Osteoporosis and celiac disease: is it useful to a new guideline?

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Celiac disease (CD) is a permanent glutensensitive enteropathy characterised by reversible small-bowel mucosal atrophy in a genetically predisposed person, resulting from an inappropriate immune response to dietary gliadin, a component of wheat proteins. CD can have a varied clinical presentation, with most symptoms being attributed to malabsorption [1]. CD has long been known to cause metabolic bone disease. Early reports focused mainly on the association with vitamin D deficiency and osteomalacia, but more recent attention has turned to Osteoporosis (OS) [2,3]. OS can be reliably detected by measurement of bone mineral density (BMD), which can be expressed as the number of standard deviation (SD) above or below the mean BMD for young adults (T score) or the mean BMD for age matched controls (Z score). A BMD more than 2.5 SD below the mean for a young adult is generally taken to indicate OS [4]. Stratification for fracture risk is possible using BMD. The risk increases roughly twofold for each SD decline in BMD below the population mean. The risk of low BMD seems to rise with increasing age at diagnosis, decreased body weight and in postmenopausal women [5]. Low BMD can be detected even in children and adolescents with newly diagnosed CD [6]. Furthermore, the need to consider CD as a pathogenetic factor in individuals presenting with OS has been emphasised; this particularly applies to those with clinical features of the disease and those who fail to respond to treatment for their OS. As the risk of sustaining an osteoporotic fracture is estimated to double with each standard deviation decrease in bone mineral density [7], various groups, including the British Society of Gastroenterology [8], have recommended that adult celiacs should have regular bone scans and if OS is detected prolonged treatment with hormone replacement therapy or bisphosphonates. The clinical significance of the observed reduction in bone mineral density associated with CD is unclear. While a small proportion of patients undoubtedly develop severe OS with multiple fragility fractures, the question of whether fracture risk is significantly increased across the clinical spectrum of CD has only recently been explored. Studies in small numbers of selected patients, drawn from hospital clinics, have suggested that fracture risk is increased; however, the clinical importance of the reduction in bone mineral density demonstrated is unclear as few studies have addressed the actual fracture risk in patients with CD. Mather et al. [9] studied 100 patients with low BMD who were screened for the presence of IgA anti-EMA. Seven patients tested positive for IgA anti-EMA at low titers (1:20). All patients underwent upper endoscopy and biopsy; none were given the diagnosis of CD. Gonzalez et al. [10] screened 127 Argentinian postmenopausal osteoporotic women and 747 control women for the presence of IgA and IgG antigliadin antibodies and IgA anti-EMA; individuals with positive serologic test results underwent endoscopy with small bowel biopsy. Fischer et al. screened the serum of 347 consecutive older patients (60 years of age) with hip fracture (74% females; age range 60–101 years, mean age 81.5 (SD 7.3) years) for the presence of IgA endomysial antibodies (EMA), IgA and IgG gliadin antibodies (IgA-AGA and IgG-AGA), and total IgA. In 13% of patients, the IgA-AGA test was positive (above 34 ELISA units) while in 11% of patients the titre of IgG-AGA was slightly elevated (above 46 ELISA units). Thomason et al. [11] found only small and statistically non-significant increases in their bone fracture risk or in their risk of “low trauma” fractures compared with the general population. In particular, fractures of the hip and forearm were reported with a similar frequency in patients with CD and controls. It is not surprising that no significant increase in fracture could be detected in this population of well treated celiacs, given previous findings. The American Gastroenterology Association recently reviewed studies of OS in gastrointestinal diseases, including CD [12], according to standard levels of evidence. All such studies have shown low mean bone mineral density (BMD) around the time of diagnosis of untreated CD, with a pooled analysis showing very low bone mass (age and sex adjusted z scores below –2) in 40% in the spine and 15% at the hip [13]. The relative and absolute risks of fractures were well described in an important population based cohort of patients with 4732 disease, using data from the UK General Practice Research Database between 1987 and 2002, by West et al. [14]. These authors also published data on the risks of malignancy in the same cohort of patients with disease over the same period. The question as to whether the severity of the symptoms relates to the degree of bone loss has been specifically dealt with only in a few studies. Väldimarsson et al. [15] reported patients with persistent villous atrophy and noticeable osteopenia. Most were not compliant with gluten free diet as their symptoms were only mild. 63 of the same group later found that in a cohort of 63 patients with CD, although very low BMD was seen in 20%, symptom severity (presence of symptoms of malabsorption) had no influence on BMD. Mazure et al. [16] however, when directly comparing symptomatic to asymptomatic patients found that the symptomatic patients had significantly lower BMD. Effective treatment strategies for OS in individuals with CD have not been defined. It seems reasonable to advocate similar lifestyle measures to those recommended in postmenopausal OS; these include vitamin D repletion where required and maintenance of adequate calcium intake, using calcium supplements if necessary. While an increase in fracture risk cannot be excluded on the basis of these studies, it can be concluded that the magnitude of any increase, if present, is small and thus for the majority of individuals with CD absolute risk of fracture is low. Furthermore, prospective studies have shown significant improvement in bone mineral density after introduction of a gluten free diet, indicating that bone densitometry at diagnosis may considerably overestimate short term fracture probability. From the above it seems logical to think and believe that CD are predisposing to an alteration of metabolic bone that can lead to OS and osteopenia. Numerous studies have been performed to show statistically the relationship between CD, OS and fracture risk, but they seem almost nonexistent histochemical studies on the relationship between values-based serological IgG antigliadin, IgA antigliadin, IgA antitissue transglutaminas, and IgA antidendymosal anti values and DEXA bone related to a set of symptoms. In short, it still seems like the open discussion of CD can be measured with blood tests can predict with statistical values influence the quality of bone tissue. So the OS would be preventable by knowing the probability that it occurs in certain situations and gastric bleeding in both patients with

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newly diagnosed CD, both in patients who experience gastrointestinal symptoms although the gluten-free diet. At the present time therefore available evidence indicates that the risk of fragility fractures is, at most, only slightly increased in coeliac disease and that the absolute risk of fracture in the majority of these individuals is low. Bone densitometry should therefore be reserved for the minority with a high fracture probability, selected on the basis of risk factors. Screening of all patients with CD, as currently recommended in the British Society of Gastroenterology guidelines, cannot be justified and represents an inappropriate use of resources. The creation of a new guideline would be a valuable tool for prevention, knowing now, CD and OS.

References