Abdominal tuberculosis: the great mimic

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INTRODUCTION

The incidence of abdominal tuberculosis is increasing in the UK. Clinicians need to have a high index of suspicion in patients with unexplained abdominal symptoms or pyrexia of unknown origin, particularly in those from high-risk groups. This article describes the case of a young woman who presented with life-threatening gastrointestinal and peritoneal tuberculosis, which highlights the difficulty in the diagnosis and management of this condition.

DISCUSSION

Once known as the 'white plague', tuberculosis continues to be a major global problem, being responsible for 7–10 million new cases each year and 6% of deaths worldwide. In India alone, tuberculosis claims over 1000 lives every day, with abdominal tuberculosis being responsible for 6% of admissions for intestinal perforation (Ahmed and Hassan, 1994; Ahmed and Scott, 1996).

Abdominal tuberculosis is also re-emerging as a clinical problem in the UK — particularly in immunosuppressed patients and those from the Indian subcontinent — but often remains underdiagnosed. In a study from a West London hospital serving a large Asian community, 90 patients with abdominal tuberculosis were identified over a period of 10 years (Underwood et al, 1992), while another study from East London reported 7.7 cases of abdominal tuberculosis per 100 000 among the Bangladeshi community compared with 0.3 cases per 100 000 in the white population (Ahmed and Scott, 1996).

Associated pulmonary tuberculosis is seen in approximately 15–20% of cases of abdominal tuberculosis. In immunosuppressed patients, isolated extrapulmonary tuberculosis is relatively common (50–60% of cases compared with 10–15% in immunocompetent patients) and can have a rapidly fatal outcome (Marshall, 1993).

As demonstrated by this case report, the diagnosis and management of abdominal tuberculosis can be difficult. Patients may present with a wide spectrum of non-specific symptoms, allowing a correct diagnosis before laparotomy in only 50% of cases. A positive tuberculin test and signs of active disease on chest X-ray are seen

CASE REPORT

A 22-year-old woman, who had immigrated from India 2 years earlier, presented to casualty in May 1997 with cough and atypical chest pain. Sputum microscopy was positive for acid-fast bacilli, there was an equivocal right apical shadow on chest X-ray and she had a grade three Heaf test. Consequently, she was commenced on triple antituberculous therapy, but this was stopped 2 months later on the advice of the Mycobacterial Reference Laboratory when *Mycobacterium fortuitum* (a presumed contaminant) was isolated from one sputum culture.

The patient remained well until Febuary 1998, when she was referred back with a 3-week history of cough, night sweats, abdominal pain and fever. At admission, she was dehydrated and pyrexial (40°C), with a microcytic anaemia (haemoglobin 9.5 g/dl, vitamin B_{12} 411 ng/litre, folate 5.1 nmol/litre), normal white cell count and raised inflammatory markers (C-reactive protein 153 mg/litre, erythrocyte sedimentation rate 110 mm/h). Chest X-ray was unremarkable. Cultures of urine, stool, blood and sputum (including sputum microscopy for acid-fast bacilli) were negative. Abdominal ultrasonography was normal apart from a small amount of culture-negative ascitic fluid, which contained a high concentration of white cells (70% polymorphs). Transthoracic echocardiography was normal. A 99m-technetium-labelled white cell scan showed increased uptake in the right iliac fossa. Ileocolonoscopy on the tenth day of admission showed a stenosed ileocaecal valve and extensive caecal ulceration (*Figure 1*), with histological features of caseating granulomata and numerous acid-fast bacilli on Ziehl–Neelsen staining. Subsequent cultures confirmed the presence of multisensitive *Mycobacterium tuberculosis*.

Immediately after the colonoscopy, the patient was commenced on quadruple antituberculous therapy with rifampicin, isoniazid, ethambutol and pyrazinamide, but over the next 2 weeks, she had ongoing fevers >40 °C with weight loss despite nasogastric feeding. Abdominal computed tomography with oral contrast showed a small amount of ascites but no paracaecal or other masses and no signs of perforation. In an attempt to control her systemic symptoms, intravenous hydrocortisone was added to her antituberculous therapy in the fourth week, with some improvement in her fevers. However, 2 weeks later, she suddenly developed painful ascites with worsening sepsis syndrome. An urgent ascitic tap showed faecal organisms, and she proceeded to laparotomy later that day. Multiple nodular caseating lesions were present over the parietal and visceral peritoneum (*Figure 2*), and a pinpoint perforation of the ileocaecum was identified. A wedge excision of the ileum was performed as her general condition and multiple adhesions mitigated against more extensive bowel resection.

The patient went on to have a complicated and prolonged intensive care stay, with the development of an ileocolonic fistula requiring a right hemicolectomy. She was finally discharged home 4 months after admission and is now well and followed up regularly in the outpatient department.

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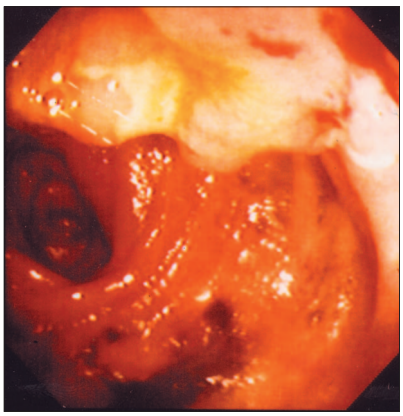

Figure 1. Endoscopic view of the caecum showing an ulcerated ileocaecal valve. Ziehl–Neelsen staining of biopsies from the ulcer revealed numerous acid-fast bacilli.

in approximately three-quarters and one-fifth of cases respectively (Marshall, 1993; Singh et al, 1995).

When the peritoneum is involved, the ascitic fluid is characteristically an exudate with a high lymphocyte concentration. Microscopy for acid-fast bacilli is positive in less than 5% of cases, with culture sensitivities ranging from 20–50% after 4–8 weeks, thus limiting its usefulness in diagnosis. In contrast, adenosine deaminase activity — an enzyme released by macrophages and lymphocytes during a cellular response — in ascitic fluid has been reported to have more than 90% sensitivity and specificity for tuberculous peritonitis (Marshall, 1993; Ahmed and Scott, 1996).

Barium contrast studies, abdominal computed tomography and white cell scans may all show abnormalities, but none of the features are pathognomonic of abdominal tuberculosis. Ileocolonoscopy provides a means of visualizing the involved bowel directly and obtaining tissue for diagnosis, although the histological appearances may be indistinguishable from Crohn's disease. Caseation, seen only in tuberculosis, is not always evident in gut wall and lymph node histology, even in proven cases (Marshall, 1993; Singh et al, 1995). Peritoneal involvement occurs in about 20% of cases, with the characteristic findings at laparoscopy or laparotomy of multiple miliary nodules ('tubercles') covering both the visceral and parietal peritoneum (Ahmed and Hassan, 1994).

Treatment for abdominal tuberculosis is usually medical, with a course of antituberculous medication for 6–9 months. Surgery is generally reserved to aid diagnosis where other methods have failed and to treat complications such as bowel obstruction, fistula formation, perforation or haemorrhage (Singh et al, 1995; Ahmed and Scott, 1996). Despite modern management, the prognosis of abdominal tuberculosis remains poor, with reported mortality rates of 11–50% (Ahmed and Hassan, 1994; Ahmed and Scott, 1996). Clinicians need to maintain a heightened awareness of the problem in order to curb the recurrence of this 'plague'. HM

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Ahmed ME, Hassan MA (1994) Abdominal tuberculosis. *Ann R Coll Engl* **76:** 75–9

Marshall JB (1993) Tuberculosis of the gastrointestinal tract and peritoneum. *Am J Gastroenterol* **88:** 989–9

Singh V, Jain AK, Agarwal AK et al (1995) Clinicopathological profile of abdominal tuberculosis. *Br J Clin Pract* **49:** 22–4

Underwood MJ, Thompson MM, Sayers RD, Hall AW (1992) Presentation of abdominal tuberculosis to general surgeons. *Br J Surg* **79:** 1077–9

Figure 2. Appearance of the peritoneal cavity at the time of laparotomy. Note the presence of multiple whitish 'miliary' nodules (<5 mm) scattered over the visceral and parietal peritoneum.