Etidronic acid prevents corticosteroid-induced osteoporosis

Intermittent etidronic acid is a *'safe'* and effective therapy for the prevention of corticosteroid-induced osteoporosis, report researchers from Canada.[1]

Patients with a variety of underlying disorders who had commenced high-dose corticosteroid therapy within the past 100 days and who were expected to continue such therapy for ≥ 1 year were followed in this multicentre study.* They were randomised to receive etidronic acid ['Didronel'] 400mg once daily (n = 67), or placebo (74), for 14 days. All patients then received 76 days' therapy with calcium carbonate ['Didrocal'] 500mg. This 90-day cycle was repeated 3 times during the 1 year duration of the study.

No change in bone density

After 52 weeks' therapy, the bone density of the lumbar spine and trochanter had not changed significantly from baseline among etidronic acid recipients, but it had declined among placebo recipients.

The between-group differences in the mean percentage change from baseline in the bone density of the lumbar spine and trochanter were significant. However, the between-group differences in the mean percentage change from baseline in the bone density of the femoral neck and the distal and midshaft radius were not.

During the study period, 9% of etidronic acid recipients developed new vertebral fractures, compared with 15% of placebo recipients; the relative risk of fractures was 0.6 for etidronic acid, compared with placebo, recipients.

Eight patients in each treatment group experienced adverse events; the majority of these involved the GI system and were of a transient and mild nature.

'Substantial step forward'

Dr Ian Reid from the University of Auckland, New Zealand, describes the above study as a *'substantial step forward'* and adds that the results of the study *'strengthen the case for bisphosphonates for the prevention of glucocorticoid-induced osteoporosis'*.[2]

Dr Reid recommends that the lumbar spine bone density should be measured in all patients who are commencing long-term corticosteroid therapy, and that prophylaxis should be offered to patients with bone densities *'at the lower end of the normal range for young adults or below'*. He concludes that the *'challenge now is to make the assessment of the risk of osteoporosis an integral part of the decision to commence glucocorticoid therapy so that therapy can be introduced before fractures occur'*.

* Patients were included if they expected to receive prednisone (or its equivalent) at a mean daily dose of ≥ 7.5mg for 90 days followed by a mean daily dose of ≥ 2.5mg. The study was supported by a grant from Procter & Gamble.

1. Adachi JD, et al. Intermittent etidronate therapy to prevent corticosteroid-induced osteoporosis. New England Journal of Medicine 337: 382-387, 7 Aug 1997.
2. Reid IR. Preventing glucocorticoid-induced osteoporosis. New England Journal of Medicine 337: 420-421, 7 Aug 1997.

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