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Celiac Disease and Impact on Bone

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Celiac disease is an immune mediated disease characterized by intolerance to gluten and other peptides from different cereals in those who are genetically susceptible to develop CD. Treatment requires life-long abstinence from gluten. CD involves the small bowel from the duodenum to the distal ileum and is characterized by villous atrophy.

Diagnosis is made by evaluating for the presence of markers in the form of antigliadin, antiendomysium and antitransglutaminase antibodies, and the appearance of the small bowel on endoscopy. The diagnosis is confirmed on the small bowel biopsy. Population screening studies estimate the prevalence of CD to be approximately 1:152 individuals in Europe and 1:250 individuals in North America. Screening first-degree relatives of those with CD have confirmed the presence of significant small bowel change without any symptoms of the condition.

CD has a number of skeletal complications including inadequate bone mineralization and development of osteoporosis and osteomalacia. In children, rickets can develop. Patients can have bone and muscle pain in association with inadequate levels of vitamin D.

Studies in children with CD have demonstrated improvements in bone mineral content following initiation of a gluten-free diet.

In adults, nearly 40% of patients with CD will have osteopenia or low bone density at the time of diagnosis. The expected prevalence of osteopenia in a comparable adult population without CD is about 16%. Osteopenia has been found in patients who have been asymptomatic, as well as those who have had symptoms. The skeletal effects of CD do not correlate with malabsorption.

Initiation of a gluten-free diet is associated with improved bone mineralization and bone density. A prospective study evaluating 21 celiac patients found increased lumbar spine bone density of 6.6% and increased hip bone density by 5.5% over one year simply on a GF diet. The impact of the GF diet is through enhanced absorption of calcium from the bowel and improved absorption of phosphorous and 25 hydroxy vitamin D. Studies demonstrate the absorption rate of calcium in those with CD is nearly 45% lower than in controls. After six months on a GF diet, calcium absorption significantly improved with increases in urinary calcium excretion by 52%.

Fractures associated with little or no trauma occur in people with CD. An 8-fold increased risk of fragility fracture appears in those with CD compared to the control population. The most common sites of fracture are wrist, forearm, pelvis, tibia and clavicle.

Individuals who present with osteoporosis and have low levels of serum calcium or urine calcium excretion or elevations in parathyroid hormone should definitely be evaluated for CD. Patients may be entirely asymptomatic and these biochemical abnormalities may be the clue to underlying malabsorption. Correction of calcium, phosphorous and vitamin D absorption will result in enhanced bone mineralization and bone density without any additional pharmacologic intervention.

CD patients may also have low bone density due to general malnutrition and decreases in body mass index.

Inadequate calcium absorption will result in elevations in parathyroid hormone which contribute to progressive bone loss as the skeletal stores are utilized for calcium requirements. Absorption of vitamin D requires vitamin D receptors in the small bowel mucosa. Studies have demonstrated that the vitamin D receptors may be present in individuals with CD even in the presence of mucosal damage. However, the vitamin D regulated proteins, known as calbindin, and calcium binding protein that actively uptake calcium from the bowel, are lost from the area of the damaged small bowel mucosa. Other factors may also contribute to the development of low bone density including zinc deficiency. This may play a role in leading to low levels of growth stimulating factors (IgF-1).

Treatment of low bone density in CD, proceeding with a GF diet, is the key to improving bone mineral density particularly in children. Currently, there is a lack of long-term follow-up studies and it is not known whether optimal peak bone mass can be achieved with adherence to a strict GF diet in children with CD.

In adults, the GF diet has been associated with significant improvements in bone density. Correction of elevations in parathyroid hormone also occur as calcium, phosphorous and vitamin D absorption normalize. It appears that calcium intake must be higher than the recommended dietary allowance in order to obtain appropriate absorption of calcium in individuals with celiac disease. Studies have not currently evaluated the exact calcium requirement in individuals with CD. The

need for additional vitamin D supplementation also appears to be greater than the recommended dietary allowance. It is necessary to monitor serum calcium and vitamin D levels, as well as the 24-hour urine calcium, to ensure that adequate calcium and vitamin D is being administered to patients with CD.

It is important for physicians to be in close communication in managing patient care. Gastroenterologists and osteoporosis specialists should be aware of the complications present. Prevention of bone fragility fractures is the goal in individuals with celiac disease and this can be achievable with early diagnosis and aggressive management. The earlier in life that the diagnosis is made, the greater the response to therapy with a gluten-free diet.

The Canadian Celiac Health Survey as conducted by Dr. Ann Cranney and colleagues demonstrates that delays in diagnosis of CD continued to be an important issue which needs to be addressed. It is necessary for physicians to be aware and follow-up on associated medical conditions such as osteoporosis and thyroid disease. Screening of first-degree relatives for CD is also an important issue which should be emphasized.