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Acute arthritis associated with acute Q fever

Sirs,

In clinical practice rheumatologists often see patients with undifferentiated arthritis. In many of these cases synovitis remains unclassified after an adequate period of follow-up (1). Acute Q fever is known to be a cause of acute pneumonia, hepatitis or self-limited fever. In addition, a variable degree of arthralgia and myalgia is also common. However, acute arthritis as the predominant and presenting manifestation of acute Q fever is exceptional (2). In this report, we describe a patient presenting with acute arthritis with serological evidence of acute Q fever which was successfully treated with doxycycline.

A 33-year-old female farm worker presented at our outpatient clinic because of pain and swelling in her right knee that had begun 6 days earlier. She denied other symptoms and, apart from the presence of severe synovitis in the right knee, the rest of the physical examination was unremarkable. Full blood cell count and hepatic and renal functions tests were normal. The erythrocyte sedimentation rate was 14 mm/hr. C reactive protein was increased (37.5 mg/L; normal < 5 mg/L). Antinuclear antibodies and rheumatoid factor were negative. Interestingly, arthrocentesis yielded an inflammatory synovial fluid (SF) (7,446 leucocytes/mm^3) with 70% macrophages, 20% lymphocytes and 10% polymorphonuclear cells (Fig. 1). The glucose level in the synovial fluid was normal (61 mg/dl; serum glucose level 87 mg/dl) and no microcrystals were seen. Cultures of the SF were also negative. Tuberculin skin test and serological tests for syphilis, brucella, yersinia, salmonella, cytomegalovirus, mononucleosis, rubella, toxoplasma, mycoplasma, and hepatitis B and C were negative. Chest radiograph was normal.

However, serology for *Coxiella burnetii* (immunofluorescence) was positive; phase II IgG positive at 1/640 and IgM positive. Antibiotic therapy with doxycycline (100 mg/bid) was given for 14 days. The patient experienced a rapid improvement of the synovitis within the first 72 hours after the onset of the antibiotic therapy. In a further study performed 3 months later, the serology for *C. burnetii* showed the following results: phase II IgG positive at 1/80 and IgM negative. Six months after the diagnosis of acute Q fever she remained asymptomatic.

This report describes a case of Q fever presenting with acute arthritis which often is not diagnosed at the right time if serological tests are not considered for early diagnosis.

Q fever is caused by an obligate intracellular parasite, *C. burnetii*, acquired through inhalation of infected dust or ingestion of unpasteurized milk from subclinically infected farm animals. A definite diagnosis of acute Q fever depends on the demonstration of an IgM antibody response. Chronic osteoarticular infections associated with Q fever have been reported (3). In acute Q fever, arthralgia is common. However, acute arthritis as presenting manifestation of acute Q fever has not been commonly reported (2, 4-7).

To the best of our knowledge, there are only 6 cases described in the literature (2, 4-7). Three of them presented as monoarthritis (2 hip, 1 knee), and the other 3 as polyarthritis. In only one of these patients were the characteristics of the SF described (5). As in our case, a predominance of macrophages was observed. Direct infection was confirmed by immunofluorescence in the SF in only one patient presenting with acute arthritis (5). In this regard, although it is not a definitive criterion, in our case the rapid response to antibiotic therapy might be supportive evidence of an infectious arthritis. However, the rarity of macrophagic synovial effusions make it difficult to determine whether this feature may be a useful indicator of the diagnosis of Q fever arthritis. Descriptions of further cases will be required to confirm whether the predominance of macrophages in the synovial fluid might be a clue for this particular infectious arthritis. Finally, we do not know whether HLA determination would have given additional information concerning the possible immunogenetic predisposition toward such a type of articular reaction to the infection.

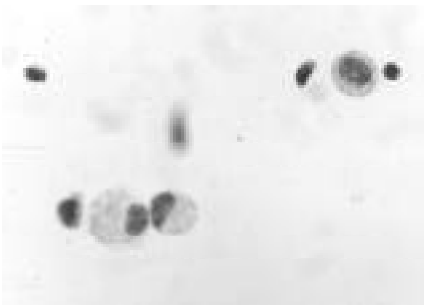

Fig. 1. Presence of histiocytes with eccentric nuclei and clear and microvacuolated cytoplasms along with some lymphocytes and polymorphonuclear cells in the synovial fluid of our patient (Giemsa x 400).

In summary, the present case may be a good example of the necessity of using serological tests in those patients presenting with acute arthritis of unclear etiology from endemic areas for Q fever. Indeed, even in atypical cases like this one, where pulmonary and hepatic findings were absent, a positive test for Q fever might allow us to make a diagnosis of this condition.

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