

## Celiac disease and osteoporosis: A frequent but unrecognized association

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Over the past decade, the introduction of new serological tests for celiac disease has dramatically increased the frequency of its diagnosis, resulting in considerable changes in our clinical perspective of this disease. In particular, our concepts of the epidemiology and clinical picture of celiac disease have changed significantly. The face of celiac disease has changed, from an uncommon disease presenting in childhood, to a common disease that can present at any age and one that is frequently asymptomatic. Osteoporosis is one of the common complications of celiac disease and more often than not, this clinical association goes unrecognized. Furthermore, osteoporosis itself is also very often asymptomatic. Since both conditions are treatable, it is important for the clinician to recognize the association, diagnose unrecognized celiac disease, and institute appropriate management as early as possible.

### Celiac disease: A new face

Celiac disease is an autoimmune inflammatory disease of the small intestine in genetically susceptible individuals due to a lifelong sensitivity to dietary gluten, a wheat protein.<sup>1,2</sup> Other related proteins from rye and barley may also result in celiac disease. The disease is associated with intestinal malabsorption of a variety of nutrients, including iron, folic acid, calcium, and vitamin D, as well as other fat-soluble vitamins. Until recently, celiac disease was thought to be rare, but the advent of new serological screening tests have established that celiac disease is extremely common. Initially, the high incidence was recognized in Europe,<sup>3,4</sup> but it has now been confirmed in North America.<sup>5</sup> These studies estimate the prevalence of celiac disease to be between 1/120 and 1/300 of the population. This has stimulated increased interest in the disease, which in turn has sparked much investigation in the last few years and expanded our knowledge of the clinical features and pathogenesis, as well as the epidemiological aspects.

The histological hallmark of celiac disease is villous atrophy seen on small intestinal biopsy. However, other findings are also part of the histological picture, including lymphocytic invasion of the intestinal mucosa and crypt hyperplasia. Indeed, villous atrophy is not universally present. The pathogenesis of the mucosal changes that result in malabsorption is complex, involving increased mucosal permeability to gliadin, the generation of antibodies to gliadin and to tissue transglutaminase, the release of tissue metalloproteinases, and the activation of specific celiac T-cell epitopes. The latter two factors are the key players in causing the mucosal damage. The pathogenesis of celiac disease has been reviewed in detail by Schuppan.<sup>6</sup> The process also involves the local release of inflammatory cytokines such as interferon- $\alpha$ , IL-2 and IL-6.<sup>7,8</sup> Indeed, serum levels of IL-6 are increased in celiac patients,<sup>9</sup> and these



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## A CASE REPORT

A 42-year-old female physician with osteopenia and a family history of osteoporosis was referred for treatment. A routine bone densitometry revealed a lumbar spine t-score of  $-2.4$ . She was an athletic woman with regular menses and her past history was negative apart from mild anemia and some very intermittent low back pain. Her 76-year-old mother had known osteoporosis, and her 46-year-old sister had a similar degree of osteopenia. The physical examination was normal except that her thyroid was slightly enlarged, soft, and non-tender. She had a moderate scoliosis. The electrolytes, creatinine, calcium, phosphorus, alkaline phosphatase, total protein, albumin, 25OH-vitamin D and PTH were normal. The hemoglobin was 11.1, hematocrit .33, and ESR 10 mm/hr. The TSH was 11.9 mU/L, the free T4 9.6 pmol/l, and the microsomal and antithyroglobulin antibodies were both elevated.

When she returned 3 months later, she was clinically euthyroid and her abnormal thyroid indices had returned almost to normal. She was considered to have a resolving mild thyroiditis and her thyroid tests normalized on subsequent testing. However, at the 3-month visit, the results of a recent hemogram became available; it revealed anisocytosis, poikilocytosis, giant platelets, and Howell-Jolly bodies. She was referred to a hematologist who suspected celiac disease and referral to a gastroenterologist confirmed the diagnosis of celiac disease on small bowel biopsy.

This diagnosis had not been suspected at the initial two visits because of her robust general health and the total lack of any symptoms of intestinal disease. She was treated with a gluten-free diet, a calcium supplement, and alendronate 10 mg daily.

One year later, a small bowel biopsy showed histological improvement. Her 4 children all had positive endomysial antibody tests and presumptive celiac disease. Her older sister was found to have celiac disease by small bowel biopsy. The patient was also found to have IgA deficiency. Her father, also a physician, was known to have dermatitis herpetiformis and "gluten sensitivity." There was a weight gain of 2.2 Kg, and an increase of 4% in spinal bone density. After 2 years of treatment, there was a 12% increase in spinal BMD. The femoral neck BMD was consistently normal. Her older sister who also had osteopenia and chronic anemia, but was otherwise in similar good health, was treated with a gluten-free diet alone for 1 year and experienced increases in bone density of 7.3% and 11.5% at the lumbar spine and femoral neck, respectively.

This patient may not have been diagnosed with celiac disease had she not spontaneously developed hyposplenism, which together with the osteopenia and the family history led to the clinical suspicion of celiac disease that was subsequently confirmed by biopsy. She has responded appropriately to therapy and her future health prospects are excellent.

cytokines, which are known to stimulate bone resorption, have been implicated in the osteopenia of celiac disease.<sup>9</sup> The genetics of celiac disease are complex and multifactorial. The disease is strongly associated with certain HLA haplotypes, particularly DW-8.<sup>10</sup> Also, celiac-specific epitopes have been identified on activated T-cells.<sup>10</sup>

The clinical presentation of celiac disease is extremely variable. It can present in childhood, adulthood, and even in old age. Indeed the majority of cases are now diagnosed in adulthood. Celiac disease is very often asymptomatic, but commonly there is low body weight and anemia. The patient may complain of a lifelong "nervous bowel;" mild flatulence and diarrhea are frequent, but the disease may progress to severe diarrhea. The common clinical features of celiac disease are listed in Table 1. It may present with abnormalities in many systems.

**Table 1: Common clinical features of celiac disease**

Diarrhea	Recurrent miscarriage
Weight loss	Recurrent aphthous stomatitis
Anemia (iron, folate deficiency)	Short stature
Anorexia	Depression
Lassitude	Bone pain
Infertility	

There are many other associated conditions including other autoimmune diseases, type I diabetes, primary biliary cirrhosis, Sjögren's syndrome, autoimmune thyroid disease, rheumatoid arthritis, and IgA deficiency, as well as dermatitis herpetiformis, Down's syndrome, epilepsy, polyneuropathy, and other neurological abnormalities. Furthermore, there are a number

**Table 2: The effect of gluten free diet on parameters of calcium and bone metabolism**

	Pre-treatment	Post-treatment	P
BMD (gm/cm <sup>2</sup> )			
Lumbar spine	0.795±0.177	0.907±0.180	<.001
Femoral neck	0.741±0.189	0.818±0.145	0.002
Alkaline phosphatase (U/L)	72±24	62±12	0.006
25OHD (ng/ml)	20.1±13.8	23.2±1.32	n.s.
Serum ionic calcium (mmol/l)	1.19±0.02	1.20±0.02	n.s.
Urine calcium (mmol/24h)	2.95±1.20	4.60±1.32	<.001

Values for bone mineral density (BMD) by dual photon absorptiometry in 41 patients with newly diagnosed celiac disease treated with a gluten-free diet for a period of one year. From Ciacci et al<sup>18</sup> with permission. Note the significant improvement in bone mineral density at both spine and hip. There is no significant change in 25OHD levels, but urine calcium has increased significantly, suggesting an increase in intestinal calcium absorption.

of infrequent, but clinically important complications of celiac disease that are now recognized. These include ulcerative jejunitis, small bowel T-cell lymphoma, splenic atrophy, and the skeletal complications of osteomalacia and osteoporosis. Ulcerative jejunitis and small bowel lymphoma are rare complications that should be suspected in patients with intestinal bleeding or obstruction, or those who are refractory to treatment with a gluten-free diet, and these complications are related to the duration of untreated disease.<sup>11</sup> With such a wide variety of clinical presentations, and a high frequency of asymptomatic cases, particularly in adults, the diagnosis is often overlooked.

### The association between celiac disease and osteoporosis

The strong association between celiac disease and osteoporosis is now apparent from a number of reports. In two recent cross-sectional studies of affected patients, osteoporosis was initially present, as determined by densitometric criteria in 26% to 34% of celiac patients.<sup>12,13</sup> Increased fracture prevalence has also been observed in patients with celiac disease. In a cross-sectional study of 165 patients with celiac disease, 25% had experienced from 1 to 5 fractures compared to only 8% of controls.<sup>14</sup> Furthermore, it has now been noted in several studies that celiac disease is more common in patients with osteoporosis by 9- to 14-fold in comparison to the general population.<sup>15-17</sup> The association is likely caused in large part by malabsorption of calcium and vitamin D, but other nutrients such as vitamin K may also be important. It

appears that in many patients, particularly those with mild disease, calcium malabsorption plays a major role, (Table 2). On the other hand, a recent study by Nuti et al<sup>15</sup> demonstrates an impressively strong correlation between serum levels of antitransglutaminase and 25-hydroxyvitamin D across a wide range of values. In addition, the release of bone resorbing cytokines such as IL-6 from the inflamed mucosa are likely to play an important role as well, since they have been implicated in other bone diseases where increased bone resorption is present.

### Clinical presentations of bone disease in celiac disease

In celiac patients with low bone density, the classic presentation is of vitamin D deficiency osteomalacia, often with secondary hyperparathyroidism. However, we now see osteoporosis much more commonly. In these patients the serum calcium, alkaline phosphatase and PTH are normal and the 25OHD may be normal or slightly low. However, frank osteomalacia is still sometimes seen, with 25OHD values clearly in the deficient range and with elevated alkaline phosphatase and serum PTH. There is a wide spectrum of abnormalities of calcium and bone metabolism that can present initially, depending on the severity and duration of the disease (Table 3). These range from mild osteopenia to severe derangements in calcium metabolism and severe pain associated with symptomatic osteomalacia and pseudofractures. In the osteoporosis of celiac disease, excess fracture prevalence occurs between the ages of 20 and 40 years of age, as well as in those over 50.<sup>14</sup>

The features of osteoporosis that occur in association with celiac disease are distinctive in some respects.

- First, the osteoporosis is very often familial.
- Second, the osteoporosis may present at any age.

When celiac disease is present in childhood, there may

**Table 3: Calcium and bone disease in celiac disease: varied initial clinical presentations**

- Osteoporosis ± fractures
- Osteopenia ± loose stools or diarrhea
- Vitamin D deficiency
- Elevated alkaline phosphatase
- Hyperparathyroidism
- Symptomatic hypocalcemia and hypomagnesemia
- Asymptomatic

be short stature and failure to attain a normal peak adult bone mass. Growth delay is partly due to a delay in linear bone growth, and growth delay usually resolves on a gluten-free diet. Celiac disease may also attenuate growth hormone secretion.<sup>19</sup>

- Osteoporosis can affect patients of either sex. One should suspect celiac disease in men or premenopausal women with osteoporosis and in familial osteoporosis.

### Diagnosis of celiac disease

This broad clinical picture, together with the high prevalence of asymptomatic patients, particularly in adults, means that the most important aspect of diagnosis is a high index of clinical suspicion. In patients with osteoporosis, a history of minor bowel complaints such as frequent stools or flatulence should be pursued, as well as those with unexplained anemia or a family history of osteoporosis. The diagnosis is usually approached in a stepwise fashion.

The earlier widespread use of antigliadin antibodies as a screening test has given way to the much more sensitive and specific antiendomysial antibody test. The current approach to diagnosis has been reviewed.<sup>1,20</sup> The autoantigen that is the basis of the endomysial antibody test is tissue transglutaminase.<sup>21</sup> Recently an ELISA for transglutaminase has been developed and is becoming more widely used since it is much simpler to carry out.<sup>22</sup> The sensitivity and specificity of both these tests are impressive. Sensitivity for the more widely available endomysial antibody test is between 85%-98%, while that of the anti-transglutaminase test is 95%-98%. The specificity of both tests is 94% or better.<sup>1</sup> If the serological tests are positive in a significant titre, the diagnosis should be confirmed by small bowel biopsy.

### Histologic findings

Total villous atrophy is no longer regarded as essential for the diagnosis, but is part of the spectrum of findings indicating mucosal damage listed above. Indeed, it has been questioned if a positive endomysial or anti-transglutaminase test is sufficient to establish the diagnosis of celiac disease. At present, the small bowel biopsy remains the diagnostic gold standard,<sup>20,23</sup> but it should be rec-

ognized that occasionally the bowel histology may be normal in patients with many other hallmarks of the disease, and a repeat biopsy is sometimes useful in patients where the index of clinical suspicion is high.

Pitfalls in the diagnosis include the ameliorating effects of dietary gluten restriction on both abnormal serological test results and the histological picture on intestinal biopsy. In addition, since the antiendomysial and antitransglutaminase antibody tests are usually IgA-based, celiac patients with IgA deficiency may have negative tests. Thus, serum IgA should be measured when necessary by immunoelectrophoresis.

### Screening tests

Bone mineral density should be assessed in all patients with celiac disease and serological testing should be carried out in family members. In patients with osteoporosis, routine serological screening for antiendomysial or antitransglutaminase antibodies is not recommended and is still a matter of debate. However, since recent studies suggest prevalences of celiac disease in the range of 10% in osteoporotic patients,<sup>15-17</sup> larger studies may change the current recommendation. Nevertheless, serological screening in patients with osteoporosis should be carried out in those with diarrhea, even if mild or episodic. A detailed history pertaining to bowel habits should be obtained in all patients. Celiac antibody testing should be performed in premenopausal women or males with no other secondary causes of osteoporosis, and in familial osteoporosis. These tests are also suggested in patients who do not respond to adequate treatment for osteoporosis and in unexplained osteoporosis occurring in patients without any obvious clinical risk factors.

### Management of patients with celiac disease and osteoporosis

The treatment of celiac disease is a gluten-free diet and in patients with osteopenia, this is often sufficient to normalize bone density as early as one year after instituting therapy.<sup>24,25</sup> On the other hand, it is advisable in patients with prevalent fractures or other important risk factors for osteoporosis to institute treatment with bisphosphonates at least for some period of time.

The British Society of Gastroenterology has recently published a useful set of guidelines for treatment of these patients.<sup>26</sup>

It is important to realize that pharmaceutical treatment of osteoporosis in celiac patients without a gluten-free diet is doomed to failure. This emphasizes the importance of diagnosis and treatment of celiac disease in osteoporosis clinics. On a therapeutic diet, clinical symptoms of celiac disease should regress and serological tests should show a favourable response. An amelioration of the osteoporosis should be expected.

### Summary

Celiac disease is now recognized as a common and often asymptomatic disease that can be of major pathogenetic importance in patients with osteoporosis. A strong index of suspicion on the part of the clinician should lead to appropriate serological screening and confirmation of the diagnosis by small bowel biopsy. A gluten-free diet is essential in the management of osteoporosis in patients with celiac disease.

### A Question

#### What are the differences in racial incidence of celiac disease?

It is said that celiac disease is rare in non-Caucasians. However, this has not been extensively investigated. In many parts of the world, wheat may not be the most frequent grain consumed. With the availability of the new anti-transglutaminase ELISA, this question should be re-examined in future studies.

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## Abstracts of Interest

### Bone density and fractures in patients with coeliac disease

DAVIE MWJ, GAYWOOD I, MASUD T. VARIOUS CENTRES, U.K.

Patients with coeliac disease have low bone mineral density, but whether this is equally low at the spine or the upper femur and whether fracture rates are increased is uncertain. We studied 85 patients (M, 19, age 59.9±9.7 yr; F, 66, 55.4±11.4 yr) with coeliac disease. Fracture details and lifestyle data were collected by questionnaire; weight and height measured and bone mineral density measured by Hologic QDR4500W at L2-4 and the femoral neck. BMD was expressed as 'z' scores. All patients had been diagnosed by small bowel biopsy.

**RESULTS:** 28 women (42.4%) had 51 fractures and 7 men (36.8%) had 11 fractures. Women with fracture had low bone density at the femoral neck (FN) (mean -0.67, -.28:-1.06, 95%CI) but this not significantly lower than women without fracture (-0.57, -.23:-0.91) although both were significantly lower than a z score of zero ( $p < 0.05$ ). At the lumbar spine BMD was not low (-0.28, 0.03:-0.59,  $p = ns$ ); in women with fracture, BMD was low (-0.6, -0.22:-0.98,  $p < 0.05$ ) but it was not significantly different from the values in women without fracture. When BMD z score was related to age, values at LS in women under 50 yr were positive ( $0.25 \pm 0.8$ ; mean±SD) and fell progressively with age to  $-0.99 \pm 0.79$  in patients over 65yr ( $p$  for trend  $< 0.05$ ). Femoral neck BMD 'z' score was positively correlated with Body mass index ( $r = 0.25$ ,  $p < 0.02$ ) and with weight ( $r = 0.39$ ,  $p < 0.01$ ). In men bone mineral density z score was  $-0.71 \pm 0.88$  at the spine and  $-0.36 \pm 1.13$  at the femoral neck ( $p = ns$ ).

**CONCLUSIONS:** BMD is low in coeliac disease in women and correlates with body weight. Women under 50 yr do not have low BMD at the lumbar spine; women over 50 yr are at risk of losing bone in excess of that which might be expected. Whether this is caused by undertreatment or delay in diagnosis is uncertain.

Abstract presented at the 2001 Meeting of the International Bone & Mineral Society and European Connective Tissue Society, Madrid. *Bone* 2001;28:S212

### The prevalence of celiac disease in an osteoporotic population.

STENSON WF, R. D. NEWBERRY RD, LORENZ R, CIVITELLI R. VARIOUS CENTRES, USA.

The purpose of this study was to compare the prevalence of celiac disease in osteoporotic and non-osteoporotic populations. We screened a group of 84 osteoporotic volunteers (T-2.5) for celiac disease. Antigliadin IgA, antigliadin IgG, antiendomysial and anti-tissue transglutaminase antibodies were assessed. Volunteers with either a positive antiendomysial antibody or a positive tissue transglutaminase antibody underwent endoscopic biopsy of the duodenum to confirm the diagnosis of celiac disease. Of the 84 volunteers with osteoporosis 4 had both a positive antiendomysial antibody and a positive anti-tissue transglutaminase. Endoscopic biopsies confirmed the diagnosis of celiac disease. Two of these 4 volunteers had the lowest T-scores among the 224 volunteers tested (T < -4.37, and T < -4.48). Among the 140 volunteers without osteoporosis only one had a positive antiendomysial antibody and a positive tissue transglutaminase antibody. This volunteer was osteopenic with a T-score of -1.78. Endoscopic biopsy confirmed the diagnosis of celiac disease. There were a total of 5 volunteers with positive antiendomysial antibodies, all five were also positive for antigliadin IgG, antigliadin IgA and anti-tissue transglutaminase antibodies. There were, however, a large number of volunteers who were positive for antigliadin IgG or antigliadin IgA antibodies who were negative for antiendomysial and anti-transglutaminase antibodies. These patients were distributed in

both the osteoporotic and non-osteoporotic groups. This study leads to the following conclusions: 1) The prevalence of celiac disease in the osteoporotic population was 4.7%. This is at least 14-fold greater than the reported prevalence of celiac disease in the United States population. If these numbers are confirmed in a larger study it would be reasonable to recommend that all patients with osteoporosis be screened for celiac disease. 2) Screening with anti-tissue transglutaminase antibody or antiendomysial antibody is more specific than screening with antigliadin antibodies.

Abstract presented at the 2001 Meeting of American Society of Bone Mineral Research, Phoenix, AZ.

*J Bone Min Res* 2001;16(suppl 1):S519

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