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Arthropathy of calcium pyrophosphate deposition disease and of haemochromatosis

SIR, The recent report by Bourqui *et al.*¹ documents again the rather specific structural joint damage seen in the hand and the wrist in patients with calcium pyrophosphate dihydrate (CPPD) crystal deposition disease. I would like to make several additional observations.

As noted in our previous article² as well as in one by Martel *et al.*,³ it is the distribution and the morphology of the articular abnormalities, especially in the hand and wrist, that are distinctive in radiographs in patients with CPPD crystal deposition disease. Although Bourqui *et al.* are indeed correct in their emphasis of the second and third metacarpophalangeal joints in this disease, their description of wrist alterations is incomplete. CPPD crystal deposition disease demonstrates a remarkable predilection for the radiocarpal joint in the wrist. Interosseous space narrowing between the distal radius and the scaphoid is most characteristic. Furthermore, the scaphoid moves proximally, leading to scalloped erosion of the distal radius, and the lunate migrates distally, with joint space narrowing mainly identified between it and the capitate. Separation, or dissociation, of the scaphoid and the lunate is subsequently observed, perhaps related to crystal deposition in the interosseous ligament.

At these articular locations as well as the metacarpophalangeal joints (and elsewhere) several morphological characteristics distinguish the arthropathy of CPPD crystal deposition disease from osteoarthritis: (1) multiple (and often large) subchondral rarefactions or cysts; (2) irregularity or 'crumbling' of the articular surface; (3) severe progressive alterations that resemble those occurring in neuroarthropathy; (4) variable osteophyte formation; (5) single or multiple intra-articular osteocartilaginous bodies.

In their article Bourqui *et al.* acknowledge prior reports that indicate the similarity of the arthropathy of CPPD crystal deposition disease to that of haemochromatosis. We have recently compared the radiographic abnormalities in these two diseases⁴ and have recognised differences between the two. In haemochromatosis, as compared with CPPD crystal deposition disease, findings include more prevalent narrowing of the metacarpophalangeal joint spaces, including those in the fourth and fifth digits, peculiar hook-like osteophytes on the radial aspect of the metacarpal heads, and less prominent separation of the scaphoid and the lunate. These radiographic differences indicate that the arthropathy of haemochromatosis is related to factors additional to the presence of CPPD crystals.

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Arthritis and angioimmunoblastic lymphadenopathy

SIR, Angioimmunoblastic lymphadenopathy is a lymphoproliferative condition of unknown aetiopathogenesis.¹ Typical features include lymphadenopathy, hepatosplenomegaly, cutaneous rash, pleuropulmonary lesions, and hypergammaglobulinaemia. Both arthralgia and arthritis have also been reported occasionally.²⁻⁴

A 49-year-old woman presented with fever, anorexia, weight loss, and symmetrical polyarthritis. These symptoms had been present for the last 11 months. There was no former history of viral infection or drug ingestion. Physical examination revealed a chronically ill patient with temperature of 39°C. Numerous small, movable, rubbery and nontender lymph nodes were palpated in the cervical region. Synovial thickening, warmth, and tenderness of wrist, elbow, and knee joints were noted, as well as moderate bilateral knee effusion. The ESR was 100 mm/h. Protein electrophoresis showed a polyclonal hypergammaglobulinaemia. The latex test for rheumatoid factor and the antinuclear antibody test were negative.

Aspiration of the left knee revealed a clear fluid with a moderate number of leucocytes, 67% of which were polymorphonuclears. Synovial fluid protein, glucose, and C3 were within normal ranges. Culture was sterile and crystals were not observed. A radiograph of the left wrist showed periarticular osteoporosis and narrowing of the radiocarpal joint space with irregular subchondral erosions.

A lymph node biopsy was performed. Histological lesions consisted of diffuse effacement of nodal architecture with a pleomorphic cellular proliferation in which immunoblasts, plasma cells, histiocytes, and lymphocytes predominated. In addition, vascular proliferation—arborising blood vessels—and prominent eosinophilic PAS-positive interstitial material were seen.

Treatment with prednisolone 80 mg per day and indomethacin 100 mg per day was begun. Within three weeks the patient had defervesced. Lymph nodes diminished in size and synovitis resolved. After six months of treatment the patient was asymptomatic.

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Bacterial confirmation of gonococcal arthritis

SIR, The case report 'Moraxella infectious arthritis: first report in an adult' by Rosenbaum *et al.*¹ describes the unexpected culture of a species of *Moraxella* from the synovial fluid of a 42-year-old woman who was thought on clinical grounds to be suffering from gonococcal septic arthritis. However, the criteria used for identification were not sufficiently stringent to exclude the possibilities that the organism might in fact have been a strain of *Neisseria gonorrhoeae* or of *Branhamella catarrhalis*.

The isolate was described repeatedly as being a Gram-negative diplococcus. Lautrop² notes that the short, plump Gram-negative rods of *Moraxella* may approach a coccoid form, but stresses nevertheless that the rod-like shape of the genus is an important characteristic distinguishing it from the Gram-negative cocci within the family *Neisseriaceae*. The apparent absence of any rod-like forms in Gram-stained smears of the isolate is indicative of *Neisseria* or *Branhamella*, not *Moraxella*.

The isolate was reported as not utilising any sugars. We assume that the sugars tested were incorporated into the cysteine (*sic*) trypticase agar mentioned in the report, and that they included glucose, maltose, lactose, and sucrose. *B. catarrhalis* does not utilise sugars, and further tests may have indicated that the isolate belonged to this species. However, a more probable explanation is that the isolate was a strain of *N. gonorrhoeae* that failed to utilise glucose in cystine trypticase agar, similar to strains described by White and Kellogg.³

Perhaps the most disturbing aspect of the bacteriological investigations was the use of the API 20E system, designed primarily for the identification of members of the *Enterobacteriaceae*, for the characterisation of a fastidious Gram-negative coccus. In our laboratory known strains of *N. gonorrhoeae* (including a WHO reference strain) were iden-

tified as *Moraxella* sp. by this system. Other procedures for identification of *N. gonorrhoeae*, such as the use of specific fluorescent antibody or coagglutination tests, were apparently not carried out on the isolate.

In their introductory paragraph Rosenbaum *et al.* note the similarities between *Neisseria gonococcus* (*sic*) and *Moraxella* species, and they conclude their report with a comment on the need for bacterial culture to confirm a clinical diagnosis of suspected gonococcal arthritis. We emphasise that the identification procedures must include methods that are appropriate to the class of organism suspected, and that, if any doubt exists, confirmation of identity by a suitable reference laboratory should be obtained.

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Immune deposits at the dermoepidermal junction in patients with rheumatoid arthritis

SIR, The deposition of immunoglobulin and complement components at the dermoepidermal junction in normal skin is well described in systemic lupus erythematosus (SLE).¹ Studies in rheumatoid arthritis (RA) have yielded conflicting results with a frequency of 0-50% reported.²⁻⁶ casting doubt on the diagnostic specificity of the 'lupus band' test. Reasons for such variation are not clear from inspection of the studies.

We have determined the prevalence of immune deposits at the dermal junction in 45 patients with RA. The study was designed to establish factors which might influence the development of deposits. Thus skin was sampled from the forearm of all patients, and from 34 an additional biopsy was taken from the leg to determine regional variation. Patients were studied as a group and by subdivision into those with articular disease alone and those with extra-articular manifestations. The influence of serological factors and of drug therapy was also examined. For comparison biopsy specimens were also taken from the arm and the leg of 14 patients with SLE and a miscellaneous group of 22 control subjects and patients with other rheumatological disorders. Sections of skin were processed for routine histological examination and by a direct immunofluorescent technique using rabbit antisera to human IgG, IgM, IgA,