



Managing Bone Health in Pediatric Celiac Disease: Effects of a Gluten-free Diet and Calcium-vitamin D Therapy

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ABSTRACT

Aim: Celiac disease is an autoimmune disease affecting individuals of all ages, causing damage to the small intestines upon consuming gluten. This study aimed to assess changes in bone mineral density among children with celiac disease following dietary intervention and treatment compared to their pre-intervention levels, and also to determine the frequency of metabolic bone disease at the time of diagnosis.

Materials and Methods: This study included pediatric patients with biopsy-proven celiac disease who underwent dual-energy X-ray absorptiometry at diagnosis and after 12 months. Anthropometric measurements, serum calcium, phosphorus, alkaline phosphatase, parathyroid hormone, and 25-hydroxyvitamin D levels were recorded. Lumbar spine (L1-L4) bone mineral density was measured using a dual-energy X-ray device. Anthropometric measurements, dual-energy X-ray absorptiometry results, and biochemical laboratory findings were evaluated before and after treatment. All patients received standardized vitamin D (400-2,000 IU/day based on their deficiency status) and calcium supplementation (age-appropriate daily intake 800-1,300 mg/day) based on their baseline deficiency status, and their adherence to a gluten free diet was verified by clinical improvement and negative anti-tissue transglutaminase IgA at follow-up.

Results: Sixty children (36 female, 24 male; mean age 8.82±3.90 years) were included in this study. At the initial evaluation, low bone mineral density was identified in 25% of the patients. During follow-up, some patients demonstrated worsening dual-energy X-ray absorptiometry findings despite adherence to the diet. Further assessment revealed that these patients had vitamin D deficiency and were non-compliant with the prescribed supplementation.

Conclusion: These findings highlight the critical role of dietary management and appropriate supplementation in managing celiac disease, emphasizing the necessity for dual-energy X-ray absorptiometry screening at diagnosis and follow-up.

Keywords: Bone mineral density, calcium, celiac disease, malabsorption, vitamin D

Introduction

Celiac disease (CD) is an autoimmune disorder in which gluten exposure leads to immune-mediated damage of the small intestine in genetically susceptible individuals. Once manifested, CD becomes a lifelong condition, and the only

effective treatment is a gluten-free diet (GFD) (1). The global prevalence of CD is approximately 1% (2).

Classical symptoms include failure to thrive, chronic diarrhea, and weight loss; however, many patients present with fatigue, bloating, constipation, abdominal pain, or metabolic

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bone disease (MBD) (3). The mechanisms underlying MBD in CD are multifactorial and not fully understood. Proposed contributors include autoimmune activity, circulating inflammatory cytokines, and impaired absorption of key nutrients such as vitamin D, magnesium, phosphorus, and calcium (4). Local and systemic inflammation, particularly involving tumor necrosis factor- α , interleukin-1 (IL-1), and IL-6, affects bone metabolism in children with CD. Alterations in the RANK/RANKL/OPG pathway have been reported, with CD patients demonstrating elevated OPG and RANK levels and a reduced OPG/RANKL ratio, which correlates with spine bone mineral density (BMD) and IL-6 levels. The clinical relevance of OPG autoantibodies remains unclear due to conflicting findings (5,6).

MBD, a potential complication of CD, encompasses disorders which disrupt bone mineral homeostasis (7). It may be asymptomatic or present with bone pain, vertebral compression, or long-bone and vertebral fractures. Diagnosis relies mainly on dual-energy X-ray absorptiometry (DXA), the gold standard for assessing bone mineral content (BMC) and BMD. BMC reflects the mineral amount in a specific region, while BMD is calculated by dividing BMC by bone area. Although DXA is recommended for monitoring children at risk of MBD, such as those receiving long-term parenteral nutrition (8), there are no specific guidelines for routine BMD assessment in pediatric CD cases.

Children with CD, who are at increased risk for impaired bone health, require careful evaluation in order to prevent MBD and guide appropriate treatment. The primary aim of this study was to assess changes in BMD after dietary and medical intervention compared with baseline values. The secondary aim was to determine the frequency of MBD at diagnosis in children with CD.

Materials and Methods

Patients were eligible for inclusion if they had a biopsy-proven diagnosis of CD and were between 2 and 17 years of age at the time of this diagnosis at Sivas Numune Hospital, Clinic of Pediatric Gastroenterology, Hepatology, and Nutrition, between the dates of January 2021 and June 2023. The inclusion criteria also required the availability of baseline laboratory tests, DXA measurements, and at least one follow-up evaluation.

Exclusion criteria included the presence of chronic systemic diseases (e.g., endocrine disorders, renal disease, inflammatory disorders) which may affect bone metabolism, the use of medications known to influence BMD, incomplete medical records, or a lack of follow-up DXA evaluation.

Adherence to a GFD was confirmed by clinical assessment and negative anti-tissue transglutaminase immunoglobulin A (anti-TTG IgA) levels at follow-up. Only those patients with confirmed compliance were included in the longitudinal analysis.

The diagnosis of patients was based on positive celiac serology results and histopathological findings from upper gastrointestinal endoscopy, where biopsy specimens were scored according to Marsh classification as being 2 or 3.

Serum levels of calcium, phosphorus, magnesium, alkaline phosphatase (ALP), parathyroid hormone (PTH), and 25-hydroxyvitamin D3 [25(OH)D3], as well as BMD z-score results at the time of diagnosis, were reassessed after 1 year of adherence to a GFD. Serum 25(OH)D3 levels below 20 ng/mL were considered indicative of vitamin D deficiency.

We conducted DXA scans. Measurements of the lumbar spine included L1-L4. The DXA scans of the spines were analyzed in order to determine BMD (g/cm^2). L1-L4 z-scores were determined using standard deviation (SD) values of areal BMD (g/cm^2) for Turkish children, adjusted for height-age and gender. Patients with a BMD z-score ≤ -2 SD were categorized as the low BMD group, those with z-scores between -1 and -2 were categorized as the high-risk of low BMD group, and those with z-scores ≥ -1 were categorized as the normal group in L1-L4 measurement area. Following baseline evaluation of BMD and serum 25(OH)D3 levels, all patients received individualized supplementation. Those patients with vitamin D deficiency [25(OH)D3 < 20 ng/mL] were prescribed 1,000-2,000 IU/day of vitamin D, while those with normal levels received 400-800 IU/day. Calcium supplementation was provided according to age-specific daily requirements (4-8 years: 800 mg/day; 9-18 years: 1,300 mg/day). Supplementation was adjusted based on follow-up biochemical results and administered under the guidance of a pediatric endocrinologist.

Anthropometric measurements including weight, height, body mass index (BMI), and z-scores for these parameters at the time of diagnosis and during follow-up were retrieved from the patient records. Weight, height, and BMI z-score values were computed using the tool provided by the Turkish Pediatric Endocrinology and Diabetes Association (CHILD METRICS, <https://www.ceddcozum.com/>).

This study was approved by the Sivas Cumhuriyet University Non-Interventional Clinical Research Ethics Committee (approval number: 2024-02/20, date: 22.02.2024), and informed consent was obtained from the parents of each pediatric patient.

Statistical Analysis

Statistical analysis was conducted using the SPSS-IBM, version 22 (SPSS Inc., Chicago, Illinois, USA). All eligible patients were included in the analysis. Comparisons between the initial visit and follow-up visit were conducted using the Chi-square test for gender, Paired Samples t-test (mean±SD) for normally distributed countable variables, and Wilcoxon test [median (min-max)] for non-normally distributed countable variables.

Correlations between BMD z-scores, anthropometric and biological parameters were evaluated using Pearson's rank correlation coefficient in the high-risk of low BMD and the low BMD groups. A value of $p < 0.05$ was considered statistically significant.

Results

Sixty children with CD participated in this study. The mean age of the children was 8.82 ± 3.90 years. The youngest child was 2.44 years old, and the oldest was 16.23 years old. Of the children included, 36 (60%) were female, and 24 (40%) were male.

The initial and follow-up median weights of the patients were 22.12 kg (range: 11.45-70.75) and 25.35 kg (range: 15.50-68.90), respectively. The initial and follow-up mean heights

of the patients were 124.82 ± 21.61 and 132.75 ± 20.35 cm, respectively. The initial and follow-up median BMI of the patients were 15.69 (range: 12.41-28.34) and 16.80 g/cm² (range: 14.05-28.3), respectively. A statistically significant differences were found between these data. Over the course of a year adhering to a GFD, there was a notable increase observed in weight, height, and BMI measures. However, there was no statistically significant difference found between the z-score of weight, height, and BMI. The anthropometric measurements of the patients which were recorded at diagnosis and at follow-up are presented in Table I.

At the initial visit and follow-up visit, the levels were as follows: calcium levels were 9.73 ± 0.55 and 9.91 ± 0.38 mg/dL, phosphorus levels were 4.84 ± 0.81 and 4.68 ± 0.50 mg/dL, magnesium levels were 1.99 ± 0.12 and 1.97 ± 0.20 mg/dL, ALP levels were 195.21 ± 74.02 and 209.55 ± 86.35 U/L, PTH levels were 37.60 (range: 11.40-176.60) and 26.85 (range: 10.10-96.60) pg/mL, and 25(OH)D3 levels were 17.55 ± 7.37 and 22.56 ± 8.79 ng/mL, respectively. Among these laboratory parameters, only the 25(OH)D3 levels were found to be statistically significantly higher at the follow-up visit when compared to the initial visit levels ($p = 0.024$). The laboratory measurements of the patients at diagnosis and at follow-up are presented in Table II.

Table I. Anthropometric measurements of the patients at diagnosis and at follow-up

	Initial visit	Follow-up visit	p value
Weight (kg)	22.12 (11.45-70.75)*	25.35 (15.50-68.90)*	<0.001
Weight z-score	-0.83±1.19	-0.68±1.15	0.300
Height (cm)	124.82±21.61	132.75±20.35	<0.001
Height z-score	-0.96±0.94	-0.79±0.95	0.101
BMI	15.69 (12.41-28.34)*	16.80 (14.05-28.3)*	0.0010
BMI z-score	-0.52±1.18	-0.22±1.11	0.147

Paired Samples t-test (mean±SD), *Wilcoxon test [median (min-max)], p value <0.05 significant
BMI: Body mass index, kg: Kilogram, cm: Centimeter, SD: Standard deviation, min-max: Minimum-maximum

Table II. Laboratory measurements of the patients at diagnosis and at follow-up

	Initial visit	Follow-up visit	p value
Calcium (mg/dL)	9.73±0.55	9.91±0.38	0.250
Phosphorus (mg/dL)	4.84±0.81	4.68±0.50	0.715
Magnesium (mg/dL)	1.99±0.12	1.97±0.20	0.224
ALP (U/L)	195.21±74.02	209.55±86.35	0.134
PTH (pg/mL)	37.60 (11.40-176.60)*	26.85 (10.10-96.60)*	0.050
25(OH)D3 (ng/mL)	17.55±7.37	22.56±8.79	0.024

Paired samples t-test (mean ±SD), *Wilcoxon test [median (min-max)], p value <0.05 significant
ALP: Alkaline phosphatase, PTH: Parathyroid hormone, 25(OH)D3: 25-hydroxyvitamin D3, SD: Standard deviation, min-max: Minimum-maximum

The mean L1-L4 BMD z-scores of the patients at the initial visit and follow-up visit were calculated as -1.09 ± 1.08 and -0.79 ± 1.29 , respectively, and no statistically significant difference was found. The BMD z-score results of the patients at the time of diagnosis and at follow-up are presented in Table III.

When the patients were categorized into 3 groups based on their L1-L4 BMD z-score, we found 24 (40%) patients were in the normal group, 21 (35%) patients were in the high-risk of low BMD group, and 15 (25%) patients were in the low BMD group at their initial visit. At the follow-up visit, there were 27 (45%) patients in the normal group, 21 (35%) patients in the high-risk of low BMD group, and 12 (20%) patients in the low BMD group (Table IV).

During the follow-up visit, analysis of BMD z-scores revealed that 3 patients improved from the high-risk of low BMD group to the normal group, and 3 patients progressed from the low BMD group to the normal group.

In the high-risk of low BMD group, there was no correlation between BMD z-scores and the other anthropometric or biochemical parameters. In the low BMD group, a strong positive correlation was observed between BMD z-scores and calcium levels (r -value=0.975, $p=0.005$).

Table III. The bone mineral density results of the patients at diagnosis and at follow-up

	Initial visit	Follow-up visit	p value
L1-L4 BMD z-score	-1.09 ± 1.08	-0.79 ± 1.29	0.098

Paired samples t-test (mean \pm SD), $p < 0.05$ significant
BMD: Bone mineral density, SD: Standard deviation

Table IV. Distribution of bone mineral density measurements in lumbar region (L1-L4)

	Normal group: n (%) ($-1 < Z$ -score < 1)	Low bone mineral density group: n (%) ($-2 < Z$ -score < -1)	High-risk group of low bone mineral density: n (%) Z -score < -2
Initial visit	24 (40)	21 (35)	15 (25)
Follow-up visit	27 (45)	21 (35)	12 (20)

Discussion

Our study shows that children newly diagnosed with CD have significantly reduced BMD, and although a GFD combined with calcium-vitamin D supplementation improves biochemical parameters, short-term recovery of BMD remains limited. These findings emphasize the importance of early DXA assessment and close monitoring of vitamin D status during follow-up in children with CD.

CD is a chronic immune-mediated disorder caused by gluten ingestion, leading to mucosal damage and nutrient malabsorption. It is associated with HLA-DQ2/DQ8 molecules which activate T lymphocytes and trigger autoimmune injury, resulting in villous atrophy (1,9). CD affects individuals of all ages; in our cohort, 60% were female and 40% male, with ages ranging from 2.44 to 16.23 years, which is consistent with previous studies (2,10,11).

CD is increasingly recognized as an important cause of MBD. Malabsorption of calcium and vitamin D contributes to secondary hyperparathyroidism and reduced bone mineralization. Osteopenia, osteoporosis, and fractures are well-known skeletal complications (12). In our study, 25% of the children had low BMD and 35% were at high risk at diagnosis, supporting earlier findings reporting osteoporosis rates between 27.5% and 44% (11,12). Consistent with these observations, Zacay et al. (13) demonstrated an increased risk of fractures among children with CD, both before and after diagnosis, indicating that impaired bone quality may precede diagnosis and may persist despite treatment. After GFD and supplementation, BMD improved modestly, with 20% having low BMD and 35% remaining high risk. Similar improvements have been described in other studies (14-17). In line with our findings, a recent systematic review and meta-analysis by Oliveira et al. (18) reported that GFD significantly increases both BMD and BMC in children and adolescents with CD, although values often remain lower than those of healthy controls.

While no correlation was identified between BMD z-scores and other parameters in the high-risk group, significant positive correlations were found between BMD z-scores and calcium levels ($r=0.975$, $p=0.005$) in the low BMD group. These findings may indicate that children presenting with low BMD may particularly benefit from targeted nutritional and therapeutic interventions to improve bone outcomes. Trovato et al. (19) similarly reported no correlation between anti-TTG IgA levels and BMD z-scores, although some studies report conflicting results, likely due to differing patient populations (20,21).

Vitamin D-dependent intestinal calcium absorption is essential for bone mineralization. In our cohort, 25(OH)D3 levels significantly increased at follow-up, likely reflecting supplementation and GFD adherence.

Bone turnover is affected by numerous biological and environmental factors beyond malabsorption. Given the limited sunlight exposure in our geographic region, vitamin D levels may not adequately improve with diet alone; therefore, supplementation appears necessary. Geographic variation should be considered when planning supportive therapy in CD.

Although improvements in height, weight, and BMI were observed, rapid linear growth may outpace bone mineralization, delaying measurable improvements in BMD. Increased needs for calcium and vitamin D, time required for bone matrix formation, and growth plate maturation may explain the slow normalization of bone parameters.

Study Limitations

This study was limited by its small sample size, single-center design, and short follow-up period. In particular, the 1-year follow-up may be insufficient to fully assess changes in pediatric BMD, as bone mineralization can require longer periods to show significant improvement. Additionally, physical activity, sunlight exposure, pubertal stage, and adherence to supplementation were not fully evaluated and may have influenced outcomes.

Conclusion

Adequate intake and absorption of calcium, phosphorus, and vitamin D are essential for maintaining bone health, especially in CD where malabsorption may occur. Regular DXA evaluation and appropriate preventive or therapeutic strategies are crucial. DXA should be performed at diagnosis and periodically thereafter in order to effectively monitor bone health in children with CD.

Ethics

Ethics Committee Approval: This study was approved by the Sivas Cumhuriyet University Non-Interventional Clinical Research Ethics Committee (approval number: 2024-02/20, date: 22.02.2024).

Informed Consent: Informed consent was obtained from the parents of each pediatric patient.

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Footnotes

Authorship Contributions

Surgical and Medical Practices: E.K.T., S.E., Concept: E.K.T., S.E., Design: E.K.T., S.E., Data Collection or Processing: E.K.T., S.E., Analysis or Interpretation: E.K.T., S.E., Literature Search: E.K.T., S.E., Writing: E.K.T., S.E.

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