Osteoporosis in rheumatological practice: questions to be answered

Osteoporosis, the most common metabolic bone disorder in western populations, is now firmly positioned on the agenda of clinical rheumatologists in Britain and the United States. While this has been the order of the day throughout mainland Europe for decades, our own commitment to the management of osteoporosis is more recent. The change has much to do with altered perceptions of the disorder: we see a lot of it; we can measure it; and we can do something about it. While there is truth in each of these observations (and support, in the rapidly growing numbers of papers, congresses, and journals devoted to osteoporosis), the challenge of translating much of this recent knowledge into coherent, effective preventive and therapeutic strategies remains. To meet this challenge, we must have a clear idea of what we do, and do not, know. This editorial addresses this problem, with regard to two issues relevant to all rheumatologists—the clinical uses of bone densitometry, and the prevention of steroid induced osteoporosis.

Although several techniques have been developed in recent years for the non-invasive measurement of bone density, the most precise and widely used method is dual energy x-ray absorptiometry (DXA). Initially developed to measure the spine and hip, the method has been extended to permit estimation of bone mineral at the forearm, whole body and lateral spine. The limitations of absorptiometric techniques have been widely aired, including their failure to measure volumetric density directly; their influence by osteophyte; vertebral deformity, and extraskeletal calcification; and the variations in reference data provided by different manufacturers. Despite these limitations, several prospective studies have now confirmed that bone mineral density values, as determined by absorptiometry, are strongly associated with the risk of future fracture. Thus the incidence of osteoporotic fractures approximately doubles with each standard deviation (SD) decline in bone density. This relationship appears slightly weaker, though still holds, when measurements are made at skeletal sites distant from the fracture site under consideration, and applies across a wide age range. Nevertheless, the most appropriate means of utilising this new technology remains contentious, and the interpretation of measurements does not follow any standardised practice. It is clear that a population-wide bone density screening programme is not justifiable on current evidence. Consensus is building, however, on a narrower range of clinical indications for bone densitometry in which results of the test influence individual clinical decision making. The most widely proposed indications include: (a) the presence of strong risk factors, such as corticosteroid therapy or a premature menopause; (b) radiographic evidence of vertebral deformity or osteopenia; (c) multiple low trauma fractures; and (d) the monitoring of therapy with newer drugs acting on bone. The service demand for densitometry on the basis of these indications is around 175 scans per 100,000 population each year.

As the gradient of fracture risk with bone density is continuous, there is no easily recognised threshold at which osteoporosis may be defined. The World Health Organisation has recently proposed two cut offs for diagnostic purposes: the first (a bone density value between 1 and 2.5 SD below the young normal mean) designated osteopenia, and the second (a bone density value more than 2.5 SD below the young normal mean) designated osteoporosis. It is intended that the higher threshold should stimulate consideration of prophylactic intervention, while the lower one should serve as a stronger indication. Although these criteria are currently only available for women, and are more suitable for use between ages 50 and 65 years, they do at least provide a consistent means whereby bone density measurements might be expressed and experiences compared between centres.

The second aspect of osteoporosis research which is directly relevant to the practising rheumatologist is that of preventing steroid induced osteoporosis. The timing and dose- and disease-dependence of steroid induced bone loss have been well characterised. This loss has now been directly linked to the risk of vertebral and hip fracture. In this issue of the Annals, Peat and colleagues review steroid prescription behaviour in a large teaching hospital. Their survey highlights the size of the problem, and confirms that co-prescription of prophylaxis is rare. However, a clear statement of the nature of such prophylaxis cannot yet be made. It is clear that this discussion hinges on how we should manage patients commencing a course of steroids likely to involve doses of greater than 7.5 mg daily of prednisolone (or equivalent) for longer than a year. It is reasonable to advise these patients to take an adequate dose of calcium, and to recommend oestrogen replacement to postmenopausal women, especially between the ages of 50 and 65 years. The crucial question, however, is whether drugs acting specifically on bone should be introduced at this stage. The agents worthy of consideration are the bisphosphonates, calcitonin, and vitamin D metabolites, but formal evidence supporting their efficacy is limited. Only one study unequivocally addressed the issue of primary prevention of bone loss with bisphosphonates. Twenty patients with temporal arteritis
treated with prednisolone were allocated randomly to receive cyclical etidronate, or no treatment, for a year. Bone mineral was measured only in the lumbar spine; the intervention exerted a beneficial effect at this site. The other randomised trial compared a pamidronate/calcium regimen with calcium alone in patients who were established on steroid therapy (12–15 mg daily for five years) but had not sustained fractures. Again, the bisphosphonate exerted a beneficial effect which persisted into the second year. The only other primary preventative trial studied 103 patients within four weeks of the onset of steroid treatment, and evaluated three regimens: calcium, calcium/calcitriol, and calcitriol/intranasal calcitonin. Spinal bone loss was significantly reduced in the two calcitriol groups during the year of treatment. In the second year of the study (in which treatment had been stopped), spinal bone loss was arrested only in the calcitriol/calcitonin group. Until these data have been supplemented by other studies, it will remain difficult to expect most rheumatologists to do other than consider these interventions carefully for the prevention of steroid induced bone loss, and to utilise bone densitometry where available to gauge an individual patient’s future fracture risk.

MRC Environmental Epidemiology Unit, Southampton General Hospital, Southampton SO16 6YD, United Kingdom

Cyrus Cooper