Case Report

Isolated Tuberculous Monoarthritis Mimicking Oligoarticular Juvenile Rheumatoid Arthritis

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ABSTRACT. Isolated monoarthritis caused by Mycobacterium tuberculosis in the absence of clinical pulmonary disease is extremely rare in North America. After decades of consistent declines in incidence, a remarkable resurgence of tuberculosis (TB) is occurring in North America. It must always be considered in the differential diagnosis of chronic monoarthritis if devastating sequelae are to be avoided. We describe 2 cases of tuberculous arthritis in young children presenting with monoarthritis of the knee. The presumptive diagnosis in each case was oligoarticular onset juvenile rheumatoid arthritis (JRA). Each had an atypical course for JRA, with lack of response to intraarticular corticosteroid. The diagnosis of TB arthritis was made only with synovial biopsy. (J Rheumatol 2000;27:204–6)

Key Indexing Terms:
TUBERCULOUS ARTHRITIS
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Isolated monoarthritis caused by Mycobacterium tuberculosis in the absence of clinical pulmonary disease is extremely rare in North America. After decades of consistent declines in incidence, a remarkable resurgence of tuberculosis (TB) is occurring in North America. Chronic monoarthritis is a relatively common pediatric problem, and the differential diagnosis includes: juvenile rheumatoid arthritis (JRA), trauma, pigmented villonodular synovitis, foreign body synovitis, Lyme disease, viral arthritis, reactive arthritis, and arthritis associated with spondyloarthopathies, malignancies, sarcoidosis, tuberculosis, and other chronic infections. In the absence of systemic manifestations or other clinical clues it may be difficult to determine the specific cause of monoarthritis in children. JRA is by far the most frequent cause of chronic monoarthritis in our community and is the usual provisional diagnosis. Two children with monoarthritis unresponsive to usual therapy for JRA were found to have tuberculous arthritis.

CASE REPORTS

Case 1. In March 1998, a 2.5-year-old Caucasian girl presented with an 8 month history of swelling of one knee following minor trauma. She had mild pain, morning stiffness, and swelling that waxed and waned but never resolved completely. She limped continuously and developed a fixed flexion deformity. There was no history of fever, cough, or weight loss. There was no history of travel to an endemic TB area and no contact with anyone known to have TB. She had not received bacillus Calmette-Guérin (BCG). When seen in our clinic, she was well grown (height 50th percentile and weight 25th percentile) and the general physical examination was entirely normal. Her left knee showed a large effusion and 10° fixed flexion deformity with pain at the end range. There was no skin erythema or bony tenderness. Laboratory investigations including a test for antinuclear antibodies (ANA) were normal apart from an elevated erythrocyte sedimentation rate (ESR) (53 mm/h). Radiographs of the knee showed a large effusion with soft tissue swelling, but no osteopenia or joint space loss. She was given naproxen for the presumptive diagnosis of oligoarticular onset JRA.

One month later, the naproxen was discontinued because she developed a rash on her legs and there was no improvement in her symptoms. She had considerable pain and stiffness, and she was prescribed prednisone 5 mg bid. Because of the history of minor trauma prior to onset and because of the poor response to initial treatment, a magnetic resonance image (MRI) was performed to look for other causes for the knee swelling. The MRI showed nodular synovial thickening with large effusions and numerous “loose bodies.” Gram stain of synovial fluid (SF) showed no organisms and the culture was negative after 48 h of incubation. In spite of intraarticular triamcinolone hexacetonide therapy on 2 occasions, 2 months apart, and the addition of prednisone and tolmetin sodium, there was no improvement of her symptoms. Arthroscopy and synovectomy yielded straw colored fluid with a large number of yellowish “rice bodies” and villous synovitis. The histologic examination showed noncaseating granulomatous synovitis. The culture of synovial tissue grew Mycobacterium tuberculosis sensitive to all the usual agents. A PPD skin test was reactive with 12 mm induration. Her chest radiograph was normal. Both parents and her 3 siblings had nonreactive PPD skin test.

She was started on triple chemotherapy (INH, rifampin and pyrazinamide). Pyrazinamide was discontinued after 3 months and the plan was to continue the other anti-TB medications for one year. She improved significantly. Three months after starting the TB treatment there were no joint effusions, and radiograph of the knee showed no bony lesions.

Case 2. In June 1998 a 6-year-old boy who had recently immigrated with his family from Somalia presented with an 11 month history of pain and swelling in his left knee that was preceded by minor trauma. He complained of knee pain that was worse at night and morning stiffness lasting 30 min daily. He was seen by several physicians including the family doctor and an orthopedic surgeon, and had started taking tolmetin sodium, and although this improved his symptoms, it was discontinued because of abdominal pains. Although TB

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forms of chronic arthritis. SF is inflammatory; white cell the SF characteristics may be indistinguishable from other fever. The usual insidious chronic course of TB arthritis and arthritis may cause pain, stiffness, effusion, and low grade particularly in areas where TB is not endemic. Clinically, TB States infected with tubercle bacillus is unknown because the patients. Delayed diagnosis of TB arthritis is not unusual. In most children with TB arthritis (including both our cases) there were no pulmonary symptoms and the chest radiographs were normal. Prior BCG vaccination is not a contraindication to tuberculin testing and, in general, a reactive area of > 10 mm in a BCG vaccinated child probably indicates infection and necessitates further diagnostic evaluation and preventive chemotherapy.

Two other cases of TB arthritis in children were seen in our hospital: a 15-year-old Asian girl who is a recent immigrant presented with a 5 month history of left hip pain. A radiograph of her hip showed loss of articular space and changes at proximal femur suggestive of osteomyelitis. Large hilar lymph nodes with calcification were seen on her chest radiograph. Her PPD skin test was strongly positive. She was started on therapy for the presumptive diagnosis of TB. She is not living in the country now and no further followup is possible. The 4th patient was a 2-year-old North American Indian boy with Ponce’s disease who presented with increasing back pain and sterile effusions of the knee and ankle, who was found to have active vertebral tuberculosis and a perivertebral abscess.

Treatment of bone and joint tuberculosis requires prolonged combination therapy, initially with isoniazide, rifampin, and pyrazinamide for the first 2 months, followed by isoniazide and rifampin administered once a day or twice a week under direct observation for a period of 9–12 months. Shorter courses of chemotherapy (6 month regimens) are associated with higher failure rate, especially if surgical intervention has not been undertaken.

Arthritis with TB may be a result of direct infection of the joint or a “reactive” arthritis secondary to visceral disease (i.e., Ponce’s disease). Arguably much of the joint damage in the former situation is mediated by activated lymphocytes, and this may be modified by steroids. Adjuvant treatment with corticosteroids is controversial, except in the treatment of tuberculous meningitis. It may also be considered in the treatment of miliary disease, endobronchial disease, and pleural and pericardial effusions. There are no studies that evaluate the role of systemic or intraarticular corticosteroid in tuberculous arthritis. In both our patients, intraarticular corticosteroid was used before the diagnosis of TB was established. Despite the considerable delay in the diagnosis (18 months), there was no clinical or radiological evidence suggestive of disease extension or complication and the outcome in both cases, once chemotherapy was started, was good. The role of intraarticular corticosteroids in preventing or exacerbating damage in TB arthritis remains unclear.
In an era of increasing incidence of tuberculosis in children, especially the extrapulmonary form, tuberculous arthritis should be considered in the differential diagnosis of monoarthritis. A history of TB exposure should be sought; and it may be that even in the absence of risk factors PPD application should be considered. In a patient with presumed JRA with a monoarticular course, TB should be considered if there is an inadequate response to usual treatment. Our 2 patients were fortunate not to have destructive joint disease, in spite of a delayed diagnosis.

REFERENCES