Celiac Disease Predisposes Patients to Bone Loss

BY MICHELE G. SULLIVAN

WASHINGTON — If you're not already taking a serious look at the bone health of patients with celiac disease, you should be, according to Dr. Peter Green, director of the Celiac Disease Center at Columbia University, New York.

The gastrointestinal disease presents a double-edged sword: Patients with celiac disease have an increased risk of osteoporosis and fragility fractures, not only because their intestines poorly absorb calcium and vitamin D, but also because the disorder induces bone-destructive inflammatory and autoimmune responses, he said.

Studies show that up to 50% of men, 40% of postmenopausal women, and 10% of premenopausal women with the disorder have osteoporosis, and up to

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DR. GREEN

30% of the people in these groups have osteopenia.

Yet only 6% of patients with celiac disease will have either osteopenia or osteoporosis as their presenting symptom.

The role of autoimmune inflammation on bone health in these patients is not as widely appreciated as is the issue of vitamin D and calcium malabsorption, he said at an international symposium sponsored by the National Osteoporosis Foundation.

Antibodies against tissue transglutaminase (tTG), which contribute to the gluten intolerance that characterizes celiac disease, appear to have a deleterious effect on bone, Dr. Green said.

"Tissue transglutaminase is a ubiquitous enzyme that is also present in bone," he said. Antibodies to tTG are also present in bone, and emerging evidence suggests they impair active mineralization.

Several studies have shown that a gluten-free diet, which decreases anti-tTG levels, directly correlates with increased bone mass in patients with celiac disease and low bone mineral density, he said.

"After 16 months on a gluten-free diet, bone mineral density increased by more than 7% at the femoral neck in celiac patients who also had osteoporosis," Dr. Green said (Am. J. Gastroenterol. 2001;96:112-9).

Proinflammatory circulating cytokines also are increased in celiac disease, and may contribute to decreases in bone density, although the exact mechanism by which this occurs is unknown, he said.

Several studies show improvements in bone density as cytokine levels diminish on a gluten-free diet (Am. J. Gastroenterol. 1998;93:413-8; Scand. J. Gastroenterol. 1999;34:904-8).

Comorbidities may also play a role. Secondary hyperparathyroidism is common in patients with celiac disease and may prevent patients from attaining their maximum bone density during childhood.

Premature menopause in women and reduced gonadal function in men also can contribute to poor bone health, Dr. Green said.

Whatever the mechanism, the bone loss associated with celiac disease results in a significantly increased risk of both peripheral and central fracture, Dr. Green said. A 2008 meta-analysis of seven studies showed that patients were up to 10 times more likely than were controls to suffer a fragility fracture (Dig. Liver Dis. 2008;40:46-53).

Strict adherence to a gluten-free diet seems to be the best way to boost bone health in these patients, Dr. Green explained.

Calcium and vitamin D supplements are important, but no studies have shown whether these patients need larger doses than those of the general population.

Bisphosphonates should be used with caution, as there have been several case reports of bisphosphonate-induced hypocalcemia in this group. ■