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Celiac Disease

Description

- Celiac sprue is an inherited digestive disease. In this disorder, gluten (protein fraction) found in wheat, rye, oats and barley damages the lining of the small intestine. The symptoms can be overt with diarrhea, weight loss and malnutrition or it can be subclinical with isolated weight loss and nutritional deficiencies, but no gastrointestinal symptoms. Diagnosis is confirmed by a biopsy of the small intestine and a rapid response to the gluten-restricted diet.[1]

Causes

- Celiac disease is caused by a genetic disorder.

Types

- Celiac disease is also referred to as gluten-induced enteropathy, gluten-induced sprue and idiopathic steatorrhea (loose, fatty stools). These conditions are lifelong. Gluten intolerance may occur as a temporary condition secondary to intestinal damage in other disorders.[1]

At Risk

- Celiac disease typically appears in infancy when cereals are added to the diet or in adults 20 to 30 years of age.

Prevention and Maintenance

- There is no prevention of celiac disease since it is a genetic disorder. Patients must follow a gluten free diet, which is essential and life-long. The gluten-free diet involves avoiding wheat, rye, oats and barley and any products made with these grains. A gluten free diet allows the intestine to heal, eliminating problems with malabsorption.[2]
- Restricting fat and avoiding lactose may be necessary initially. Once the intestinal tract heals, fat and lactose may be reintroduced.
- In one study, patients on a gluten free diet with documented magnesium depletion showed increased bone mineral density with magnesium supplementation.[3]
- Bone mineral density in the celiac patient has been shown to be significantly lower than that of age-matched controls when <u>not</u> following a gluten free diet.[4]

Sources of Additional Information

- Gluten Intolerance Group of North America/PO Box 23053/Seattle WA 98102-0353
- Celiac Disease Foundation/13251 Ventura Blvd #3/Studio City, CA 91604
- http://cpmcnet.columb.edu/dept/gi/celiac.html
- http://www.mc.vanderbilt.edu/peds/pidl/gi/celiac.html

Abstracts

Corazza GR, Di Sario A, Cecchetti L, Jorizzo RA, Di Stefano M, Minguzzi L, Brusco G, Bernardi M, Gasbarrini G. Influence of pattern of clinical presentation and of gluten-free diet on bone mass and metabolism in adult celiac disease. Bone 1996 Jun;18:525-30. Since no information is available on bone derangements in subclinical celiac disease (CD), we evaluated bone mineral density (BMD, expressed as z score) at lumbar spine, by x-ray dual-photon absorptiometry and serum indices of bone metabolism and remodeling in 14 subclinical or silent patients, 10 classical patients and 15 healthy volunteers all on a gluten-containing diet. In the subclinical group, BMD at lumbar spine was significantly higher than in the classical group (-1.3 +/ 0.8, 73% vs. 2.6+/- 0.6, 88%, respectively; $p<0.001$), but significantly lower than in volunteers (+0.4 +/- 1.1, 104%; $p<0.001$). Similar changes were observed in serum calcium, whereas, as regards parathyroid hormone, no significant difference was found between subclinical and classical patients. 25-vitamin D was significantly lower, and 1,25-vitamin D was significantly higher in subclinical and classical patients than in healthy volunteers. Indices of bone remodeling were higher in the subclinical and classical groups than in the volunteers, but lower in the subclinical than in classical patients. Eight subclinical and 8 classical patients were reexamined after a period of gluten-free diet (GFD) and in both groups BMD had significantly improved. Our results show that osteopenia is a frequent feature also in subclinical CD, although the extent of bone and mineral metabolism derangements is lower than in classical CD> GFD is able to normalize BMD in subclinical and to significantly improve it in classical patients.

References

1. Zeman FJ. Clinical Nutrition and Dietetics. 2nd Ed. New York:MacMillan Publishing Company; 1991. p 240-241.
2. Kemppainen T, Uusitupa M, Janatuinen, Jarvinen R, Julkunen R, Pikkarainen P. Intakes of nutrients and nutritional status in coeliac patients. Scand J Gastroenterol. 1995;30:575-9.
3. Rude RK, Olerich M. Magnesium deficiency: possible role in osteoporosis associated with gluten-senstive enteropathy. Osteoporos Int. 1996;6:453-61.
4. Valimnarsson T, Lofman O, Toss G, Strom M. Reversal of osteopenia with diet in adult coeliac disease. Gut 1996;38:322-7.