

currently afebrile, in remission from her leukaemia, and continues on her chemotherapy.

Comment

The incidence of imported malaria in the UK has steadily increased during the past decade,¹ with 1909 cases reported in 1978. At St Thomas's Hospital *P falciparum* malaria is much commoner than *P vivax* and is mostly acquired in tropical Africa.² Most of the malaria reported in Asian immigrants is *P vivax*,² and, although the World Health Organisation has reported an increase in the incidence of falciparum malaria in Bangladesh, India, and Sri Lanka,³ we have not previously seen a falciparum infection in an Asian. Chloroquine-resistant falciparum malaria has been reported in South-east Asia and South America for many years, but its occurrence in the Asian subcontinent and in East Africa is much more recent. Two cases of imported chloroquine-resistant falciparum infections have recently occurred in the UK; both were from Kenya and one was fatal.⁴

Our patient would appear to have shown RI resistance to chloroquine in that parasites reappeared after 23 days, although the initial course of chloroquine had apparently eradicated the infection. We made no attempt to culture the malarial parasites to confirm in-vitro resistance. This patient probably acquired her malaria from blood transfusion in Bangladesh, and there have been no reports of chloroquine-resistant *P falciparum* being transmitted by this route, although transfusion malaria is known to be common and under-reported in the developing countries, and many cases of falciparum are described.⁵

Malaria in our patient was initially diagnosed by chance when blood films were being examined. The patient at this time was relatively well and afebrile, and malaria was not suspected clinically. The resurgence of the falciparum was unexpected and delayed diagnosis in view of the many other possible causes of fever in a neutropenic postoperative patient with leukaemia. Malaria was not initially entertained as a likely diagnosis because of the apparently successful treatment with chloroquine. It is of interest that the clinical response to intravenous quinine was slow, particularly defervescence, and perhaps "quinine fever" may have been contributory. The disappearance of parasites was also slow; indeed, the parasitaemia appeared unchanged after two infusions of quinine.

Although we are unaware of the effect of severe neutropenia and immunosuppression on the clinical course of malaria, it seems likely that this patient was infected with a chloroquine-resistant strain of *P falciparum*.

We thank Dr A P Hall, Hospital for Tropical Diseases, for his help and advice on the management of this patient.

¹ Bruce-Chwatt, L J, Southgate, B A, and Draper, C C, *British Medical Journal*, 1974, **2**, 707.

² Ellis, C J, *et al*, *British Medical Journal*, 1979, **1**, 385.

³ World Health Organisation, *Malaria Control Strategy*. Unpublished document, WHO A31/19, 1978.

⁴ Malaria Reference Laboratory, London School of Hygiene and Tropical Medicine. Personal communication.

⁵ Bruce-Chwatt, L J, *Tropical Diseases Bulletin*, 1972, **69**, 825.

(Accepted 25 July 1979)

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Treating irritable bowel syndrome with peppermint oil

The irritable bowel syndrome is characterised by recurrent attacks of colicky abdominal pain, feelings of distension, and altered bowel habit. Although the cause is not fully understood, manometric studies have shown disordered bowel motility, which may cause some of the symptoms. Useful treatments include increasing dietary fibre, and drugs such as anticholinergics, antispasmodics, and sedatives¹; their effect is often disappointing and their use limited by side effects. Peppermint oil is a naturally occurring carminative, which is included

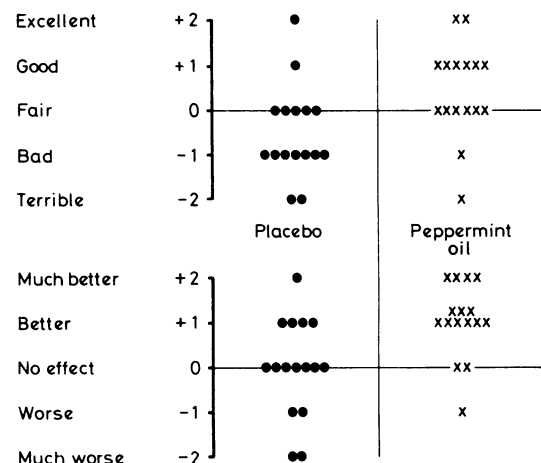
in many drug preparations and relaxes gastrointestinal smooth muscle both in vitro and in vivo.³ We have examined its effect in patients with irritable bowel syndrome in a double-blind cross-over trial.

Patients, methods, and results

Eighteen patients who had active symptoms of the irritable bowel syndrome participated in the trial. The peppermint oil was prepared by placing 0.2 ml in gelatin capsules (Elanco LOK caps), which were then coated with a cellulose acetate-phthalate solution to prevent disintegration within the stomach; identical placebo capsules that contained arachis oil were prepared. Capsules were dispensed in containers with free peppermint oil injected under the cap to ensure that both types of container smelt strongly of peppermint. Patients were asked to take one or two capsules thrice daily, depending on the severity of symptoms.

Patients were given either peppermint oil or placebo capsules. During each treatment period of three weeks, patients recorded daily the severity of abdominal symptoms graded from 0 (asymptomatic) to 3 (severe symptoms); stool frequency and side effects were also noted. Overall symptoms were graded on a 5-point scale ranging from +2 (excellent) to -2 (terrible) after each treatment, and the success of treatment was similarly graded from +2 (much better) to -2 (much worse). Each treatment began when active symptoms developed in order to minimise the effect of spontaneous remissions.

Two patients failed to complete the trial and their results are excluded from the analysis. Paired observations in each subject were analysed using the paired *t* test. The overall assessment of each period shows that patients felt significantly better while taking peppermint oil capsules compared with placebo ($P < 0.01$), and considered peppermint oil better than placebo in relieving abdominal symptoms ($P < 0.005$) (figure). Analysis of the symptom



Overall assessment of severity of symptoms (above) and change in symptomatology (below) after treatment with placebo or peppermint oil in 16 patients.

grades showed a lower total and mean daily score with peppermint oil treatment, but the values were not significantly different from placebo. Patients experienced more symptom-free days (grade 0) and fewer severe symptoms (grade 3) on peppermint oil, but the differences from placebo were not statistically significant. There was no significant effect on stool frequency.

Two patients developed heartburn, which may have been caused by premature release of oil in the stomach and relaxation of the lower oesophageal sphincter.

Comment

Peppermint oil is a carminative with potent antispasmodic properties and our double-blind cross-over trial shows that it reduces abdominal symptoms in the irritable bowel syndrome. The simple preference or help scores are more sensitive in detecting differences between the preparations than more complex scoring systems.⁴

Although the irritable bowel syndrome is a benign disorder that causes no deaths, it is a chronic relapsing disease with appreciable morbidity, which affects mostly young and middle-aged individuals. Until the precise cause is understood, treatment must aim at relieving symptoms. Current treatment is unsatisfactory and, although some respond, many patients experience little or no improvement, and peppermint oil may be particularly valuable for them. The relative absence of side effects favours this preparation, but, because oil is

