part, seven did not know how the disease was caught, and three had not heard of it.

Have you suffered from malaria? If so, when?—Seven respondents (16%) had previously suffered malarial attacks. One of these was a British resident travelling abroad; in the six others the attacks had occurred before immigration.

What immunisations did you receive before travel?—Forty respondents (89%) had received typhoid, cholera, or smallpox immunisation before travelling.

Did you take tablets to prevent malaria? If no, why not?—Prophylactic measures taken against malaria had been satisfactory in three cases (7%) and irregular or stopped on return to this country in nine (20%); 33 respondents (73%) had not taken any prophylactic measures. Two of the three taking satisfactory precautions were doctors. Thirteen respondents thought that the risk did not warrant medication, four thought no malaria existed where they were going, and three thought that injections against other diseases had included malaria. Thirteen had not considered the problem at all.

Whom would you approach in the future for advice on malarial prevention?—All respondents said that their source of advice would be their general practitioner.

Comment

Most travellers did not take precautions against malaria, although immunisation was considered and accepted for other diseases. Some did not consider malaria to be dangerous. Others thought that it was not present in the areas they visited, implying ignorance of the upsurge of malaria throughout the Indian subcontinent over the past 10 years, which remains a cause for concern despite a fall in notifications recently.^{1 2}

The presence of malaria and the value of prophylactic measures might be discussed by the general practitioner when other immunisations are given, though now that smallpox has been eradicated international certificates are rarely required and there may be less opportunity for this. Other methods of contacting travellers such as through travel agencies or airlines may become increasingly important.

Reference centres with updated information on regional differences in parasite prevalence and resistance patterns might usefully be more widely advertised. A source of such information in Scotland is the Communicable Diseases (Scotland) Unit at Ruchill Hospital, Glasgow (telephone 041 946 7120).

We thank Mr I A Tervez, manager of the National Bank of Pakistan; Mr T Bedi, Bedi's Continental Food Company; and all others in Glasgow who co-operated and helped in collecting data.

Notifications for Scotland were to the Information Services Division of the Common Services Agency of the National Health Service, Edinburgh.

¹ WHO. Synopsis of the world malaria situation 1978. *WHO Weekly Epidemiological Record* 1980;55:193-200.

² Ray AP. Some aspects of *Plasmodium falciparum* containment programme. *Indian J Med Res* 1979;70:1-13.

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Isolation of *Clostridium difficile* from the small bowel

The association of *Clostridium difficile* and its cytotoxin with pseudomembranous colitis^{1 2} has stimulated interest in the presence of this organism in the gut. While the organism has been isolated from the faeces of both healthy infants and adults with antibiotic-associated disease and in intimate association with rectal mucosa,³ there has been no report of the isolation of *C. difficile* from the upper small intestine. We report the isolation of *C. difficile* from the proximal jejunum of a patient with diarrhoea.

Case report

An 80-year-old woman presented with a three-month history of diarrhoea. loss of 12 kg over the preceding year, and episodic left-sided abdominal pain,

Bowel actions of yellow, watery stools, with no blood or mucus, ranged from three to 10 times daily. She had had several episodes of diarrhoea in the past but extensive investigations had disclosed no cause, and it had responded to various antidiarrhoeal agents. In 1950 she had undergone laparotomy for perforation of the appendix and in 1965 cholecystectomy for gall stones.

Physical examination showed an enlarged spleen but no other abnormality. Sigmoidoscopy and rectal biopsy showed mild oedema but no other abnormality. Results of all laboratory investigations were normal. Appearances on barium-enema and barium meal and follow-through examinations were also normal. Examination of stools showed no ova, cysts, parasites, *shigellae*, or *salmonella*. Glucose-hydrogen and ¹⁴C-glycolate breath tests for small-intestinal colonisation were negative.

Jejunal fluid was aspirated by the method of Taylor and Waterman⁴ and cultured using the media and methods described by Borriello *et al.*⁵ Culture yielded three *Clostridium* spp: *C. perfringens* 10⁶/l, *C. difficile* 10⁵/l, and unidentified *Clostridium* spp 10⁶/l. No other organisms were isolated. The isolates were identified from colony and cellular morphology, carbohydrate fermentation patterns, and gas chromatographic analysis of volatile fatty acids.

Before the results of culture were known the patient was treated symptomatically with loperamide 2 mg four times daily, which controlled the diarrhoea. She gained 5 kg over the next six months and two years later remained well with no further treatment.

Comment

In view of the known association between faecal carriage of *C. difficile* and diarrhoea, this organism may have been a factor in the causation of diarrhoea in this patient. Unfortunately stool samples were not cultured for *C. difficile* or analysed for the cytotoxin at the time.

In a further group of 27 patients presenting with diarrhoea, culture of jejunal aspirates yielded *Clostridium* spp from only four specimens. Isolates were identified as *C. perfringens* (2), *C. subterminale* (2), *C. sporogenes* (1), and *C. formicoaceticum* (1). The highest count of *clostridia* found was 6.8 × 10⁶/l. *C. difficile* was not isolated from any other patient.

C. difficile resident in the small intestine may act as a reservoir for recolonisation of the large bowel in the phases immediately after treatment with the concomitant toxin production and symptomatic relapse that is increasingly noted with pseudomembranous colitis. To answer questions posed by the finding of *C. difficile* in the upper small bowel and to increase our understanding of the pathogenesis of *C. difficile*-mediated disease, we suggest that small-bowel specimens should be cultured for this organism whenever the opportunity is presented.

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¹ Larson HE, Price AB, Honour P, Borriello SP. *Clostridium difficile* and the aetiology of pseudomembranous colitis. *Lancet* 1978;ii:1063-6.

² Borriello SP, Larson HE. Antibiotic and pseudomembranous colitis. *Antimicrob Agents Chemother* 1981;7, suppl A: 53-62.

³ Borriello SP. *Clostridium difficile* and its toxin in the gastrointestinal tract in health and disease. *Research and Clinical Forums* 1979;1:33-5.

⁴ Taylor RH, Waterman S. "One swallow" test for the investigation of malabsorption and diarrhoea. *Gut* 1977;18:A425.

⁵ Borriello SP, Hudson MJ, Hill MJ. Investigation of the gastrointestinal bacterial flora. *Clin Gastroenterol* 1978;7:329-49.

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Correction

Aquagenic pruritus

An error occurred in this paper by Dr M W Greaves and others (20 June, p 2009). In the section headed "Morphological studies" the sentence starting on line 10 of the second paragraph should have read: "The percentage degranulation was not significantly increased after challenge ($t=1.91$; $0.1 > p > 0.05$; $df=2$; paired t test)."