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Nutritional Deficiencies in Celiac Disease: A Cross-Sectional Study of Iron and Vitamin B12 Status in Diyala, Iraq

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ABSTRACT

Background: Celiac disease (CD) is an autoimmune disorder triggered by gluten ingestion, leading to chronic inflammation and small intestinal damage. The prevalence of CD in Iraq, particularly in Diyala province, has significantly increased in recent years. Nutritional deficiencies are common among CD patients due to impaired nutrient absorption resulting from intestinal damage .
Objective: This study aimed to characterize iron and vitamin B12 status in Diyala's CD population.
Methods: This cross-sectional study assessed iron and vitamin B12 status in 90 CD patients and 30 healthy controls at AL Shams Medical Labs, Diyala, between October 2024 and May 2025.
Results: The mean age of CD patients was 48.03 ± 1.84 years, while controls had a mean age of 36.33 ± 2.32 years. The results showed a significant increase in anti-tissue transglutaminase antibodies (tTG-IgA and tTG-IgG) and I-FABP levels in CD patients compared to healthy controls. Additionally, serum iron and ferritin levels were significantly lower, while total iron-binding capacity (TIBC) was significantly higher in CD patients ($P < 0.05$). Although mean vitamin B12 levels were lower in the CD patient group, this difference was not statistically significant ($P = 0.114$).
Conclusion: Overall, CD significantly affects iron metabolism, whereas vitamin B12 levels appear less consistently impacted, emphasizing the need for targeted nutritional monitoring.

Keywords: Celiac Disease; I-FABP, Nutritional Deficiencies; Iron; Vitamin B12.

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INTRODUCTION

Celiac disease (CD) is a chronic autoimmune enteropathy triggered by gluten consumption in genetically predisposed individuals. Because the only effective treatment is permanent adherence to a gluten-free regimen, the challenge of managing the condition becomes apparent, along with the necessity to evaluate health-related quality of life in CD patients [1]. Globally, its prevalence is estimated at around 1% [2], but in many developing countries, including Iraq, the disease remains underdiagnosed and poorly managed. Limited awareness among healthcare providers, lack of screening programs, and similarities with other gastrointestinal disorders pose significant challenges to timely diagnosis and treatment in Iraq. Since CD is associated with injury to the small intestinal mucosa, its typical presentation primarily involves gastrointestinal nutrient malabsorption. Common symptoms include persistent diarrhea, abdominal bloating and discomfort, weight loss, and growth reduction in infants [3,4].

Nevertheless, individuals with CD often face various extraintestinal manifestations and disorders, such as persistent fatigue, depression, osteoporosis, anxiety, impaired fertility and sex desire, especially among women [3]. Although CD typically involves a wide range of symptoms, some individuals remain without noticeable symptoms, even when intestinal mucosal injury is present [5]. Those individuals are at an increased risk of complications, as they often fail to perceive the clinical progression of CD and are generally less responsive to the therapy [6].

Iron deficiency anemia (IDA) remains the most prevalent extra-intestinal manifestation, affecting approximately 40% of CD patients at diagnosis, while vitamin B_{12} deficiency occurs in 41% of untreated cases [7,8]. These deficiencies stem not only from malabsorption due to mucosal damage but also from chronic inflammation-mediated mechanisms, including hepcidin dysregulation and cytokine-driven iron sequestration [8].

Recent studies in Middle Eastern populations reveal distinct patterns of micronutrient depletion. In Iraq, CD patients with Marsh III histological severity demonstrated a 59% prevalence of low ferritin levels and significant hemoglobin reductions, particularly among those with poor dietary compliance [9]. This geographical variation highlights the interplay between genetic predisposition, dietary habits, and environmental factors in shaping nutritional outcomes. Despite histological recovery post-GFD, up to 33% of pediatric patients continue showing iron deficiency, and 21.1% exhibit vitamin D insufficiency, suggesting persistent enterocyte dysfunction or inadequate dietary compensation [10]. The nutritional landscape in Iraq presents unique challenges for CD management. Traditional Iraqi diets heavily reliant on wheat-based staples may complicate GFD adherence, while limited access to fortified gluten-free products exacerbates deficiency risks. This study therefore aims to characterize iron and vitamin B_{12} status in Diyala's CD population.

METHODOLOGY

This cross-sectional comparative study was conducted on celiac patients and healthy controls at Al Shams Medical Labs in Diyala City between 1 October 2024 and 10 May 2025. Diagnosis of celiac disease (CD) in patients was confirmed using serological tests for anti-tissue transglutaminase (anti-tTG) antibodies and intestinal fatty acid-binding protein (I-FABP). (I-FABP). Participants were excluded if they did not have confirmed celiac disease or had conditions that could confound iron or vitamin B_{12} levels, such as chronic kidney disease, liver disease, hematological disorders, or pernicious anemia. Individuals who had received blood transfusions or taken iron or B_{12} supplements within the past three months were also excluded. Additionally, pregnant or lactating women, patients with malignancies or severe chronic illnesses, and those who did not provide informed consent were not included in the study.

Blood Samples Collection

Nearly 4 mL of blood was collected from the CD patients and an equal volume from healthy controls under strictly aseptic conditions. The blood samples were then transferred into gel-containing tubes and left to clot at normal room temperature. Subsequently, they were centrifuged at 2000 rpm for 15 minutes, after which the resulting serum was aliquoted into 4 Eppendorf tubes. These tubes were then stored at −20°C to be analyzed later for the detection of iron, ferritin, total iron-binding capacity (TIBC) as well as vitamin B12.

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Serological Assessments

The tTG-IgA and tTG-IgG antibodies were detected using the Chorus® instrument (Diesse Diagnostica Senese, Italy), which employs an immunoenzymatic method based on mono-test devices, following the manufacturer's instructions. Intestinal Fatty Acid-Binding Protein (I-FABP) levels were determined using a commercial ELISA kit (manufacturer: DIALAB, Austria), according to the enzyme-linked immunosorbent assay protocol provided by the manufacturer. Serum iron and TIBC levels were measured using the cobas® c 311 analyzer (Roche Diagnostics, Germany) following the manufacturer's protocol. Serum ferritin levels were evaluated using the cobas® E411 analyzer (Roche Diagnostics, Mannheim, Germany), which utilizes the electrochemiluminescence immunoassay (ECLIA) technique. Serum vitamin B_{12} levels were measured using the VIDAS® system (bioMérieux, France), which is based on an enzyme-linked fluorescent assay (ELFA) technique, following the manufacturer's instructions.

Ethical Approval

The study was approved by the Ethics Committee of the College of Medical and Health Techniques, University of Bilad Alrafidain (No. E-503/2024-8-15), and conducted in accordance with the Declaration of Helsinki. All participants provided informed written consent. Data were collected at Al Shams Medical Laboratories, licensed by the Iraqi Ministry of Health, from patients who voluntarily underwent diagnostic testing as part of routine clinical care.

Statistical Analysis

The data were analyzed using SPSS version 26.0. Descriptive statistics were presented as mean ± standard error of the mean. An independent t-test was employed to compare means between groups, with a p-value < 0.05 considered statistically significant.

RESULTS

Demographic characteristics of the study groups

A total of 120 individuals were enrolled in this study, comprising 90 patients diagnosed with celiac disease (CD) and 30 clinically healthy controls. The average age of CD patients was 48.03 ± 1.84 years, while that of the control group was 36.33 ± 2.32 years.

Serological Assessment of tTG-IgA, tTG-IgG, and I-FABP in Celiac Disease and Control Groups

The findings indicate a significant elevation in TTG-G and TTG-A antibody levels among celiac disease patients compared to healthy controls. Specifically, the TTG-G levels in patients (40.04±3.38 IU/mL) were markedly higher than in controls (8.89±1.09 IU/mL, P = 0.001). Likewise, TTG-A levels were significantly increased in patients (45.78±4.64 IU/mL) compared to controls (3.75±0.42 IU/mL, P = 0.001) (Table 1). In addition, serum levels of I-FABP were significantly higher among patients (5.10±2.33 ng/mL) as compared to control groups (1.50 ± 0.73 ng/mL, P = 0.001) (Figure 1).

Table 1: Serum levels of Tissue Transglutaminase Antibody Levels among Study Groups.

tTG Tests (Normal Range)	Group	N	Mean	Std. Error Mean	*P*-value
TTG-G **Negative: < 20 IU/mL** **Positive: ≥ 20 IU/mL**	Patients	90	40.0412	3.38725	0.001
	Controls	30	8.8967	1.09040	
TTG-A **Negative: < 9 IU/mL** **Equivocal:10-15 IU/mL** **Positive > 16 IU/mL**	Patients	90	45.7880	4.03553	0.001
	Controls	30	4.6407	0.62913	
***P*i value <i 0.05**				* i *Independent.-samples* i *T test*	

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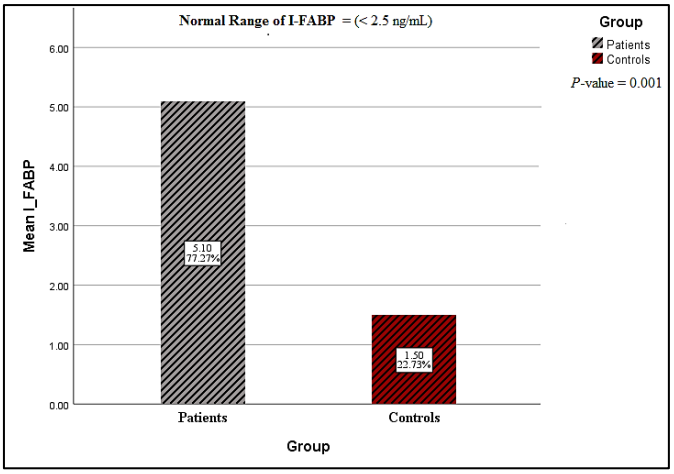

Figure 1: Serum Levels of I-FABP in Celiac Disease Patients Compared to Healthy Controls

Serological Assessment of Iron, TIBC, and Ferritin in CD Patients and Controls

The results of this study demonstrate significant alterations in iron metabolism in patients with celiac disease. Specifically, iron levels were significantly lower in the patient group (10.96±0.63 µg/dL) compared to healthy controls (17.86±1.05 µg/dL), indicating a potential deficiency in iron status (*P*=0.002). Similarly, ferritin levels were significantly reduced in patients (31.73±6.06 µg/L) compared to controls (82.76±5.19 µg/L), reflecting diminished iron reserves (*P*=0.001). Additionally, TIBC was significantly elevated in the patient group (76.44±3.22 µg/L) compared to controls (45.86±1.63µg/L) (*P*=0.001), suggesting increased iron-binding capacity, likely due to impaired iron utilization (Table 2).

Table 2: Serum levels of Iron, Ferritin, and TIBC Levels among Study Groups.

Iron Panel (Normal Range)	Group	N	Mean	Std. Error Mean	*P*-value
IRON	Patients	90	10.9662	0.63201	0.002
(10 -30 µg/dL)	Controls	30	17.8667	1.05671	
FERRITIN	Patients	90	31.7388	6.06057	0.001
(20 -300 µg/L)	Controls	30	82.7667	5.19298	
TIBC	Patients	90	76.4417	3.22072	0.001
(10 -70 µg/dL)	Controls	30	45.8667	1.63351	
***P* value < 0.05**				* *Independent-samples T test*	

Serological Assessment of Vitamin B_{12} in CD Patients and Controls

The mean vitamin B_{12} levels were insignificantly lower in the celiac disease (CD) patient (267.17±10.11 pg/mL, *P* = 0.002) compared to the control group (295.06±14.25 pg/mL) (*P* > 0.05), indicating a potential association between reduced vitamin B_{12} levels and CD (Figure 2).

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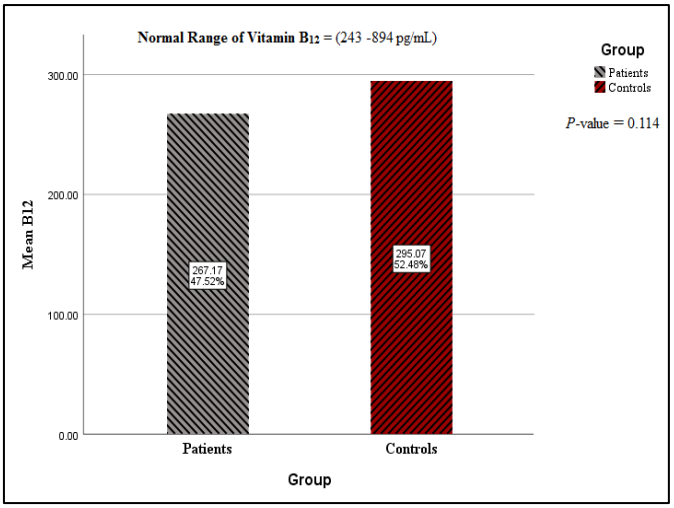

Figure 2: Serum levels of Vitamin B_{12} Levels in Celiac Disease Patients Compared to Healthy Controls.

DISCUSSION

Celiac disease (CD) is an autoimmune condition marked by the body's immune response to gluten, causing inflammation and harm in the small intestine. The diagnosis and monitoring of CD often involve serological tests, including anti-tissue transglutaminase (anti-tTG) IgA and IgG antibodies as well as intestinal fatty acid-binding protein (I-FABP). The current results revealed a statistically significant increase in serum levels of anti-tissue transglutaminase antibodies and I-FABP among CD patient groups. These findings are consistent with numerous studies conducted in various regions of the world [11–14]. Serological testing is widely recognized as a reliable, non-invasive standard for the diagnosis and follow-up of celiac disease. The mechanism behind this elevation in tTG-IgG and tTG-IgA levels involves an immune response triggered by gluten ingestion in genetically predisposed individuals. Gluten peptides cross the intestinal barrier and are deamidated by tissue transglutaminase 2 (TG2), enhancing their affinity for HLA-DQ2 or DQ8 molecules on antigen-presenting cells (APCs). This leads to activation of gluten-specific T cells, which stimulate B cells to produce anti-TG2 antibodies [15].

Fatty acid-binding proteins (FABPs) are low-molecular-weight (14–15 kDa) intracellular proteins that play a role in the metabolism of cholesterol and phospholipids, facilitate the transport of long-chain fatty acids, and help regulate lipid homeostasis [16]. The intestinal type (I-FABP) is exclusively produced in the small intestinal tract and encoded by the FABP2 gene located on chromosome 4 [17]. Intestinal fatty acid-binding proteins (I-FABPs) are distributed throughout the intestine, with highest expression in the jejunum, and are more concentrated in enterocytes located at the villous tip compared to those in the crypts. In celiac disease, elevated levels of intestinal fatty acid-binding protein (I-FABP) are due to enterocyte damage caused by the immune response to gluten. Specifically, the gluten-tTG (transglutaminase) complex triggers inflammation and damage to the villi of the small intestine, causing enterocytes to release I-FABP into the bloodstream. This damage and the subsequent release of I-FABP are reflected in the increased serum levels observed in untreated celiac patients [18,19].

Key observations of this study include lower iron levels, reduced ferritin concentrations, and elevated Total Iron-Binding Capacity (TIBC) in celiac disease (CD) patients compared to healthy controls. Similar findings were reported in an Iraqi study by Hasan *et al*. [9], which found an increase in TIBC and a decrease in iron and ferritin levels among CD patients. Another recent study conducted in Basrah, Iraq, indicated a highly significant decrease ($p \leq 0.001$) in serum iron levels in both age categories of CD patients compared to the healthy group [20].

The findings of this study are also consistent with global data around the world, which found similar results. Several international studies have reported significant alterations in iron metabolism in patients with celiac disease [21–24].

Iron deficiency anemia is a common complication of celiac disease, affecting up to half of newly diagnosed patients [25]. This condition arises primarily due to impaired nutrient absorption resulting from villous atrophy in the small intestine. The proximal duodenum, where most iron absorption occurs, is particularly affected by celiac disease [21]. Ferritin reflects body iron stores. Lower ferritin levels indicate diminished reserves and are often seen alongside iron deficiency anemia (IDA) [8, 26]. In our study, significantly reduced ferritin levels suggest compromised iron storage capacity among celiac patients. This aligns with previous studies indicating that only about 50% of patients recover their iron stores even after adhering to a gluten-free diet (GFD) [8, 25].

In celiac disease, elevated TIBC is mainly a compensatory response to iron deficiency resulting from impaired iron absorption. The aberrant expression of transferrin receptors (TfR) in enterocytes reflects this iron-starved state and further supports the biological demand for iron, prompting increased transferrin (and thus TIBC) production by the liver [24, 27].

Vitamin B_{12} is a water-soluble molecule with a complex chemical structure. Higher animals cannot synthesize vitamin B_{12} because they lack the genes required for cobalamin (vitamin B_{12}) production. In contrast, bacteria, yeasts, and certain algae are capable of synthesizing vitamin B_{12}. Interestingly, some of these bacteria inhabit the upper gastrointestinal tract of herbivores, which explains why herbivorous animals typically do not exhibit vitamin B_{12} deficiency despite having minimal dietary intake [28]. This study reveals an insignificant decrease in vitamin B_{12} levels in patients with celiac disease (CD) compared to healthy controls. This finding aligns with previous research indicating that vitamin B_{12} deficiency is not very common in CD patients, with prevalence ranging from 5% to 12% [7, 29–32]. Similarly, in a recent study conducted in the United kingdom, McGrogan et al. [33] found that vitamin B_{12} was normal among children with celiac disease. On the other hand, a recent study conducted in Baghdad, Iraq, revealed that vitamin B_{12} was significantly decreased among patients with CD [34].

Vitamin B_{12} deficiency in celiac disease (CD) is relatively uncommon compared to other micronutrient deficiencies. This is primarily because the terminal ileum (where B_{12} is absorbed) is typically unaffected in CD. Additionally, many gluten-free products are fortified with B_{12}, and patients often receive supplementation after diagnosis. However, B_{12} deficiency can still occur in CD patients with severe or widespread intestinal damage, slow mucosal healing (especially in adults), poor dietary intake, or coexisting conditions such as autoimmune atrophic gastritis [35]. Therefore, while B_{12} levels are often normal, persistent deficiency should prompt evaluation for additional causes beyond CD alone.

CONCLUSION

CD is an autoimmune disorder that mainly targets the small intestine, triggered by gluten ingestion in genetically predisposed individuals. Its onset involves a complex immune reaction to gluten proteins. This cross-sectional study highlights the presence of nutritional deficiencies, particularly in iron status, among patients with celiac disease (CD) in Diyala City. While a significant reduction in serum iron levels was observed, vitamin B12 levels showed only a minor and statistically insignificant decrease. However, clinical monitoring remains advisable.

Conflict of Interest

The authors declare that no conflict of interest.

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