Sequential study of bone mineral density in patients with systemic lupus erythematosus

A loss of bone mineral density has been reported in premenopausal women with systemic lupus erythematosus (SLE), but its pathogenesis is uncertain. Studies on the sequential changes in bone mass in these patients are scarce. Recently, we studied 74 premenopausal women with SLE without any complications or treatment (except for glucocorticoids) that could interfere with bone mineral density. We excluded nine osteoporotic patients according the established World Health Organisation criteria, because they were on treatment for low bone density. We repeated the measurement of bone mineral density (L2-L4) and femoral neck (FN) by dual energy x ray absorptiometry (DXA), using a densitometer (Hologic QDR 1000). Measurement of the bone mineral content, calibrated with the Hologic X-iestimator linked to anthropomorphic spine, the phantom of the known mineral content, was accurate to 0.5%. The precision measurement was better than 0.01 g/cm² (coefficient of variation = 1.0% at bone mineral density 1.0 g/cm²). Disease activity was assessed with the University College Hospital/Middlesex SLE scoring system, by a numerical score graded from 0 to 4 (inactive to severe active disease).

The results were expressed as the mean (SD). For all conventional analyses we used the SPSS/PC software package. A t test was used and the correlations were calculated by linear regression analyses. Results were considered significant at P < 0.05.

At the time of the first densitometry, the mean age was 31.7 (6.8) years, and disease duration was 91 (64) months. During 18 months there was no significant decrease in bone mineral density, despite glucocorticoid treatment (table), in either the lumbar spine or the femoral neck. No fractures were found. Serum calcium, phosphate, alkaline phosphatase, and 24 hour urine calcium and phosphorus did not change during this period.

Using linear regression, there was no correlation between bone mineral density or changes in bone mineral density and the prednisone dose (cumulative and baseline). We found no correlation between disease duration or mean disease activity grade and lumbar spine, and femoral neck. No other correlations with bone mineral density were found.

This study shows that in premenopausal SLE patients lumbar and femoral bone mineral density did not change with respect to baseline values after 18 months, despite continuous glucocorticoid treatment (the mean dose was 9.2 mg/day during the 18 months). Recently, Pons et al. reported similar results in a study of 31 premenopausal women followed for a mean of 36.6 months. Kalla et al. also found no changes in bone mineral density in 56 SLE patients over 18 months.

The effect of glucocorticoids on SLE osteoporosis is controversial. We did not find any correlation between prednisone dose and bone mineral density in chronic steroid users. The effects of glucocorticoids on the bone mass are more pronounced early in the course of steroid treatment. The reported reduction in bone mineral density in these patients may occur at the onset of the disease. Nevertheless, future studies to demonstrate a small loss of bone mineral density in SLE will probably require more patients and a longer period of study.

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