differentiated adenocarcinoma which had completely replaced the node. The patient subsequently deteriorated rapidly and died in February 1968.

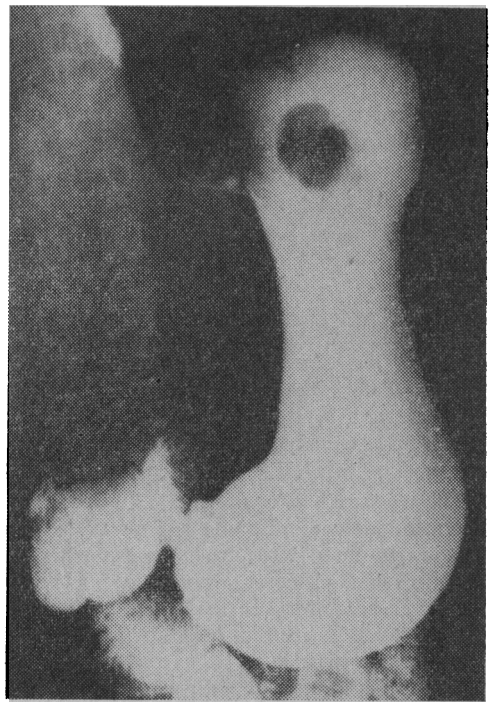

FIG. 3.—Radiograph showing large polypoid carcinoma of stomach.

Necropsy showed a fundal carcinoma of the stomach with regional lymph node involvement and deposits in the liver and lungs. There was bilateral bronchopneumonia. A careful search was made for lymph nodes, but the only ones found were those to which metastasis had occurred. The spleen was small, weighing only 74 g. Macroscopically there was no evidence of leukaemic infiltration of any organ, nor did histological examination show leukaemic deposits. The red marrow had extended to fill the whole shaft of one femur examined. Similarly, red marrow was very prominent in all the vertebral bodies. Numerous microscopic deposits of carcinoma were found in two vertebrae and in the femur.

COMMENT

There can be little doubt that when this patient was first seen she was suffering from pernicious anaemia. She died from a carcinoma of the stomach, a complication arising in about 10% of cases (Zamchek *et al.*, 1955). The nature of her lymphocytic response is, however, open to question. The findings in the marrow and peripheral blood when she first presented were very suggestive of a lymphatic leukaemoid reaction. Her subsequent course with a poor response to vitamin B_{12}, the need for repeated blood transfusions in the absence of haemolysis or blood loss, and, in particular, the dramatic response to chlorambucil suggested that this was a leukaemic process. The absence of post-mortem evidence of leukaemia, however, makes this hard to sustain. The coexistence of pernicious anaemia and chronic lymphatic leukaemia has been described (Rich and Schiff, 1936 ; Mason and Schwartz, 1949 ; Ikkala and Kaipainen, 1962). Although this association may be coincidental, a significantly higher incidence of pernicious anaemia in the families of leukaemic patients has been described (Videbaek, 1946). Cases of myeloid leukaemia and pernicious anaemia have also been described (Sterne *et al.*, 1941 ; Woolley, 1944).

It is tempting to speculate whether the presence of a long-standing lymphocytic leukaemoid reaction was a marker of subsequent malignant change in the stomach.

We would like to thank Dr. A. E. Read for permission to report this case.

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Relapsing Amoebic Colitis of 12 Years' Standing Exacerbated by Corticosteroids

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A case of amoebic colitis undiagnosed for 12 years and exacerbated by systemic corticosteroid therapy is described. The difficulties in differentiating amoebic colitis from non-specific ulcerative colitis have been previously reported (Wright, 1966). However, misdiagnosis still occurs, and cases of amoebic colitis are treated with corticosteroids which may be followed by grave consequences. It is therefore hoped that this case report will draw further attention to the problem.

CASE REPORT

A 33-year-old Englishman who had lived in Malaya and Singapore in 1955 and 1956 developed diarrhoea with blood and mucus in the stools six months after his return to the United Kingdom. Stool microscopy was said to have shown only *Giardia lamblia* cysts and he was treated with mepacrine with some temporary improvement. Six months later his symptoms had become worse and he was admitted to a general hospital, where sigmoidoscopy showed proctocolitis. Stool microscopy on this occasion was negative and he received symptomatic treatment. However, within 10 months a further admission to hospital was necessary and on this occasion *G. lamblia* cysts were again seen in the stool samples. He received "small doses of cortisone" and more mepacrine. After this he improved and remained asymptomatic until March 1965, when he began passing frequent loose stools with blood and mucus. Several

stool microscopies, carried out elsewhere, were "negative" and during the following three years he received Salazopyrin (sulphasalazine) and Metamucil, without relief. A barium enema in November 1967 was reported to show no abnormalities. In July 1968 he received a 12-day course of oral corticosteroids with a marked exacerbation of his symptoms.

He was first seen by us at the Hospital for Tropical Diseases in August 1968, when he gave a history of passing four or five blood-stained stools a day and of having lost 1 stone (6·4 kg.) in weight during the preceding year.

Physical examination showed an ill-looking man with evidence of recent loss of weight. His blood pressure was 125/70 and there were no other remarkable physical findings. At sigmoidoscopy 18 cm. of the bowel was visualized and showed marked mucosal hyperaemia and oedema with contact bleeding and a moderate amount of blood-stained exudate; in addition several small superficial ulcers were seen. Scrapings obtained from the ulcers were examined microscopically in a drop of saline on a warm stage and showed numerous haematophagous *Entamoeba histolytica* trophozoites. Stool cultures were negative for salmonellae and shigellae. His haemoglobin was 13·3 g./100 ml., W.B.C. 6,100/cu. mm., eosinophils 2%, and E.S.R. 2 mm. in 1 hour. A Malmo barium enema showed normal haustrations and no abnormalities were demonstrated. He was treated with emetine 60 mg. intramuscularly daily for four days, followed by Furamide (diloxanide furoate) 500 mg. t.d.s. for 10 days. By the third day of treatment he was symptomatically improved and an endoscopic examination at the end of therapy showed mild mucosal hyperaemia confined to the distal 5 cm. of the bowel only, the rest of the mucosa visualized being normal.

He was discharged from hospital and was seen by us again in November and December 1968. On both occasions he reported no symptoms, sigmoidoscopic examinations were completely normal, and, in addition, microscopy of stool and scrapings obtained from the bowel mucosa were negative.

Discussion

Autochthonous amoebiasis is extremely rare in the United Kingdom (Wilmot, 1962). We believe that this patient's entire illness was due to amoebiasis acquired in the Far East. Untreated amoebic colitis is known to exhibit periods of quiescence. Giardiasis, if it was indeed present, could not have caused his dysenteric symptoms. Mepacrine, which is used in the treatment of Giardiasis, has been reported to have some amoebicidal properties (Radke, 1951) and may have been partly responsible for inducing a remission in our patient.

The previous unsuccessful attempts at demonstrating *E. histolytica* in this patient illustrate the diagnostic difficulties facing a physician who does not have access to a competent parasitological service. Sigmoidoscopic examination is obligatory in the evaluation of any case of diarrhoea with blood and mucus in the stools, and if there is even a distant history of residence in the tropics scrapings must be obtained from the lesions for microscopical examination, which should be carried out immediately. "Negative stool microscopy" does not exclude amoebiasis.

McAllister (1962) suggested that biopsy and fresh stool samples should be taken from all cases of idiopathic ulcerative colitis. While fully endorsing his cautious approach, we would emphasize the importance of microscopical examination of fresh scrapings obtained from the lesions. We do not practise biopsy for the diagnosis of amoebiasis; if one is performed the material should be examined immediately as a squash preparation as described by Manson-Bahr (1957), who stressed that amoebae must be seen in the living state. Examination of fixed histological material not only delays diagnosis but is unsuitable for definite identification of *E. histolytica* unless they are very numerous. Differentiation from macrophages may be impossible, especially when they contain erythrocytes.

At endoscopy the mucosal appearances may closely resemble those of idiopathic ulcerative colitis, and Wilmot (1962) stated that even normal endoscopic appearances do not exclude acute ulcerative amoebiasis, as the lesions may be beyond the range of the instrument.

The effects of corticosteroids on amoebic infection have been documented before. The first of the cases reported by McAllister (1962) was one of autochthonous amoebiasis in which the patient deteriorated markedly on oral corticosteroids and nearly underwent a total colectomy, but fortunately a definitive diagnosis of amoebic colitis was reached preoperatively.

Mody (1959) reported two patients who received hydrocortisone enemata and prednisolone suppositories respectively for supposed idiopathic proctocolitis; both were made worse by such therapy and numerous *E. histolytica* trophozoites were later found in scrapings obtained from the bowel mucosa. Eisert *et al.* (1959) described a case of *Pemphigus vulgaris* treated with systemic corticosteroids; the patient died from fulminating intestinal amoebiasis with perforation of the bowel. Any patient who develops diarrhoea while on corticosteroids prescribed for whatever condition should be carefully investigated for amoebiasis. Morton *et al.* (1951) estimated that 1·6% of the population of the British Isles are amoebic carriers. Among the immigrant population the figure is likely to be much higher. It is theoretically possible for any symptomless carrier to develop symptomatic amoebiasis following steroid therapy.

We wish to thank Professor A. W. Woodruff for allowing us to publish this case and also for his helpful advice during the preparation of this paper. We are also grateful to Dr. D. S. Ridley and Mr. A. V. H. Allen for the pathological reports.

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