

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-osteitis 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 $7.8 \times 10^9/l$. The pain worsened and the patient lost 8 kg.

The first x rays were taken in July 1987, showing a T8-9 spondylodiscitis 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 spondylodiscitis and osteosclerosis predominating in L5.

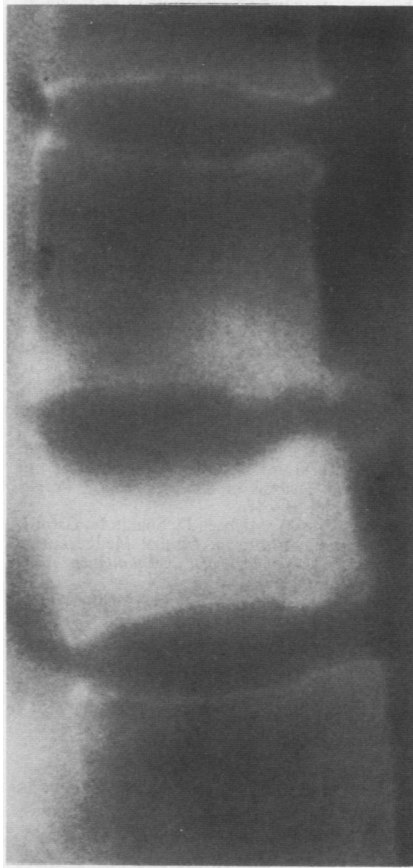


Figure 1: Lateral tomogram of the segment T7-10 showing erosion of vertebral plateau T8-9 and underlying osteosclerosis, mainly on T9.

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 spondylodiscitis 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-osteitis (also called sternocostoclavicular hyperostosis),² though extraspinal skeletal changes were not found. In addition to palmoplantar pustulosis³ and a history of psoriasis², the above spondylitis groups together some features which have been found in classical cases of sternocostoclavicular hyperostosis—namely, radiological erosions of the vertebral plateaus associated with bone sclerosis,^{2 4-7} histological changes suggesting an infectious origin,^{4 7 8} and a presumed infection with *Propionibacterium acnes*.¹

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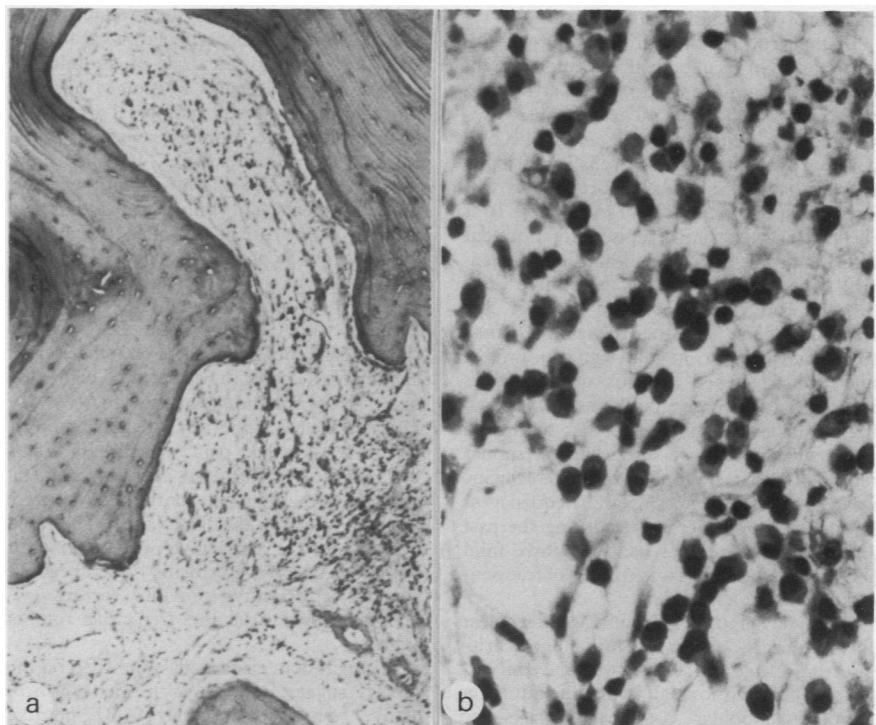


Figure 2: Biopsy specimen from the T8-9 region. Haematoxylin and eosin. (a) Bone marrow inflammatory fibrosis with signs of an old remodelling in adjacent bone trabeculae indicated by numerous cement lines. (b) Detail of the bone marrow space: chronic inflammation is shown by numerous lymphocytes and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

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Cricothyroid arthritis in a child with familial Mediterranean fever

Sir: We describe for the first time the occurrence of cricothyroid arthritis in a girl who first presented with migratory polyarticular arthritis but eventually developed the classical features of familial Mediterranean fever.

A 9 year old Palestinian Arab girl was admitted in January 1979 with fever and migratory polyarticular arthritis of the large joints. The heart was normal. The erythrocyte sedimentation rate was 110 mm/h and the antistreptolysin O titre was 400 Todd units. A diagnosis of acute rheumatic fever was made and treatment was started with secondary prophylaxis. During the following six years she had several episodes of arthritis, which were interpreted as recurrence of acute rheumatic fever due to irregular prophylaxis, and occasional fever and abdominal pain.

In January 1985 the girl was admitted with fever and arthritis of both elbows and the right wrist. Next morning she developed arthritis of the cricothyroid joint. The diagnosis was verified by indirect laryngoscopy. She also developed arthritis of the interphalangeal joints of both hands. She became better after five days of aspirin treatment. Two months later she had another similar episode of transient arthritis of the cricothyroid and interphalangeal joints. During the following three years the girl had several episodes of fever and abdominal pain, with the frequency progressively increasing to one to two attacks a week. She also developed arthritis of the ankles associated with erysipelas-like erythema. Family history disclosed that her mother, a maternal aunt, and two sisters had had similar recurrent episodes. Prophylaxis with colchicine was effective in decreasing the frequency of febrile and painful episodes; during the past 12 months the girl has had only three mild abdominal attacks and one episode of transient arthritis of the left ankle.

The synovial attack of familial Mediterranean fever typically appears as acute monoarthritis affecting a large joint of the lower extremity.¹⁻⁴ Involvement of the small joints, including the temporomandibular, sternoclavicular, and metatarsophalangeal joints, has been described in a minority of patients with familial Mediterranean fever,¹⁻⁴ whereas involvement of the interphalangeal joints has been reported

to be most unusual.¹ Cricothyroid arthritis in the course of familial Mediterranean fever has not been previously described.

The presentation with migratory polyarticular arthritis, the involvement of the interphalangeal joints, and the long period before the appearance of the classical manifestations of familial Mediterranean fever are other unusual features in this case.

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Trauma and seronegative spondyloarthropathy

Sir: We would like to offer what we believe to be a necessary reply to Professor Panayi's letter published in the *Annals*.¹ Professor Panayi considers that in the two B27 positive patients we described, who developed peripheral arthritis immediately after trauma,² physical injury and the onset of peripheral arthritis were only coincidental. The first case represents, in his opinion, a reactive arthritis following gastroenteritis, and the second case, arthritis of the knees begun by chance after the trauma.

If other articles on this subject³⁻⁶ are not taken into account this may seem to be the most logical conclusion, partly because no evidence of causality may be produced other than the immediate onset of peripheral arthritis after trauma, and the lack of an infective trigger. Wisnieski³ and Masson *et al*⁴ have reported other cases of peripheral arthritis in B27 positive subjects immediately after physical injury. In some of these, like our patient 1,² there was also urethritis with negative urethral smears and culture, in addition to arthritis. Our patient also had a diarrhoea with negative stool culture, which subsided in two days without any treatment. In 1982 Jacobs *et al* reported that five of their 58 patients with juvenile onset B27 positive spondyloarthropathy had a trauma severe enough for a doctor to be consulted before the onset of peripheral arthritis.⁵ In 1988 we reported the cases of two B27 positive subjects who had never had pain to peripheral joints before, but developed an erosive peripheral arthritis of the right hip shortly after a severe physical injury to the same joint.⁶ The rapid evolution of the destructive process, which is not usual in erosive arthritis of seronegative spondyloarthropathy, provides further evidence in favour of the triggering role of trauma.

In conclusion, the articles published on the subject suggest that as in psoriatic arthropathy,^{7,8} physical injury may, in B27 positive subjects, trigger the onset of a peripheral arthritis predominantly affecting the injured joints. We hope that others will report similar cases and perform studies on the synovial fluid

and blood of patients with B27 associated peripheral arthritis following trauma, in an attempt to understand the pathogenetic mechanisms. We appreciate the comments of Professor Panayi and thank him for drawing attention to this topic of seronegative spondyloarthropathy.

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Chondroprotective drugs and osteoarthritis

Sir: I read with interest the leader article by Doherty on 'Chondroprotection by non-steroidal anti-inflammatory drugs' published in the *Annals*.¹

Although I am in general agreement with the views expressed by Dr Doherty, he raised some issues which I consider deserve further comment.

In his article Dr Doherty questions the relevance of certain laboratory derived data on non-steroidal anti-inflammatory drugs (NSAIDs) to their clinical use in osteoarthritis. He considers the standard for assessing these drugs is the long term symptomatic and functional improvement in patients 'rather than individual biochemical or structural characteristics'. It should be noted, however, that most NSAIDs are also powerful analgesics and may effectively relieve the symptoms of osteoarthritis without necessarily influencing its progression. Pain relief and improvement of joint mobility are thus inadequate criteria for distinguishing between an NSAID acting only as an analgesic and an NSAID which is also positively influencing the underlying osteoarthritic disease. More objective methods of clinical assessment of patient response to drug treatment are therefore required before this matter can be resolved. Such methods are presently under investigation, and promising findings have been reported with biochemical markers of cartilage breakdown in synovial fluid²⁻⁴ and serum,^{5,6} x ray microfocus (Buckland-Wright *et al*, unpublished data)