment of patients with *C. difficile* has been well described and may contribute to transmission of the organism in hospital or nursing home settings.[1,33,35] Contaminated commodes, telephones, and rectal thermometers have been implicated as potential sources of *C. difficile* in outbreaks.[32,37]

HOST RESPONSE TO *C. DIFFICILE* IN OLDER ADULTS

Antibiotic use alone is not sufficient for the acquisition of *C. difficile*, or for the development of CDAD. Antibiotics disrupt the normal gastrointestinal flora, but other factors, such as exposure to toxigenic strains of the organism and host susceptibility, are necessary for disease to occur.[38] The currently accepted hypothesis for the pathogenesis of infection with *C. difficile* is that there is negligible risk until an individual receives a systemic antimicrobial agent. If the patient is exposed to the organism during or shortly after the completion of antibiotic therapy, colonization without diarrhea may occur, or the patient may develop CDAD.[39] There are also data to indicate that once asymptomatic colonization with *C. difficile* is established, there is subsequently a decreased risk of developing CDAD.[40,41] Even toxigenic and apparently virulent strains of *C. difficile* are more likely to be associated with asymptomatic carriage rather than CDAD, suggesting that there must be other factors, perhaps related to host susceptibility, for disease to occur.[40] Virulence factors contributing to the epidemic transmission of *C. difficile* in hospitals and nursing homes are also uncertain, but may include enhanced fecal shedding or ability to persist on environmental surfaces.[35]

The observed increased risk of *C. difficile* infection in older adults may relate to defects in phagocytic and humoral host defenses. The ability of neutrophils to phagocytose and kill *C. difficile* in vitro is impaired in older adults when compared with that in younger volunteers.[42] This defect is specific, as serum from elderly patients added to neutrophils from young adults leads to declines in phagocytosis and killing of *C. difficile* but not other bacteria. In contrast, serum from younger individuals improves the phagocytic function of neutrophils from older adults. A potential candidate for this protective serum factor is IgG against toxin A. Other studies suggest that serum IgG to *C. difficile* toxin is reduced in healthy older subjects compared with younger adults.[43,44]

There is increasing evidence that serum IgG and IgM responses to toxin A are important for protection from *C. difficile* infection and prevention of recurrences.[45] Almost 70% of the general adult population has antibodies to toxin A or toxin B, suggesting that exposure is common and likely related to some environmental factor.[46] Although antibody concentration appears to rise with increasing age, the capacity of serum to neutralize *C. difficile* toxins decreases.[47] Hospitalized patients who develop high levels of anti-toxin IgG following acquisition of *C. difficile* do not develop diarrhea.[48] These high antibody responses generally occur within days of acquisition of the organism, suggesting prior exposure to a related organism or toxin. Failure to develop a serum IgG or IgM response to toxin A appears to be associated with an increased risk of recurrent disease.[45] The role of secretory IgA in protection from CDAD remains uncertain.

CLINICAL FEATURES AND OUTCOME IN THE ELDERLY

A spectrum of disease has been associated with *C. difficile* infection, ranging from mild diarrhea to potentially fatal pseudomembranous colitis with toxic megacolon. Clinical criteria for the diagnosis of CDAD generally include: (1) increased frequency of watery, loose, or unformed stools (at least three bowel movements per day for 2 or more days) not attributable to another cause; and (2) use of an antimicrobial or antineoplastic agent within the prior 4 to 6 weeks.[49] Patients are often febrile with crampy lower abdominal pain. Symptoms in the presence of pseudomembranous colitis tend to be more severe, with abdominal tenderness or peritoneal signs. The disease is most difficult to diagnose in the presence of abdominal pain and ileus, but little or no diarrhea. Symptoms typically begin during or shortly after antimicrobial therapy, but may occasionally be delayed several weeks.

There are no data to suggest that the clinical features of CDAD in the elderly are any different than those in younger individuals. During a hospital outbreak, *C. difficile* infection in older adults was associated with mild to moderate diarrhea lasting less than 10 days in 60% of patients, whereas 32% had a more prolonged illness (mean duration of diarrhea,

18 days) and 8% had severe colitis with complications.[10] Protein-losing enteropathy associated with CDAD has also been described in nursing home residents.[50,51] Increased mortality has been reported in elderly individuals with CDAD, but most deaths have been unrelated to infection.[14,24,30,52] In one study, there was no difference in early mortality rates in nursing home residents with or without *C. difficile* infection, but 12-month mortality was higher in those infected, suggesting that *C. difficile* may be a marker for increased debility and risk of death.[23]

However, elderly residents of LTCFs with *C. difficile* are most often asymptomatic.[14,15,21,23] This would suggest that age, per se, may not be a risk factor for development of CDAD. In one study, older adults (> 60 years of age) were more likely to develop CDAD than were younger adults, but there was no apparent difference in severity of infection, outcome, or mortality.[52] A delay in diagnosis was more likely to have occurred in the elderly patients. Recurrence of symptomatic infection has been reported in up to 26% of elderly nursing home residents despite appropriate treatment.[28,30] One study also found that recurrent CDAD was more likely to occur in older adults (mean age, 67 years) than in those who were younger,[53] but in most other studies, age did not appear to be a prognostic factor for failure to respond to treatment, recurrence, or other complications.[52,54-57]

DIAGNOSIS

C. difficile infection should be suspected in any adult with antibiotic-associated diarrhea. Abdominal imaging studies may reveal an ileus with dilated colonic segments or "thumbprinting" suggestive of mucosal edema. These changes are consistent with, but not pathognomonic of, pseudomembranous colitis. *C. difficile* can be detected in approximately 95% of patients with pseudomembranous colitis confirmed by endoscopy. But endoscopy may only reveal a nonspecific colitis without the development of pseudomembranes, or may miss proximal or right-sided disease.[49] Flexible sigmoidoscopy or colonoscopy identifies only approximately 50% of cases of pseudomembranous colitis in patients with *C. difficile* and cytotoxin in their stools. Therefore, the diagnosis of *C. difficile* infection generally relies on laboratory testing for the organism or its toxins.

The optimal diagnostic test(s) for the diagnosis of CDAD remains uncertain. Some investigators have recommended the use of stool toxin detection only, whereas others have recommended a combination of stool culture in addition to toxin detection. The relative merits and disadvantages of the various diagnostic tests were reviewed in a recent SHEA position paper,[13] and are only summarized here. The appropriate laboratory specimen for *C. difficile* testing is a watery or loose stool.[13,49] A single specimen is generally sufficient and multiple stool specimens are not usually required for establishing the diagnosis.[58] There is no value to testing stools of asymptomatic individuals (including for "test of cure"), unless part of an outbreak investigation.

The specificity of stool culture appears to be less than that of the cytotoxin assay, probably because asymptomatic carriage of *C. difficile* in hospitalized adults and LTCF residents is relatively common.[1,40,49] Therefore, a positive culture alone should not be used to establish a diagnosis of *C. difficile*–associated disease. However, if a culture is the only positive test result in a patient suspected of having CDAD (diarrhea and a history of antibiotic exposure), the isolate should be tested for toxin production in vitro; if positive, this is sufficient evidence to diagnose CDAD.[49] Stool cultures for isolation and typing of the organism may also be valuable as part of an outbreak investigation.

Detection of *C. difficile* cytotoxin (toxin B) has been considered to be the "gold standard" for diagnosis because of its high specificity (> 95%). However, the test may lack sensitivity (75% to 90%). Both stool culture and cytotoxin assay require a minimum of 48 hours for a result to be available. Several commercial enzyme immunoassays have been developed for the detection of toxin A alone, or for both toxins A and B, and are able to provide results within hours. Sensitivities for these assays have been reported to range from 60% to 90%, with specificities ranging from 75% to 100%. The vast majority of *C. difficile* strains produce either both toxins A and B or neither toxin, although strains that appear to produce only one of the two toxins have been reported to cause disease.[59-61] Perhaps for this reason, assays able to detect either toxin appear to be more sensitive than tests detecting only toxin A.[62-64]

Several typing methods have been described for characterizing strains of *C. difficile* as part of epidemiologic investigations during outbreaks. Pulsed-field gel electrophoresis (PFGE), arbitrarily primed polymerase chain reaction (AP-PCR), and random amplified polymorphic DNA (RAPD) techniques appear to have adequate discriminatory power and reproducibility.[31,33,65-67] PFGE may be easier to interpret, but not all strains are typeable by this method because of DNA degradation; AP-PCR appears to have greater discriminatory power but may be somewhat less reproducible.

TREATMENT

General treatment measures applicable to all patients with CDAD include replacement of fluid and electrolyte losses, avoidance of antiperistaltic agents and opiates, and discontinuation of antimicrobial therapy if possible. These measures may suffice in approximately 15% to 20% of patients, particularly in those with mild disease (ie, in those with mild diarrhea without fever, leukocytosis, or other signs of systemic toxicity).[68] However, in patients with CDAD not responding to these conservative measures within a few days, in those in whom the offending antimicrobial agents cannot be discontinued, or in those with more severe symptomatic disease, specific therapy for *C. difficile* will be required. Guidelines from the American College of Gastroenterology[69] and the Society for Healthcare Epidemiology of America[13] recommend use of either orally administered metronidazole or vancomycin. Treatment with either one of these agents results in symptomatic improvement in 85% to 90% of patients, and in comparative trials, these agents appear to be equally effective.[70,71] Clinical improvement generally occurs within 2 to 4 days, and symptoms remit within 7 to 10 days.

Metronidazole is recommended as the agent of first

choice for most patients with CDAD because it is less expensive than vancomycin, and is less likely to promote emergence and spread of vancomycin-resistant enterococci.[13,69,72,73] Metronidazole administered orally in dosages of 250 mg four times a day or 500 mg three times a day for 10 to 14 days is most commonly used as initial therapy, although some authorities prefer oral vancomycin (250 mg four times a day) for those with more severe illness.[69] Vancomycin is also recommended for patients who remain symptomatic while being treated with metronidazole, or for those who cannot tolerate metronidazole.

Patients with an ileus associated with *C. difficile* infection and those unable to take oral medications pose a difficult therapeutic challenge. Parenteral administration of vancomycin usually fails to provide adequate concentrations of the drug in the bowel lumen, and parenteral vancomycin has no role in the treatment of CDAD. Although controlled treatment trials are lacking, most authorities recommend the use of intravenous metronidazole for such patients.[74]

Patients who cannot tolerate or do not respond to metronidazole or vancomycin have few therapeutic options. Treatment with bacitracin has resulted in symptomatic improvement in approximately 75% of patients, but recurrence rates exceeding 40% have been reported.[75,76] Although not available in the United States, teicoplanin given orally (100 to 400 mg twice a day for 10 days) and oral fusidic acid (500 mg three times a day for 10 days) have yielded rates of improvement of greater than 90%, although a recurrence rate of 28% was reported following treatment with fusidic acid.[71,77,78] Agents that bind to and block the biological activities of *C. difficile* toxins have been found to be effective in animal models, although safety and efficacy still need to be demonstrated in human clinical trials.[79]

Unfortunately, even in patients who have been successfully treated with either metronidazole or vancomycin, CDAD may recur in approximately 5% to 20% of cases.[69,80] Relapse and reinfection each account for approximately half of the recurrent episodes.[81] Relapses may arise from germination of vegetative forms persisting after a course of therapy. Re-acquisition of spores contaminating the environment of patients likely predisposes patients to reinfection. Metronidazole remains the drug of choice for treatment of an initial recurrence, even if this was the original drug used.[68,69,80] A second course of metronidazole has cured more than 90% of patients with an initial recurrence.[68] A small proportion of patients will experience more than one episode of recurrent disease. The optimal approach for managing this problem is uncertain. The combination of oral vancomycin and rifampin has been found to be effective in a relatively small number of patients.[82] In two randomized, controlled trials, adjunctive therapy with the probiotic agent *Saccharomyces boulardii* significantly reduced the recurrence rate in patients with recurrent CDAD.[83,84] Treatment with *Lactobacillus GG* has also been reported to be efficacious in the management of recurrent CDAD.[85,86] Further evaluation of the role of probiotic agents for the management of recurrent CDAD is required, and no commercial preparations of these probiotics are currently available in the United States.

Less than 1% of patients with *C. difficile*–associated colitis require surgical intervention for the management of perforation or toxic megacolon.[80,87,88] Signs of peritonitis or of organ failure may indicate the need for imaging studies and surgical exploration.

INFECTION PREVENTION AND CONTROL

The knowledge of the epidemiology of *C. difficile* infections in acute care institutions should serve as the basis for the management of these infections in LTCFs pending availability of data specific to the long-term–care setting. As noted in an earlier SHEA position paper regarding CDAD,[13] there are two components to managing disease caused by this organism in the healthcare setting: (1) efforts designed to prevent spread of the organism to the patient; and (2) attempts to reduce the likelihood of clinical disease.

Prevention of Transmission of C. difficile

Patients with diarrhea caused by *C. difficile* typically have large numbers of organisms in their stool. Management of CDAD in healthcare facilities is based on the assumption that these symptomatic patients are the major reservoir for the organism. In addition, the hands of healthcare providers are frequently contaminated with *C. difficile* following patient contact,[1] and wearing gloves can significantly reduce the spread of *C. difficile* in hospitals.[36]

Information about hand washing is not definitive. One study reported that the use of chlorhexidine eliminated *C. difficile* and its spores from the hands of healthcare workers more effectively than soap.[1] However, in another study there was no difference in the effectiveness of a liquid soap compared with 4% chlorhexidine gluconate in decontaminating bare hands inoculated with *C. difficile*.[89] Larson et al.[22] noted that regular hand washing and wearing of vinyl gloves together were effective in preventing hand carriage by healthcare workers. The efficacy of alcohol-based antiseptics alone or in combination with gloves in reducing *C. difficile* infection has not been specifically addressed. Most commonly used hand hygiene products, including alcohol-based products, are not sporicidal.[90,91] It is presumed that increased use of alcohol-based antiseptics will improve compliance with hand washing, decrease transient contamination of hands, and reduce infection rates.

To reduce spread, it has been recommended that patients with *C. difficile* diarrhea be placed in isolation until their diarrhea has resolved.[13] The use of private rooms or cohorting for patients with CDAD and enteric or contact precautions has been successful in limiting transmission of *C. difficile* in hospitals and on a geriatric ward.[28,92,93] However, because these measures have been implemented along with other infection control interventions, it is not possible to determine the specific effectiveness of isolation techniques or the use of a private room. There are data to suggest that transmission of *C. difficile* to roommates may occur. Therefore, although of unproven value, it may be useful to care for patients with CDAD in a private room, especially if they have fecal incontinence.

Given the widespread dissemination of *C. difficile* spores from patients with diarrhea, what are appropriate actions to take in the patient's environment? It has been suggested that disinfectants that do not contain chlorine cause increased spore production by *C. difficile*.[94] As with the use of isolation, there is considerable circumstantial evidence about the role of disinfection, but few interventions with documented efficacy. Extensive environmental disinfection with a variety of agents appeared to decrease rates of nosocomial CDAD in several studies,[28,92,93,95] but because other interventions were implemented at the same time, it is not possible to ascertain the benefit of environmental disinfection or to determine the optimal cleaning agents. Environmental cleaning with solutions containing hypochlorite has decreased surface contamination with *C. difficile*,[96] and Mayfield et al. showed that the use of unbuffered 1:10 hypochlorite solution decreased the prevalence of CDAD on a bone marrow transplant unit.[97] In the United Kingdom, the tendency to use detergents rather than disinfectants containing chlorine has recently been questioned.[98]

Gowns are often used as a method of preventing contamination of healthcare workers and their uniforms. Gowns and uniforms may become contaminated with *C. difficile*.[99] Attempts to determine the efficacy of gowns in limiting transmission are confounded because gown use is generally implemented together with other infection control measures. There is no evidence that the use of gowns by itself is an efficacious control measure.

Management of the asymptomatic carrier of *C. difficile* has been evaluated. Carriers of the organism appear to be resistant to acquisition of outbreak-associated strains.[100] Individuals with asymptomatic colonization are no more likely to develop CDAD than are those with negative stool cultures.[40,41] Metronidazole has not been found to be effective in eradicating asymptomatic carriage with *C. difficile*, and treatment with oral vancomycin resulted in only transient elimination of fecal carriage of the organism.[24,101] Therefore, antibiotic treatment of asymptomatic patients excreting *C. difficile* is not recommended.

Methods to Reduce the Development of C. difficile*–Associated Diarrhea*

After exposure to antimicrobials, development of CDAD requires exposure to a toxigenic strain of *C. difficile*.[39] Virtually every antimicrobial agent has been implicated, although the most common causes have been broad-spectrum penicillins and cephalosporins. There is also a high risk for CDAD associated with clindamycin therapy. Restricting the use of clindamycin was associated with a marked decrease in the rates of nosocomial CDAD at two hospitals,[102,103] and restrictions on the use of broad-spectrum antibiotics were shown to reduce the frequency of CDAD in a long-term–care unit.[29] Therefore, development of programs that encourage the proper use of antibiotics is essential, particularly in LTCFs.[104]

As noted above, probiotic agents have been evaluated for the treatment of patients with recurrent CDAD. There have also been several studies of the prophylactic administration of probiotics to prevent the development of CDAD in patients receiving antibiotics. In one study, the prophylactic use of *S. boulardii* appeared to prevent antibiotic-associated diarrhea in hospitalized patients,[105] but was found to be ineffective in elderly adults in another study.[106] The prophylactic administration of *Lactobacillus* species does not appear to be effective in preventing the development of antibiotic-associated diarrhea.[107,108]

RESEARCH NEEDS

Relatively little information describing *C. difficile* infection has focused on variations associated with aging. Although an increased risk of infection has been reported with increasing age, it is unclear whether this is a result of changes associated with aging or an increased frequency of comorbidities and antimicrobial use in older adults. As our understanding of the organism's virulence factors and host characteristics promoting transmission increases, the impact of age, if any, will need to be better defined. Are older patients truly at increased risk of acquiring *C. difficile* or CDAD? If there is an age-related variability in frequency of infection acquisition or of disease manifestation, what determinants are responsible? Are therapeutic strategies equally effective in older populations and in younger adults?

The epidemiology and clinical impact of *C. difficile* in LTCFs need to be better understood. Given the wide variety of patient populations residing in LTCFs, there should be differential risks for outbreaks or disease acquisition determined by levels of acuity and care. Facilities providing chronic hospital care may be at greater risk than are skilled nursing facilities. If so, are these differences explained by variations in antibiotic exposure, or are there other factors? Variables that influence transmission of *C. difficile* between residents in the long-term–care setting need to be identified. Important questions regarding the role of environmental contamination and patient care practices (eg, feeding method) in the acquisition of infection need to be addressed specifically in the long-term–care setting. What level of environmental cleaning, hand hygiene, or glove use is optimal to limit transmission of the organism? Are infection control recommendations different for patients with diarrhea compared with those without? Identifying determinants of colonization and disease would provide insights into interventions that should be evaluated for use in different LTCF settings and in different patient populations.

SPECIFIC RECOMMENDATIONS

Although diarrhea is a relatively common problem in LTCFs, the development of a cluster of cases of acute diarrheal illness requires a careful search for the etiology. It is recognized that private rooms for isolation are frequently not available in LTCFs, and there is often a lack of convenient hand washing facilities. These considerations may affect a facility's ability to optimally care for residents with CDAD. The following recommendations are based on criteria previously used in other Society for Healthcare Epidemiology of America position papers (Table),[109] and are intended to guide

TABLE
Classification of the Strength and Quality of Evidence of Each Recommendation*

Category	Definition
Categories reflecting the strength of the recommendation	
A	Good evidence to support the recommendation
B	Moderate evidence to support the recommendation
C	Poor evidence to support the recommendation
Categories reflecting the quality of evidence for the recommendation	
I	Evidence from at least one properly randomized, controlled trial
II	Evidence from at least one well-designed clinical trial without randomization, from cohort or case-controlled analytic studies (preferably from more than one center), from multiple time-series studies, or from dramatic results in uncontrolled experiments
III	Evidence from opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees

*This classification scheme has been used for Society for Hospital Epidemiology of America position papers since 1994.[109]

the management of both sporadic and epidemic disease associated with *C. difficile* in LTCFs.

Surveillance and Diagnosis

1. Effective surveillance for CDAD should be done in every long-term–care setting. *Category BIII.*

2. Surveillance should include appropriate and prompt diagnostic testing of LTCF residents with antibiotic-associated diarrhea or an acute diarrheal illness not otherwise explained. *Category AII.*

3. LTCFs must have accessible laboratory support to facilitate prompt identification of CDAD. *Category BIII.*

4. Tests for *C. difficile* or its toxins should be done only on diarrheal (unformed) stool specimens, unless ileus due to *C. difficile* is suspected. *Category BIII.*

5. Testing of stool specimens from asymptomatic LTCF residents for *C. difficile* or its toxins (including "test of cure" after treatment) is not recommended, except as part of epidemiologic investigations. *Category BII.*

Treatment

1. If clinically appropriate, discontinuation of the offending antimicrobial agent(s) is recommended if CDAD is suspected. *Category AI.*

2. Oral metronidazole should be considered the treatment of choice for CDAD. Treatment with oral vancomycin should be reserved for therapy for CDAD only if there has been a failure to respond to metronidazole, or if the resident cannot tolerate or is allergic to metronidazole. *Category AI.*

3. Treatment of asymptomatic residents with *C. difficile* colonization is not recommended. *Category AI.*

4. Residents with a first recurrence of CDAD following treatment should be re-treated as for the initial episode (generally with metronidazole). *Category BIII.*

Prevention and Control

1. Implement policies in the LTCF for the prudent use of antimicrobial agents. *Category AII.*

2. Surveillance of antimicrobial utilization in the facility should be conducted. *Category BIII.*

3. Healthcare providers in the facility should be educated about the clinical features, transmission, and epidemiology of CDAD. *Category BIII.*

4. Care for LTCF residents with CDAD and fecal incontinence should be in a private room. If facilities are available, a private room should be considered for all residents with CDAD until the diarrhea has resolved. *Category BIII.*

5. Meticulous hand hygiene with soap or an antimicrobial agent is recommended after contact with residents, their body substances, or their potentially contaminated environment in the LTCF. *Category BIII.*

6. Healthcare providers should wear gloves for contact with LTCF residents with CDAD, and for contact with their body substances and environment. *Category AI.*

7. Use of disposable, single-use thermometers (rather than shared electronic thermometers) is recommended. *Category AII.*

8. For a resident with CDAD, patient care items and equipment such as stethoscopes and blood pressure cuffs should be dedicated and not shared with other residents. If such items must be shared, they should be carefully cleaned and disinfected between residents. *Category BIII.*

9. Disinfection of the environment (eg, room surfaces) of a resident with CDAD should be done using sporocidal agents, such as a diluted hypochlorite solution. *Category BII.*

10. Residents with CDAD may be removed from isolation when their diarrhea has resolved. *Category BIII.*

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