Fortnightly Review

Diagnosis and management of *Clostridium difficile* infection

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Clostridium difficile is the commonest enteric pathogen in patients in hospital. In 1978 *C difficile* was first recognised as the main cause of pseudomembranous colitis and antibiotic associated colitis and diarrhoea.[1-4] Since then, extensive studies have helped to elucidate the role of this organism in human disease, but there are still some issues to be resolved. In this paper we review the work that led to the recognition of *C difficile* and discuss our current knowledge on the pathogenesis, diagnosis, and management of disease associated with *C difficile*.

Summary points

- *Clostridium difficile* is a major nosocomial pathogen causing illness ranging from antibiotic associated diarrhoea to antibiotic associated colitis and pseudomembranous colitis
- Antibiotic treatment is an important predisposing factor to *C difficile* associated disease, and elderly patients and those with serious underlying disease are especially at risk
- *C difficile* is nosocomially acquired and may cause outbreaks of illness by spreading directly from patient to patient or being acquired from the environment and from healthcare workers
- Diagnosis depends on clinical presentation and laboratory investigations (culture of *C difficile* and demonstration of toxins in stools), while sigmoidoscopy is occasionally helpful
- Treatment of *C difficile* infection should include stopping the implicated antibiotic, rehydration, and oral treatment with metronidazole or vancomycin, while severe complications may require emergency surgical intervention
- Control measures include isolation of infected patients, implementation of infection control practices, and introduction of strict antibiotic policies

Historical background

Pseudomembranous lesions of the intestine were first described in 1893 by Finney in a postoperative patient,[5] but pseudomembranous colitis was relatively rare until the 1950s, when it became a common complication of antibiotic treatment after the introduction of penicillin, tetracycline, and chloramphenicol. At that time *Staphylococcus aureus* was thought to be the organism causing this condition, and the condition was called staphylococcal enteritis and was later treated with oral vancomycin.[6] This view was not challenged until the 1970s, when a resurgence of interest in pseudomembranous colitis occurred after a report that, in a prospective study of 200 patients treated with the antibiotic clindamycin, 21% developed diarrhoea and 10% were shown to have pseudomembranous colitis by endoscopy.[7] Subsequent testing of stool specimens showed the presence of *C difficile* toxin.[7] This strong association with clindamycin led to the introduction of the term clindamycin associated colitis[8] and to a challenge to *Staphylococcus aureus* as the cause of pseudomembranous colitis.

CLOSTRIDIUM DIFFICILE

In 1935 Hall and O'Toole first isolated this organism, designated *Bacillus difficilis*, from the meconium and faeces of newborn infants.[9] The organism was shown to produce a lethal toxin in experimental animals, but since it was commonly found in the stools of healthy neonates it was classified as commensal and subsequently attracted little attention until 1974, when a comprehensive study showed that *C difficile* was widespread in nature and could be isolated from the stools of several animal species and from patients' faeces and genitourinary tracts.[10,11] It was also noted that most strains of *C difficile* produced a lethal toxin, but no further work was undertaken.

The link between clindamycin associated colitis and *C difficile* was not made until 1977. Larson *et al* showed that stool fitrates from a patient with pseudomembranous colitis had a cytotoxic effect on tissue culture cells, suggesting the presence of a toxin of undefined source.[12] At the same time investigators in the United States showed that clindamycin and other antibiotics induced a lethal caecitis in hamsters; the caecal contents contained a filterable toxin that was cytopathic in a cell culture assay and would reproduce the typical lesions when injected intracaecally.[13] An organism identified as *C difficile* was isolated from the animals and was shown to be the source of the toxin, and Koch's postulates were satisfied. Soon after, *C difficile* and its toxins were detected in the stools of patients with pseudomembranous colitis,[14] and oral vancomycin was shown to be effective treatment in animal models and in patients.[14] *C difficile* has since become established as a major cause of nosocomial diarrhoeal infection.

Pathogenesis

In order to cause disease, *C difficile* has to establish itself in the colon and produce toxins that cause mucosal damage, inflammation, and fluid secretion. Host factors are important in determining the clinical outcome of the disease.

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Pathogenesis of *C difficile* associated disease

- Antibiotic usage
- Changes in normal colonic flora
- Establishment and proliferation of *C difficile*
- Production of toxins A and B
- Effect on colonic mucosa and fluid secretion
- Other virulence factors

USE OF ANTIBIOTICS

It has been suggested that the use of broad spectrum antibiotics leads to changes in the normal intestinal flora and to disturbance in the control mechanisms of the bacterial populations in the gut, thus allowing *C difficile* to become established and to proliferate. The precise nature of the changes in the normal flora that permit colonisation by *C difficile* are not clearly defined. The source of *C difficile* may be endogenous if the patient is a carrier or, most commonly, exogenous, acquired nosocomially from the environment.

The antibiotics most commonly implicated are the broad spectrum drugs that have a large impact on the normal intestinal flora, particularly when given orally. These include the penicillins and those incorporating β lactamase inhibitors, the cephalosporins, and clindamycin. *C difficile* associated disease can be induced by antibiotics to which the organism is sensitive in vitro (for example, broad spectrum penicillins). Nearly all antibiotics have been implicated in inducing *C difficile* associated diarrhoea, though to a lesser extent with intravenously administered aminoglycosides when they are given singly.[15] Combinations of antibiotics have a higher risk of inducing colonisation by *C difficile* and the development of disease. The relative risk of an antibiotic inducing *C difficile* associated disease depends on how often and for how long it is used.

Other factors—Although antibiotic use is the most important predisposing factor for *C difficile* induced disease, other factors also play a part, such as a susceptible host treated with chemotherapeutic and immunosuppressive agents, patients with leukaemia, or patients in intensive care. Pseudomembranous colitis can occasionally occur without exposure to antibiotics.

Antibiotics and *C difficile* infection

- Almost all antibiotics have been implicated in *C difficile* infection, including oral, intravenous, intravenous, intramuscular, and topical antibiotics
- Relative risk data for antibiotics depends on how extensively they are used
- Antibiotics implicated in *C difficile* infection include:

Penicillins	Erythromycin
Cephalosporins	Tetracyclines
Clindamycin	Trimethoprim
Quinolones	Sulphonamides

TOXIN PRODUCTION

Toxigenic strains of *C difficile* produce two large protein exotoxins: toxin A is a 308 kDa enterotoxin, and toxin B is a 250/270 kDa cytotoxin. Almost all toxigenic strains possess the genes for, and produce, both toxins. Toxin A causes fluid secretion and mucosal damage, resulting in diarrhoea and inflammation. The precise mechanism of action has not yet been determined, but the toxin seems to bind to specific mucosal receptors and then enters the cell, causing rounding of the cell by altering the cell's actin cytoskeleton. Similar cellular changes occur with toxin B in cultured cells. Toxin B is not enterotoxigenic in animals but is a powerful cytotoxin, 1000 times more potent in tissue cultures than toxin A. Toxin A also attracts human neutrophils in vitro.[16] Both toxins activate the release of cytokines from human monocytes,[17] and this effect may be responsible for the colonic inflammation seen in pseudomembranous colitis. Other possible virulence factors have been described but are less well understood.

Some strains of *C difficile* are non-toxigenic and lack the genes for toxins A and B. It is generally accepted that non-toxigenic strains are also non-pathogenic. However, we recently studied an outbreak of diarrhoea among elderly patients in a hospital ward that was associated with a non-toxigenic strain of *C difficile* (unpublished data), and similar associations have been reported.[18] Thus, other pathogenic mechanisms may be present and should be investigated.

Epidemiology

There has been a pronounced rise in the number of reports of *C difficile* infection, particularly in the past decade. This may be due in part to better isolation methods and improved reporting. Most reports of infection have been in patients over 50. The increasing numbers of elderly patients in Western countries means that *C difficile* infection in hospital patients is likely to increase. Other patients who seem particularly susceptible to *C difficile* associated disease include surgical patients, those with chronic renal disease, and those with cancer. This may reflect the use of antibiotics by these patients and, in the latter two groups, the use of immunosuppressive drugs. Outbreaks of *C difficile* associated disease are usually restricted to wards where there is clustering of susceptible patients and heavy environmental contamination with *C difficile* due to the presence of patients with diarrhoea caused by the organism.

CARRIAGE RATES

The reported carriage rates of *C difficile* in healthy adults have varied from 0-3% in Europe to up to 15% in Japan.[19] These differences reflect variations in the sensitivity of methods of culture and in the selection of subjects who have not previously received antibiotics. Asymptomatic carriage in healthy volunteers increased to 46% after they were given antibiotics.[15] A more accurate assessment of the carriage and acquisition rates in patients is provided by prospective surveillance studies carried out over 3-11 months, in which patients were screened for *C difficile* shortly after admission into hospital and once or twice weekly thereafter.[20] In these studies the carriage rate, which reflects the level of carriage in the community, varied from 1·4% in general medical patients to 8·6% in patients with haematological malignancies, reflecting the repeated hospital admissions of the second group of patients.[20,21]

Carriage rates in healthy neonates are much higher (35-65%)[19] *C difficile* is acquired from the environment[22,23] or from the birth canal of the colonised mother.[23] Neonatal colonisation is almost always asymptomatic despite the presence of toxin producing strains of *C difficile* and high toxin titres in infants' stools. The absence of symptoms is thought to be due to the immature nature of the intestinal flora and lack of development of the toxin receptors in the intestine. After the age of 1-2 years the rate of colonisation decreases, and children become increasingly susceptible to *C difficile* associated disease.

ACQUISITION OF C DIFFICILE

While nosocomial acquisition and cross infection with *C difficile* had been suspected for several years, convincing evidence for nosocomial acquisition was provided only when reliable typing methods became available. Several fingerprinting and typing methods have been used successfully for epidemiological studies.[20] The acquisition rate of *C difficile* in hospital patients varies with the patient population studied, the use of antibiotics, and the presence or absence of an outbreak of *C difficile* associated diarrhoea or colitis in the wards studied. For example, patients with haematological malignancies have an acquisition rate of 2·6% in the absence of an outbreak and 21·5% during an outbreak.[20] The increased rate of acquisition

is due to the inevitable heavy environmental contamination and cross infection. These studies have been reviewed elsewhere.[20]

Environmental contamination—Colonisation is thought to result from ingestion of spores, which can survive in extreme environmental conditions and persist for months or years. Ingested spores survive the acidity of the stomach and convert to vegetative organisms when they reach the colon. Contamination with *C difficile* is common in hospital, and infected patients are an important reservoir of the organism. *C difficile* has been isolated from floors, toilets, bedpans, and furniture, especially from areas where patients with *C difficile* infection have been nursed.[24] *C difficile* has also been isolated from healthcare workers' hands, and hand transmission is thought to be important in the acquisition and spread of *C difficile* in hospital patients.[25] Symptomatic *C difficile* infection has been reported in healthcare workers.[26]

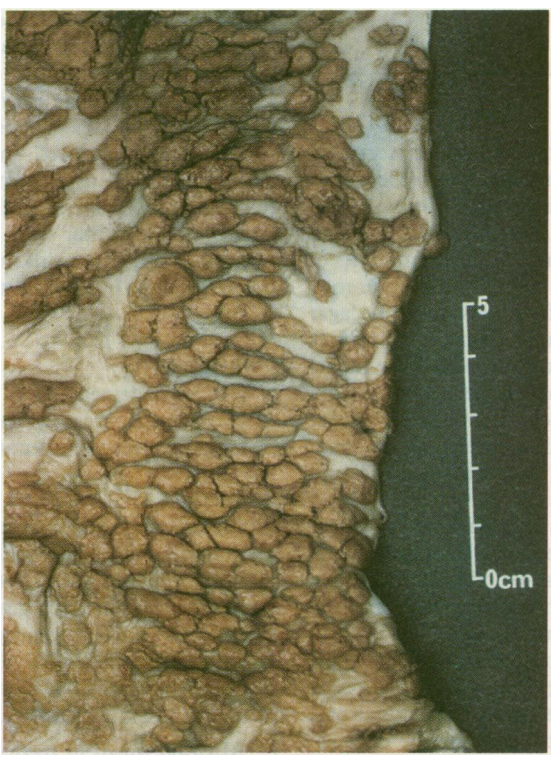

FIG 1—*Postmortem appearance of colon of patient with pseudomembranous colitis*

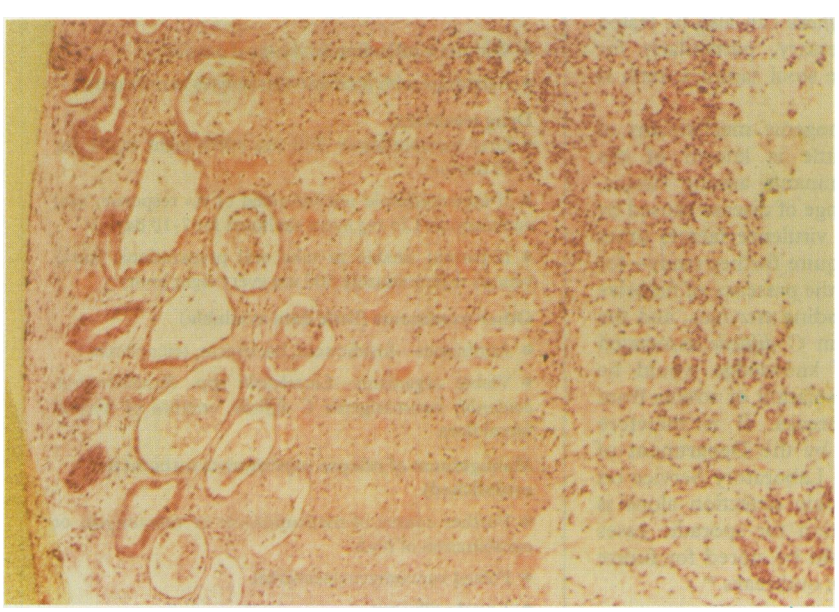

FIG 2—*Endoscopic biopsy specimen from patient with pseudomembranous colitis showing considerable epithelial necrosis overlaid by pseudomembrane consisting of inflammatory cells, mucin, and cellular debris (haematoxylin and eosin stain)*

Diagnosis

The diagnosis of *C difficile* associated disease is based on clinical presentation and laboratory investigations.

Clinical diagnosis of *C difficile* associated disease

- Unexplained diarrhoea, especially if watery
- History of antibiotic treatment
- Endoscopic appearance
- Response to withdrawal of implicated antibiotic and to treatment with metronidazole or vancomycin

CLINICAL PRESENTATION

C difficile causes a spectrum of disease ranging from mild diarrhoea to life threatening pseudomembranous colitis with toxic megacolon and possible perforation. The most severe forms of the infection are the least common.

The commonest presenting symptom is the sudden onset of unexplained diarrhoea (unformed, loose, or watery stools more than twice daily). The stools are foul smelling and contain mucus but generally no blood. Patients may be feverish. A history of antibiotic use can usually be ascertained. Symptoms may occur at varying times after drug use, as early as the first or second day of treatment or weeks after the antibiotics have been discontinued. Physical examination in mild cases of antibiotic associated diarrhoea is often unremarkable apart from slight tenderness in the lower abdomen. Sigmoidoscopy in such patients is often normal and is thus of no diagnostic use.

Patients with antibiotic associated colitis have profuse diarrhoea and abdominal pain and distension accompanied by nausea, fever, and dehydration. The peripheral white blood cell count is often raised. Sigmoidoscopy usually reveals a non-specific colitis which may be diffuse or patchy.

Patients with pseudomembranous colitis have more pronounced systemic symptoms. Sigmoidoscopy reveals characteristic raised yellow plaques (fig 1), which are pathognomic of *C difficile* infection and which consist of inflammatory cells, fibrin, mucus, and debris (fig 2). The plaques vary in diameter from 2 mm to 10 mm, and the intervening mucosa is usually normal or only slightly erythematous. In severe cases the rectum and colon are also affected, but in up to 10% of cases the lesions are confined to the proximal colon and are therefore inaccessible unless colonoscopy is performed. It is worth noting that pseudomembranes do not form in neutropenic patients because of lack of neutrophils, and diagnosis is mainly based on clinical history and laboratory investigations.

Patients with *C difficile* infection may occasionally present with fulminant colitis and paralytic ileus. Toxic dilatation of the colon may also occur, which could lead to a decrease in diarrhoea. Colonic perforation, peritonitis, and secondary Gram negative bacteraemia may result. An abdominal radiograph may reveal a dilated colon and oedema of the colonic mucosa, which manifests as "thumbprinting." Sigmoidoscopy or colonoscopy are contraindicated in cases of fulminant colitis because of the risk of bowel perforation. These severe conditions present as medical and surgical emergencies, may require emergency surgical intervention, and are associated with a high mortality if action is delayed. Diagnosis is based on clinical grounds, history of antibiotic use, and laboratory diagnosis of *C difficile*.

C DIFFICILE IN NON-INTESTINAL DISEASE

C difficile has occasionally been isolated from wounds and pleuropulmonary infections. It has also

been implicated in inflammatory bowel disease and the sudden infant death syndrome, but the evidence remains unconvincing.

LABORATORY DIAGNOSIS

The laboratory methods for the diagnosis of *C difficile* infection are essentially detection of the organism and demonstration of its toxins.

Laboratory diagnosis of *C difficile* infection

- Culture of *C difficile* on selective medium
- Detection of toxins in stools:
  - Toxin B—cytopathic effect on tissue cell lines, neutralisation with *C sordellii* or *C difficile* antitoxins
  - Toxin A—enzyme linked immunosorbent assay (ELISA) kit (less sensitive than toxin B assay but more convenient)
- Polymerase chain reaction research development
  - Directly on stool extract
  - On clinical isolates

Culture of C difficile

C difficile is a Gram positive, spore forming, anaerobic bacillus. It can be isolated from stools with a selective medium—cefoxitin, cycloserine, and fructose agar (CCFA) (Oxoid, Basingstoke). Improved recovery is obtained by reducing the concentrations of cefoxitin and cycloserine to 8 mg/l and 250 mg/l respectively. Also the use of fastidious anaerobe agar (FAA) seems to enhance fluorescence of *C difficile* colonies. Several other modifications of culture media have been described.[27] *C difficile* is best identified by its colonial morphology, smell, fluorescence under long wave ultraviolet light (yellow-green), Gram stain, and specific pattern of volatile fatty acids on gas liquid chromatography.

A commercial kit based on latex particle agglutination has been introduced for the identification of *C difficile* (Mercia Diagnostics, Guildford), but cross reactions occur with other clostridial species and the kit is relatively expensive for routine use.

Culture of stool specimens provides the most sensitive method of detecting *C difficile*. Isolation of the organism is essential for the typing of strains as part of epidemiological studies to distinguish between sporadic cases and cross infection with single strains in outbreaks. The strains of *C difficile* can also be tested for toxin production if necessary, particularly for symptomatic patients in whose stool samples toxin is not detected.

Our knowledge of the pathogenic mechanisms of *C difficile* is incomplete. Little is known of the variations in virulence determinants among strains, which may explain the wide range of disease caused by this organism. There are also virulence factors other than toxins A and B which require further study—for example, the ability to adhere, the presence of fimbriae and capsule, other tissue degrading enzymes, and the role of non-toxigenic strains in *C difficile* associated disease. No advancement of knowledge would be possible without culture and isolation of strains from sporadic infections and outbreaks. It is therefore disappointing that the report by the Department of Health and the Public Health Laboratory Service on *C difficile* recommends that toxin detection alone is adequate laboratory investigation for sporadic cases and that stool specimens should be stored for future culture only for investigations of outbreaks.[28]

Demonstration of C difficile toxins

Because of its high sensitivity and specificity, many consider the ideal method of detecting *C difficile* toxin B to be the demonstration of its cytopathic effect in tissue culture and the specific neutralisation of cytotoxicity by *Clostridium sordellii* antisera. This method, however, takes 48-72 hours, and not all routine microbiology laboratories have access to tissue culture facilities. More rapid methods of toxin detection have been developed and are commercially available.[29]

Latex agglutination was developed for detecting toxin A in stool specimens but has subsequently been shown to detect an unrelated antigen (glutamate dehydrogenase) which is present in both toxigenic and non-toxigenic strains and which cross reacts with other organisms. This method is not recommended because of poor sensitivity and specificity.[27]

Enzyme immunoassays to detect toxin A have recently become commercially available (such as the Premier Kit, Meridian Diagnostics, Cincinnati, USA). These are not as sensitive as tissue culture methods but have high specificity and are rapid, allowing a result to be obtained on the same day. However, their cost means that it is common practice to perform batch tests rather than individual ones, thus defeating the purpose of having a rapid test.

The polymerase chain reaction has been used to detect *C difficile* in stool specimens[30] and to identify toxigenic strains among isolates, and directly from primary culture plates, by using primers based on the genes for toxins A and B.[31] These methods are highly specific and sensitive, but further work on the direct detection of *C difficile* and its toxins in stool samples is needed before they can be used routinely.

Management

The management of *C difficile* infection consists of two aspects, treating individual patients and controlling the spread of infection.

TREATMENT

The most important strategy for treating infection is to stop the inducing antibiotics and rehydrate the patient. Sometimes it is not possible to stop antibiotic treatment if it has been given for serious infection. In mild cases the symptoms will usually settle after stopping the antibiotics. If a patient is ill and the diarrhoea persists, antibiotic treatment for *C difficile*

Management of patients with *C difficile* associated disease

- Withdraw offending antibiotic if possible
- Rehydrate with fluid and electrolytes as necessary

Drug treatment

- Oral metronidazole 400 mg every eight hours for 7-10 days *or*
- If metronidazole not tolerated or no response, oral vancomycin 125 mg every six hours for 7-10 days *or*
- If patient unable to take oral preparations, intravenous metronidazole 500 mg every eight hours

Other treatments (little data available)

- Antibiotics—fusidic acid, bacitracin, teicoplanin
- Viable organisms—*Lactobacilli* spp, *Saccharomyces boulardii*, non-toxigenic *C difficile*, enemas of normal faecal flora

Management of relapse (optimum treatment not established)

- Further course of metronidazole or vancomycin or combination of both
- Pulsed vancomycin treatment
- *Lactobacillus* spp
- *Saccharomyces boulardii*

infection is indicated. Antidiarrhoeal agents should not be given as they may aggravate the condition and occasionally predispose to toxic megacolon.[32]

The purpose of the antibiotic treatment against *C difficile* is to achieve effective antimicrobial concentrations in the bowel. Initial antibiotic treatment is with oral metronidazole (400 mg every eight hours) or oral vancomycin (125 mg every six hours) for 7-10 days. There is anecdotal and theoretical support for the suggestion that vancomycin may be superior to metronidazole in treating *C difficile* infection. Vancomycin is not absorbed from the bowel after oral administration and thus achieves concentrations many times higher than the known minimal inhibitory concentration of *C difficile*. However, the one prospective randomised trial to compare the two drugs did not show any difference between them.[33] Vancomycin is more expensive than metronidazole and is thus reserved for patients who cannot tolerate metronidazole, who do not respond to metronidazole, or who are severely immunocompromised and have severe antibiotic associated diarrhoea or pseudomembranous colitis. Patients should respond to treatment within 48 hours and return to normal within 4-7 days, but severe conditions may take longer and may require higher doses of vancomycin (up to 500 mg every six hours).

Patients with ileus or any other contraindication to oral treatment should receive intravenous metronidazole. This is excreted into the bile and secreted into the intestine and achieves bactericidal concentrations in the colon. Intravenous vancomycin should not be used as there is inadequate excretion of this agent into the bowel lumen.

Other drugs that have been used in initial treatment include bacitracin, fusidic acid, and teicoplanin. There is, however, little experience in their use, and data are insufficient at present to recommend them.

RELAPSE

About a fifth of patients will have a symptomatic relapse after completing initial treatment. The diagnosis of recurrent *C difficile* infection should be confirmed by reculturing for *C difficile* and demonstrating *C difficile* toxin in the stools. Possible mechanisms for relapse include reinfection with the same or another strain of *C difficile*, germination of residual spores in the colon, and further antimicrobial treatment. Relapse as a result of strains being resistant to antibiotics seems to be rare.

A mild symptomatic relapse may resolve spontaneously, and antimicrobial treatment is not indicated. Most relapses will respond to a further course of treatment with metronidazole or vancomycin. However, some patients have numerous attacks, and there have been various methods of treating recurrent infection.[34] These have included using prolonged courses of oral vancomycin, a combination of vancomycin and metronidazole, and pulsed vancomycin. The intervals between pulses (2-7 days) allows residual spores to germinate to vegetative forms, which are then killed by vancomycin. Cholestyramine, which has previously been used as treatment, is not recommended because, although it binds the *C difficile* toxins in the intestine, it also binds vancomycin. Another approach has been the administration of microorganisms that antagonise *C difficile*. Oral preparations have included non-toxigenic *C difficile*, *Lactobacillus* spp, and the yeast *Saccharomyces boulardii*. Other investigators have used enemas of normal faecal flora. The management of clinical relapses of *C difficile* infection has not been investigated extensively, and the optimum treatment is not yet known.

INFECTION CONTROL

Control measures are aimed essentially at control of spread. Patients with *C difficile* diarrhoea are the main source of infection, but occasionally asymptomatic carriers may serve as reservoirs for nosocomial acquisition and environmental contamination.

Prevention of cross infection and spread

- Isolate patients with diarrhoea
- Institute full enteric precautions
- Thorough handwashing by all attending staff after contact with patients and their environment
- Reduce environmental load of spores by thorough daily cleaning

Routine infection control procedures are the most important aspect of control. Any patient with suspected infectious diarrhoea should be moved to a single room, and enteric precautions which entail wearing disposable aprons and gloves and strict handwashing, should be observed until the cause of the diarrhoea is identified. If the diarrhoea is due to *C difficile*, treatment can be started. Patients should be kept isolated until they have formed stools and these have been tested for the presence of toxin. If there is an outbreak, it may be possible to keep affected patients in one area of the open ward with full precautions and have a designated group of nurses to care for them (cohort nursing). The ward should be closed to transfers and admissions during the outbreak.

Handwashing by all staff before and after contact with patients is essential, and this should be enforced and monitored by the infection control team. Soap or detergent may be used for handwashing as there is no evidence that alcohol based hand rubs are more effective at eliminating *C difficile* spores. *C difficile* spores are inherently resistant to the antiseptic hypochlorite and are able to persist for long periods in the environment. However, it is generally agreed that some attempt should be made to reduce the environmental spore load by thorough daily cleaning of the areas occupied by patients with symptomatic *C difficile* infection.

Appropriate use of antibiotics is essential to prevent *C difficile* infection. Prescribing of antibiotics should be monitored constantly, and an antibiotic policy should be adopted in hospitals. Detailed recommendations for preventing and managing *C difficile* infection have been published by a joint Department of Health and Public Health Laboratory Service working group in 1994.[28]

Prevention of *C difficile* infection

- Prompt isolation and treatment of symptomatic patients
- Control of antibiotic use
- Treatment of asymptomatic carriers who may act as a reservoir is undesirable and ineffective
- Future prospects for prevention—by development of vaccine and immunisation

FUTURE DEVELOPMENTS

Research into the development of a vaccine against *C difficile* to immunise high risk patients is currently under way. Serum antibodies to the toxins are present, but it is unlikely that they play a part in modifying the disease. However, IgA antibodies to toxin A are secreted into the colonic lumen by most patients with *C difficile* associated disease, and these may block the binding of toxin A to its receptor.[35] This suggests that immunisation against *C difficile* toxins may be a useful

means of preventing the disease. Passive immunisation may be achieved by using antisera to toxin A or to synthetic peptides based on specific amino acid sequences of toxin A, and active immunisation may be induced by cloning specific sequences from toxin A in a suitable delivery system to provoke a potent local immune response in the colon. Both of these approaches are being investigated.

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A PATIENT WHO CHANGED MY PRACTICE

The zero option

The patient who changed my practice was a woman in her mid-70s. I was a senior registrar and I met her during a preoperative ward round. She was due to have a vaginal hysterectomy and repair the next day. Following my chief's practice I was interviewing patients in a small side room off the main ward.

I can remember explaining to her that the meeting was intended to give her an opportunity to ask any questions that she might have about the operation. It also gave me the chance to make sure that she understood what we were going to do and to get her informed consent to the procedure. She did not ask many questions, but she did seem to understand my description of the operation. Before signing the consent form I noticed that her brow was furrowed and that she really did not seem happy at all with what was going on. I confronted her with this, to which she replied that she did not understand why she was there. I then asked her to tell me how she had come to be admitted for a major surgical procedure.

Responding to a cervical smear campaign, she had gone to her general practitioner for her first smear, which was duly taken. She had no other complaint. After the examination her doctor mentioned that she had a slight prolapse and suggested that it would be best if she saw a gynaecologist. My chief recommended a ring pessary and made a follow up appointment. The ring did not make her feel any more comfortable as she had not felt uncomfortable to start with. It did, however, produce stress incontinence. She mentioned this at the second clinic visit when she saw the registrar; he fitted a larger ring, which made her incontinence worse. At her third visit she saw the senior house officer, who noted the worsening symptoms despite the ring pessary and gave her a date for admission for vaginal hysterectomy and repair.

Her fears and anxiety were understandable. She had no symptoms, had seen her doctor only for a smear and had ended up on a conveyor belt leading to a major procedure, which would not have improved the quality of her life; indeed, it may have had the opposite effect. I asked her why she had not called a halt much earlier, to which she replied, "People of my generation don't argue with doctors, Doctor."

What did I learn from this encounter? Firstly, do not create potential problems for the patient and unnecessary work for the NHS by bringing to the notice of asymptomatic patients benign conditions which are not affecting the quality of life. Secondly, ensure that the way in which the outpatient clinic is organised is such that junior members of the team are not responsible for booking patients for inappropriate procedures. Thirdly, always offer the patient the zero option—that is, the opportunity to have nothing done. In the surgical specialties we are taught that management may be conservative or surgical, but it is not emphasised often enough that doing nothing may often be the best management.

That lady went home shortly after our chat, considerably relieved. She had taught me a major lesson: give patients the chance to say "no."—BEVERLEY WEBB *is a consultant obstetrician and gynaecologist in Stevenage*