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THE ABSORPTION OF CALCIUM AND ITS INCORPORATION INTO BONE
DURING CORTICOSTEROID THERAPY.

Allan Geoffrey Need

Department of Medicine

University of Adelaide

Adelaide

and

Division of Clinical Chemistry

Institute of Medical and Veterinary Science

Frome Road

Adelaide

South Australia

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ABSTRACT

Previous studies indicate that corticosteroid osteoporosis is unique in that it is marked by both an increase in bone resorption and a decrease in the bone formation rate. The increased bone resorption has been demonstrated in this series by an increase in urinary hydroxyproline excretion which has been shown to be associated with decreased radiocalcium absorption and increased urinary calcium excretion. An index (radiocalcium absorption - calcium excretion) discriminated between osteoporotic (OP) and non-osteoporotic (N) corticosteroid-treated cases better than any parameter alone.

When radiocalcium absorption was regressed on the serum 1,25-dihydroxycalciferol (1,25D) level it was found that the slope was normal for the N patients (0.0050 ± 0.0010) but significantly flatter for the OP patients (0.0024 ± 0.0008 ; $p < 0.001$). This indicates a decreased efficiency in the gut response to 1,25D in the OP patients.

Treatment of the calcium malabsorption with 1,25D caused the hourly fractional radiocalcium absorption to rise from 0.37 ± 0.04 to 0.64 ± 0.07 of the dose per hour ($p < 0.01$) and, when combined with a calcium supplement, a fall in urinary hydroxyproline excretion to normal.

The radiokinetic bone formation rate was found to be decreased in patients on corticosteroids, when they were compared with postmenopausal osteoporotic women. Treatment with nandrolone decanoate increased the bone formation rate

from 45 ± 17 to 134 ± 37 mg Ca/d ($p < 0.025$) in those given nandrolone alone.

The effects of nandrolone on the forearm bone mineral density, measured by photon absorptiometry, were investigated in a cross-over trial. There was a significant (time-weighted) gain in bone density on nandrolone ($+1.6 \pm 0.6$ mg/ml/month; $p < 0.05$) and a significant (time-weighted) loss off the drug (-1.3 ± 0.3 mg/ml/month; $p < 0.01$). The difference between these 2 rates was highly significant ($p < 0.001$).

The results confirm that corticosteroid osteoporosis is associated with a) decreased intestinal calcium absorption and b) increased urinary calcium excretion and a new index, combining both parameters, has been developed which discriminates well between osteoporotic cases and those with normal spines ($p < 0.0001$). This may be useful for predicting the risk of the disease in any individual patient on corticosteroid therapy. The biochemical response to calcitriol and calcium therapy suggests that this is a useful combination for treatment and the response of bone mineral density to nandrolone suggests that further improvement may be gained from anabolic steroid therapy which appears to correct the abnormality in bone formation.