PERITONEAL TUBERCULOSIS PRESENTING AS PYREXIA OF UNKNOWN ORIGIN IN IMMIGRANTS

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Introduction

Peritoneal tuberculosis remains a comparatively rare presentation of tuberculosis in this country. In 1963, Lougheed found 40 cases among 3,755 patients with tuberculosis. The diagnostic difficulties have been well reviewed in the United States where it is much commoner in the Negro population (Sochachy, 1967). In this series of 100 cases, 55 had abnormal chest x-rays compatible with pulmonary tuberculosis or pleural or pericardial effusion. Twenty-three of the patients had tubercle bacilli grown from the sputum. Involvement of other abdominal organs appears to be uncommon (Singh, Bhargava and Jain, 1968). There have been occasional reports from Britain (Grant, Grunberg and Blair, 1951; Salmon, 1951; Hyde, 1958; Dowling, 1967; Basserby, 1967).

The following three case reports were seen between 1971 and 1973.

Case 1

A 26-year-old Ugandan African microbiology student, resident in the U.K. since 1965, was admitted in March 1971. He presented to the surgical department with a one month history of fever, central abdominal pain and melaena. On examination, there was generalized abdominal tenderness and slight guarding. No ascites was detected. Investigations: the chest x-ray was normal. The ESR was elevated 98, 100, 133, 98 Westergren units. The blood picture showed a persistent anisochromia and anisocytosis with a haemoglobin range between 7·6 and 8·6. The total white cells were between 2,700, 8,900. The alkaline phosphatase was elevated between 180 and 250 i.u. (n.r. 30 - 80), as was the lactic dehydrogenase (LDH), 420 to 600 i.u. (n.r. 100 - 200), and the serum glutamyl oxalic transaminase (SGOT), 80 - 270 i.u. (n.r. 20 - 50); the bilirubin and serum albumin/globulin fractions were normal. A Mantoux test (concentration not given) was negative. A laparotomy on the seventeenth day of admission showed a large number of creamy white nodules on the peritoneum with numerous fibrous adhesions; there was no ascites. The diagnosis was confirmed histologically and tubercle bacilli were cultured. In addition to standard antituberculous therapy, prednisone was given for the first six months. He made an uneventful recovery.

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Case 2

An Indian schoolboy of 16 had been resident in England since 1969. His first admission in June 1971 was with a pyrexia of unknown origin (PUO). Anorexia and symptoms of fever with night sweats had been present for 10 days. There were no physical signs. The chest x-ray showed fibrosis of the left upper lobe and a white cell count was 7,400 with 30 per cent lymphocytes. The ESR was 61 Westergren units. The Tine test was positive. Alkaline phosphatase and lactic dehydrogenase were both slightly elevated. Further investigations were unhelpful. The fever settled without treatment and he was discharged.

The second admission was four months later for fever and slight generalized abdominal tenderness. Investigations: the blood film was normal and the haemoglobin between 11·8 and 12·9 G. Total white cells were between 4,600 and 8,200. The ESRs were 60, 58 and 63. Alkaline phosphatase was elevated between 120 and 180 i.u., the lactic dehydrogenase slightly elevated, 225 - 330 i.u., and the SGOT, bilirubin and albumin/globulin fractions were normal. A liver biopsy showed inflammatory changes which were non-specific. A laparotomy on the 27th October, 1971, showed peritoneal tubercular deposits confirmed histologically. There were extensive adhesions but no ascites was evident. Tubercle bacilli were cultured.

Case 3

A 48-year-old West Indian man, resident in the U.K. since 1960, was admitted in May, 1973. He complained of a four-month history of night sweats and weight loss and, shortly before the admission, a haemoptysis. The chest x-ray was clear. During the first week of admission he developed severe ascites. The protein

content of the fluid was 4·5 G/100 ml.; SG 1019; no cells or acid-fast bacilli were seen. The Mantoux test was negative. The blood film showed anisochromia and anisocytosis. The haemoglobin was 9·6 to 11·8 G, white cells 3,200 to 9,000, ESRs 30, 20, 36, 80 and 35. Alkaline phosphatase was normal. The lactic dehydroganase was elevated, 258 to 789, as was the aspartic transaminase, 69 to 177 i.u. (n.r. 10 - 50). A liver biopsy showed a few collections of histocytes in the parenchyma. The patient refused laparotomy and laparoscopy was unsuccessful because of widespread adhesions and ascites causing failure of adequate visualization. Anti-tuberculous treatment was begun empirically three weeks after admission. The tubercle bacillus was grown from the ascitic fluid.

Discussion

These three cases illustrate some of the difficulties in the diagnosis of peritoneal tuberculosis. Abdominal pain and tenderness may be minimal and ascites is often absent (Hyman *et al.*, 1971); this may lead to delay in diagnosis (Kahr, 1952). Investigations may also be misleading. The white cell count is commonly below 10,000 in the presence of a severe fever, there may be a secondary anaemia and the tuberculin skin test may be negative. Only the third patient had ascites and the protein content was 4·5 grams per cent; it is very rare to find the concentration below three grams per cent (Sochachy, 1967). The alkaline phosphatase, LDH and SGOT were persistently abnormal but the bilirubin and albumin/globulin fractions were normal. Although intrahepatic tuberculosis cannot be excluded in these patients, abnormal liver enzyme tests are commonly found in extrahepatic bacterial infections and may be unhelpful in the differential diagnosis of intrahepatic and extrahepatic infections (Neale *et al.*, 1966). Liver biopsy in cases 2 and 3 was unhelpful showing non-specific inflammatory changes. Blind needle peritoneal biopsy has been recommended to make the diagnosis (Levine, 1968) and although this was tried in the third patient the changes were of non-specific chronic inflammation. Diagnostic laparotomy in cases of obscure fever can be very helpful as a final investigation (Barr, Magnusson and Wallensten, 1972). The preoperative diagnosis is commonly not made and most patients have required laparotomy to make the diagnosis (Basserby, 1967). In the first two cases who came to laparotomy the only alternative way of making the diagnosis was by laparoscopy. In the third patient laparoscopy was unsuccessful, mainly because of extensive adhesions, and since all three were shown to have adhesions this procedure may be less valuable than laparotomy when this diagnosis is suspected. No other abdominal organs were shown to be involved in these three patients. In the 45 cases reported by Basserby, only three had other intrabdominal involvement; two had ileocaecal tuberculosis and the third an ileal stricture alone. However, the need to exclude other organ involvement is another advantage of laparotomy over laparoscopy. All three patients made a full recovery on a standard anti-tuberculous regime of streptomycin, paraaminosalicylic acid and isoniazid. The hospital mortality in the Basserby series was 6·6 per cent during a 10 year period before 1967. In the pre-chemotherapy era several large series had a hospital mortality rate between 25 and 55 per cent (Wichelhausen and Brown, 1950).

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Summary

Three immigrant patients with peritoneal tuberculosis seen over a three year period are discussed. All three patients presented with fever and minimal abdominal signs. One patient developed ascites while under investigation. In all cases, the fever was prolonged, liver enzymes were elevated and the ESR was raised. Two patients required laparotomy to make the diagnosis, and in the third tubercle bacilli were grown from the ascitic fluid. The difficulties in diagnosis are discussed.

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two of the adult patients, liver biopsy provided the first positive histological evidence. If lymph glands are enlarged, aspiration of gland juice may provide material for smear or culture; moreover, an accessible gland may be removed for histological examination.

The specific treatment is sodium stibogluconate (Pentostam). We give a much higher dose than that usually recommended. We give daily intravenous injections for 15 days, with 300 mg. and working up quickly to 600 mg. daily, giving a total dose of 8·3 G. All the six adult patients did well and no repeat courses were necessary. The temperature subsided within three to four days.

In those cases where the temperature is initially high, a further rise in temperature not uncommonly follows the start of treatment with Pentostam. This will often result in a further aggravation of the toxaemic state. We have used steroids, usually prednisone 40 mg. daily, in order to mitigate the undesirable effects of the toxaemia. The E.S.R., the spleen and the electrophoretic pattern may take up to three to four months to return to normal.

In conclusion, it is worth noting that visceral leishmaniasis may be acquired whilst holidaying in the Mediterranean. Indeed, in the last few years case reports have appeared of patients who had developed symptoms of leishmaniasis in their own countries after having returned from endemic areas (Buckner 1966; Treske and Stansic, 1968; Heilmann, Dolmert and Wohlenberg, 1971; Clayton, 1972). With mass tourism to areas where the disease is endemic, this phenomenon is likely to recur and possibly increase.

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