consistent with the competitive inhibition of glucosamine by glucose for transport from the gastrointestinal tract. Furthermore, higher serum levels might be expected because glucose would competitively inhibit the uptake of glucosamine into the liver, thus allowing a higher concentration of glucosamine to enter the peripheral circulation. We do not believe that the minimal raising or extending glucosamine levels would be of significant therapeutic benefit.

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High association of mycobacterial infections with polymyositis in a non-endemic country for tuberculosis

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The susceptibility to mycobacterial infections of immunosuppressed subjects, for example, those treated actively for rheumatic diseases, is widely recognized.^{1,2} However, the co-existence of mycobacterial infection (caused either by *Mycobacterium tuberculosis* or other mycobacterial infections) and polymyositis (PM) has been reported only in small series of patients^{2–5} predominantly from countries where tuberculosis (TB) is endemic^{2,3} or in immigrants in a Western country.^{4,5}

We assessed the frequency of mycobacterial infections in patients with PM and dermatomyositis (DM) in Finland, a non-endemic country for TB that has kept detailed registry for TB. All patients with an ICD-8 code for PM (716.00) and DM (716.10) who had been discharged from any Finnish hospital during the years 1969 to 1985 were identified by using the information from the National Discharge Registry. During this period, diagnostic studies for PM and DM were usually performed on inpatients. Therefore, the present series of patients represents the vast majority of PM and DM cases in Finland during this period.

We found 176 patients with PM and 72 with DM who fulfilled the diagnostic criteria of Bohan and Peter.^{6,7} We followed their progress by studying the records until they died or until the end of August 1995. At the time of the diagnosis of myositis, 26 (10%; 19 with PM and 7 with DM) of the whole series of 248 patients had a history of TB. In addition, 13 out of 176 patients (7.4%) with PM and 2 out of 72 patients (2.8%)

with DM were documented to have a mycobacterial infection after a median time (range) of 20 (3–111) months from the diagnosis of myositis. Only one of them (who had PM) was in the group with a history of TB (57 years earlier).

Cumulative probability of mycobacterial infections among patients with PM was 4.8% (95% CI 2.4 to 9.4%) in 5 years and 9.3% (95% CI 5.4 to 15.6%) in 10 years (fig 1). On the basis of acid fast stain and mycobacterial culture, we determined that 11 out of the 15 mycobacterial infections were caused by *M. tuberculosis* (nine of them in PM patients) and four by other mycobacteria (*M. avium intracellulare* or mycobacteria group IV). At the time of the diagnosis of the mycobacterial infection, all the patients used corticosteroids, and seven PM patients also used or had used azathioprine. The risk factors for developing mycobacterial infections did not differ between patients with and without the infection.

Altogether, in 40 out of 248 (16%) patients with myositis — in 31 out of 176 (18%) with PM and 9 out of 72 (13%) with DM — there was a former or diagnosed mycobacterial infection noticed during the follow up. To the best of our knowledge, the present study is the first one reporting a high frequency of mycobacterial infections in a large, systematically collected population of subjects with PM and DM in a Western non-endemic area for TB.

It may be supposed that, at least in part, the high incidence of mycobacterial infections during the treatment of myositis is caused by the reactivation of a former infection. The association

Figure 1 Cumulative probability of mycobacterial infections in 176 patients with polymyositis after the diagnosis of myositis. The incidence of tuberculosis was 110 out of 100 000 people in Finland in 1969. It was 47 out of 100 000 in 1980, and 11 out of 100 000 in 1995.⁸ The incidence of mycobacterial infections (*M. tuberculosis* and other mycobacteria) in PM patients was remarkably high when compared with that in the general population, especially considering the downward trend in infection over the period of this study. The risk of infection even seems to increase during the follow up after the diagnosis of PM. *M. tuberculosis* had been coded as either the main cause (N=3) or immediate cause (N=1) of death for four patients with PM.

between mycobacterial infections and PM is interesting, especially if there was a latent infection present in most of the cases, although this cannot be confirmed from their

records. A weak causal relationship with 'reactive myositis' caused by a former or latent mycobacterial infection cannot be ruled out.

We conclude that the possibility of mycobacterial infection has to be kept in mind when diagnosing and treating patients with myositis.

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Fat-suppression magnetic resonance imaging in the diagnosis of late-onset axial spondyloarthritis

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Although late-onset spondyloarthritis (SpA) is not rare, it is usually associated with peripheral joint involvement, which is relatively easier to recognise.^{1–3} We report a case of an elderly woman with late-onset SpA who presented with axial disease, where the diagnosis was established by magnetic resonance imaging (MRI). This shows the potential of MRI for defining the full spectrum of SpA-related diseases.

The patient was a 75-year-old female who presented with a one-year history of vague pain in the thoracic spine left posterior chest radiating to the left mid-axillary line and associated early morning stiffness for 2–3 hours. Her respiratory and cardiac systems were unremarkable, and the pain did not alter when breathing or sneezing. She had no pain in bed at night and had mild relief by taking anti-inflammatories. There was no history of peripheral enthesopathy, iritis or psoriasis or of precipitating infections. The family history was relevant in

that her daughter has ankylosing spondylitis and associated ulcerative colitis.

Upon examination, the only positive findings were discomfort on rotation of the thoracic spine. On some occasions, the patient did have some restriction of thoracic rotation with occasional discomfort adjacent to the paraspinal regions, especially on the left side, which were variable, but no spinous process tenderness. She had no clinical enthesopathy or peripheral synovitis. Routine investigations showed an erythrocyte sedimentation rate of 7 mm/hour and a C-reactive protein of 3 mg/l (<10 mg/l), negative rheumatoid factor and negative antinuclear antibodies, negative HLA-B27 and a negative myeloma screen. X-rays of her thoracic spine and a bone scan were normal.

During a rheumatology review, a diagnosis of inflammatory back pain was suspected and an MRI scan with fat suppression

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