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*Article*

# Biochemical Alterations of Trace Elements and Lipid Profile in Patients with Celiac Disease

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**Abstract: Background**: Chronic inflammatory status of the intestine with malabsorption is a characteristic condition of celiac disease, resulting in great impairment of trace elements and lipid metabolism. Assessment of these biochemical changes can be useful for disease diagnosis and monitoring of nutritional and inflammatory status of patients affected. **Aims of the study:** To assess biochemical changes in the levels of trace elements and lipid profile parameters in patients with celiac disease and correlate them with markers of inflammation. **Methodology:** This case–control study was conducted from January 2025 to March 2026 in Iraq with 100 patients with celiac disease and 50 healthy controls matched by age and sex. The diagnosis was confirmed by the presence of clinical symptoms, anti-TTG serology and intestinal biopsy. Hematological, biochemical, inflammatory and lipid profile parameters were analyzed by automatic analyzer and ELISA method. The concentration of serum iron, ferritin, zinc, copper, magnesium, calcium, CRP and lipid profile markers were determined in standard laboratory conditions. Data were analyzed statistically using SPSS version 26 and included analyses such as t-tests, Pearson correlation, and ROC curve analysis. **Result:** Patients with celiac disease had significantly lower BMI, higher rates of chronic diarrhea, weight loss, and positive family history, when compared with controls. The serum level of iron, ferritin, zinc, magnesium, calcium, total cholesterol, HDL-C, LDL-C, hemoglobin and albumin was significantly reduced, while copper, WBC count, platelet count, ESR and CRP were significantly increased ($p < 0.001$). There were significant correlations between inflammatory and nutritional biomarkers. The ROC analysis showed that zinc, ferritin and magnesium had good diagnostic values, and they could be used as markers for the evaluation of celiac disease. **Conclusions;** Celiac disease is associated with significant disturbances in trace elements, lipid profile, and inflammatory biomarkers. Zinc, ferritin, and magnesium demonstrated valuable diagnostic potential for assessing nutritional and inflammatory alterations in affected patients.

**Keywords:** Celiac disease, Trace elements, Lipid profile, Zinc deficiency, Inflammatory biomarkers.

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## Introduction

Celiac disease is a chronic immune-mediated enteropathy that is caused by the ingestion of gluten-containing grains in a genetically predisposed host. Characterised by inflammation of the mucosa of the small bowel with villous atrophy, crypt hyperplasia and impaired absorption. Celiac disease was once thought to be a rare gastrointestinal disease, but recent epidemiological studies have shown that it is a widespread disease that affects about 1% of the global population [1]. The manifestation of the disease can be at any age and can have a wide range of gastrointestinal and extraintestinal symptoms that include diarrhea, abdominal pain, anemia, osteoporosis, weight loss, fatigue, and metabolic disorders. With many patients, the diagnosis is delayed and this can result in high levels of nutritional deficiencies and subsequent systemic complications [2,3].

Celiac disease pathogenesis is a combination of environmental, genetic and immunological influences. Wheat, barley and rye contain gluten peptides that trigger an abnormal response from the immune system in individuals with predisposition to the disease (HLA-DQ2 and HLA-DQ8 alleles) [4]. This immune activation results in the synthesis of pro-inflammatory cytokines and autoantibodies (in particular anti-tissue transglutaminase antibodies) that gradually cause damage to intestinal mucosa. Chronic intestinal inflammation causes disruption of the absorption of nutrients, but also leads to systemic metabolic and inflammatory abnormalities. Thus, celiac disease is now known not to be a solely gastrointestinal disease but rather a multisystems disease [5].

One of the most serious effects of celiac disease is the malabsorption of micronutrients and trace elements. Iron, calcium, zinc and magnesium are mainly absorbed in the proximal small intestine, and a defect in the proximal small intestine frequently leads to a deficiency in these minerals [6]. Iron deficiency anemia is one of the most frequent hematologic findings in celiac disease and is a condition that can manifest without any gastrointestinal symptoms. Ferritin is depleted when iron levels in the body are low and it is common with chronic intestinal inflammation and decreased iron absorption [7]. Likewise, zinc deficiency is commonly seen in people with celiac disease and could lead to immune problems, slow healing and decreased antioxidant defenses. Although very rarely, magnesium and calcium deficiency can also occur as a result of the malabsorption over a long period of time, and this may be associated with osteopenia and osteoporosis in affected individuals [8].

The inflammatory responses and the oxidative stress may also affect the levels of serum ceruloplasmin and copper in celiac disease, which may affect copper metabolism. This imbalance of trace elements might therefore be a sign of nutrition deficiency and a reflection of systemic inflammation in celiac disease. The measurement of these micronutrients can offer valuable insights into disease severity, nutritional status and treatment response in individuals with CD [9,10].

Celiac disease is not only accompanied by micronutrient perturbations, but also by significant changes in lipid metabolism. Villous atrophy of the intestine can affect the digestion and absorption of dietary lipids, causing a decrease in serum cholesterol and lipoproteins [11]. Previous studies have found that untreated patients with celiac disease have lower levels of total cholesterol, low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C). But changes in lipid profiles can change in severity, depending on the length of time the disease has been present, the severity of damage to the intestine, eating habits and compliance with a gluten-free diet. Another possible role of abnormal lipid metabolism in celiac disease is that it may induce systemic inflammatory reactions and metabolic imbalance [12,13].

Other inflammatory and hematological abnormalities are also frequently seen in the patients of celiac disease. The inflammatory mediators and acute-phase proteins are released into the blood with chronic intestinal immune activation, leading to increased levels of ESR and CRP. In addition, chronic inflammation and nutritional deficiencies can cause anemia and thrombocytosis and changes in immune cell numbers. These changes in the laboratory can be due to intestinal damage and/or systemic inflammatory burden of active disease [14,15].

Although there are increased awareness levels regarding celiac disease, many patients are not diagnosed or diagnosed at a late stage where metabolic and nutritional complications have already occurred. Thus, the measurement of the trace elements, lipid profile parameters and inflammatory markers could become a key part of the evaluation and monitoring of the nutritional status of patients with the disease. An understanding of the biochemical changes and inflammatory activity may also help to identify those patients at higher risk of complications and help plan therapeutic management strategies [16].

The purpose of the present study was, therefore, to examine the biochemical changes of trace elements and lipid profile parameters in patients suffering from celiac disease and to determine their correlation with inflammatory and hematological parameters in comparison to the healthy controls.

**Methodology**

The study was a case-control study carried out from January 2025 to March 2026 in the Gastroenterology Unit, and Clinical Biochemistry Laboratory of a specialized teaching hospital in Iraq. The study involved 100 patients with celiac disease and 50 apparently healthy people as a control group matched for age and sex. The diagnosis was confirmed by clinical features and by positive serological markers (anti-tissue transglutaminase antibodies [anti-tTG]) and by intestinal biopsy, in accordance with the modified Marsh classification system. Clinical symptoms were chronic diarrhea, abdominal pain, weight loss, bloating, and symptoms of nutritional deficiency, including fatigue. The inclusion criteria were patients with newly diagnosed or untreated CD (either gender) and exclusion of patients with chronic liver disease, chronic renal disease, diabetes mellitus, autoimmune disorders other than CD, malignancies, inflammatory bowel disease, pregnancy, acute infections, lipid lowering therapy, mineral supplementation, and long-term corticosteroid treatment. Venous blood (about 5 mL) was drawn aseptically from all the subjects after a 12-hour fasting period and was stored in EDTA tubes for hematological examination and plain gel tubes for serum separation. After centrifugation at 3000 rpm/for 10 minutes, samples of the serum were collected and stored at −20 °C for analysis. An automated hematology analyzer was used to measure haemoglobin concentration, white blood cell count and platelet count, and erythrocyte sedimentation rate (ESR) was measured by the Westergren method. Biochemical assay was performed for serum iron, ferritin, zinc, copper, magnesium, calcium, total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) using commercially available biochemical assay kit following the manufacturer instructions. The concentration of serum C-reactive protein (CRP) was measured by enzyme linked immunosorbent assay (ELISA) tests under standardized laboratory conditions. The data were analyzed statistically with SPSS version 26 (IBM Corporation, Armonk, NY, USA). Mean ± SD was used to present quantitative variables, independent-sample t-tests were used to compare quantitative variables between groups, Pearson correlation analysis was used to assess relationship between selected biomarkers and diagnostic performance of selected biomarkers was assessed by receiver operating characteristic (ROC) curve analysis. P value of $< 0.05$ was used as statistically significant.

**Statistical analysis:**

Quantitative data were analyzed using SPSS version 26. Results are presented as frequencies and percentages. For normally distributed variables, independent and dependent t-tests (two-tailed) were used. For non-normally distributed variables, the Mann-Whitney U test, Wilcoxon test, and Chi-square test were applied. A p-value of $< 0.05$ was considered statistically significant.

**Ethical approval:**

The study was approved by the human ethics committee of Al-Imamain Alkadhimain Medical City, Everyone who took part in the study was told about it and asked to sign a consent form. The patient was also guaranteed that his information would be kept private.

## Results

### Comparison of demographic features and clinical manifestations between study groups

The findings presented in Table 1 showed no statistically significant differences between celiac disease patients and healthy controls regarding age and gender distribution ($p > 0.05$), indicating appropriate matching between the study groups. However, body mass index (BMI) was significantly lower in celiac disease patients compared with controls (21.4 ± 3.1 vs. 24.8 ± 2.9 kg/m², $p < 0.001$). The mean disease duration among patients was 4.6 ± 2.3 years. Chronic diarrhea and weight loss were highly prevalent among celiac disease patients, affecting 74.0% and 63.0% of patients, respectively ($p < 0.001$). Furthermore, more patients had a positive family history than controls (27.0% vs. 8.0%, $p = 0.008$), which also pointed to a genetic predisposition towards celiac disease.

**Table 1.** Sociodemographic and Clinical Characteristics of Celiac Disease Patients and Healthy Controls.

| Variables | Celiac Disease Patients (n=100) | Healthy Controls (n=50) | P-value |
|---|---|---|---|
| Age (years) Mean ± SD | 31.8 ± 10.4 | 30.5 ± 9.7 | 0.448 |
| Male, n (%) | 42 (42.0%) | 21 (42.0%) | 1.000 |
| Female, n (%) | 58 (58.0%) | 29 (58.0%) | 1.000 |
| BMI (kg/m²) Mean ± SD | 21.4 ± 3.1 | 24.8 ± 2.9 | <0.001 |
| Duration of disease (years) | 4.6 ± 2.3 | N/A | N/A |
| Chronic diarrhea, n (%) | 74 (74.0%) | 0 | <0.001 |
| Weight loss, n (%) | 63 (63.0%) | 0 | <0.001 |
| Family history of celiac disease, n (%) | 27 (27.0%) | 4 (8.0%) | 0.008 |

### The concentrations of serum trace elements found in both celiac disease patients and healthy individuals were compared.

As seen in the results in Table 2, there were significant differences between the trace element levels of the serum in the celiac disease patients and the healthy control group ($p < 0.001$). Patients with celiac disease had significantly lower serum levels of iron, ferritin, zinc, magnesium and calcium, suggesting that intestinal absorption is impaired and that these nutrients are deficient in celiac disease because of chronic mucosal damage. Patients, by contrast, had significantly higher levels of copper in the serum compared to controls (129.4 ± 21.8 vs. 103.6 ± 17.2 µg/dL, $p < 0.001$); this may be due to inflammatory and oxidative stress related to the active disease. The results presented here show that celiac disease has a significant effect on micronutrient metabolism and trace element homeostasis.

**Table 2.** Serum Trace Element Levels in Celiac Disease Patients and Healthy Controls.

| Parameters | Celiac Disease Patients | Healthy Controls | P-value |
|---|---|---|---|
| Serum Iron (µg/dL) | 54.7 ± 15.6 | 88.3 ± 17.4 | <0.001 |
| Ferritin (ng/mL) | 19.8 ± 8.5 | 52.6 ± 14.3 | <0.001 |
| Zinc (µg/dL) | 61.5 ± 12.1 | 91.7 ± 13.6 | <0.001 |
| Copper (µg/dL) | 129.4 ± 21.8 | 103.6 ± 17.2 | <0.001 |
| Magnesium (mg/dL) | 1.54 ± 0.31 | 2.08 ± 0.27 | <0.001 |
| Calcium (mg/dL) | 8.12 ± 0.74 | 9.34 ± 0.61 | <0.001 |

**A comparison of the levels of serum lipids in celiac patients and healthy people is performed.**

As indicated in the findings presented in Table 3, there were significant changes in the various lipid profile parameters for the celiac disease patients as compared to healthy controls. In celiac disease patients, there was a significant decrease in total cholesterol, HDL-cholesterol and LDL-cholesterol levels ($p<0.001$), which was indicative of the impaired intestinal lipid absorption and metabolic disturbances caused by the mucosal injury. Triglyceride and VLDL-cholesterol were not significantly different in the study groups ($p > 0.05$). The findings indicate that the celiac disease primarily interferes with the metabolism of cholesterol and with lipoprotein transport, but not with triglyceride metabolism.

**Table 3.** The lipid profile parameters of celiac disease patients and normal controls were compared.

| Lipid Parameters | Celiac Disease Patients | Healthy Controls | P-value |
|---|---|---|---|
| Total Cholesterol (mg/dL) | 151.6 ± 29.4 | 182.8 ± 24.7 | <0.001 |
| Triglycerides (mg/dL) | 118.3 ± 33.5 | 109.7 ± 27.8 | 0.116 |
| HDL-Cholesterol (mg/dL) | 36.9 ± 7.8 | 49.4 ± 8.1 | <0.001 |
| LDL-Cholesterol (mg/dL) | 89.7 ± 24.1 | 111.2 ± 21.5 | <0.001 |
| VLDL-Cholesterol (mg/dL) | 23.7 ± 6.7 | 21.9 ± 5.5 | 0.098 |

**Comparison of hematological and inflammatory parameters between celiac disease patients and healthy individuals**

Celiac disease patients showed significant hematological and inflammatory changes when compared with healthy controls ($p < 0.001$) as seen in the results presented in Table 4. The patients had significantly reduced levels of hemoglobin and albumin, which indicates nutrition deficiency, chronic inflammation and impaired intestinal absorption due to celiac disease. Celiac disease patients, however, had significantly higher levels of white blood cells, platelets, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) which were markers of systemic inflammation and immune activation. These findings validate the inflammatory and malabsorptive effects of celiac disease and the effects on hematological and biochemical homeostasis.

**Table 4.** Hematological and Inflammatory Biomarkers in Celiac Disease Patients and Healthy Controls.

| Parameters | Celiac Disease Patients | Healthy Controls | P-value |
|---|---|---|---|
| Hemoglobin (g/dL) | 10.9 ± 1.8 | 13.6 ± 1.2 | <0.001 |
| WBC ($\times10^3/\mu L$) | 8.9 ± 2.4 | 6.8 ± 1.7 | <0.001 |
| Platelets ($\times10^3/\mu L$) | 356.2 ± 74.5 | 271.4 ± 48.7 | <0.001 |
| ESR (mm/hr) | 38.5 ± 11.7 | 12.8 ± 4.6 | <0.001 |
| CRP (mg/L) | 18.4 ± 7.9 | 4.2 ± 1.8 | <0.001 |
| Albumin (g/dL) | 3.41 ± 0.54 | 4.36 ± 0.47 | <0.001 |

**Pearson correlation analysis among trace elements, hematological parameters, inflammatory markers, and lipid profile variables**

The data from the correlation analysis, displayed in Table 5, showed significant relationships between biochemical, inflammatory and hematological markers in CD patients. The level of hemoglobin displayed significant positive correlations with ferritin, zinc, and HDL-cholesterol and significant negative correlation with CRP levels, suggesting a relationship between worsening inflammation and anemia and nutritional deficiencies. Ferritin level was positively correlated with the zinc level and with HDL-cholesterol while both ferritin and zinc levels were significantly inversely correlated with CRP and ESR, indicating that the chronic inflammatory activity in celiac disease patients may induce depletion of trace elements and affect the metabolic status. In addition, CRP showed a good positive correlation with ESR and negative correlation with ferritin, zinc and HDL-cholesterol, indicating a close correlation between systemic inflammation and nutritional imbalance in active CD.

**Table 5.** Correlation Analysis Between Biochemical and Inflammatory Biomarkers in Celiac Disease Patients.

| Variables | Ferritin | Zinc | CRP | HDL-Cholesterol |
|---|---|---|---|---|
| Hemoglobin | r = 0.521** p < 0.001 | r = 0.462** p < 0.001 | r = -0.417** p < 0.001 | r = 0.308* p = 0.002 |
| Ferritin | 1 | r = 0.438** p < 0.001 | r = -0.392** p < 0.001 | r = 0.281* p = 0.005 |
| Zinc | r = 0.438** p < 0.001 | 1 | r = -0.391** p < 0.001 | r = 0.286* p = 0.004 |
| CRP | r = -0.392** p < 0.001 | r = -0.391** p < 0.001 | 1 | r = -0.344** p = 0.001 |
| ESR | r = -0.365** p < 0.001 | r = -0.334** p = 0.001 | r = 0.582** p < 0.001 | r = -0.276* p = 0.006 |

**Receiver operating characteristic (ROC) curve analysis of biochemical and inflammatory biomarkers for predicting celiac disease**

ROC curve analysis in Table 6 showed that a number of biochemical and inflammatory markers had an acceptable to excellent diagnostic value in the distinction between patients with CD and healthy controls. Serum zinc showed the highest diagnostic accuracy with an AUC of 0.912 (95% CI: 0.861–0.963), sensitivity of 88.0%, and specificity of 84.0% at a cut-off value below 72.5 μg/dL ($p < 0.001$). Ferritin and magnesium also showed very good diagnostic performance with AUC of 0.881 and 0.874, respectively. Furthermore, good diagnostic values such as significant $p<0.001$ and sensitivity and specificity values for CRP and HDL-cholesterol were obtained. The results indicate that the laboratory measures of trace element deficiencies and inflammatory biomarkers may be useful as markers for celiac disease diagnosis and management.

**Table 6.** Diagnostic Performance of Selected Biomarkers in Celiac Disease Patients.

| Biomarker | Cut-off Value | Sensitivity (%) | Specificity (%) | AUC (95% CI) | P-value |
|---|---|---|---|---|---|
| Zinc | <72.5 μg/dL | 88.0 | 84.0 | 0.912 (0.861–0.963) | <0.001 |
| Ferritin | <28.0 ng/mL | 84.0 | 80.0 | 0.881 (0.821–0.941) | <0.001 |
| CRP | >9.5 mg/L | 81.0 | 78.0 | 0.846 (0.778–0.914) | <0.001 |
| HDL-Cholesterol | <41.5 mg/dL | 79.0 | 74.0 | 0.812 (0.739–0.885) | <0.001 |
| Magnesium | <1.75 mg/dL | 83.0 | 82.0 | 0.874 (0.808–0.940) | <0.001 |

**Discussion**

In the present study, significant biochemical and inflammatory alterations have been found in celiac patients, reflecting the complexity of the metabolic and immunological abnormalities that take place in the background of chronic intestinal inflammatory disease and nutritional malabsorption in celiac disease. There were no significant differences in the distribution of age and gender between the two groups of celiac patients and the healthy controls, indicating that the two samples were well matched. BMI however was significantly reduced in those with celiac disease, perhaps reflecting chronic malabsorption, villous atrophy and impaired nutrient assimilation that occurs due to gluten induced mucosal damage [17]. The same has been observed in previous research where untreated celiac disease patients had lower BMI scores, attributed to length of gastrointestinal symptoms and nutritional deficiencies [18]. Other studies, however, indicated that children with celiac disease had normal/even high BMI, even when diagnosed early in the course of the disease or while on a GFD. These differences may be attributed to differences in diet, length of time a patient has had the disease, genetic differences or adherence to treatment modalities [19].

The trace element analysis showed that during celiac disease, the serum iron, ferritin, zinc, magnesium and calcium levels were significantly lower than in healthy controls. Iron deficiency is one of the most prevalent extraintestinal complications of celiac disease, and is primarily attributed to the reduction of iron availability for absorption in the proximal small intestine, which is most affected by the disease [20]. Iron depletion as reflected by decreased ferritin levels seen in this study is further evidence of depleted iron stores secondary to chronic malabsorption and intestinal inflammation. The same was reported in multiple studies which showed that there is a high rate of iron deficiency anemia among celiac disease patients without treatment [21].

Several studies, however, have found less severe iron deficiency, which could be attributed to earlier diagnosis, iron supplementation and partial mucosal recovery with gluten elimination [22]. In the present study, as well, serum zinc was also significantly decreased. In the case of celiac disease, zinc deficiency can be caused by reduced intestinal absorption, chronic diarrhea and excessive loss through the intestine [23]. Because of its critical role in the regulation of the immune system, antioxidant activity and repair of the intestinal epithelium, zinc deficiency could exacerbate the mucosal damage and systemic inflammation. Other studies also showed a decreased zinc level in celiac disease patients, especially in those with active disease [24]. On the other hand, no significant Zn deficiency were observed in some studies and this might be due to various factors such as nutritional intake and supplementation, and disease severity [25].

Intriguingly, there was a significant increase in the copper concentration of the patients with celiac disease. This elevation could be a compensatory effect caused via chronic inflammation as copper containing proteins are acute phase reactants in inflammatory situations [26]. An elevation of serum copper levels has been linked to oxidative stress and activation of inflammatory cytokines in autoimmune gastrointestinal diseases [27]. The magnesium and calcium deficiencies that were noted in the present study might be attributed to the impairment of intestinal absorption, vitamin D deficiency, and chronic inflammatory injury to mineral metabolism. Long-standing celiac disease is associated with well-known associations of bone demineralization and osteoporosis, which may be attributable to hypocalcemia in celiac disease [28].

The outcome of lipid profile showed that patients with celiac disease had significantly lower levels of total cholesterol, HDL-cholesterol and LDL-cholesterol than the control group. These changes are likely due to dysfunctional lipid absorption due to villous atrophy and impaired intestinal transport of dietary fats [29]. A corresponding decrease in these cholesterol fractions has been reported in untreated celiacs and correlated with malabsorption and defective lipoproteins production. Others, however, found that after starting gluten-free diets lipid levels normalized or even rose after the start of gluten-free diets as a result of intestinal recovery and increased caloric consumption. In the present case, no significant differences were observed regarding the triglyceride and VLDL; this could suggest that the triglyceride metabolism is not so prominently affected in celiac disease as the cholesterol transport pathways [30].

The hematological and inflammatory parameters showed marked decreases in hemoglobin (Hb), and increases in white blood cell count (WBC), platelet count, ESR, and CRP concentration. Celiac disease associated anemia is multifactorial and could be caused by iron-deficiency, chronic inflammatory process, folate deficiency and impaired erythropoiesis [31]. Increased platelets could be a reactive response to cytokines released during inflammation, or due to iron deficiency [32]. Higher ESR levels and CRP levels are indicative of ongoing systemic inflammation and immune activation that is linked with gluten-induced damage to the intestine. The inflammation seen here was similar to what had been reported in patients with active celiac disease. However, it was reported that those patients with treated celiac disease on a strict GFD had lower levels of inflammatory markers, suggesting that a strict gluten-free diet is indeed important for reducing immune activation [33].

After performing correlation analysis, it was found that there were significant positive correlations between ferritin and zinc, ferritin and HDL-cholesterol, and hemoglobin and nutritional biomarkers, and significant negative correlations between inflammatory markers and nutritional biomarkers [34]. The results indicate that the worsening nutritional deficiencies are associated with rise in inflammatory activity and deterioration of the metabolic status in celiac disease. The inverse correlation between zinc and CRP can be seen as a redistribution of the zinc from serum to tissue during acute phase response [35]. Moreover, the positive correlation between CRP and ESR indicates the activation of inflammatory pathways and that of acute-phase protein synthesis [35].

The ROC curve analysis revealed a good diagnostic accuracy for zinc and ferritin for the prediction of celiac disease. Zinc had a higher area under the curve (AUC) which indicated that abnormal levels of trace elements could be used as markers of the activity of inflammatory disease and intestinal malabsorption. These results add to the previous studies highlighting the importance of micronutrient deficiencies in the assessment of celiac disease as a diagnostic and prognostic tool [36]. Others, however, proposed that a single biomarker would not be sufficient since other malabsorptive or inflammatory diseases can also cause a similar deficiency. Thus, looking at the inflammatory/hematological markers in addition to trace element analysis could be useful to enhance the diagnosis of severity and increase accuracy [37].

**Conclusion**

In conclusion, the results of the present study point to the strong relationship between chronic intestinal inflammation, nutritional deficiencies, disrupted lipid metabolism and systemic immune activation in celiac disease. The biochemical disturbances may play a role in the progression of the disease and in long-term complications if not diagnosed and treated properly. These changes that are observed highlight the need for comprehensive laboratory monitoring in patients with celiac disease especially in the active disease phase prior to the complete resolution of the mucosa.

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