

Microbe of the month

Breaking The Chain of Infection

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Newsletter

Compiled by Helen Loudon, Independent IPC Practitioner



Featured
this
month:

Clostridioides difficile

A new name for an old foe

Hello readers!

Did you know that the bacterium '*Clostridium difficile*' ('C. diff.') is now referred to as '**Clostridioides difficile**'?

The change follows a decision in 2018 by the Clinical and Laboratory Standards Institute (CLSI) to call the organism '*Clostridioides difficile*', after global backlash and resistance to a proposal in 2016 to reassign *Clostridium difficile* to the genus 'Peptoclostridium'.

However, to move the nomenclature over to '*Peptoclostridium difficile*' was not straightforward! *C. difficile* is a very familiar name with recognition that extends far beyond medical practice, so drastically changing the name would have led to a significant financial burden in terms of relabelling, laboratory reporting, etc.

The solution was to create a new genus that was sufficiently similar to *Clostridium* to minimise any confusion. This involved creating a genus that began with the letter 'C', so that relevant abbreviations such as 'C. diff' and CDAD (*Clostridium difficile*-associated diarrhoea) remained unchanged. Consequently, '**Clostridioides difficile**' was coined.

BACKGROUND and EPIDEMIOLOGY

C. difficile is a Gram-positive, **spore-forming anaerobic bacillus**, found everywhere in nature, and is especially prevalent in soil.

It was first described in 1935 as part of the intestinal flora of new-born infants, and is believed to colonise the gut in approximately 1-15% of healthy individuals.

However, arising from outbreaks in the late 1970s, *C. difficile* is now recognized as one of the most important **opportunistic pathogens** in hospital and community healthcare settings.

The transmission of *C. difficile* within the healthcare environment is an example of our collective failure to heed basic infection control policies and practices designed to interrupt or prevent such transmission.

It constitutes +/- 20% of cases of antibiotic-associated diarrhoea ('AAD'), and in 2019, the CDC designated *C. difficile* as an **urgent threat** because of its association with antibiotic use, high mortality and morbidity.

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The vegetative cells of *C. diff* are rod-shaped and occur in pairs or short chains.



Clostridioides difficile endospores are extremely hardy to ensure the organism's survival outside the body.

- 'Clostridium' is derived from the Greek word 'kloster', meaning 'spindle', to describe its unusual 'drumstick' appearance.
- The 'bulge' at the terminal ends are where the spores are formed.
- To be cultured successfully in the laboratory setting, *C. difficile* must be provided with specific conditions to meet its anaerobic and nutritional requirements - hence the other half of its scientific name, 'difficile', from the Latin for 'difficult'.
- Interestingly, synonyms for 'difficile' include: 'troublesome, tough, problematic, onerous, demanding, severe, burdensome, painful, challenging, heavy, terrible, complicated and serious ...' [<https://www.dictionary.com/browse/difficile>]

RISK FACTORS FOR *C. DIFFICILE* INFECTION (CDI)

It is estimated that about two thirds of CDI cases are related to a stay in a hospital or nursing home, and the other third are community-associated, involving people with no recent hospital or nursing home exposure. **90% of deaths from *C. difficile* infections occur in people 65 years and older.**

- ✓ Broad spectrum and/or prolonged antibiotic exposure (esp. cephalosporins, fluoroquinolones and Clindamycin)
- ✓ Extended length of stay in a healthcare setting
- ✓ Serious underlying illness and/or immunocompromising conditions
- ✓ Advanced age
- ✓ Inflammatory bowel disease
- ✓ Gastric acid suppression (proton pump inhibitors ('PPI's') available over the counter and on prescription for heartburn and gastric reflux)
- ✓ Tube feeding (faecal-oral transmission from contaminated feeds / hands of healthcare workers)
- ✓ Improperly cleaned and disinfected colonoscopes following their use in a patient with *C. diff* colitis

PATHOGENESIS OF CDI - the direct link to antibiotic therapy

The complications caused by *C. difficile* are an example of a secondary infection that is directly related to antibiotic use.

Broad-spectrum antibiotic therapy can affect 30% of the gut communities, causing a rapid and significant drop in **species diversity** and the elimination of many of the species of intestinal bacteria which normally keep species such as *C. difficile* in check. As protective flora diminishes with antibiotic therapy, the clostridia have the unopposed space in which to multiply.



Antibiotics which may induce *Clostridioides difficile* diarrhoea and colitis

Frequently associated	Occasionally associated	Rarely associated
Fluoroquinolones	Macrolides	Aminoglycosides
Clindamycin	Trimethoprim	Tetracyclines
Broad-spectrum penicillins	Sulphonamides	Chloramphenicol
Broad-spectrum cephalosporins		Metronidazole
		Vancomycin

Clinical relevance?

The alteration of the human microbiome ('dysbiosis') from exposure to antibiotics has an almost immediate effect on gut health. The development of resistant opportunistic pathogens occurs as a result of 'selective pressure', and therefore the gut has long been established as a significant '**reservoir**' of antibiotic resistance.

Mode of transmission

A patient acquires CDI via the faecal-oral route most often by touching a surface contaminated with *C. difficile* spores and then touching their mouth with the contaminated hand. The spores are ingested and travel unharmed through the acidic environment of the stomach and germinate into the vegetative (active) bacterial form in the colon.

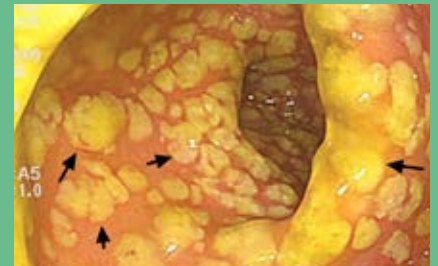
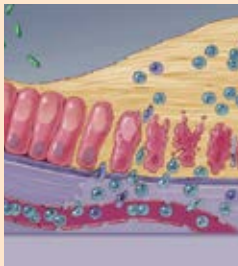


Every time a toilet is flushed without the lid closed, bioaerosols contaminate nearby surfaces!

The highest number of *C. difficile* were recovered from air sampled immediately after flushing at heights up to 25cm above the toilet seat, and up to 90 minutes after flushing, at concentrations 12-fold greater with the lid up than with the lid down.⁷

In 2003, a severe outbreak of CDAD occurred in both the United States and Canada due to a clone designated as BI/NAP1/027. Since the original identification of this strain, cases have been documented across Europe, Asia, Africa, and the Middle East.

Studies have demonstrated that this strain (027) produces **toxins A and B** more quickly and in larger quantities. This highly-virulent toxigenic organism is also capable of producing a **binary toxin**, which facilitates its adherence to the colonic mucosa, causing extreme inflammation, ulceration and in severe cases, perforation (i.e., pseudomembranous colitis, also known as 'toxic megacolon').



C. difficile is ingested.

Toxic megacolon and pseudomembranous colitis.

C. diff toxins cause severe ulceration of the colon tissue, resulting in perforation and septicemia.

WHAT ABOUT ASYMPTOMATIC *C. difficile* CARRIAGE? ²

The role of asymptomatic *C. difficile* colonisation (i.e., *C. diff* 'carriers') as part of the clinical spectrum of CDI is complex, because many risk factors are common to both disease and asymptomatic states. Asymptomatic *C. diff* colonisation is a condition where the bacillus is detected in the stool in the absence of symptoms of infection.

Epidemiologic studies have reported that 10–16% of hospital inpatients in high-risk units became carriers after receiving antibiotics, acting as an infection reservoir and presenting a risk to other vulnerable patients.

SIGNS AND SYMPTOMS OF *C. DIFFICILE* INFECTION (CDI)

Symptoms include watery diarrhoea, fever, nausea, and abdominal pain - complications include pseudomembranous colitis, toxic megacolon, perforation of the colon, and sepsis.



CDI case definition or 'clinical prediction rule'

- Significant diarrhoea (i.e., 'the new onset of more than three partially formed or watery stools per 24-hour period')
- Recent antibiotic exposure
- Abdominal pain
- Fever (up to 40.5°C)
- A distinctive odour to the stool resembling horse manure

DIAGNOSIS ^{3,4}

CDI is diagnosed based on the patient's medical history, signs and symptoms, combined with test results, but is believed to be under-reported and/or under-diagnosed due to incorrect test ordering practices. The optimal method for laboratory diagnosis of *C. diff.* is the subject of debate and depends on how carefully patients are selected for testing.

- Previously, most laboratories relied on testing the stool for *C. difficile* toxin A/B enzyme immunoassays, but these have a low sensitivity rate of 70-80%.
- PCR-based molecular assay:
 - Pros – it is very sensitive; results are available within hours and also detect the epidemic strain.
 - Cons – the sensitivity of the test may lead to over-diagnosis; therefore, it is recommended that a *C. diff.* common antigen test and a stool toxin test (such as an immunoassay) be used as part of a two or three-step test process.
- Anaerobic stool culture (disadvantage is the long turn-around time of 48-96 hours).
- Colonoscopic findings of pseudomembranous colitis.

TREATMENT ⁵

Recently-updated recommendations are:

- Initial episode of CDI – **oral** vancomycin or fidaxomicin.
- Limit the use of metronidazole to cases in which neither vancomycin nor fidaxomicin are unavailable or contraindicated.
Note: this is a change from the 2017 guidelines which recommended metronidazole as the preferred antibiotic for initial mild-moderate CDI.
- Faecal microbiota transplantation (FMT) is recommended for the treatment of people with two or more recurrences of *C. diff.* and for whom traditional antibiotic treatment has not worked.

Once antibiotic treatment has stopped, the microbiota may demonstrate a degree of resilience and return to a composition similar to the original one – however, the initial state is often not totally recovered. **Antibiotic-induced dysbiosis** can persist for months or even years. The use of probiotic bacteria and faecal transplantation aimed at re-establishing the gut microbiota after antibiotic treatment is a promising approach.



***C. difficile* colitis should be suspected in any patient with diarrhoea who has received antibiotics in the previous 2 months and/or when unexplained diarrhoea occurs 72 hours or more after hospitalisation.**

10-35% of successfully treated patients experience a relapse of *C. difficile* colitis with recurrence of diarrhoea, abdominal cramps, and abdominal pain. Relapses typically occur days or even weeks after treatment is stopped.



Faecal microbiota transplantation (FMT) is a clinical procedure that replaces and restores healthy bacteria in the colon by introducing stool via enema or colonoscopy from a healthy human donor. Potentially fatal '*C. diff.*' infections have been cured within days using FMT.



Watch this fascinating video (4 and a half minutes) on the role of faecal microbiota transplantation for *Clostridium difficile* colitis.

<https://www.youtube.com/watch?v=Awn3haOpfcl>

C. DIFF FACTSHEET

Clostridioides difficile (formerly known as *Clostridium difficile*) is a bacterium that causes diarrhea and colitis (an inflammation of the colon). *C. diff* infections can be deadly.

IMPACT



C. diff causes close to half a million illnesses each year and can affect people of all ages.¹



1 in 5 patients will get *C. diff* at least once more.¹



One in 11 people over 65 diagnosed with a healthcare-associated *C. diff* infection die within a month.¹

RISK



People on antibiotics are 7 to 10 times more likely to get *C. diff* while on the drugs and during the month after.²



Extended stays in healthcare settings, especially hospitals and nursing homes, also increase risk.



More than 80% of *C. diff* deaths occur in people 65 and older.

SPREAD



C. diff spreads when people touch surfaces that are contaminated with poop from an infected person.



Or when people don't wash their hands with soap and water.



It can also happen when one healthcare facility fails to notify another when it transfers a patient with *C. diff*.

Healthcare professionals can help PREVENT *C. diff* by:



Improving the way they prescribe antibiotics.



Using the tests that give the most accurate results.



Rapidly identifying and isolating patients with *C. diff*.



Wearing gloves and gowns when treating patients with *C. diff*—and remembering that hand sanitizer doesn't kill *C. diff*.



Cleaning surfaces in rooms where *C. diff* patients are treated with EPA-approved, spore-killing disinfectant (see List K).

cdc.gov/cdiff

¹ Table 3 from Lessa FC, Mu Yi, Bamberg WM et al. N Engl J Med 2015;372:825-34. DOI: 10.1056/NEJMoa1408913

² Hensgens MPM, Goorhuis A, Dekkers OM, Kuijper EJ. J Antimicrob Chemother 2011. DOI: 10.1093/jac/dkr508



U.S. Department of Health and Human Services
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LESSONS LEARNED FOR INFECTION PREVENTION AND CONTROL



1. *C. difficile* is found readily on hospital surfaces (especially ICU's) as a result of faecal contamination.
2. Symptomatic patients are known to excrete a large number of the bacilli in their stool, either as vegetative organisms or as spores.
3. ***C. difficile* spores can survive in the environment for up to two years** and withstand extreme conditions
4. *C. diff* is resistant to many disinfectants, and **the spores are not inactivated by alcohol-based hand rubs.**
5. Isolate the patient as soon as possible and ensure the use of a dedicated toilet / bathroom.
6. Use strict contact precautions (gloves and plastic disposable aprons).
7. Ensure frequent hand hygiene with antibacterial soap and water.
8. Clean and disinfect environmental surfaces with sodium hypochlorite (chlorine)-based detergents or disinfectants with proven sporicidal efficacy.
9. Wash bedpans and faecal receptacles in automated washers which exceed 85°C.
10. Ensure a laboratory-based alert system for immediate notification of positive test results.
11. Implement and/or review your antimicrobial stewardship programme.
12. Educate HCW's, housekeeping, facility management, patients and families about *C. diff* infection.

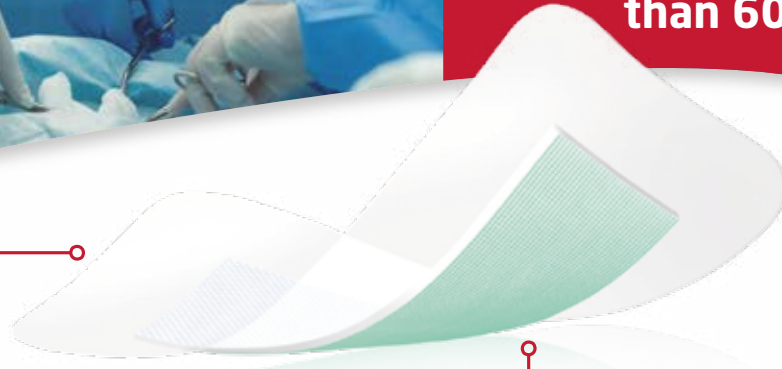
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* P.J. Stanirowski, et al. Dialkylcarbamoyl chloride-impregnated dressing for the prevention of surgical site infection in women undergoing cesarean section: a pilot study. Arch Med Sci 2016; 12, 2

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