Parathyroid-Mediated Bone Loss

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Among the many causes of diminished skeletal mass, parathyroid hormone (PTH)-mediated bone resorption has been most widely studied experimentally, and age-related osteoporosis is the least understood. Because of histologic heterogeneity, it has been assumed that the etiology of osteoporosis, either postmenopausal (Type I) or senile (Type II), is multifactorial (Riggs, et al, pp. 337 ff.). This session examined the possible contribution of PTH-mediated bone loss to the pathogenesis of osteoporosis.

Ed. Note - This overview was originally presented at the International Symposium on Clinical Disorders of Bone and Mineral Metabolism, May 9-13, 1983. The following list indicates the presentations given in this session at the Symposium and the contents of the corresponding chapter in the Proceedings of the Symposium published by Excerpta Medica. The numbers in parentheses refer to pages in this volume. Complete information about the contents of the Proceedings can be found at the back of this issue.

Estimation of trabecular bone resorption by histomorphometry: Evidence for a prolonged reversal phase with normal resorption in post menopausal osteoporosis and coupled increased resorption in primary hyperparathyroidism. R. Baron, S. Magee, A. Silverglate, A. Broadhus, and R. Long (191)

Mechanisms of parathyroid hormone mediated bone loss. L.G. Raisz (196)


Vitamin D deficiency and hip fractures: Cause or coincidence? P. Lips, M.J.M. Jongen, F.C. van Giunkel, W.J.F. van der Vijgh, R. Bouillon, W.H.L. Hackeng, and J.C. Netelenbos (204)

Osteoclastic resorption of osteoid in secondary hyperparathyroidism. Ming Cai Qiu, C. Mathews, and A.M. Parfitt (209)

Among the many causes of diminished skeletal mass, parathyroid hormone (PTH)-mediated bone resorption has been most widely studied experimentally, and age-related osteoporosis is the least understood. Because of histologic heterogeneity, it has been assumed that the etiology of osteoporosis, either postmenopausal (Type I) or senile (Type II), is multifactorial (Riggs, et al, pp. 337 ff.). This session examined the possible contribution of PTH-mediated bone loss to the pathogenesis of osteoporosis.

Raisz (pp. 196 ff.) reviewed the possible contribution of PTH-mediated bone loss to the pathogenesis of osteoporosis.

Baron (pp. 191 ff.) reviewed the histologic assessment of bone resorption. Osteoclasts occupy only a small fraction of the total crenated (Howship's) lacunar surface. The remainder are covered by various kinds of mononuclear cells that appear in the time interval between the end of resorption and the beginning of formation and identify the so-called reversal surface. In hyperparathyroidism and other states of increased bone turnover, the increase in total resorption surface reflects an increase in osteoclast surface. On the other hand, in postmenopausal and other types of osteoporosis, the increase in total resorption surface reflects only an increase in reversal surface. Accordingly, increased total resorption surface is not a
valid index of increased resorption rate in osteoporosis. Qiu (pp. 209 ff.) reviewed another facet of the histologic assessment of bone resorption and found that osteoclasts normally avoid osteoid and resorb only unmineralized bone. However, osteoclastic resorption of osteoid may occur with the stimulus of severe hyperparathyroidism. Kleerekoper (pp. 200 ff.) demonstrated that chronic hyperparathyroidism, whether primary or secondary, accelerates age-related loss of cortical bone. Loss of trabecular bone, observed in only a small subset of patients with primary hyperparathyroidism, was more substantial in patients with secondary hyperparathyroidism due to gastrointestinal malabsorption of calcium. However, even in these patients, the relative deficit was significantly greater for cortical than for trabecular bone. This conclusion differs from that of other investigators, partly because of different interpretation of measurements by single photon absorptiometry at the distal radius. Although this measurement is generally believed to indicate the loss of trabecular bone, our studies indicate that trabecular bone at this site is metabolically inert and that, as at the proximal site, losses are due predominantly to cortical thinning. None of the studied patients had sustained a fracture, but in another session Rao demonstrated a significant increase in fracture risk for patients with secondary hyperparathyroidism even without osteomalacia (Rao, et al, pp. 224 ff.).

Lips (pp. 204 ff.) examined the relationship between subclinical vitamin D deficiency and hip fractures in elderly patients. Iliac histomorphometry of the patients with hip fracture was divided into three groups: increased turnover (increased resorption and formation); impaired coupling (increased resorption and normal formation); and low turnover (depression of both resorption and formation). This analysis was based on static measurements not on kinetic studies with tetracycline labeling. In the high turnover group, the plasma calcidiol level was low and cortical thickness in the ilium was significantly reduced. None of the patients had osteomalacia as defined elsewhere (Rao, et al, pp. 224 ff.). The data were consistent with the view that the early osseous effects of vitamin D deficiency are mainly those of secondary hyperparathyroidism, leading to accelerated loss of cortical bone. In general, PTH-mediated bone loss probably makes little contribution to postmenopausal osteoporosis (Type I) with vertebral compression fractures, but may significantly contribute to senile osteoporosis (Type II) leading to hip fractures.