TB or not TB? That should be the question

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Introduction

In the first half of the twentieth century Tuberculosis (TB) was a common and often fatal disease. Advances in radiological diagnosis and antibiotics then led to the decline in all forms of TB in developed countries [11]. With fewer cases diagnostic thresholds inevitably altered. Consequently, there has been resurgence in the incidence of TB. Noted cases have increased from 5000 to 7000 in England and Wales since 1987 [5]. London in particular has seen TB double in the past decade, and now accounts for more than 40% of UK cases [5]. Skeletal involvement occurs in approximately 1-2% of patients with TB [2]. We report on three patients with different forms of skeletal TB – spondylitis, arthritis and osteomyelitis, in the hope of stimulating a high index of suspicion to facilitate early diagnosis.

Case 1

A 23 yr old site worker presented to the casualty with a four-week history of left knee pain and swelling associated with fevers. He had immigrated to the UK from India one year
earlier. He had undergone incision and drainage of a popliteal abscess in the ipsilateral knee in casualty two weeks prior to presentation.

On examination he had a temperature of 37.4°C and was tachycardic (120 beats/min). The affected knee was hot, swollen and exhibited generalised tenderness. Flexion was limited to 10° and caused acute pain.

Radiography (Fig. 1) showed a generalised reduction of joint space and gross soft tissue swelling. Markers of inflammation were high (Erythrocyte sedimentation rate 62 mm in first hour; C reactive protein 127 mg/l). Staphylococcus aureus cultured from a joint aspirate taken at the time of admission revealed sensitivity to flucloxacillin. However, microscopy for acid-fast Bacilli was negative.

Figure 1:
Plain radiograph of left knee. There is loss of joint space, juxta-articular osteopenia and bony erosion, particularly in the lateral compartment (marked arrow).
A diagnosis of septic arthritis was made and he underwent arthroscopy and washout of the knee, during which frank pus was drained. His symptoms improved following two weeks of splintage and intravenous therapy with flucloxacillin and fucidic acid and he was discharged home on oral antibiotics.

He presented five days later with worsening pain, stiffness and swelling in the knee. A further arthroscopic washout was performed and intravenous antibiotics were recommenced. Histology on synovial biopsies showed necrotising granulomatous inflammation strongly suggestive of a tuberculous aetiology. A presumptive diagnosis of tuberculous arthritis was made and antituberculous quadruple therapy consisting of rifampicin, isoniazid, pyrazinamide and ethambutol was started. The knee was placed on skin traction for four weeks, followed by functional knee bracing. Culture of the original washout fluid confirmed growth of Mycobacterium tuberculosis sensitive to rifampicin six weeks later. Chemotherapy was continued for six months.

At nine month follow-up there was no clinical or biological evidence of active TB. However, there were degenerative changes in the knee joint reflected by a reduced range of flexion (0-90°).

Case 2

A 28 yr old male refugee from Afghanistan was referred to the orthopaedic clinic with a four-week history of a painful, tender right forearm following a trivial injury. A radiograph taken in casualty at the time of the injury did not show any obvious abnormality.

On examination, his temperature was normal, and apart from a slight reduction in supination he had a full range of movements in the forearm, wrist and fingers. The patient was followed up in the outpatient clinic for continued pain. Subsequent radiography revealed a lytic area in the distal third of the ulna (Fig. 2). His markers of inflammation were raised (Erythrocyte
Figure 2: Six weeks after the first consultation there was a lytic lesion in the midshaft of the ulna with surrounding periosteal reaction (marked arrow)

sedimentation rate 28 mm in first hour; C reactive protein 15 mg/l). A Tc-\textsuperscript{99m} bone scan confirmed increased vascularity in the distal third of the ulna, with normal activity throughout the remainder of the skeleton. An open biopsy of the cystic lesion revealed frank pus. Histology showed focal necrotising granulomatous inflammation strongly suggestive of TB, although no acid-fast bacilli were seen on Ziehl-Nielsen staining.

The patient was commenced on anti-TB quadruple therapy for a total of six months. Four weeks later Mycobacterium tuberculosis was cultured. At ten months follow-up, there was a clinical and radiological improvement with normalisation of inflammatory markers.

Case 3

A 53 yr old Caucasian man was referred with a long standing history of intermittent low back pain which had progressively worsened in the preceding two years despite physiotherapy. More recently he had experienced weight loss, night sweats and fevers. He was born in the UK, had no TB contacts and possessed a BCG vaccination scar. There was no history of
foreign travel and no chronic illnesses. He was married and had the same sexual partner for over 20 years.

Clinical examination revealed mild upper lumbar tenderness with dysaesthesia over the L1 dermatome with a negative sciatic stretch. Examination of the power and reflexes was unremarkable. The inflammatory markers were raised (Erythrocyte sedimentation rate 46 mm in first hour; C reactive protein 68 mg/l). His HIV test was negative.

Plain radiography (Fig. 3) showed end plate changes in the L1/L2 disc suggestive of discitis. MRI of the spine (Fig. 4) showed destruction of L1-2 disc and the adjacent end plates with bilateral psoas abscesses extending from L1 to L3.

![Figure 3: Plain lateral radiograph of the lumbar spine showing loss of disc height and end plate changes suggestive of discitis (marked arrows).](image)

He underwent ultrasound guided aspiration of the psoas abscesses, which were auramine stain positive for acid fast bacilli. Subsequent culture of the pus confirmed Mycobacterium
tuberculosis. Treatment consisted of a three-point brace with quadruple antituberculous chemotherapy.

Figure 4: Subsequent MRI scanning showed bilateral psoas abscesses (marked arrows) originating from the L1/L2 disc and extending to L4.

At nine months follow-up, there was a resolution of symptoms with normalisation of inflammatory markers. Radiographs and MRI confirmed improvement of the tuberculous lesions with no progression of vertebral collapse.

Discussion

The resurgence of TB in the developed world poses a diagnostic challenge to clinicians in all specialities. In Asia, Africa and India, skeletal TB is usually encountered in children and young adults. In the western world there is a higher incidence in adults than in children [11]. In the UK, notified cases of extrapulmonary TB have risen by over 25% since 1993 [5]. Two of our cases were young immigrants from areas where TB is endemic. Both presented with a history of unexplained skeletal pain. The third patient of British indigenous origin presented at the age of 53, two years after the initial onset of symptoms. Halsey et al also reported a
similar age related discrepancy between immigrants and the indigenous Caucasian population in Britain [6].

Predisposing factors for TB include: recent TB contact, previous pulmonary TB, lower socio-economic class, heavy alcohol consumption, trauma and previous steroid therapy, and immunodeficiency [4,6]. Our first two cases were immigrants from endemic areas of low socio-economic class. However, the third patient in our report had no risk factors for TB. We speculate that this may have contributed to the delay in diagnosis.

The first case illustrates TB septic arthritis. Radiography of a tuberculous joint may reveal the characteristic features of Phemister’s triad [1]. This consists of juxta-articular osteopenia, peripherally located osseous erosions and gradual narrowing of the joint space. In the early stages of TB osteomyelitis, plain radiography may not show any abnormalities, as seen in our second patient. With disease progression radiographs reveal lesions that are radiolucent and well-defined, round or oval in shape and have variable amounts of sclerosis [8].

It has been proposed that MRI is the best diagnostic modality for spinal TB [3,10]. It is more sensitive than radiographs and more specific than CT in the diagnosis of spinal TB. It can provide the diagnosis of spinal TB four to six months earlier than conventional methods, and is useful in determining the spread of the disease to the soft tissues and spinal canal [3].

In our series, none of the patients had a tuberculin skin test at presentation. This can be a valuable diagnostic tool [4]. A positive skin test in the appropriate clinical setting supports the diagnosis leading to earlier chemotherapeutic intervention. This is useful for patients where initial tissue biopsy or pus is smear negative for acid fast bacilli. One further diagnostic obstacle exists in the delay in culturing mycobacteria, which may take up to six weeks. Anti-TB chemotherapy is the cornerstone in the management of all cases of skeletal TB [7,8,11]. It seems appropriate that in the right clinical setting, histological characteristics consistent with TB justify the initiation of presumptive treatment while the results of cultures are pending.
In conclusion, these cases highlight the challenge remaining to clinicians in the diagnosis of skeletal TB. In 1968, Walker stated that: ‘It is a truism to say that to make a diagnosis of TB one must consider it as a possibility’ [9]. This has been restated more recently by Rodger et al with regard to pulmonary TB [7]. We propose that the diagnosis of TB be entertained in any patient with skeletal pain. Histological and microbiological examination of synovial fluid or biopsy of the bone or synovium should be obtained early. In addition, a tuberculin skin test can be helpful. Timely treatment of TB can avoid extensive investigations, treatment delays and adverse long-term outcomes, such as serious bone and joint destruction and neurological deficits in the case of spinal TB.

References

5. From the Public Health Leadership Society website:


