tightness may be found (Menelaus 1964, Spiegel *et al.* 1972, Wenger *et al.* 1978). The ESR is usually but not invariably raised and the white blood count may be moderately elevated (Spiegel *et al.* 1972).

X-ray changes develop from about two weeks after onset (Doyle 1960). Initially there is disc space narrowing followed by vertebral end-plate erosion and subchondral sclerosis (Menelaus 1964, Spiegel *et al.* 1972). Involvement of mainly the lumbar or lower thoracic spine at a single level is typical (Wenger *et al.* 1978). Disc space narrowing is not specific; the differential diagnosis includes tuberculosis, brucellosis, typhoid, osteomyelitis and tumour (Bonfiglio *et al.* 1973, Spiegel *et al.* 1972). Trauma and particularly osteochondritis (Scheuermann's disease) may cause confusion particularly in the older child, although the latter typically affects several vertebrae (Spiegel *et al.* 1972, Wenger *et al.* 1978).

Isotope bone scanning can be useful in early diagnosis, as radiological changes are sometimes delayed for several weeks (Fischer *et al.* 1978, Wenger *et al.* 1978). Blood cultures may be positive and biopsy of disc material, using needle or open techniques, has yielded variable results (Doyle 1960, Kemp *et al.* 1973, Spiegel *et al.* 1972, Wenger *et al.* 1978).

Management variously comprises bed rest, spinal immobilization and antibiotic administration. Use of back supports is dictated to some extent by symptoms, but treatment with antibiotics is debateable where bacteriology is negative (Doyle 1960, Menelaus 1964, Spiegel *et al.* 1972, Wenger *et al.* 1978).

The picture of discitis in adults and children differs clinically, possibly reflecting aetiological differences. In adult cases, patients are often predisposed to infection by conditions such as diabetes, renal failure, alcoholism or rheumatoid arthritis. Spinal cord involvement and abscess formation are relatively common, and a bacterial cause (most frequently *Staph. aureus*) is found in the majority (Bonfiglio *et al.* 1973, Kemp *et al.* 1973, McCain *et al.* 1981).

Children are usually well prior to the disease, which tends to be self-limiting and generally free of complications. There has been one recently reported case of psoas abscess secondary to a septic discitis in a girl aged 13 years (Holliday *et al.* 1980). However, of almost 300 cases reviewed in the literature, there was evidence of paraspinal abscess formation in only 3 children (Bonfiglio *et al.* 1973, Fischer *et al.* 1978, Menelaus 1964, Spiegel *et al.* 1972, Wenger *et al.* 1978). This rarity of abscess formation is surprising and difficult to explain if bacterial infection is the major aetiological factor. Although an infective cause is assumed by most authors, bacterial confirmation is obtained only in a minority (Fischer *et al.* 1978, Spiegel *et al.* 1972, Wenger *et al.* 1978). Previous antibiotic usage, low-virulence organisms or viral infection could account for negative cultures. Alternatively, other processes such as trauma, avascular necrosis or dislocation of epiphyses may be relevant aetiologically in many cases of childhood discitis (Spiegel *et al.* 1972, Wenger *et al.* 1978).

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Sarcoidosis associated with Crohn's disease of ileum, mouth and oesophagus[1]

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Crohn's disease and sarcoidosis are both granulomatous diseases of unknown aetiology. Crohn's disease principally affects the gastrointestinal tract, whilst sarcoidosis rarely, if ever, involves the gut. However, the two diseases share similar systemic manifestations, histological appearances and defects in cellular immunity. It is, perhaps, surprising that the two diseases have been reported in the same patient on only three previous occasions (Morland 1947, Dines *et al.* 1971, Padilla &

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Sparberg 1972). We report a further case. Our patient also had the unusual manifestations of oral and oesophageal Crohn's disease.

Case report

The patient, a Caucasian woman, was 32 years old when first seen at this hospital in August 1973 with a four-month history of intermittent cramping abdominal pain and distension. There was clinical and plain X-ray evidence of a distal small bowel obstruction and barium studies suggested Crohn's disease as the cause. At operation a 90 cm segment of ileum with the typical macroscopic appearances of Crohn's disease was resected. The microscopic features were considered classical for Crohn's disease.

In March 1974 she was admitted to a different hospital with pain and swelling of the feet, knees, elbows and shoulders, associated with general malaise and lassitude. The spleen and liver were enlarged, but there was no peripheral lymphadenopathy. She had erythema nodosum on her legs. A chest X-ray showed gross hilar lymphadenopathy, but no other changes. Tubercle bacilli were not detected in the sputum or urine. The Kveim test was strongly positive and a diagnosis of sarcoidosis was made. There was no evidence of recurrent Crohn's disease in the bowel at that time. She was treated initially by bed rest and phenylbutazone, with diminution of her symptoms and reduction in size of the hilar lymphadenopathy. By November 1976 her physical examination and chest X-ray had returned to normal.

She remained well until late in 1978 when she presented again at this hospital complaining of a painful mouth, nausea and dysphagia. There were ulcers in the buccal mucosa with underlying submucous swellings, but physical examination was otherwise normal. A barium swallow, meal and follow-through demonstrated symmetrical narrowing of the distal half of the oesophagus with moderate proximal dilatation (Figure 1). The stomach, duodenum and small bowel were normal. At endoscopy the distal oesophagus showed marked inflammatory changes and ulceration. Biopsies of the inflamed area showed that the mucosa was completely absent, and there was a dense inflammatory infiltrate throughout the muscle layers. Ill-defined epithelioid granulomas and giant cells were seen. The appearances were thought consistent with Crohn's disease. Biopsy of the lesions in the buccal mucosa revealed numerous epithelioid granulomas beneath the squamous mucosa. No caseation was seen, but Langhans' giant cells were abundant (Figure 2). These appearances were also thought to be consistent with oral Crohn's disease.

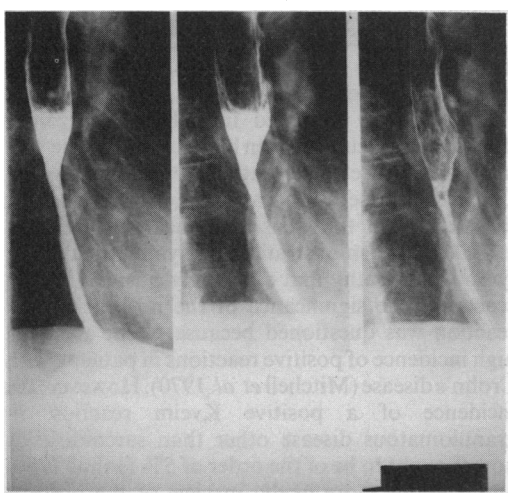

Figure 1. Barium swallow (November 1978) demonstrating narrowing of distal oesophagus, with proximal dilatation

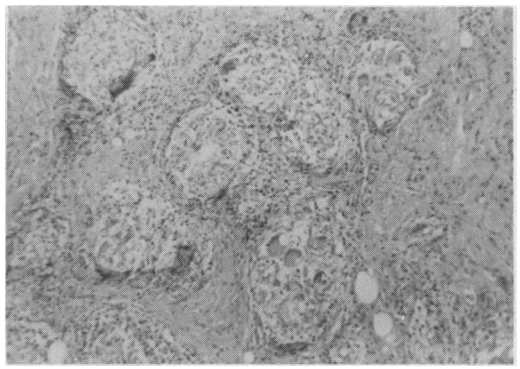

Figure 2. Numerous epithelioid granulomas in buccal mucosa. (× 128)

At this time her chest X-ray was normal with no radiological evidence of sarcoidosis. Her initial treatment was conservative and largely symptomatic, but over the next six weeks her dysphagia worsened and a further barium swallow examination showed a more severe stricture with ulceration (Figure 3). She was given oral prednisolone 10 mg three times a day. After two months the dosage was reduced to 10 mg twice a day; by the third month of treatment with steroids, her dysphagia and mouth lesions had almost disappeared and a further barium swallow showed a marked improvement (Figure 4).

Discussion

This patient is reported because of the unusual coexistence of two rarely described manifestations of Crohn's disease (in the mouth and oesophagus) and because of the unusual association with sarcoidosis.

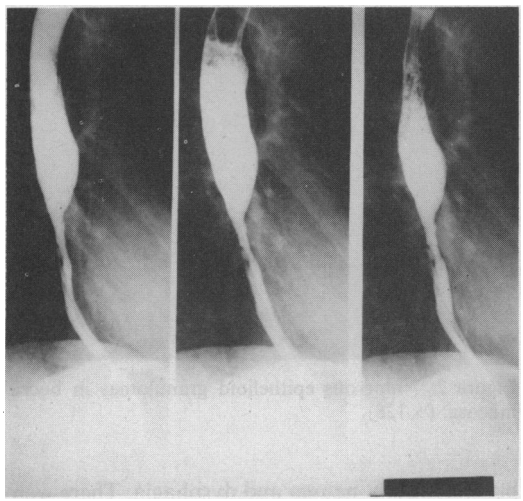

Figure 3. Barium swallow (February 1979) demonstrating increased stricturing of the oesophagus

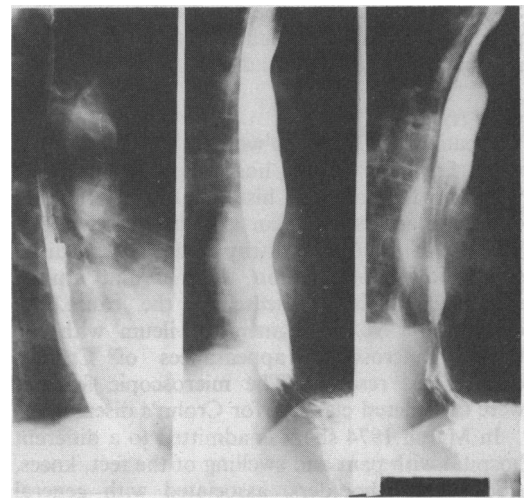

Figure 4. Barium swallow (April 1979) showing improvement after steroid treatment

Dudeney & Todd (1969) first reported Crohn's disease involving the mouth. Since then several reports linking mouth lesions with intestinal Crohn's disease have appeared (Issa 1971, Bishop *et al.* 1972, Croft & Wilkinson 1972). Crohn's disease has also been reported in the stomach (Martin & Carr 1953) and duodenum (Roberts *et al.* 1954). Franklin & Taylor (1950) first reported an inflammatory oesophagitis similar to Crohn's disease, and subsequent reports of a nonspecific granulomatous oesophagitis were published by Achenbach *et al.* (1956), Madden *et al.* (1969), Turina *et al.* (1969), and LiVolsi & Jaretski (1973). None of these cases was linked with intestinal disease. Heffernon & Kepkay (1954), Gelfrand & Krone (1968), Legge *et al.* (1970) and Bagly *et al.* (1972) have linked oesophagitis with intestinal Crohn's, but the oesophageal biopsy was either nonspecific or not performed. However, more conclusive evidence of Crohn's disease of the oesophagus in association with gastric, duodenal, ileal and colonic Crohn's disease has been obtained after surgery or autopsy in several patients (Dyer *et al.* 1969, Haggitt & Meissner 1973, Miller *et al.* 1977, Cynn *et al.* 1975).

In the present case, although absolute biopsy confirmation of oesophageal Crohn's disease is lacking, the biopsies are very suggestive, and the radiological features correlate well with those described by Legge *et al.* (1970) and Bagly *et al.* (1972). We therefore consider that this patient did have Crohn's disease of the oesophagus and the mouth simultaneously. This case appears to be the first in which this probable association has been documented, although Dyer *et al.* (1969), and Miller *et al.* (1977) suggest that mouth ulcers may be a common accompaniment of Crohn's disease of the oesophagus, and may precede the oesophageal lesions by many months or years.

Crohn's disease and sarcoidosis share similar systemic manifestations (Maycock *et al.* 1963, Rankin *et al.* 1979) and histological appearances. Both conditions are associated with impairment of cell-mediated immunity (Kraft 1971) and there is some evidence to implicate a transmissible agent in both diseases (Mitchell 1976). Despite this overlap, only 4 other patients have previously been reported as having the two diseases occurring together (Morland 1947, Dines *et al.* 1971, Padilla & Sparberg 1972). Apart from an uncommon, but well recognized, incidence of minor salivary gland involvement (Tarpley *et al.* 1972, Greer & Sanger 1977, Nessan & Jacoway 1979), sarcoidosis rarely affects the alimentary tract.

Our patient presented initially with Crohn's disease of the ileum, proven histologically. One year later, without evidence at the time of recurrent Crohn's disease, she was diagnosed as having sarcoidosis on the basis of clinical features, the radiological demonstration of symmetrical hilar lymphadenopathy and a strongly positive Kveim reaction. The significance of the positive Kveim reaction was questioned because of the reported high incidence of positive reactions in patients with Crohn's disease (Mitchell *et al.* 1970). However, the incidence of a positive Kveim reaction in granulomatous disease other than sarcoidosis is now thought to be of the order of 5% (Johns 1980) and it seems more likely, in view of the positive clinical and radiological appearances (which resolved simultaneously), that the positive Kveim reaction was, in fact, another manifestation of

sarcoidosis. By the time our patient presented 3½ years later with oral and oesophageal lesions, there was no clinical or radiological evidence of sarcoidosis. We therefore consider, on the basis of clinical, radiological and histological appearances, that this patient is another example of the rare association of sarcoidosis with Crohn's disease.

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Simultaneous occurrence of actinomycosis and lymphocytic lymphoma[1]

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Infection with actinomycosis is not common but may occur more frequently in patients with malignant disease. The simultaneous occurrence of actinomycosis and lymphoma has not, to our knowledge, been described previously. A patient is now reported who presented with a painful sternal lesion due to opportunistic infection by actinomycosis of lymphomatous tissue.

Case report

A 60-year-old Caucasian man presented in January 1981 with central chest pain and sternal discomfort unrelated to exercise. He had lost 6 kg in weight over the previous 12 months. In 1968 he had undergone a left pneumonectomy for squamous cell carcinoma of the bronchus and in 1978 had developed mild hypertension. On examination there was an area of tender erythema over the centre of the sternum measuring 4 × 3 cm.

Haemoglobin was 12.7 g/dl, white cell count 9.3×10^9/l, ESR 99 mm/h, alkaline phosphatase 272 iu/l; other liver function tests were normal. A technetium bone scan showed an area of increased uptake in the sternum. There was soft-tissue swelling, bone destruction and periostitis on X-rays of the sternum. Chest X-ray showed only the changes of the left pneumonectomy. A sternal aspirate showed lymphocytes and gram-positive coccal forms suggestive of degenerative actinomycetes. Subsequent anaerobic culture grew *Actinomycosis israeli.*

The patient was treated with phenoxymethyl-penicillin 2 g per day for two months with no improvement in either the symptoms or appearance of the sternal lesion. Antibiotic therapy was continued for a further two months with high-dose tetracycline. During this time he developed axillary and cervical lympha-denopathy. A further biopsy of the sternum showed poorly-differentiated lymphocytic lymphoma and at that time actinomycosis was again cultured.

He received ten courses of cyclophosphamide, adriamycin, vincristine and prednisolone, at

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