Clostridium difficile in association with sporadic diarrhoea

R P BRETTLE, I R POXTON, J McC MURDOCH, R BROWN, MARIE D BYRNE, J G COLLEE

Abstract

A total of 154 patients admitted to an infectious diseases unit were included in a year's prospective survey of sporadic diarrhoeal disease. Stools from 10 of them yielded Clostridium difficile, generally on more than one occasion. Twelve of these patients were assessed as having a severe or moderately severe gastrointestinal illness: CI difficile was the only pathogen isolated from 10 of them, and two had an associated salmonella infection. Seven had each a recent course of antibiotics, but five had not taken antibiotics. Faeces from seven patients with moderate or mild gastroenteritis illness yielded CI difficile, and two of these patients also had an associated salmonella infection. Two patients in this group had no antibiotic history.

From these findings, the occurrence of C difficile in faeces could not be described as antibiotic-associated. Faecal Cl difficile cytotoxin was detected in only six patients, and generally at low levels. In such patients a more relevant pathogenic index might take account of the numbers of Cl difficile present and of their toxigenic potential.

Introduction

Though a pathogenic association between Clostridium difficile and pseudomembranous colitis is accepted, the role of this organism in intestinal health and disease in infants and adults is still uncertain.1 The development of better selective culture procedures has greatly facilitated the isolation of Cl difficile from stools.2 We have therefore conducted a prospective study to determine the range of circumstances in which Cl difficile may be detected and possibly implicated in sporadic cases of diarrhoea in adults admitted during one year to an infectious diseases hospital.

Patients and methods

We included in the survey all patients admitted to the infectious diseases unit during November 1979 to October 1980 who presented with diarrhoea or developed diarrhoea. During the study period there was no recognised epidemic of gastroenteritis or diarrhoeal...
**Bacteriological Investigations**

**Specimens of Faeces—**Samples were taken with care to avoid contamination and submitted in sterile plastic containers without transport medium. When *Clostridium difficile* was isolated follow-up specimens were submitted.

**Microscopy—**Suitable preparations of each specimen were examined by phase-contrast microscopy; a Gram smear was prepared and examined by light microscopy. Pus cells, red blood cells, and Gram-positive rods with spores were noted.

**Culture—**Approximately 0·1 g or 0·1 ml of faeces was plated on cefoxitin cycloserine fructose agar medium (CCFA)\(^1\) and incubated anaerobically in 90% hydrogen and 10% carbon dioxide for 24 hours at 37°C. Another, similar sample was inoculated into Robertson's cooked meat broth and incubated in the same way. The CCFA plate was examined for typical colonies, and the number of *Clostridium difficile* in the faeces estimated semi-quantitatively: \(+\) = growth in well only (10\(^4\)-10\(^5\) organisms/g faeces), \(++\) = growth in well and on primary streaks (10\(^5\)-10\(^6\)), and \(++++\) = growth in well, primary streaks, and secondary streaks (>10\(^5\)). The cooked meat broth culture was plated on CCFA medium and this plate subsequently examined for *Clostridium difficile* and scored \(\pm\) if positive only after enrichment. All CCFA plates were incubated for 48 hours before being discarded as negative. All presumptive isolates were confirmed by gas chromatography of the volatile fatty-acid products of metabolism. The acidified supernate from a 24-hour culture in proteose peptone, yeast extract, serum, and glucose medium\(^2\) was examined on a column of 15% Supelco SP1220, 1% H\(_2\)PO\(_4\) on chromasorb W acid washed in a Pye Unicam model 104 gas chromatograph. A typical fatty-acid profile with pronounced peaks of acetate, N-butyric, isobutyric, N-valeric, isovaleric, and isocaproic acids was taken as diagnostic.

**Toxin assay—**Toxin was assayed essentially according to Bartlett\(^3\) by observing a cytopathogenic effect on monolayers of human embryonic fibroblast cells. All faecal assays were performed retrospectively on specimens that had been stored at \(-18°C\). Before centrifugation, solid faecal specimens were mixed with a minimum of physiological saline to give a fluid suspension; liquid specimens were used without further dilution. Neutralisation tests were performed with *Clostridium sordellii* antitoxin (10 \(\mu\)l of a one in 25 dilution added to 90 \(\mu\)l medium over the monolayer just before adding 10 \(\mu\)l of faecal extract). The toxicity of fresh isolates of *Clostridium difficile* was estimated by assaying the supernate from five-day cultures in 3.5% (w/v) brain-heart infusion medium supplemented with 1% (w/v) proteose peptone (both from Oxoid).

### Results

A total of 154 patients were included in the survey, 145 of whom were referred by their general practitioners and nine transferred from other hospitals. All were admitted for investigation and treatment of illnesses that either presented with or developed a diarrhoeal phase. Of these, 39 had medical or surgical conditions that might reasonably be associated with diarrhoea and not regarded as primarily infective. Of the remaining 115 patients, 27 (23%) yielded enteropathogenic bacteria, 12 (10%) had salmonella infections, and 15 (13%) yielded campylobacters. A reovirus may have caused another one diarrhoeal episode. From four of our 115 patients a *Salmonella* sp and *Clostridium difficile* were isolated together. Eighty-seven patients had diarrhoea that might have been infective and was unexplained; from 15 of these we isolated *Clostridium difficile* alone.

During the survey 35 patients were admitted who did not fulfil the criteria of the study—that is, they did not produce three or more loose stools daily for one day or more. Stools from these patients were also investigated and contained no *Clostridium difficile*. Since other patients resident in hospital are not entirely equivalent, the 35, like the study patients, were in the main admitted directly from the community, were regarded as matched controls.

*Clostridium difficile* was isolated from a total of 19 patients (group 1). Of these, 17 came from their homes and two were from other hospitals. The age range was 19 to 88 years, with a mean of 56; the male to female ratio was 1:4; two patients died; 15 of the patients presented during May to October, and one had travelled abroad recently. There was no case-to-case spread of *Clostridium difficile*. On only one occasion were two patients with the organism in the ward at the same time. A check on the ward area and clean bedpans during the survey yielded no *Clostridium difficile*.

Toxin that could be neutralised by *Clostridium sordellii* antitoxin was detected in the faeces of six patients in group 1. No cytotoxin was detected in any of the specimens that were available for testing (106 tested out

### Details of 19 patients from whom *Clostridium difficile* was isolated (group 1)

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age and sex</th>
<th>Severity of illness</th>
<th>Antibiotic or steroid history</th>
<th>Other enteric pathogens isolated</th>
<th>Other illnesses diagnosed</th>
<th><em>Clostridium difficile</em> isolated</th>
<th>In-vitro toxin titre</th>
<th>Faecal cytotoxin titre</th>
<th>Treatment and comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42 F</td>
<td>++ + + +</td>
<td>Co-trimoxazole†</td>
<td></td>
<td>Rheumatic heart disease</td>
<td>+ +</td>
<td>1 000</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>2</td>
<td>69 F</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td>Diabetic, chronic obstructive airways disease</td>
<td>+ +</td>
<td>100</td>
<td>1 024</td>
<td>Vancomycin, died</td>
</tr>
<tr>
<td>3</td>
<td>85 F</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td>Ischaemic heart disease</td>
<td>+ +</td>
<td>10 000</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>4</td>
<td>77 F</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td>Ischaemic heart disease, chronic obstructive airways disease</td>
<td>+ +</td>
<td>16</td>
<td></td>
<td>Vancomycin, cholestyramine</td>
</tr>
<tr>
<td>5</td>
<td>88 F</td>
<td>++ + + +</td>
<td>Ampicillin, fluoloxacin</td>
<td></td>
<td>Gastrointestinal atony</td>
<td>+ +</td>
<td>1 000</td>
<td>ND</td>
<td>Vancomycin, cholestyramine, vancomycin, chloramphenicol</td>
</tr>
<tr>
<td>6</td>
<td>40 M</td>
<td>++ + + +</td>
<td>Erythromycin</td>
<td><em>Salmonella panama</em></td>
<td>Cerebral cortical atony</td>
<td>+ +</td>
<td>1 000</td>
<td>1 024</td>
<td>Vancomycin, cholestyramine, vancomycin, chloramphenicol</td>
</tr>
<tr>
<td>7</td>
<td>24 F</td>
<td>++ + + +</td>
<td><em>Salmonella derby</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>65 F</td>
<td>++ + + +</td>
<td>Prednisone, co-trimoxazole†, cefuroxim†</td>
<td><em>Salmonella derby</em></td>
<td>Rheumatoid arthritis</td>
<td>+ +</td>
<td>10 000</td>
<td>1 024</td>
<td>Vancomycin, cholestyramine, vancomycin, chloramphenicol</td>
</tr>
<tr>
<td>9</td>
<td>87 F</td>
<td>++ + + +</td>
<td>Ampicillin</td>
<td></td>
<td>Chronic obstructive airways disease</td>
<td>+ +</td>
<td>100 000</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>10</td>
<td>19 F</td>
<td>++ + + +</td>
<td>Penicillin, cephradine, penicillin</td>
<td></td>
<td>Panhypopituitarism</td>
<td>+ +</td>
<td>10</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>11</td>
<td>85 F</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td>Ischaemic heart disease, hypothyroidism</td>
<td>+ +</td>
<td>100 000</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>12</td>
<td>44 F</td>
<td>++ + + +</td>
<td>Doxycycline</td>
<td></td>
<td></td>
<td>+ +</td>
<td>100</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>13</td>
<td>50 F</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td>Colostomy, diverticulosis</td>
<td>+ +</td>
<td>100 000</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>14</td>
<td>84 F</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td>Secondary myocardial infarction</td>
<td>+ +</td>
<td>100</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>15</td>
<td>37 M</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Died from chronic obstructive airways disease</td>
</tr>
<tr>
<td>16</td>
<td>79 M</td>
<td>++ + + +</td>
<td>Ampicillin, fluoloxacin†, chloramphenicol, amoxyacin,† erythromycin</td>
<td><em>Salmonella agona</em></td>
<td>Femoral popliteal bypass</td>
<td>+ +</td>
<td>10</td>
<td>16</td>
<td>Died from chronic obstructive airways disease</td>
</tr>
<tr>
<td>17</td>
<td>75 M</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td></td>
<td>+ +</td>
<td>10</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>18</td>
<td>19 F</td>
<td>++ + + +</td>
<td>Flucloxacillin, lincomycin†</td>
<td><em>Salmonella virchow</em></td>
<td></td>
<td>+ +</td>
<td>10</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>19</td>
<td>19 F</td>
<td>++ + + +</td>
<td></td>
<td></td>
<td></td>
<td>+ +</td>
<td>10</td>
<td>ND</td>
<td>Vancomycin</td>
</tr>
</tbody>
</table>

ND = Not done.
†Organism also isolated from blood cultures.
*Neutralised by *Clostridium sordellii* antitoxin.
§Not detected in undiluted extract.
§§Diarrhoea developed while taking drug.
of a possible 135) from patients who did not have Cl difficile in the faeces (group 2). Twelve of the 19 patients in group 1 and 17 of the 135 in group 2 had courses of antibiotics during the six weeks before illness was reported to hospital, results of one of the 15 patients with campylobacter diarrhea and none of the eight with salmonella infections alone had received antibiotics during that time.

There was no appreciable difference between the two groups in age, sex, mortality, seasonal incidence, contact history, or travel abroad, and the groups were indistinguishable in terms of presentation, biochemical and haematological profiles, and clinical course. Group 2 included a wide variety of acute surgical cases (appendicitis, perforated visisus, obstructed hernia, ischaemic ileitis, and colitis) and medical conditions (diverticular disease, carcinomatosis, pneumonias, ulcerative colitis, and renal failure).3 There was no obvious correlation with faecal excretion of Cl difficile in patients with diarrhoea other than recent antibiotic treatment, which was not an invariable association.

Climacteric assessment—The table summarises relevant data for the 19 patients in group 1. The patients were divided retrospectively into four groups based on the severity of their illness; duration, frequency, and persistence of diarrhoea; occurrence of blood in the stools; fever; signs and symptoms of dehydration; increased white cell counts; and low serum albumin concentrations. The four groups were clinically severe (+ + + +), moderately severe (+ + +), moderate (+ +), and mild (+). We considered that 12 patients had appreciable enteropathy, and it was among these that the high white cell counts and low serum albumin values were recorded. The symptoms included diarrhoea, vomiting, and abdominal pain; three patients had frank blood in the stools.

Microbiological assessment—Semi-quantitative assessments of the isolation of Cl difficile from the 19 patients in group 1 showed many examples of cross-contamination with clinical severity. The reproducibility of our assessments of in-vitro toxigenicity of the Cl difficile isolates and it was largely true of our estimations of toxin in the faeces of these patients, though with one exception (case 16) all of those with detectable faecal toxin were severely ill. Five non-cytotoxic strains of Cl difficile were isolated; four were associated with diarrhoea in patients from whom no other presumptive pathogen was isolated. Pathogens other than Cl difficile were isolated from two severely ill patients (cases 7 and 8). One of these (case 7) had an associated salmonella bacteraemia, and presented with septicaemic shock initially considered to be secondary to an Escherichia coli urinary tract infection. With the exception of these two patients, 12 had a clinically severe illness in the absence of any presumptive cause other than Cl difficile. Seven of the 12 had received antibiotics within the previous six weeks, but the other five patients had not received antibiotics.

Management—Vancomycin was given to 10 of the 12 patients with clinically severe and moderately severe disease and to one patient with moderate disease. The decision to treat was clinical, though it was influenced by finding Cl difficile in the stools. At the time of each patient’s illness we did not have the results of the faecal toxin assay; in retrospect it could be argued that several of our patients did not merit specific treatment. Cholestyramine was used only twice; it failed to improve one patient (case 12), who eventually received a course of vancomycin.

Sigmoidoscopy was performed on 11 patients (cases 1-8 and 10-12). Moderate inflammation with contact bleeding was seen in four (cases 1, 3, 7, and 8), but none had pseudomembranes or ulcers; the rest were normal. Rectal biopsy in six patients (cases 1, 3, 5, 6, 7, and 8) showed oedema with chronic or acute inflammatory infiltrates. Barium enemas were performed on two patients, and one showed severe diverticulosis.

Discussion

In a recent survey no Cl difficile was isolated from a group of 62 healthy adults, but in an earlier one up to 3% of healthy adults were found to be carrying Cl difficile.8 Most of this work, however, was done before the introduction of the selective CCFA medium.8 Hence these surveys may have underestimated the carriage rate of Cl difficile if some normal adults had carried small numbers of organisms in their stools. Similarly, in our 35 controls we were unable to isolate any Cl difficile on CCFA. Cl difficile had been identified in the genital tract of man and women,9 occurs commonly in the faeces of neonates,4,11 and has an accepted causative association with pseudomembranous colitis in adults. The organism has been associated with 6-48% of cases of antibiotic-associated diarrhoea9-13; the assumed association is based on faecal cytotoxin assays or isolations of the organism on selected media. Twelve of our 29 patients with antibiotic-associated diarrhoea yielded Cl difficile, but none had pseudomembranous colitis diagnosed. Mogg et al14 and Keighley et al15 have demonstrated the inadequacy of sigmoidoscopy and rectal biopsy and the greater reliability of faecal cytotoxin determinations in diagnosing pseudomembranous colitis.

Evidence is now appearing implicating Cl difficile in non-antibiotic-associated colitis,16-18 exacerbations of chronic inflammatory bowel disease,19,20 and postoperative diarrhoea.19,20 The clustering of some cases suggests that cross-infection may occur.

Falsen et al11 found that Cl difficile was the second commonest enteropathogenic isolate in a survey of many stool specimens submitted to a laboratory. Our hospital-based survey would have excluded many young patients with salmonella and campylobacter infections treated by general practitioners without referral to hospital. In our series Cl difficile was the commonest presumptive enteric pathogen. Unknown or under-diagnosed cases of pathogen-associated diarrhoea were probably included in cases of presumed infective diarrhoea. For example, the occurrence of rotaviruses and other enteropathogenic viruses in an adult population has not been adequately assessed and merits further consideration. With these provisos, Cl difficile seems to be associated with some cases of diarrhoea requiring admission to hospital and may be acting as a primary pathogen in a proportion of these. In common with the findings of others,11,12 we were able to isolate an accepted infective agent from only 28 (24.5%) of 115 patients. Our suggestion that Cl difficile may be an additional accepted cause of infective diarrhoea is supported by recent reports of hospital studies21,22 and animal studies.23,24

The titres of faecal cytotoxin detected in our survey (from 0-1024) were appreciably lower than those obtained in classic cases of pseudomembranous colitis (500-400 00004, 1000-5000.25 and 1000-200026). Our patients may have had illnesses at the lower end of the range of clinical severity. The Cl difficile isolates from five of our patients were non-cytotoxic, which raises the question whether non-cytotoxic Cl difficile is invariably non-pathogenic. Recent work suggests that the cytotoxin presently assayed is not the enterotoxin that causes the diarrhoea.27,28

When Cl difficile was isolated, a decision to treat was made on clinical grounds with oral vancomycin28 or with cholestyramine.29 That non-cytotoxic strains of Cl difficile might be implicated in diarrhoea led us to regard vancomycin as the first choice when specific treatment seemed to be indicated. The criteria required to implicate Cl difficile as an enteric pathogen are not yet clear. It may be helpful to avoid the terms toxigenic and non-toxigenic until the role of the cytotoxin and enterotoxin in relation to enterotoxicity is defined. It is important to determine whether a relation might exist between the numbers of organisms excreted, their ability to produce one or more toxic factors, the concentrations of these factors in the faeces, and the clinical condition of the patient. The epidemiology is complex,28 and the enteropathogenic range of Cl difficile is as yet undefined. We suggest that the range of illness produced by Cl difficile may include sporadic infective diarrhoea and that oral vancomycin should be considered for patients whose clinical condition causes anxiety.

We thank Dr Margaret Calder and the staff of the City Hospital's bacteriology department, Dr Hamish Inglis and his colleagues at the Regional Virus Laboratory, and Professor Sir Alastair Currie and his staff at the University Department of Pathology for their help; the nursing staff of wards 16 and 16A without whose help we could not have organised and collected the specimens; Mr Garry Hay for technical work; Upjohn Ltd for financial support, equipment, and help with references; the Scottish Home and Health Department (Grant No K/MRS/50/2247) for the financial support of Dr I R...
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Keighley
Poxton; and Mrs J Collins for tests was
weight loss they could
confirmed
therefore report
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Until
Babies
neonatal
Antibiotic-associated
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Deacon AG, Duerten BI, Holbrook WP. Gas-liquid chromatography
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(Accepted 2 October 1981)

SHORT REPORTS

Discharge of preterm babies from neonatal units

Until 1978 very low-birthweight (< 1500 g) and very preterm babies
in our neonatal unit had to reach 2200 g before being discharged home.
A controlled trial, however, showed that provided conditions at
home were satisfactory and the babies were well and passed the nadir of post-
natal weight loss they could be discharged whatever their weight. This
confirmed observations in other countries.2,3

Such weight criteria, however, are still widely used in Britain, and
we therefore report our experience.

Methods and results

From January 1978 to June 1980, 103 babies of 32 weeks’ gestation or less
or under 1500 g at birth were discharged home when clinically well, passed
the nadir of postnatal weight loss, and feeding satisfactorily; home conditions
were satisfactory; and the parents wanted the baby home. After discharge they
were regarded regularly. We present information up to six months beyond their corrected age of term.

Weights of the babies at discharge ranged from 1300 to 3400 g (mean 1830 g). Average stay in hospital was five weeks (range three to seven). Of the 103 babies, 88 were discharged weighing under 2200 g, and 13 weighed
1500 g or less: half of these were “light for dates.” Fifteen were discharged
weighing 2200 g or more, most of these delays resulting from social problems
—the mother subnormal, mother in hospital, etc. Eleven babies were readmitted
during follow-up (table). Only one weighed less than 2200 g, admission being for
a transfusion for anaemia. Except for one baby with failure to thrive, all
had gained weight satisfactorily. None of the others would have avoided readmission had they remained in hospital till reaching 2200 g. Readmissions
were unrelated to early discharge.

Comment

Delaying discharge of small babies from neonatal units until they
reach a certain weight is difficult to justify. Health, progress, and
home conditions should be the essential determinants. Success depends on helping the parents form the bond with the baby which would have developed had separation not been enforced. On admission to the unit a photograph of the baby is taken for the mother to keep in
her bedside. The mother is visited regularly, and as soon as she is well
enough encouraged to see her baby frequently. Brothers and sisters
are also encouraged to visit. For two years wearing gowns has been
abandoned, with no increase in infection. The emphasis is on a relaxed environment and helping the parents look after their baby as soon and completely as possible. Physical contact is encouraged at an early stage—cleaning the baby’s mouth, changing nappies, tube feeding, etc. Generally the earlier a bond is forged the more often will the parents visit. Participation continues as the baby progresses—bathing, feeding, making up feeds, choosing clothes. Babies are encouraged to pass as quickly as possible to breast

| Babies discharged from neonatal unit and needing admission to hospital six to nine months after delivery |
|---|---|---|
| No of babies | Reason for admission | Place of admission |
| 1 | Bronchitis | Children’s ward |
| 3 | Repair of inguinal hernia | Neonatal unit |
| 2 | “Top-up” transfusion for anaemia | Neonatal unit |
| 1 | Suspected non-accidental injury | Neonatal unit |
| 1 | Confirmed non-accidental injury | Children’s ward |
| 1 | Enucleation of left eye (planned admission) | Children’s ward |
| 2 | Investigation of congenital heart disease | Children’s ward |
| 1 | Failure to thrive | Children’s ward |