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Abdominal tuberculosis—a disease revived

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Summary

Abdominal tuberculosis was common in the United Kingdom in the 18th and 19th centuries and in the first half of the 20th century. During the 1950's the recognition of Crohn's disease, the use of streptomycin and other drugs, and the pasteurisation of milk led to the virtual disappearance of abdominal tuberculosis in the western world.

During the last two decades a new type, mycobacterium tuberculosis hominis, has appeared mainly in the immigrant population, especially in those from the Indian subcontinent.

A retrospective review of 68 patients with abdominal tuberculosis is presented. The pathology, diagnosis and management of these cases is discussed, together with the differential diagnosis of Crohn's disease.

It is suggested that the immigrant brings the disease into the United Kingdom in his mesenteric glands and that the disease is reactivated or 'revived' at some later date due to some modification of the immune process.

Introduction and historical review

It was almost 100 years after the death of John Hunter that Robert Koch, in 1882, discovered the tubercle bacillus when working in the Imperial Health Office in Berlin. He thus established the identity of a number of conditions previously regarded as distinct clinical entities such as lupus, scrofula, phthisis and Pott's disease. Whilst working at his brother William's anatomy school in London, John Hunter had worked with Percival Pott at St. Bartholomew's Hospital between 1748 and 1759, although it was 1779 before Pott first described tuberculous disease of the spine which still bears his name. Like others at that time, John Hunter was unaware of the infective nature of Pott's disease and also scrofula, although during the same period his observations and research into the mode of transmission of both syphilis and gonorrhoea are very well known. John Hunter probably had his suspicions regarding the cause of phthisis, as about November 1759 he is said to have contracted some infection affecting his lungs, and believed that he might be threatened with tuberculosis. There is no doubt that he suffered a severe illness and on advice took a complete rest from his active life during the second half of 1760.

Historical records reveal that tuberculosis had been recognised as a disease from the earliest time, and Hippocrates in the 4th century BC bestowed the name phthisis, meaning wasting, upon the disease as it affected the lungs. It is also interesting to note that one of the aphorisms of Hippocrates warned that 'phthisical persons die if diarrhoea sets in'. In other words, he had made the observation that patients with open pulmonary tuberculosis could develop intestinal complications. The association of intestinal ulceration, perforation and peritonitis, was vividly described in 1643 in the autopsy report of Louis XIII *(1)*. This is considered one of the earliest reports of secondary intestinal tuberculosis.

Tuberculous glands of the neck, which were likened in appearance to a sow's bulging neck, were given the name scrofula, from the Latin scrofa meaning sow. This was also known as King's Evill from the superstition that a touch of the royal hand of French and English monarchs conveyed a cure to the affected person. Since the 5th century, the kings of France were believed to receive this healing power from God at the time of their anointment; Edward the Confessor in the 11th century had also claimed this healing power for the English kings. Charles II is said to have touched some 90 000 patients suffering from scrofula, with reputedly good results, and this practice continued well into the 19th century. In 1696 Richard Wiseman in his Fourth Treatise *(2)* described scrofula, and from his post mortem examinations he recognised the same pathology in the mesentery, lungs, testes and pericardium, as well as lymph nodes, bones and joints. In other words, he depicted surgical tuberculosis. Careful reading of John Hunter's words reveal observations that were very relevant to surgical tuberculosis. In 1786 *(3)* he wrote in his Lectures on the Principles of Surgery, that scrofula was 'a specific disease but without a morbid poison'. He also observed that other animals besides the human subject were liable to it and probably from the same causes. He then writes 'the true scrofula seems attended with little or no inflammation; or if it is so, its sedative quality prevents it from producing pain; [presumably he was describing a cold abscess] but when there is less of the true scrofula, it may give pain. However, it is attended with many of the effects of inflammation, as extravasation of coaguable lymph producing swelling, and also adhesion of the cellular membrane or of the sides of circumscribed cavities'. John Hunter then goes on to describe the clinical features of abdominal tuberculosis. 'I have seen the whole of the intestine adhering to the peritoneum seemingly from a scrofulous cause, and having scrofulous tumours and suppurations in them also; the symptoms were tightness of the belly without pain, and costiveness; at other times, purging'. He also stated that adhesions in the lungs of consumptive people were probably of this kind, and like Richard Wiseman, he recognised that the disease process of scrofula could affect not only lymph glands but the lungs, uterus, testicle and bones.

During the 18th and 19th centuries, abdominal tuberculosis was probably as common as pulmonary tuberculosis and other types of the disease, and in his monograph in 1828 *(4)* John Abercrombie from Edinburgh gives a very lucid description of chronic peritonitis which is clearly identifiable with tuberculous peritonitis. He records the disease as 'occurring in young persons as an insidious affection of utmost danger yet extremely obscure in its symptoms'. He observed that chronic peritonitis was generally combined with disease of mesenteric glands and tubercular disease of

Based on a Hunterian lecture delivered at the University of Leeds on 5 May 1982

the lung. Then follows a description of an interesting condition called tympanites which he attributed to chronic peritonitis (*5*). This disease occurred in females, who presented with a firm, tense abdomen, usually associated with amenorrhoea and often mistaken for pregnancy, especially in those ladies who had married late in life. If treated at an early period the condition generally disappeared in a short time under a course of mild purgatives such as 'Harrowgate water', a therapeutic agent located not very far from Leeds.

During the second half of the 19th century, surgeons were beginning to operate on the abdomen and abdominal tuberculosis is reported to have been most commonly found after malignant and acute inflammatory conditions. At the beginning of the 20th century it had become customary to describe three forms of abdominal tuberculosis:

1) ulcerative tuberculosis or tuberculous enteritis;
2) tuberculous peritonitis;
3) hyperplastic tuberculosis.

William Boyd (*6*) in 1925, described the hyperplastic group as being a separate type occurring in young women under 40 years of age. There was great formation of tuberculous granulation tissue in the ileocaecal region, with abundant giant cells, few tubercles and absent caseation. The affected part of the bowel was thickened, narrow and stiff and formed a tumour-like mass resembling a carcinoma. The neighbouring glands were usually enlarged and in these cases the tubercle bacilli were rarely demonstrated and there was usually no evidence of tuberculosis in the rest of the body. In 1928, in his paper on chronic intestinal tuberculosis, Matthew Stewart (*7*) Professor of Pathology at Leeds University, stated that tuberculous ulceration of the intestine was present in half the cases of pulmonary tuberculosis which came to autopsy. He offered the hypothesis that the hyperplastic type was caused by a bovine strain of mild virulence in a patient with a high resistance to tubercle, which resulted in marked fibrosis in the ileocaecal region. Because of these features in hyperplastic tuberculosis, some observers at that time doubted the specific nature of the disease, and there is no doubt that many cases of hyperplastic ileocaecal tubercle were in fact Crohn's disease. In some instances, text book descriptions of the lesions were word for word perfect descriptions of Crohn's lesions which had yet to be described. In 1902 Mayo Robson, a surgeon at Leeds General Infirmary, presented a paper at the Clinical Society of London on the surgical treatment of chronic intestinal tuberculosis. From his reports and descriptions, 3 of his 7 patients had in fact Crohn's disease. Even at the beginning of this century, reports of ileocaecal tuberculosis without lung involvement were common, and in the 1920's surgeons were beginning to realise that there were lesions other than abdominal tuberculosis that could cause thickening and narrowing of the small intestine. Matthew Stewart commented that 'whether there is such a thing as a hyperplastic enterocolitis of non-tuberculous origin still remains to be seen. I am inclined to think there is, in spite of arguments already put forward'. And so, in 1932 Crohn, Ginsberg and Oppenheimer (*8*) published their classical paper on the disease of the small intestine which was to become known as Crohn's disease. The classification of abdominal tuberculosis remained, however, as before.

During the 1950's the recognition of Crohn's disease, the use of streptomycin and other drugs, and the pasteurisation of milk led to the virtual disappearance of abdominal tuberculosis caused by human and bovine strains in the western world. It is still a common disease on the continent of Africa and the subcontinent of India. In the 1960's and 1970's a new type, mycobacterium tuberculosis hominis, emerged with many reports in the British literature describing the disease as it is seen in Britain today where the vast majority of tuberculosis occurs in immigrants, particularly those from the Indian subcontinent. As the immigrant population has increased, so has the incidence of abdominal tuberculosis. Between 1967 and 1980 I reviewed 68 patients with abdominal tuberculosis, in a city of 400 000 people with 50 000 immigrants. The interval occurring between immigration and clinical presentation of the disease varied from 6 months to 16 years and on average was just a little over 5 years. The precise reason for such an interval is unknown. It is more than likely that the immigrant brings the disease into this country and it is reactivated at some later date. There is experimental evidence to suggest that there is some alteration in immune patterns that may occur as a result of the stress of immigration. Whilst it is true that the majority of patients who now present with abdominal tuberculosis are from the Indian subcontinent originally, it is not always the case, as 14% of the patients in our 1979 series (*9*) were of the indigenous population. In other series, white subjects were also reported to suffer from this disease (Table 1). Of 68 patients reviewed, 41 were male and 27 were female. The age range of the patients was from 8 to 52 years. The mean age of the males was 30 years and the females 32 years.

TABLE I

Series		*No. of white subjects*
Howell & Knapton (*10*)	(1964)	5 (42%)
Anscombe *et al* (*11*)	(1967)	8 (88%)
Mandal & Schofield (*12*)	(1976)	3 (20%)
Shukla & Hughes (*13*)	(1978)	5 (62%)
Findlay & Addison (*9*)	(1979)	7 (14%)
Lambrianides *et al* (*14*)	(1980)	2 (8%)

Pathological features

From a clinicopathological view, I would propose two main groups, (a) tuberculous peritonities, (b) gastrointestinal tuberculosis, and there may be a combination of both types in the same patient.

In tuberculous peritonitis there is usually a straw coloured ascites, more marked in the acute type. The peritoneum is studded with fine white tubercles and there is thickening of the walls of the intestine and other organs and the greater omentum is greatly infiltrated and thickened. There may be caseous masses of varying size in the peritoneal cavity.

Histological appearances reveal classical granulomas with epithelioid cells, Langhan's giant cells, central necrosis and an outer rim of lymphocytes scattered through the omentum and mesenteric fat. Acid fast bacilli are not always demonstrable.

In the gastrointestinal group, gastro-duodenal tuberculosis is rare and in this series there have been 4 patients, 1 with a lesion in the pylorus and 3 with lesions in the duodenum. Mandal and Schofield (*12*) reported 3 patients in 1976, and in 1979 Mukerjee and Singal (*15*) had an incidence of 2.8% in 500 patients operated on for abdominal tuberculosis.

Tuberculous enteritis is commonest in the ileocaecal region, seen in my series (68%) and others, but may involve any part of the small or large intestine. The wall of the intestine is thickened due to granulomatous infiltration and fibrosis, and forms a mass in the right iliac fossa (Fig. 1). There are enlarged lymph nodes and tiny miliary tubercles may be found over the peritoneal surface of the bowel and mesentery. There are no classical features of tuberculosis on naked eye appearances and the lesion may be indistinguishable from Crohn's disease as the mesenteric fat is increased and there is 'fat wrapping' around small bowel strictures as in Crohn's disease. Tuberculous lesions in the small intestine not only mimic Crohn's disease, but lymphoma and ischaemic strictures, and there can be multiple

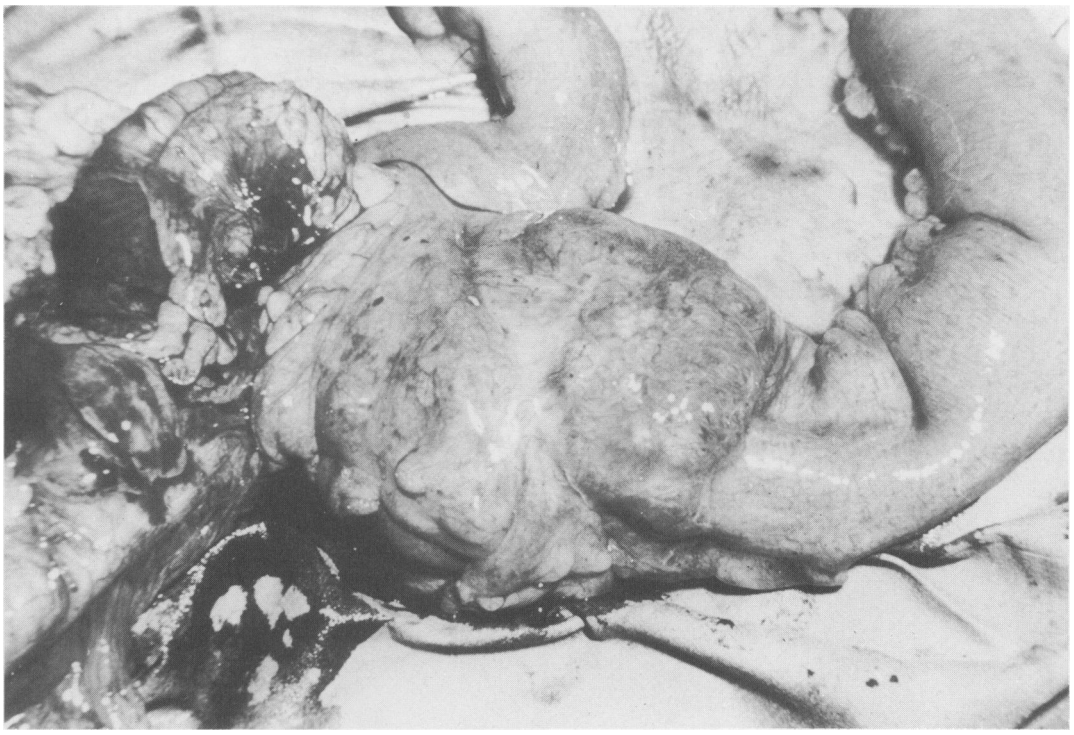

FIG. 1 Naked eye appearances of ileocaecal tuberculosis with thickened wall of intestine due to granulomatous infiltration and fibrosis and associated enlarged lymph nodes.

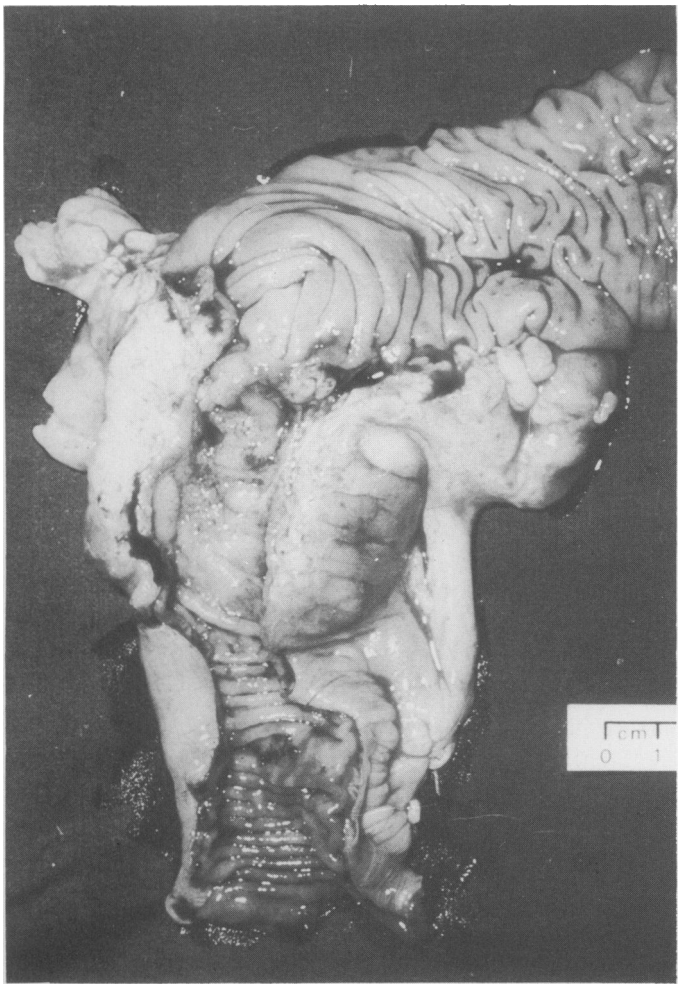

FIG. 2 Ileocaecal tuberculosis with excess amount of fibrosis and mucosal ulceration simulating malignant disease.

skip lesions. There is always superficial ulceration of the mucosa with irregular and undermined edges, associated with pseudopolyps giving rise to the appearance of a cobblestone mucosa like Crohn's and this leads to narrowing of the lumen.

On histological examination there is little difference between ulcerative lesions in the small intestine and the hypertrophic mass found in the ileocaecal region. In this region there is an excess amount of fibrosis in the thickened bowel wall and the hard white tissue associated with ulceration mimics malignant disease (Fig. 2). It has been suggested that secondary infection of tuberculous ulcers with pyogenic organisms may contribute to this appearance.

The mucosal ulcers do not usually penetrate the muscularis mucosa, but deep to this there are masses of granulomas which may coalesce and often show central necrosis with caseation (Fig. 3). The granulomata are scattered in all layers of the intestine and may involve the serosa. Caseation tends to be more common in the indigenous population especially in this series. Sometimes caseation is not seen in those patients from the Indian subcontinent and so the histological distinction between tuberculosis and Crohn's may be difficult. In non-caseating tuberculosis the granulomas are more numerous, larger and well defined, and may coalesce (Fig. 4). In Crohn's disease of the small intestine granulomas are certainly fewer in number, and the submucosa is widened due to oedema, whereas it is reduced or obliterated in tuberculosis. Inflammation in both Crohn's and tuberculosis has a transmural spread. In Crohn's it is a nonspecific inflammatory exudate, whereas in tuberculosis as we have seen, there are characteristic granulomas in all layers. Abscess and fistula formation are uncommon in tuberculosis, unlike Crohn's disease. The only certain way of distinguishing between Crohn's and tuberculosis is the actual demonstration of the tubercle bacillus within the section under examination, but this is not always possible even though the lesion histologically is typically tuberculous (*16*). The bacilli are more likely to be found when there is caseation necrosis. In my series, 58% were found to have tubercle bacilli of the hominis type. The incidence of

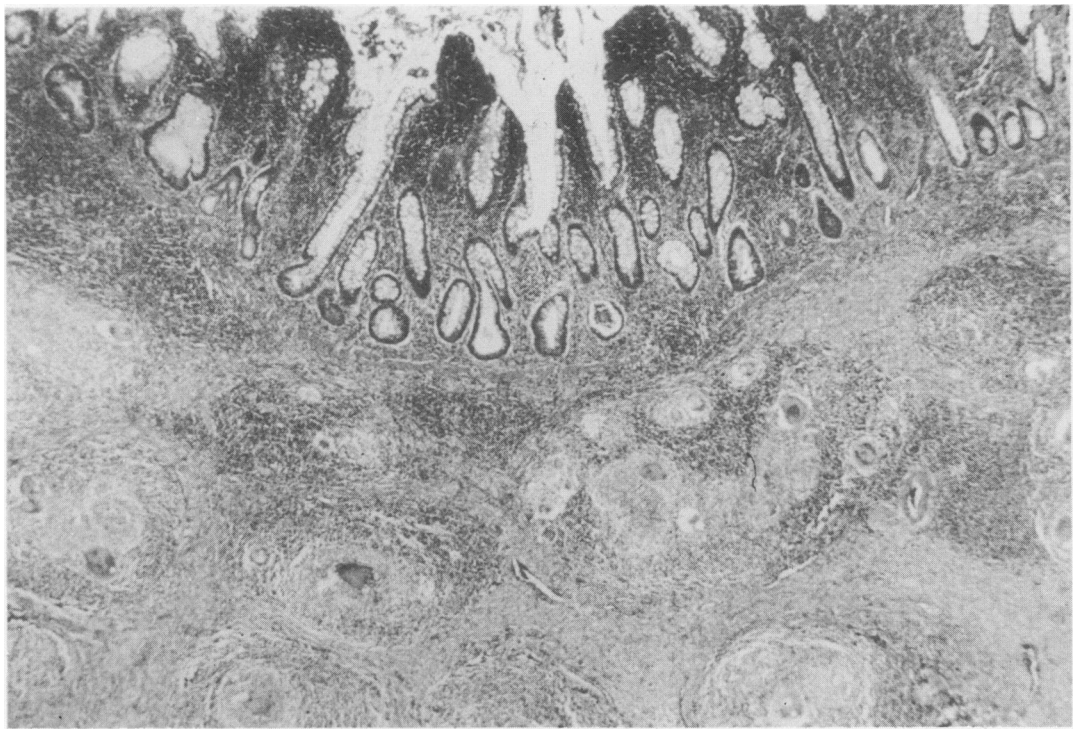

FIG. 3 Typical tuberculous enteritis with masses of granulomas, central necrosis and caseation.

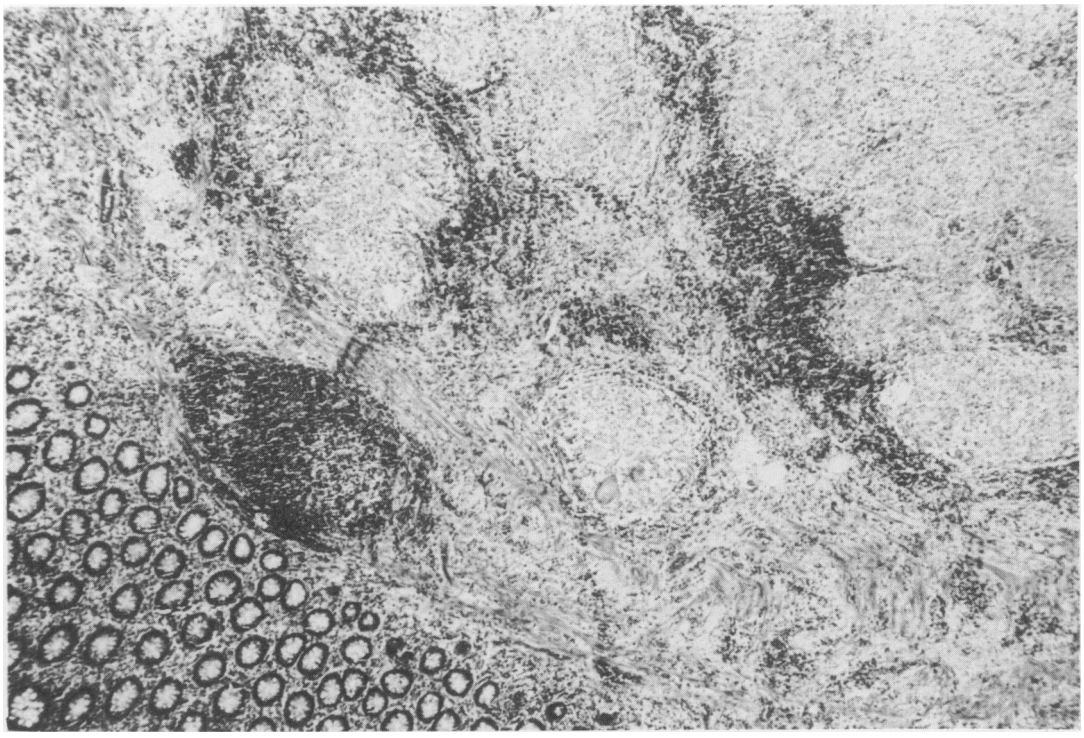

FIG. 4 Non-caseating intestinal tuberculosis with numerous granulomas in all layers undergoing coalescence.

identification of tubercle bacilli in other series is shown in Table II.

Usually the majority of lymph nodes in the mesentery contain granulomata with central caseation and occasionally acid fast bacilli are identified. The lower incidence of caseation particularly in mesenteric lymph nodes (Fig. 5) in patients from the Indian subcontinent was a feature of this study and may be due to modification of the immune process or partial attenuation of the disease. The final stage in the granulomatous inflammatory reaction in the small intestine is fibrosis, uncommon in Crohn's, which leads to multiple stricture formation and eventually intestinal obstruction.

Clinical features and diagnosis

There are no classical clinical features in abdominal tuberculosis, and it can mimic many other diseases the most common in Britain being Crohn's disease. Tuberculous peritonitis can

TABLE II

Series		*% tubercle bacilli identified*
Howell & Knapton (*10*)	(1964)	58%
Anscombe *et al* (*11*)	(1967)	22%
Prakash (*16*)	(1975)	6%
Shukla & Hughes (*13*)	(1978)	75%
Lambrianides *et al* (*14*)	(1980)	4%
Present series		58%

is said to occur in up to 85% of patients with intestinal tuberculosis alone.

Diarrhoea occurs in 10% of patients with abdominal tuberculosis and is due to small bowel ulceration. There is occasionally blood and mucus in the stools suggesting other types of inflammatory bowel disease and acute tuberculous colitis can occur (Fig. 6). In my experience ano-rectal lesions are rare in tuberculosis and if present are in fact due to Crohn's disease.

In the nonacute case, there are no pathognomonic clinical features of abdominal tuberculosis and the accuracy of

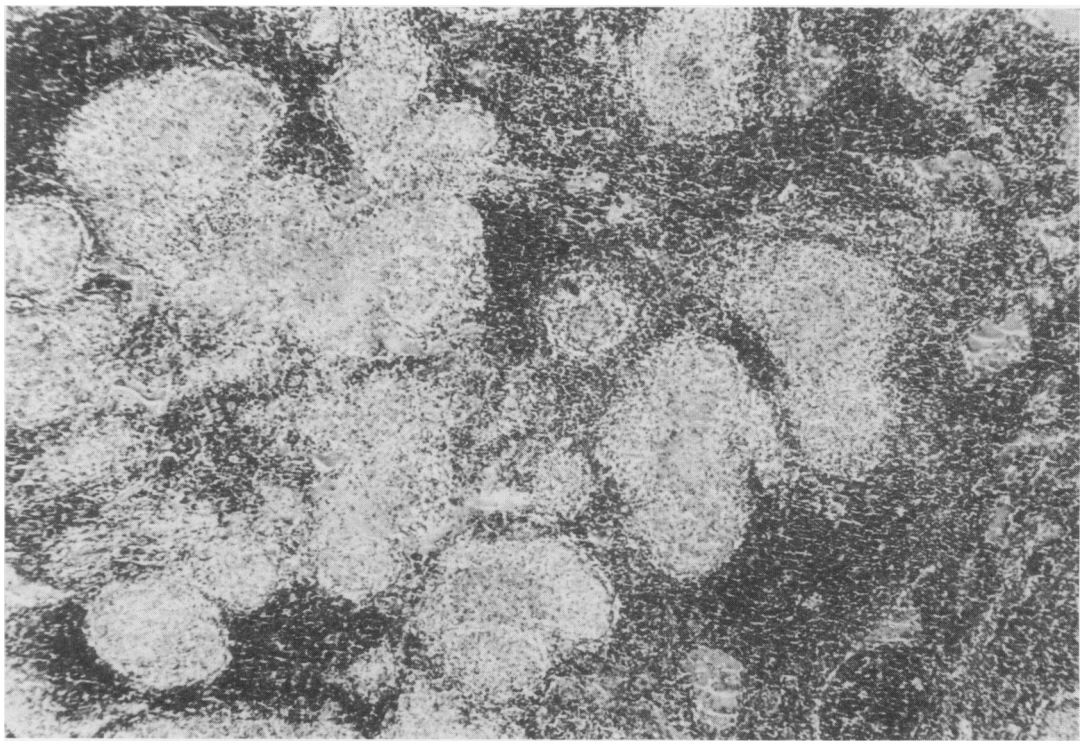

FIG. 5 Classical tuberculous granulomata in mesenteric lymph nodes.

be extremely obscure in its symptoms, as John Abercrombie pointed out in 1828.

Abdominal pain is the commonest symptom and was seen in 81% of patients in this series. In my experience, tuberculous peritonitis usually presents as an acute abdomen mainly in young children and adolescents of both sexes and mimics acute appendicitis. In 2 female Asian patients chronic tuberculous peritonitis was only discovered at the time of routine cholecystectomy for gallstones.

Ascites with abdominal pain and distension may be the first indication of tuberculous peritonitis either in the acute or chronic phase. As well as ascites there may be an associated palpable mass in the right iliac fossa and thus malignancy is considered. In my experience, carcinoma of the colon, like diverticulitis, is extremely rare in Asians, even in those who have lived in the United Kingdom for many years.

Acute or chronic obstruction with vomiting is probably the most frequent clinical presentation encountered and this would certainly appear to be the case in India (*15, 17*).

Following abdominal pain the next most common group of symptoms was pyrexia, night sweats, abdominal distension and weight loss. Pulmonary tuberculosis was present, either open or healed, in 32% in this series and there was gynaecological involvement in 11% of the females.

Ileocaecal tuberculosis usually presents as a mass in the right iliac fossa which may simulate either Crohn's disease, an appendix mass, a malignant lesion of the right colon, or an amoeboma in Africa and India. This clinical presentation clinical diagnosis is only 50% in India where the disease is endemic (*18*). Abdominal tuberculosis should, therefore, be considered in any patient, especially of Asian origin, who has obscure abdominal symptoms. The presentation can simply be that of vague ill health, lethargy and loss of weight.

Investigation

A plain X-ray of the abdomen may show small bowel fluid levels with or without ascites and there may be diffuse calcification or evidence of a localised abscess. In ileocaecal tuberculosis there are characteristic radiological appearances on barium enema examination. The caecum disappears and the ascending colon shortens; the ileum may retain its normal calibre but passes vertically upwards into the colon (Fig. 7).

In the colon a typical tuberculous lesion may simulate carcinoma with characteristic shouldering. Occasionally the lesion may be in the descending colon and may be multiple. Rarely disease of the oesophagus, stomach and duodenum may be demonstrable on barium meal examination. Patients with gastrointestinal tuberculosis may present with evidence of an iron deficiency anaemia. The ESR may be elevated and was in 56% of patients in this series. On the other hand, the ESR is frequently within normal limits. Heaf and Mantoux tests are not particularly helpful and in this series were only positive in 31%. The identification of the tubercle bacillus is extremely important and was actually detected on culture of stools, ascitic fluid or tissue in 58% of patients, but as previously mentioned, this varies in different series in the

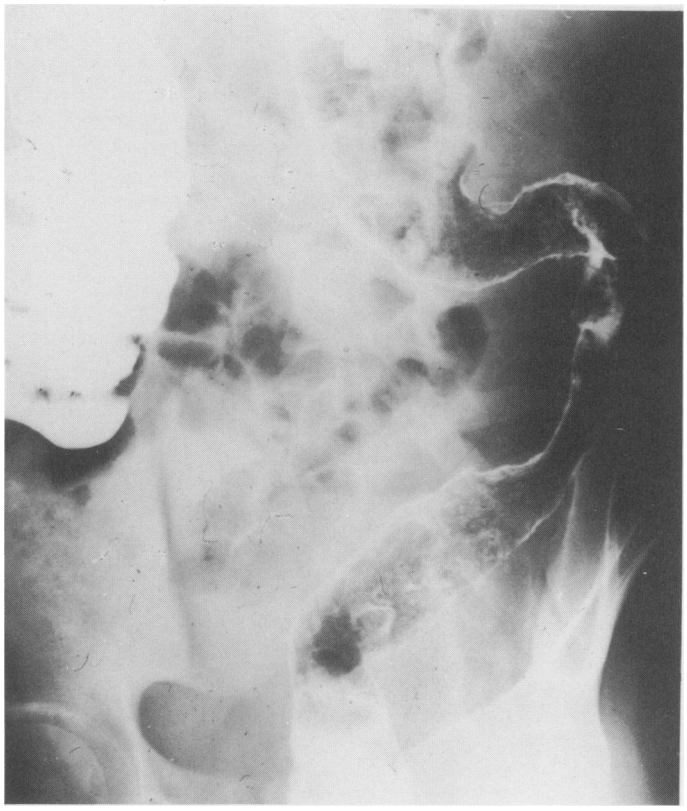

FIG. 6 Barium enema demonstrating acute tuberculous colitis affecting the pelvic colon and confirmed by biopsy.

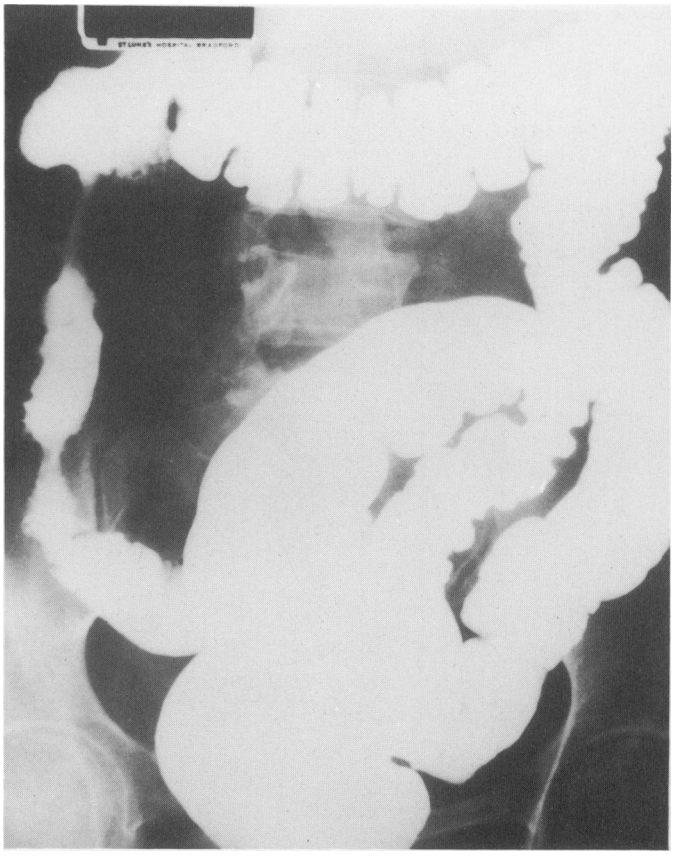

FIG. 7 Classical radiological appearances of ileocaecal tuberculosis. The ileum passes vertically upwards into the ascending colon which has shortened.

United Kingdom. Abdominal laparoscopy is now becoming a diagnostic procedure of choice and in 1979 Wolfe, Behn and Jackson (*19*) reported a positive biopsy in 8 of 11 patients and thus diagnostic laparotomy was avoided. In 1978 Udwadia in Bombay (*20*) found 29 tuberculous lesions in 384 laparoscopies in patients with abdominal symptoms and signs but in whom all the standard investigations for tuberculosis were unhelpful. In a recent series (personal communication) of 321 laparoscopies he confirmed the diagnosis in 218 and stated that peritoneal tubercles were seen in 206 patients. Colonoscopy is now becoming a very useful diagnostic procedure even in ileocaecal tuberculosis. Finally, laparotomy may be the only means of making a definite diagnosis of abdominal tuberculosis. Like the clinical symptomatology we have seen that there are no diagnostic features of tuberculosis on naked eye examination. An ileocaecal mass, small bowel strictures, enlarged mesenteric lymph nodes, thickened bowel wall and mesentery are all common findings in both tuberculosis and Crohn's disease, and similar appearances may be seen in lymphomata. A hard tuberculous caecal mass is commonly mistaken for carcinoma.

Surgical management

The surgical management of abdominal tuberculosis depends on whether the condition is acute or nonacute. Young children and adolescents may present as an acute appendicitis and the abdomen is explored. If there is no mechanical obstruction of the bowel present, then an omental or gland biopsy should be carried out and the abdomen closed. Ascitic fluid should be sent for culture and animal inoculation for tubercle bacilli. Once the diagnosis has been established a course of antituberculous drugs should be given. In the case of acute intestinal obstruction, localised resection of the solitary stenotic nodule or multiple lesions in a short segment of bowel is the treatment of choice although ileoplasty for strictures has been advocated (*21*).

For ileocaecal lesions, a limited right hemicolectomy is the operation of choice in my experience and good results have been reported (*22*). Seventeen patients underwent this procedure in my series, and again, this should be followed by a course of antituberculous drugs. In addition six patients were treated by small bowel resection and three had a large bowel resection. By-pass of the lesion with an ileotransverse colostomy should be avoided, as patients tend to fare badly after this procedure just as they do in the treatment of Crohn's disease. Short circuiting procedures like gastrojejunostomy can be used in duodenal tuberculosis. A stenotic lesion in the colon should be treated by local resection if a preoperative diagnosis of tuberculosis is made, but if the lesion is thought to be a carcinoma then a more radical partial colectomy should be carried out. The results of colonic resection are equal to those following right hemicolectomy, but should always be followed by a course of adjuvant antituberculous drugs. Specific antituberculous drugs should include Rimactazid and ethambutol.

Crohn's disease and gastrointestinal tuberculosis

Crohn's disease and gastrointestinal tuberculosis occur both in the immigrant and in the indigenous population and both diseases may present in a very similar way. Tuberculosis should always be considered in the differential diagnosis of a mass in the right iliac fossa. Similarly, Crohn's disease and ulcerative colitis must be considered in the differential diagnosis of gastrointestinal tuberculosis in immigrants. Since the recognition of Crohn's disease in 1932, there is no doubt that many cases of so-called primary hyperplastic ileocaecal tuberculosis were in fact Crohn's and many of these cases without recognisable caseation proved on further histological examination to be cases of Crohn's disease. Caseating granulomas are considered an essential criterion in the diagnosis of tuberculosis but may be difficult to demonstrate as we have seen in the Asians in this study. The

only absolutely certain way of distinguishing between Crohn's disease and tuberculosis is the actual demonstration of tubercle bacilli within the section under examination. However, we have seen in Prakash's series (*16*) of 92 patients with confirmed ileocaecal tuberculosis, that positive bacterial evidence was only found in 6 patients. Granulomas are present in both Crohn's disease and tuberculosis. In tuberculosis it is not unusual to find them in the mesenteric lymph nodes while they are absent in the intestine, whether or not the patient has received any form of chemotherapy. In Crohn's disease, if the lymph nodes do not show granulomas then none are seen in the intestine. Histologically, in Crohn's the transmural disease is a nonspecific inflammatory exudate whereas in tuberculosis there are characteristic granulomas in all layers.

Conclusions

Abdominal tuberculosis is no longer a very rare disease in parts of the United Kingdom and in fact is now 'a disease revived'. This is largely due to the arrival of the Asian immigrant, but the disease still occurs in the indigenous population. In the past, abdominal tuberculosis developed after swallowing the bovine bacillus in infected milk, and the human bacillus in infected sputum. Bovine tuberculosis has now disappeared in the United Kingdom and most patients with abdominal tuberculosis did have evidence of the disease elsewhere in the body, usually the lungs. The exception to this was the group described as 'hyperplastic ileocaecal tuberculosis' where true caseation was usually absent and the tubercle bacillus was never isolated. As there is now little open pulmonary tuberculosis one speculates as to how the intestine and peritoneum become involved in the absence of overt disease elsewhere in the body. In my series 68% had no evidence of pulmonary tuberculosis.

It is doubtful whether true primary lesions occur in the small intestine but they certainly do occur in mesenteric lymph nodes. The human bacilli pass through the intestinal mucosa of Peyer's patches without producing a local lesion and, transported by macrophages, enter the intestinal lymphatics and settle in the mesenteric lymph nodes where they may multiply or remain dormant for long periods of time, sometimes for life.

It is a distinct possibility that ulcerative lesions in the bowel are the result of retrograde lymphatic spread from mesenteric nodes. Matthew Stewart in 1928 said 'local retrograde extension from tuberculous mesenteric glands is perhaps more common than we suppose'. The more advanced tuberculous lesions with caseation in the lymph nodes compared with the bowel lends some support to this suggestion, although in the Asians in my series there was a low incidence of caseation even in the lymph nodes. Like typhoid, lymphoid tissue in the ileum develops hypersensitivity rapidly to the tubercle bacillus, resulting in ulceration which is secondary to primary infection in lymph nodes. Tuberculous peritonitis may develop in a similar manner to that of tuberculous enteritis.

It is more than likely the immigrant brings the tuberculosis into the United Kingdom in his mesenteric lymph glands, and that the disease is reactivated at some later date due to some modification in the immune process. With these facts in mind, tuberculosis must be considered in the differential diagnosis of acute or chronic abdominal pain in patients of Asian origin, remembering that it can mimic many diseases, the commonest in Britain being Crohn's disease. When the diagnosis is in doubt, surgical exploration is indicated but even at laparotomy the ultimate diagnosis may only be achieved after histological and bacteriological studies.

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