

Glucocorticoid-induced osteoporosis

(AUGUST 2010)

TO THE EDITOR: I have to say I am disappointed, but not surprised, at Dr. Dore's article, "How to prevent glucocorticoid-induced osteoporosis" in your August issue.¹ The section "Estrogen is being used more selectively" was shorter and had older and out of date references compared with the section "A role for testosterone?" and it was actually blatantly sexist: the comment in the estrogen section is that "...the consensus...that hormone replacement therapy should be restricted to women with menopausal symptoms or to older women who cannot tolerate other therapies or who express a strong preference for hormone replacement therapy *despite being informed about potential adverse events*" [my italics],¹ while the comment in the testosterone section is that males who "...are hypogonadal, and have no contraindications to androgen replacement therapy (eg, prostate cancer) *be offered testosterone therapy to preserve lean body mass and bone mineral density*" [my italics].¹

While I am not arguing that menopausal hormone therapy should be used first-line for the prevention or treatment of glucocorticoid-induced osteoporosis, I would like to note the following:

First, the referenced 2002 Women's Health Initiative study² was a prevention trial, not a therapeutic menopausal trial, and to reference it as a position statement on the use of hormone therapy is ridiculous and perpetuates misinformation about the role of menopausal hormone therapy.

Next, there has been updated information from the Women's Health Initiative, as well as updated position statements on the use of hormone therapy—the 2010 position statement on the use of estrogen and progestogen in menopausal women³ as well as the 2008 American Association of Clinical Endocrinologists position statement⁴ noting that the benefits of hormone therapy outweigh the risks for most women under age 60. So Dr. Dore's reference citation from 2004⁵ is hopelessly outdated.

And lastly, females, unlike males, routinely become hypogonadal at midlife. When faced with a medical condition that requires glucocorticoids that further intensifies the hypogonadal state by suppressing adrenal androgens, females may face a "triple whammy" on the bone.

The Women's Health Initiative actually showed fracture reduction in postmenopausal women who did not even carry the diagnosis of osteoporosis, while the referenced studies in Dr. Dore's article related to males admittedly "cannot be considered conclusive in view of their small size and the lack of fracture data..."¹

So what is bad (actually potentially good) for the goose is apparently just fine for the gander.

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REFERENCES

1. **Dore RK.** How to prevent glucocorticoid-induced osteoporosis. *Cleve Clin J Med* 2010; 77:529–536.
2. **Writing Group for the Women's Health Initiative Investigators.** Risks and benefits of estrogen plus progestin in healthy postmenopausal women. Principal results from the Women's Health Initiative Randomized Controlled Trial. *JAMA* 2002; 288:321–333.
3. **Estrogen and progestogen use in postmenopausal women: 2010 position statement of The North American Menopause Society.** *Menopause* 2010. www.menopause.org. Accessed October 28, 2010.
4. **American Association of Clinical Endocrinologists.** Position statement on hormone replacement therapy and cardiovascular risk. www.aace.com/pub/pdf/guidelines/HRTCVIRISKposition_statement.pdf. Accessed October 28, 2010.
5. **Compston JE.** The risks and benefits of HRT. *J Musculoskelet Neuronal Interact* 2004; 4:187–190.

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IN REPLY: I could find references for the use of testosterone in glucocorticoid-induced osteoporosis and could not find any references for the use of estrogen in this condition, except for the outdated American College of Rheumatology guidelines from the 1990s, which included Dr. Nancy Lane's work. So perhaps it is the research that is gender-biased rather than my article. I agree that in osteoporosis that is not glucocorticoid-induced, estrogen has great fracture efficacy even in those without osteo-

porosis, as you stated, but I tried to keep my article evidence-based and on-topic regarding glucocorticoid-induced osteoporosis. As usual, topics that involve estrogen are highly volatile, and I did not mean to fuel the fire.

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Bariatric surgery, vitamin C, and kidney stones

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TO THE EDITOR: I read with interest the excellent review by Dr. Kashyap and coauthors of bariatric surgery for patients with type 2 diabetes.¹ I am writing to contribute an additional caveat to their otherwise detailed list of post-operative complications—the increased risk of nephrolithiasis.^{2,3} The majority of kidney stones that develop after bariatric surgery tend to be composed of calcium oxalate. Increased intestinal absorption of oxalate appears to promote hyperoxaluria.^{2,3}

Vitamin C deficiency is not usual after bariatric surgery, and the dietary reference intake of vitamin C for adults is no more than 90 mg. Therefore, I was surprised to see Dr. Kashyap recommend supplementation with vitamin C 500 mg daily (in **TABLE 4** of her article). In my practice I have avoided supplemental vitamin C, other than that in a multivitamin, because of the risk of increasing urinary oxalate and stone formation.⁴

Iron deficiency can be a challenge after bariatric surgery. Although they do not state it in the review, the authors may believe

that additional vitamin C can improve iron absorption. However, there are no compelling data of which I am aware for this belief in patients who have undergone gastric bypass,⁵ and the benefit of taking vitamin C along with iron in otherwise normal people with iron deficiency remains controversial.⁶

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REFERENCES

1. Kashyap SR, Gatmaitan P, Brethauer S, Schauer P. Bariatric surgery for type 2 diabetes: weighing the impact for obese patients. *Cleve Clin J Med* 2010; 77:468–476.
2. Lieske JC, Kumar R, Collazo-Clavell ML. Nephrolithiasis after bariatric surgery for obesity. *Semin Nephrol* 2008; 28:163–173.
3. Asplin JR, Coe FL. Hyperoxaluria in kidney stone formers treated with modern bariatric surgery. *J Urol* 2007; 177:565–569.
4. Massey LK, Liebman M, Kynast-Gales SA. Ascorbate increases human oxaluria and kidney stone risk. *J Nutr* 2005; 135:1673–1677.
5. Rhode BM, Shustik C, Christou NV, MacLean LD. Iron absorption and therapy after gastric bypass. *Obes Surg* 1999; 9:17–21.
6. Hunt JR, Gallagher SK, Johnson LK. Effect of ascorbic acid on apparent iron absorption by women with low iron stores. *Am J Clin Nutr* 1994; 59:1381–1385.

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IN REPLY: Although some nutritional guidelines advocate the use of vitamin C in post-bariatric patients, most data are now suggesting that it may not be indicated. We appreciate the comments provided and are in agreement with regards to the supplementation of vitamin C.

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