Correspondence

Rheumatic manifestations of brucellosis

SIR, We read with interest the article by Norton on the rheumatic manifestations of brucellosis.¹ We reported our experience with 304 patients infected with *Brucella melitensis* in 1982.² Recently we have completed a prospective evaluation of 194 additional patients. Of these 498 patients, 180 (36%) presented with arthritis. In contrast to Norton's findings, sacroiliitis and peripheral arthritis were the most common forms of joint involvement, accounting for 90% of all the arthritis cases. Spondylitis was seen in only 18 patients (10%). As reported by others³⁻⁴ these patients were predominantly middle aged and older males with chronic brucellosis.

The discrepancy can be explained by the different age distribution of the populations studied. Only one of the 55 patients reported by Norton was younger than 15, while in our group 354 patients (71%) were younger than 35 and 125 (25%) younger than 15.

We agree with Norton in that immune abnormalities occur in human brucellosis, particularly during periods of active disease. Low titres of antinuclear antibodies were present in 25% and rheumatoid factor in 37.5% of a group of patients evaluated by us.⁵ Circulating immune complexes were detected by the Raji cell assay in 91.5%.

As in other infectious diseases⁶⁻⁷ infectious and reactive arthritis occur in human brucellosis. In our series, using a selective culture medium for brucella,⁸ one third of the patients had sterile synovial fluids fulfilling the definition of reactive arthritis.⁹ Furthermore, the arthritis was non-destructive and resolved spontaneously in most of these patients, an event not commonly seen in infectious arthritis.¹⁰ Interestingly, in one of the patients described by Norton the arthritis resolved after an intra-articular injection of corticosteroids.¹ The immune abnormalities described above strongly suggest that an immune mechanism plays a part in the arthritis of these patients.

Merck Sharp & Dohme,
Research Laboratories,
Rahway, NJ 07065,
USA

TOMÁS S. BOCANEGRA

Universidad Peruana,
Cayetano Heredia,
Lima, Peru

EDUARDO GOTUZZO
OSWALDO CASTAÑEDA

The University of Alabama
at Birmingham,
Birmingham, ALA 35294,
USA

GRACIELA S ALARCÓN

University of South Florida,
Tampa, FLA 33612,
USA

LUIS R ESPINOZA

References

- 1 Norton W L. Brucellosis and rheumatic syndromes in Saudi Arabia. *Ann Rheum Dis* 1984; **43**: 810-5.
- 2 Gotuzzo E, Alarcón G S, Bocanegra T S, *et al*. Articular involvement in human brucellosis: a retrospective analysis of 304 patients. *Semin Arthritis Rheum* 1982; **12**: 245-55.
- 3 Lifeso R M, Harder E, McCorkell S J. Spinal brucellosis. *J Bone Joint Surg [Br]* 1985; **67B**: 345-51.
- 4 Ariza J, Gudiol F, Valverde J, *et al*. Brucellar spondylitis: a detailed analysis based on current findings. *Rev Infect Dis* 1985; **7**: 656-64.
- 5 Gotuzzo E, Bocanegra T S, Alarcón G S, *et al*. Humoral immune abnormalities in human brucellosis. *Allergol Immunopathol Madr* 1985; **13**: 417-24.
- 6 Carroll W L, Balistreri W F, Brill R, Parish R A, Greenfield D J. Spectrum of salmonella-associated arthritis. *Pediatrics* 1981; **68**: 717-20.
- 7 Goldenberg D L. 'Post infectious' arthritis. New look at an old concept with particular attention to disseminated gonococcal infection. *Am J Med* 1983; **74**: 925-8.
- 8 Ruiz-Castañeda M. Improved equipment for the isolation of brucella, salmonella, etc. by blood culture. *Bol of Sanit Panam* 1957; **42**: 564-5.
- 9 Aho K, Leirisalo-Repo M, Repo H. Reactive arthritis. *Clin Rheum Dis* 1985; **11**: 25-40.
- 10 Goldenberg D L, Reed J I. Bacterial arthritis. *N Engl J Med* 1985; **312**: 764-71.

Further observations upon HLA-B27, *Yersinia enterocolitica*, and ankylosing spondylitis

SIR, Some time ago we reported that the observations we had made, in our search for evidence for the molecular mimicry hypothesis of ankylosing spondylitis (AS), on serological cross reactions between *Yersinia enterocolitica* types 0:3, 0:9, and NCTC 10460 and lymphocytes from HLA B27⁺AS⁺ subjects, with a haemagglutination technique, were in fact nothing more than a demonstration of an anti-yersinia factor present in normal human sera, quite distinct from a lymphocytotoxicity causing antibody.¹ We concluded that this did not rule out the possibility of the existence of some other undefined antigenic factor common to *Y enterocolitica* and HLA-B27. Subsequently we did detect such a factor in *Y enterocolitica* type 0:9, which was not present in type 0:3, and in lymphocytes from B27⁺AS⁺ subjects as opposed to B27⁺AS⁻ ones, by the use of an absorption/haemagglutination technique.² The numbers examined, however, were too small for us to draw any conclusions about the existence of any serological differences between the two groups of lymphocytes.

Later we found that the relevant class of antibody in these tests was IgG. We titrated erythrocytes coated with *Y enterocolitica* 0:9 against homologous antiserum, i.e.,