April 1969 because of severe pain in the lower abdomen. On admission she was afebrile and well-nourished. There was tenderness in the right iliac fossa, but no mass was palpated. Investigation disclosed hypochromic anaemia, normal white blood cell count, and normal chest radiograph. Sigmoidoscopy detected nothing abnormal and there were no perianal lesions.

One month later she underwent operation in the gynaecological department for suspected torsion of a left ovarian cyst. At laparotomy enlargement of mesenteric lymph nodes and many white nodules covering the serosal surface of the ileum were found. The bowel wall itself was not diseased. The nodules were macroscopically similar to tubercles. Some of them were taken for microscopical examination, which showed granulomatous reaction without caseation (see figure). No starch particles known to induce granulomatous peritonitis were shown in the biopsy material with the use of polarised light.

Histological specimen of serosal nodule showing granulomatous reaction without caseation.

A postoperative diagnosis of intestinal tuberculosis was made and treatment with streptomycin and isonicotinic acid hydrazide was started. Antituberculous chemotherapy was continued for three months but proved ineffective. Because of this, as well as negative results for tuberculin skin tests, and the absence of tubercle bacilli in stool culture, doubts arose about whether the diagnosis of tuberculosis was correct.

Radiological examination of the small intestine performed in November 1969 showed changes typical of ileal Crohn's disease. Several courses of oral corticosteroids (prednisone 40–15 mg daily) were given but her symptoms gradually worsened. The diagnosis of Crohn's disease was confirmed three years later after resection of the terminal ileum, which was found at operation to be the only affected segment of the bowel. Pathological study of the specimen showed noncaseating, sarcoid granulomas and fissures characteristic of Crohn's disease. At the second laparotomy serosal nodules were not seen. Four years later the patient remained well without specific treatment.

Discussion

In our patient intestinal tuberculosis was diagnosed at laparotomy. The diagnostic error was due to the peritoneal nodules that resembled tubercles and resulted in unnecessary treatment with antituberculous drugs. Identical errors occurred in two of three cases described by Heaton *et al.*³ Final diagnosis in these cases was the result of radiological, microscopical, and bacteriological studies. In our patient the diagnosis of Crohn's disease was established after typical changes were found on radiological examination, which was performed several months after the first operation. Definitive confirmation of this diagnosis took place three years later as the result of pathological study of the resected ileum.

The young age of our patient at the onset of the illness and the evolution of the serosal lesions into typical Crohn's disease of the terminal ileum suggest that the miliary form is not distinct, but an early evolutionary phase of classic Crohn's disease, as has been emphasised by other authors.^{3,5} Because of such cases it is important to

draw the attention of surgeons to miliary Crohn's disease and to the necessity of distinguishing it from intestinal tuberculosis. To exclude tuberculosis it is essential to perform biopsies on the miliary lesions. The absence of caseation and acid-fast bacilli makes tuberculosis unlikely and suggests the diagnosis of Crohn's disease.

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Departments of Gastroenterology and Pathology, Medical Centre of Postgraduate Education, Warsaw, Poland

W BARTNIK, MD, senior research assistant

J KRYŃSKI, MB, research assistant

E BUTRUK, MD, associate professor

J ORŁOWSKA, MB, senior research assistant

Malaria—the great mimic

Malaria in the United Kingdom is often diagnosed by epidemiological history and a high level of suspicion rather than by textbook signs. In view of the ever-rising trend of imported malaria in the United Kingdom—688 cases were notified in the first 32 weeks of 1976¹—these facts must be constantly kept in mind. Various haematological abnormalities have been associated with malaria, though these have not been emphasised in the textbooks. Thrombocytopenia is a comparatively recent finding and is probably more common than is generally appreciated. Haemorrhage in disseminated intravascular coagulation (DIC) is a common feature but in its absence evidence of bleeding is extremely rare. Purpura as a presenting symptom of malaria has not, to our knowledge, been reported.

Case report

A Swiss seaman, aged 23, was seen on 13 May 1976. He complained of a rash over the lower parts of both legs and fever. During the previous two weeks, he had experienced bouts of evening fever with associated chills, accompanied by anorexia, headache, and bodyache, but recovered almost completely by the daytime. A week after onset he noticed a rash over both legs around the ankles, which worried him more than the fever. He had joined his ship at Lagos on 5 March 1976 and was anchored there for between four and six weeks before reaching London on 5 May 1976.

His general condition was satisfactory but hepatosplenomegaly was present. Profuse, discrete petechial rash covered both ankles and adjoining areas of lower legs and feet. The white cell count was $5.0 \times 10^9/l$ ($5000/mm^3$), platelets $50 \times 10^9/l$ ($50\,000/mm^3$). Blood film was positive for malaria parasites, identified as *Plasmodium malariae*. On admission to hospital on 15 May 1976 the clinical features were no different. There was no evidence of anaemia, jaundice, or DIC. He had not been taking antimalarial prophylaxis or any other medication. No relevant past or family history was obtained.

Investigations included: haemoglobin 12.4 g/dl; platelets $43 \times 10^9/l$ ($43\,000/mm^3$). Test results for blood urea, electrolytes, liver function, transaminases, and glucose-6-phosphate dehydrogenase were normal. Serum total proteins and albumin and globulin concentrations were normal, but gammaglobulin was increased; IgG concentration was normal, but IgA and IgM were raised to 4.19 and 4.60 g/l, respectively. Antibodies to smooth muscle and mitochondria were not detected, but antinuclear factor was present at a mildly positive titre of 1/20.

Antimalarial treatment and a short course of prednisolone were given. The blood film became negative by the ninth day and the platelet count was $203 \times 10^9/l$ ($203\,000/mm^3$). Clinical improvement was rapid and good but the purpura, though considerably improved, had not completely disappeared at discharge on 25 May.

Comment

Haematological changes in malaria are varied² and well known, though surprisingly thrombocytopenia in malaria has been recognised as a common haematological complication only lately. Beale *et al.*³ reported considerable thrombocytopenia in 32 out of 33 cases of

naturally occurring malaria. The mechanism of thrombocytopenia is controversial, but is probably multifactorial. In our patient it might have been due to production of antiplatelet antibody of IgM class.³

Evidence of bleeding without DIC is rare. Beale *et al*³ reported one case of subconjunctival haemorrhage among 17 people with thrombocytopenia, three cases of epistaxis, and one of haematuria, but we believe our report to be the first record of cutaneous purpura in malaria. We should like to emphasise that thrombocytopenia and haemorrhage may occur in malaria without DIC. Controversy over the use of heparin in DIC is rife,⁴ but clearly unequivocal evidence of DIC must be obtained before heparin treatment is started.

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Infectious Diseases Unit, West Lane Hospital, Middlesbrough TS5 4EE

SALIL K GHOSH, MB, MRCP, consultant physician

North Ormesby, Middlesbrough

I NICOLSON, MB, CHB, general practitioner

Glibenclamide and syndrome of inappropriate secretion of antidiuretic hormone

The oral hypoglycaemic drug glibenclamide has been reported to cause a diuresis both in healthy people and in patients with diabetes mellitus.¹ This effect may be due to antagonism to the action of antidiuretic hormone (ADH).² We have used glibenclamide to treat a patient with the syndrome of inappropriate secretion of ADH (SIADH) with the following results.

Case report

A 30-year-old woman with SIADH of unknown aetiology was admitted to the metabolic unit. There was no evidence of intracranial disease, renal disease, chest disease, or neoplasia, and her management had consisted in fluid restriction since the diagnosis was made five years earlier. After a baseline period of three days a 12-hour water deprivation test with measurement of maximum urine osmolality was performed. Next day a four-hour water tolerance test was carried out with a challenge of 1 litre of water, the urine volume and minimum urine osmolality being measured. The patient was then given oral glibenclamide 2.5 mg each morning, the water deprivation and water tolerance tests being repeated on the third and fourth days of treatment, starting two hours after the dose of glibenclamide. The dose was then increased to 5 mg daily and subsequently to 10 mg daily and the procedure repeated. Finally, the investigations were repeated after two days only on 20 mg daily. Plasma osmolality was measured before each water test, and plasma ADH was measured³ during the baseline period and before the water tests two hours after giving the 10-mg and 20-mg doses. During the entire study period the daily intake of sodium, potassium, and fluid remained constant. Plasma glucose was monitored throughout the study.

Plasma ADH was raised during the baseline period at 12.3 ng/l (normal range 4-8 ng/l) and remained raised during the treatment with glibenclamide. In addition there was no significant change in any of the variables measured during treatment compared with the baseline period (figure). In particular, no increase in urine flow was seen, in contrast to previous reports.^{1,2}

Comment

In this patient glibenclamide did not modify the circulating concentration of ADH and did not have a diuretic effect. Our findings suggest that the diuretic effect of glibenclamide may not be through antagonism of ADH or through suppression of ADH secretion. Alternatively, the ADH concentrations in this patient may have been

Absence of significant change in variables measured during treatment with glibenclamide.

Conversion: SI to traditional units—Urine and plasma osmolalities: 1 mmol/kg = 1 mOsm/kg.

too high for antagonism of ADH and consequent diuresis to be apparent.

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Metabolic Unit, University Department of Medicine, Western General Hospital, Edinburgh EH4 2XU

P L YAP, MB, CHB, senior house officer

A D B HARROWER, MRCP, senior registrar

N McD DAVIDSON, MRCP, lecturer

J A STRONG, MD, FRCP, professor of medicine

MRC Blood Pressure Unit, Western Infirmary, Glasgow G11 6NT

J J MORTON, PHD, non-clinical scientist

Bronchial carcinoma metastasising to the ischiorectal fossa

Carcinoma of the bronchus is noted for its tendency to metastasise extensively. Nevertheless, it rarely metastasises to muscle and it has never been reported as spreading to fat. We report a case of carcinoma of bronchus which presented with a secondary deposit in the fat and muscle of the ischiorectal fossa.

Case report

A 72-year-old man presented with a two-week history of perianal pain exacerbated by the passage of stool. He had been well, specifically denying any weight loss and respiratory symptoms. He was a heavy smoker and had not been on antibiotics preceeding admission. He was afebrile, with a large