LETTERS TO
THE EDITOR

Amyloidosis in rheumatic diseases

Sir: I read with interest the excellent review on amyloidosis in rheumatic diseases by Dhillon et al.1

In Israel we have had an opportunity to witness the outstanding results obtained with colchicine in preventing amyloidosis in familial Mediterranean fever2 and the relative safety of this drug. Furthermore, there are some reports on the beneficial effect of colchicine in primary amyloidosis3 and secondary amyloidosis of ulcerative colitis.4 There is also experimental evidence that colchicine inhibits amyloid synthesis.5

As stated by Dhillon, children with systemic onset, juvenile chronic arthritis and persistent disease activity are at high risk for developing amyloidosis. I feel, therefore, that, at least in this high risk group, treatment with colchicine should be added in an attempt to prevent this life threatening complication.

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Propionibacterium acnes in a spondylitis with palmoplantar pustulosis

Sir: Edlund et al have recently reported the presence of Propionibacterium acnes in seven of 15 biopsy specimens taken from affected bones in 15 patients with sternocostoclavicular arthro-osteoarthritis in association with palmoplantar pustulosis.1 This prompts us to relate a case with the same dermatological condition which also suggested that this germ might involve osteoarticular tissue.

An HLA-B27 negative 45 year old woman suddenly in October 1986 had dorsolumbar pain and stiffness simultaneously with a pustulosis of the soles and palms but without fever or chills. It should also be noted that—like her daughter—the patient had had psoriasis. Erythrocyte sedimentation rate was 21 mm/1st h and white cell count 7.8 x 10^9/l. The pain worsened and the patient lost 8 kg.

The first x rays were taken in July 1987, showing a T8-9 spondylosiscitis associated with sclerosis and collapse of T9 (fig 1). A roentgenogram of the lumbar spine taken in January 1989, because of a local painless stiffness, showed an L4-5 spondylosiscitis and osteosclerosis predominating in L5.

Figure 1: Lateral tomogram of the segment T7-10 showing erosion of vertebral plates T8-9 and underlying osteosclerosis, mainly on T9.

Each of these two radiological observations was followed by a punch biopsy; examinations of bone specimens showed an old and active chronic inflammatory remodelling, the features of which suggested an infectious process (fig 2). In the first biopsy specimen, taken before any antibiotic treatment, cultures on anaerobic media (Columbia blood agar and Schaedler broth) showed numerous colonies of typical Propionibacterium acnes. In the second biopsy specimen, taken after prolonged treatment with amoxicillin clavulanic acid, cultures were sterile. The following points suggested strongly that the dorsal spondylosiscitis was due to Propionibacterium acnes: the histological image was evocative of osteomyelitis and the germs did not seem to be related to a skin contamination (the punch biopsy was performed after a surgical skin incision and later the growth of numerous colonies of the germ argued against such a contamination).

In our case, also, attention should be paid to a possible link with sternocostoclavicular arthro-osteoarthritis (also called sternocostoclavicular hyperostosis),2 though extraspinal skeletal changes were not found. In addition to palmoplantar pustulosis3 and a history of psoriasis4, the above spondylosis groups together some features which have been found in classical cases of sternocostoclavicular hyperostosis—namely, roentgenological erosions of the vertebral plates associated with bone sclerosis,5-7 histological changes suggesting an infectious origin,8 and a presumed infection with Propionibacterium acnes.1

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Figure 2: Biopsy specimen from the T8-9 region. Haematoxylin and eosin. (a) Bone marrow inflammatory fibrosis with signs of an old remodelling in adjacent bone trabeculae indicated by numerous cement lines. (b) Detail of the bone marrow space: chronic inflammation is shown by numerous lymphocytes and plasmaocytes.
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