Hypercalcemia

(AUGUST 2005)

TO THE EDITOR: We enjoyed the recent article by Aladesanmi and colleagues, "A 56-year-old man with hypercalcemia,"[1] about an interesting case. However, we would like to make some corrections to their paper and summary.

The authors did not cover all causes of malignancy-induced hypercalcemia. Malignant tumors can also cause a 1,25 dihydroxyvitamin D_3-mediated hypercalcemia. This has been found with B-cell lymphomas, Hodgkin disease, and lymphomatoid granulomatosis.[2,3] In patients with hypercalcemia and a low intact parathyroid hormone (PTH) level and no other obvious pathology (this patient was different, having an enlarged liver at initial presentation), we should consider checking the PTH-related peptide and 1,25 dihydroxyvitamin D_3 levels to determine the cause and to direct evaluation.

When physicians see an unexpectedly elevated serum calcium, they should always correct for the albumin, since this is readily available on most serum chemistry panels. If the calcium corrects to normal, there is no need to repeat or perform further testing. For every 1.0 g/dL decrease in albumin below 4 g/dL, the calcium is corrected by adding 0.8 mg/dL, and for every 1.0 g/dL increase in albumin above 4 g/dL, the calcium is corrected by subtracting 0.8 mg/dL.

The clearance of biologically inactive fragments of PTH is reduced in renal failure. These fragments may interfere with intact PTH measurements and may falsely increase the PTH result. A new assay is available to measure only the biologically active fragment of PTH.[4] It is called Bio-intact PTH and may be useful in patients with renal failure.

Finally, we would like to clarify the summary statement of recommendations for care of bone health in patients with sarcoidosis. Not only perimenopausal women with sarcoidosis being treated with steroids should be screened for and educated about osteoporosis. All patients being treated with steroids (glucocorticoids) are at risk for bone loss, osteoporosis, and fracture: men, women, and children. The current recommendations are that patients being treated with more than 5 mg of prednisone daily for more than 3 months should be treated with a bisphosphonate to prevent steroid-induced bone loss and fracture.[5] Bone density testing should be repeated at 1 year as long as steroid therapy continues.[6]

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REFERENCES

1. **Aladesanmi O, Jin XW, Nielsen C.** A 56-year-old man with hypercalcemia. Cleve Clin J Med 2005; 72:707–712.
2. **Seymour JF, Gagel RF.** Calcitriol: the major humoral mediator of hypercalcemia in Hodgkin's disease and non-Hodgkin's lymphomas. Blood 1993; 82:1383–1394.
3. **Schienman SJ, Kelberman MW, Tatum AH, Zamkoff KW.** Hypercalcemia with excess serum 1,25 dihydroxyvitamin D in lymphomatoid granulomatosis/angiocentric lymphoma. Am J Med Sci 1991; 301:178–181.
4. **Inaba M, Nakatsuka K, Imanishi Y, et al.** Technical and clinical characterization of the Bio-PTH (1-84) immunochemiluminometric assay and comparison with a second-generation assay for parathyroid hormone. Clin Chem 2004; 50:385–390.
5. **American College of Rheumatology Ad Hoc Committee on Glucocorticoid-Induced Osteoporosis.** Recommendations for the prevention and treatment of glucocorticoid-induced osteoporosis. Arthritis Rheum 2001; 44:1496–1503.
6. **The Writing Group for the ISCD Position Development Conference.** Indications and reporting for dual-energy x-ray absorptiometry. J Clin Densitom 2004; 7:37–44.