Mycobacterium tuberculosis as a cause of chronic periaortitis

Sir, We read with great interest the comprehensive review of chronic periaortitis (CP) by Jois and colleagues [1]. We wish to highlight another cause of this condition not mentioned in the review, as illustrated by a recent case.

A 36-yr-old Pakistani man resident in the UK for 13 yr presented with a 2-month history of intermittent pain in his epigastrium, myalgia, drenching night sweats and a non-productive cough. He also reported a 2-kg weight loss over 2 months. His previous medical history was of peptic ulcer disease with no known history of tuberculosis (TB). He was a market trader by profession and had not travelled abroad for at least 2 yr. He lived with his wife and two daughters, all of whom were in good health. Clinical examination revealed low-grade pyrexia and minimal epigastric tenderness without organomegaly. Cardiorespiratory and locomotor examinations were normal. His ESR was raised at 93 mm/h with CRP of 48 mg/l (normal range <10 mg/l). Autoantibody profiling showed the presence of anticardiolipin antibodies (IgG 28 IU; normal range 0–16 IU), but was otherwise normal. The following investigations were normal: full blood count, serum glucose and cholesterol, renal, liver, muscle and bone profiles, coagulation, electrocardiogram, chest radiograph, gastroscopy and cultures (of blood, stool and three early-morning urines for acid alcohol fast bacilli). An abdominal ultrasound revealed a fatty liver, but no other abnormality was noted.

MRI of the thorax and abdomen (Fig. 1a) showed circumferential encapsulation of the upper abdominal aorta with secondary aortic narrowing by an abnormal soft tissue mass. This extended 7 cm below the diaphragm and unfortunately was inaccessible for biopsy. A diagnosis of CP was made and the patient was commenced on 40 mg of prednisolone daily. However, following 3 months of treatment with only minor symptomatic improvement, repeat MRI scanning did not demonstrate any resolution of the periaortic mass.

One month later, the patient’s persistent dry cough became productive of green sputum. A clinical diagnosis of Mycobacterium tuberculosis infection (smear negative) was made and 6 months of quadruple antituberculous therapy (rifampicin, pyrazinamide, ethambutol and isoniazid, with pyridoxine cover) commenced. A dramatic improvement occurred in both his respiratory and systemic symptoms in conjunction with normalization of inflammatory markers. Repeat abdominal MRI scanning (Fig. 1b) showed significant regression of the periaortic soft tissue, and the patient is well at 12 months follow-up.

We propose that this patient’s CP be due to infection with M. tuberculosis, which, though resistant to corticosteroids, responded to antituberculous therapy. As highlighted by Jois et al. [1], chronic inflammation is the primary pathogenetic factor in the development of retroperitoneal fibrosis and periaortitis. A radiological diagnosis of CP is often made but determination of the exact aetiology may prove difficult [2, 3]. Chronic infections such as tuberculosis, actinomycosis and schistosomiasis can initiate this inflammatory reaction and are recognized causes of CP [4]. Even though microbiological confirmation of M. tuberculosis could not be made in this case, the patient’s clinical history, radiological findings and therapeutic response were consistent with this being the causative organism.

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M. tuberculosis is a rare cause of retroperitoneal disease and when it does occur more commonly gives rise to tubercular pseudotumours and retroperitoneal abscesses [5, 6]. Confluent peritoneal lymphadenopathy may cause secondary oedema and thus ureteric obstruction, mimicking CP [6, 7]. CP may also occur secondary to spinal, peritoneal or haematogenous spread of distant M. tuberculosis infection [3, 8]. In this case the initial tubercular focus may have been pulmonary, with precipitation of overt clinical symptoms following systemic corticosteroid therapy. TB-related CP has a good outcome with adequate antitubercular therapy [3, 7], making this a treatable cause of CP.

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Methotrexate in pregnancy

Sir, We read with interest the report by Kinder et al. [1] of a patient who conceived while on methotrexate. However, we were concerned that our conclusions may have been misinterpreted. Our review [2], which was the largest of its kind at the time, concluded that exposure to methotrexate in pregnancy is associated with a roughly 25% risk of fetal malformation. We were not able to conclude that there is a threshold dose at which methotrexate is safe in pregnancy, partly because the numbers of cases of fetal exposure below 10 mg weekly are so few. This conclusion came from other authors and remains speculative [3, 4]. The National Teratology Information Service (NTIS) has a report of a pregnant woman who received a single dose of methotrexate 7.5 mg between 4 and 7 weeks of gestation. An emergency Caesarean section was performed at 28 weeks for placenta praevia and the baby had a number of abnormalities, including positive sweat test, ileal perforation and respiratory distress syndrome.

We would advise contacting the NTIS in any case of methotrexate exposure during pregnancy.

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