men was distended, and tenderness and guarding were noted in both iliac fossae. Sigmoidoscopy was performed, and red, haemorrhagic rectal mucosa was noted. Visibility was hampered because of the diarrhoea. Subsequent histology was compatible with an "infective aetiology." 

C difficile toxin was detected by tissue culture of stools on the eighth day of her diarrhoeal illness. She was treated with oral metronidazole 500 mg three times a day and oral vancomycin 125 mg four times a day for one week. She made an impressive recovery; her confusion and disorientation improved within 48 hours, the diarrhoea was greatly reduced in quantity and frequency, and her abdominal distension and tenderness resolved.

Four days after starting the course of vancomycin and metronidazole she became febrile once more. She was confused again and for the first time during her admission was peripherally "shut down" and hypotensive (90/60 mm Hg). The white cell count was still raised at 18 x 10^9/l. Her clinical condition was thought to be compatible with a Gram negative septicemia. Blood cultures were taken, and she was started empirically on netilimycin 150 mg twice daily. Subsequent doses and frequency were determined by careful monitoring of her serum concentrations. She was alert and normotensive within 24 hours, and Enterobacter cloacae was isolated from her blood. This organism was resistant to ampicillin and cephalaxin but sensitive to netilimycin, cefotaxime, and cefazidime. Her white cell count subsequently fell to 8.8 x 10^9/l. After a prolonged convalescence she made a complete recovery, although C difficile toxin was detected in her stools for a further six weeks. Throughout this period she was nursed in a single room with enteric precautions that hampered her rehabilitation both psychologically and physically.

Two recent reports also highlighted septicaemia as a possible complication of pseudomembranous colitis, although the white cell count is not mentioned in either patient. Both these patients also had septicaemias of faecal organisms, like our patient and those reported on by Rampling et al. Interestingly, both patients reported on by Franson et al. and Spencer et al., our patient, and six of nine patients reported on by Rampling et al. had received a cephalosporin. Whether this represents a true association between cephalosporins and bacteraemia or merely reflects prescribing habits requires further study.

The action of C difficile enterotoxins A and B on colonic mucosa can presumably facilitate bacteraemia of gut origin. Although it seems that this complication is more likely in neutropenic patients, we feel that this potential complication should also be considered in non-neutropenic patients with pseudomembranous colitis. This is especially true if their condition deteriorates or they become febrile once an initial response to oral vancomycin or metronidazole has been documented.

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References

Anaerobic media and pH changes during growth

In the last few years attention has been paid to the growth characteristics of anaerobes in different media, with special attention to the results when susceptibility to antibiotics is tested. Several problems, however, have arisen. Watt and Brown described a drop in surface pH of several media related to bacterial growth. The change in pH could not be controlled by the use of buffers, but new media with a stable pH had greatly inferior growth properties. This fall in pH might influence the outcome of the sensitivity testing of anaerobes to antibiotics such as erythromycin. Eley et al added to this problem by observing morphological changes in Bacteroides, including filamentation and formation of spheroplasts, after growth in Wilkins-Chalgren, thioglycollate, and Schaedler broths but not in brain-heart infusion broth. We also observed a fall in pH in liquid anaerobic media during growth of B fragilis and B vulgatus, which was accompanied by a sharp loss in viability. After 24 hours of incubation the percentages of viable cells in BM broth and thioglycollate broth were as low as 1-0 and 1-3 respectively; brain-heart infusion broth and Wilkins-Chalgren broth gave much better results (about 80% viability). The addition of a phosphate buffer to the BM medium and the omission of glucose resulted in a pH of 5.0 and 5.2, respectively, after 24 hours of incubation, whereas in combination the pH did not drop below 6.2. The viability in this last medium exceeded 50%.

We do not have any evidence of a change in the growth supporting properties of BM medium after these changes; and the morphological changes described by Eley et al were not observed. We believe that careful consideration of the concentration of fermentable sugars in relation to the buffering capacity of the media might reduce the fall in pH during growth. We agree with Watt and Brown that changes in pH in media and loss of viability are highly undesirable. More research on the growth characteristics of media designed for anaerobic bacteria, especially when testing susceptibility to antibiotics, is clearly required.

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