studies showed granulomatous reaction and AF infection. Important destruction was seen and it was very difficult to find any evidence of leukaemic infiltration. Only 12 cases of septic arthritis due to AF have been reported [2, 4–9] and just one without systemic infection [9]. Two cases in renal transplant recipients have been reported as arthrocentesis and corticosteroid complication [8, 9]. We report a new case of septic arthritis by AF after corticosteroid infiltration in a woman with AML. This patient presented some risk factors for fungal infection, such as immunosuppression and joint manipulation (arthrocentesis and corticosteroid infiltration). Furthermore, joint manipulation was carried out during hospital construction projects. It is known that large numbers of Aspergillus spores may be generated during construction and high levels of air contamination have resulted in nosocomial epidemics of invasive infections among susceptible patients [10]. Treatment of AF infection includes a combination of surgical debridement and antifungal therapy. Itraconazole and AB are the most effective of theazole antifungal drugs for the treatment of invasive aspergillosis [11, 12]. We initially chose itraconazole because it has less toxic effects, oral administration, the site where the Aspergillus infection was located and the successful results in other patients published in the literature. Our patient was given oral itraconazole 600 mg daily without side-effects. After 5 weeks of treatment, SF culture was still positive for AF. For this reason, we changed the antifungal therapy and started i.v. AB.

We should alert clinicians to potential dangers of arthrocentesis and corticosteroid infiltration in immunosuppressed patients. In these cases, we suggest carrying aseptic conditions to extremes, avoiding performing joint manipulation during hospital construction work, and always processing SF for fungal culture.

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The Importance of High-Resolution Computed Tomography in the Diagnosis of Interstitial Lung Disease

Str—We read with interest the recent articles on methotrexate lung complications. There are conflicting results and opinions on whether pre-existing lung disease predisposes to methotrexate pneumonitis. Our concern lies with the method of investigation used to assess this possibility.

Over the last 5yr, the role of chest HRCT (high-resolution computed tomography) scanning has become established as an important investigation in diagnosing interstitial lung disease (primary or secondary to connective tissue disease) [1]. HRCT has been shown to identify disease before abnormality is apparent on a chest radiograph [2] and is superior to chest radiograph in diagnostic accuracy [3, 4]. It has been shown in rheumatoid arthritis (RA) that some degree of abnormality on HRCT is common even in patients without apparent lung disease [5]; this is partly as a result of RA patients presenting late with interstitial lung disease as their exercise level is limited by arthritis.

The paper by Beyeler et al. [6] relied on information from pulmonary function tests. It has been shown by McDonagh and colleagues [5] that in RA pulmonary function tests fail confidently to predict abnormalities on HRCT and in their paper the specificity of a low (>1 s.d. below predicted) forced expiratory volume in 1 s and transfer factor using indicator gas CO (TLCO) was 59 and 71%, respectively. We are undertaking a prospective study of RA-associated lung disease; preliminary results comparing HRCT and pulmonary function tests confirm these findings.

We suggest that chest HRCT scanning needs to be incorporated into studies that assume the hypothesis that interstitial lung disease predisposes to the development of methotrexate pneumonitis, as it is not possible confidently to exclude interstitial lung disease on the basis of symptoms, chest radiograph and full pulmonary function tests.

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Reply

Sir—We thank Dr Dawson and Dr Lynch for their valuable comments.

We agree that high-resolution computed tomography (HRCT) is a sensitive method for the detection of interstitial lung disease and should be incorporated into studies analysing pulmonary involvement in RA. However, HRCT was not yet available in our hospital during recruitment of our patients from 1987 to 1988.

We were well aware of the limitations of lung function tests [forced expiratory volume in 1 s (FEV1), transfer factor using mechanics forced vital capacity (FVC)] during exercise allowing the exclusion of interstitial lung disease. We included in our study the measurement of gas exchange at rest and, if possible, during exercise with calculation of the alveolar–arterial PO2 gradient (P(A-a)O2). To our knowledge, a normal P(A-a)O2 gradient during exercise allows the exclusion of interstitial lung disease such as fibrosing alveolitis, pneumonitis and fibrosis with a high probability similar to a normal HRCT.

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Cyclical Etidronate Prevents Spinal Bone Loss in Early Post-menopausal Women

Sir—We wished to address the possible use of cyclical etidronate therapy in the prevention of bone loss associated with the menopause. We therefore performed a double-blind placebo-controlled study to determine whether 2 yr treatment with cyclical etidronate is effective at preventing bone loss in early post-menopausal women with spinal osteoporosis.

Forty-six post-menopausal women within 7 yr of the menopause, who had a lumbar spine bone mineral density (BMD) (L2-4) within the lowest quartile of our local reference range, were recruited over 12 months. No subject had received treatment with hormone replacement therapy (HRT) within the previous 6 months, or had evidence of a vertebral fracture on thoracolumbar spine radiographs. Patients were randomized to receive eight cycles of 14 days treatment with etidronate 400 mg or placebo, followed by calcium 500 mg (Cacit) for 76 days. Bone mass was measured by Lunar DPX at the lumbar spine and hip at baseline, and subsequently repeated at 6 monthly intervals.

Baseline characteristics of the two treatment groups were similar (placebo group: mean 49.9 yr of age, 3.7 yr post-menopausal, 1.62 m in height, 63.1 kg in weight; etidronate group: mean 49.1 yr of age, 4.0 yr post-menopausal, 1.61 m in height, 61.9 kg in weight). The study medication was generally well tolerated. One patient, who was in the etidronate group, withdrew from the study at month 15 due to the development of bone pain and general malaise. Four subjects withdrew voluntarily due to the commencement of HRT for worsening climacteric symptoms, of whom one was in the placebo group and three in the etidronate group. In addition, one patient in the etidronate group left the study at month 9 for reasons that were not specified.

Treatment with cyclical etidronate was associated with a higher lumbar spine BMD as compared with placebo-treated women (Fig. 1). At 24 months, lumbar spine BMD in women receiving placebo had decreased by 2.2% [significantly different from zero (P = 0.04), one-sample t-test], while a non-significant increase was evident in etidronate-treated women (+0.5%). In addition, after 2 yr, BMD was higher in the etidronate than in the placebo group by 2.1% at the femoral neck, 4.4% at Ward’s triangle and 1.9% at the greater trochanter. However, these differences at the three hip regions did not reach significance, possibly reflecting the relatively low precision of hip BMD measurements as compared with those obtained at the spine.

Fig. 1.—Results for lumbar spine BMD (mean ± s.e.m.) in patients treated with etidronate (n = 24) or placebo (n = 22) over 2 yr. Repeated measures analysis of variance indicated a significant treatment effect overall (P = 0.03).