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Septic arthritis due to *Bacteroides fragilis*

SIR, We read with interest the report of Hart *et al.*¹ of a case of *Bacteroides fragilis* septic arthritis of the wrist in a patient with rheumatoid arthritis (RA). They made the claim that this was probably the first case report of wrist joint involvement due to this organism. By coincidence we reported a similar case of septic polyarthritis due to *Bacteroides fragilis* in a patient with RA earlier in 1982.² This patient had bacteroides sepsis in 9 joints including both wrists, and also responded well to metronidazole treatment.

A joint infection in a patient with RA should not only be considered when one joint suddenly flares up, but should also be borne in mind with a generalised flare-up, and anaerobic cultures should be done after joint aspirations as a routine investigation.

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Polymorphonuclear function in Behçet's syndrome

SIR, The recent report by Fordham *et al.*¹ has drawn attention to the role of polymorphonuclear leucocytes (PMN) in the pathogenesis of Behçet's disease. While the aetiology of Behçet's disease remains unknown, immune complex-induced vasculitis with associated systemic inflammatory lesions are recognised key events in its pathogenesis. The occurrence of systemic inflammatory lesions has prompted studies of PMN function in Behçet's disease. Several investigators have found an increased PMN chemotactic response,¹⁻⁵ although a reduced chemotactic response to inulin-activated serum and a normal chemotactic response to casein have also been described.⁶ Phagocytic and adherent properties of Behçet PMN are normal,¹ while leucocyte oxygen consumption in the resting state and after stimulation by phagocytosis of *Staphylococcus albus* are diminished.³ Spontaneous nitroblue tetrazolium reduction

is normal, whereas reduction after endotoxin stimulation is depressed.³

These studies have prompted us to study the production of the toxic superoxide radical O₂⁻ in response to stimulation with the chemotactic peptide N-formylmethionyl-leucyl-phenylalanine (FMLP) in the PMN isolated from a 27-year-old female suffering from Behçet's disease of 8 years' duration. She had a present or past history of mouth and genital ulcerations, arthritis, and vasculitic skin lesions, thus fulfilling 3 major and one minor diagnostic criteria for Behçet's disease.⁷ At no time did she have eye or neurological involvement. Current treatment consisted of 15 mg of prednisone and 20 mg of piroxicam daily. For the assay of O₂⁻ produced by the activated PMN, a ferricytochrome C reduction procedure was used.⁸ The production of O₂⁻ was measured on 2 separate occasions comparing results with those obtained in 2 concurrently studied age and sex-matched healthy controls and with the normal ranges of the rate and quantities of O₂⁻ production earlier established in our laboratory for 33 normal controls.⁹ After prewarming the isolated PMN for 30 minutes 6-8 duplicate assays of O₂⁻ production were performed during an approximate 3-hour period. Results are as follows:

	Rate of O ₂ ⁻ production (nmol O ₂ ⁻ / 10 ⁶ cells/min)	Total quantity of O ₂ ⁻ produced (nmol O ₂ ⁻ / 10 ⁶ cells)
Behçet patient (1st experiment)	9.42, SD 3.68	22.0, SD 4.58
Concurrent control	9.46, SD 2.33	23.9, SD 3.13
Behçet patient (2nd experiment)	9.0, SD 0.42	21.5, SD 0.74
Concurrent control	9.11, SD 0.81	23.08, SD 2.08
Normal controls (33)*	8.90, SD 1.7	22.8, SD 5.7

In this Behçet's patient both the rate as well as the total quantity of O₂⁻ produced in response to stimulation with FMLP was clearly within normal limits when compared with both concurrent and previously tested controls. Abnormal production of toxic O₂⁻ radical, a facet of PMN function, which to the best of our knowledge has not been studied before, may therefore not contribute to the generation of the systemic inflammatory lesions encountered in Behçet's disease.

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