AMERICAN JOURNAL OF BIOMEDICINE AND PHARMACY
https://biojournals.us/index.php/AJBP
ISSN: 2997-7177

Article

Evaluation of Human Leucocyte Antigen (HLA) and Immunological Markers in Iraqi Associations between Family History and BMI in Celiac Disease Patients

Asraa Faisal Hassan[1]

Citation: Hassan A. F. Evaluation of Human Leucocyte Antigen (HLA) and Immunological Markers in Iraqi Associations between Family History and BMI in Celiac Disease Patients. American Journal of Biomedicine and Pharmacy 2026, 3(11), 32-44.

Received: 10th Oct 2026
Revised: 11th Sep 2026
Accepted: 10th Oct 2026
Published: 04th Nov 2026

Copyright: © 2026 by the authors. Submitted for open access publication under the terms and conditions of the Creative Commons Attribution (CC BY) license (https://creativecommons.org/licenses/by/4.0/)

Department of Biochemistry, Salah Al-Din Directorate of Education- Al-Dur Division ,Tikrit, Salah Al-Din Governorate, Iraq
*Correspondence: 07710632480i@gmail.com

Abstract: Objective : Celiac disease (CD) is a chronic gastrointestinal disorder of immune-mediated pathogenesis that occurs in genetically susceptible individuals due to gluten. The most important genetic factors influencing susceptibility are the molecules called Human Leukocyte Antigen (HLA). In Iraq, there is a lack of data on celiac disease patients, which is needed to provide a complete picture of the condition. Details including their family history, body mass index (BMI), nutritional deficiencies, serological markers, and HLA status are among these data.
Aim: The purpose of this study is to investigate the immunological markers and the frequency of positivity for HLA risk in Iraqi patients with celiac disease. Levels of endomysial antibody (EMA-IgA), anti-tTG IgA and anti-DGP IgG were investigated as well as their correlations with BMI, dietary factors and family history. Material and methods: Eighty Iraqis took part in this prospective case-control study, forty of them had celiac disease and forty served as healthy controls. HLA molecules were typed by PCR-SSP. The serological biomarkers were measured by Elisa and indirect immunofluorescence. Various dietary and hemostatic factors were assessed. Extensive records were kept on medical history and BMI estimates.
Results: Demonstrated that the frequency of HLA risk allele positivity was significantly higher in celiac patients (85.0% vs. 25.0%; OR = 16.3, $p<0.001$). EMA-IgA positive was more common (80% vs. 5%), anti-DGP IgG levels were significantly lower (98.4 ± 28.6 vs. 6.2 ± 2.1 U/mL) and anti-tTG IgA levels were significantly higher (142.6 ± 38.4 vs. 4.8 ± 1.6 U/mL). All of these findings were statistically significant ($p<0.001$). The BMI of the patients was significantly lower (18.6 ± 3.2 kg/m² vs. 22.4 ± 3.6 kg/m²; $p<0.001$) and it had an inverse relationship with anti-tTG IgA ($r = -0.62$, $p<0.001$).
Conclusions: There was a definite deficiency of iron, folate, vitamin B12, and hemoglobin. A positive family history was detected in 42.5% of patients and was related to increased HLA positivity and greater serological markers. Iraqi celiac disease is characterized by very low diagnosis rates, a strong inverse correlation between body mass index and nutritional deficiencies, and high immunological .

Keywords: Celiac Disease; HLA; Anti-tTG IgA; Anti-DGP IgG; Iraq; BMI; Family History; Nutritional Deficiency; Immunological Biomarkers; Consanguinity

Introduction

Gluten is a protein found in wheat, barley and rye . Some people have a genetic predisposition to celiac disease (CD), a chronic autoimmune disorder that affects the small intestine . Among the most common chronic immune-mediated disorders, celiac disease is underdiagnosed in Iraq and other countries where it is prevalent (Lebwohl et al., 2018). Some of the more common symptoms of the disease include chronic diarrhea, abdominal distention, and malabsorption; less common symptoms include anaemia, short stature, infertility, and neurological symptoms; and there are also atypical and silent forms of the disease [1].

The HLA system, a set of genes on chromosome 6p21.3 that encode for MHC class II molecules, is the genetic backbone of celiac disease.These molecules are on the surfaces of antigen-presenting cells and their role, according to Sollid and Jabri, is to present immunogenic peptides derived from gluten to CD4+ T-helper cells, which in turn triggers the adaptive immune cascade that leads to damage of the intestinal lining. The presence of a heterodimer of HLA-DQ2 and HLA-DQ8 in nearly all celiac patients highlights the critical role of HLA in the disease's progression, but it is clear that additional genetic, environmental and immunological factors are necessary for clinical expression as the disease does not develop in 30-40% of individuals who carry these HLA variants [2].

Serological markers play an important role in screening and diagnosing celiac disease. Anti-tissue transglutaminase IgA (anti-tTG IgA) is the first choice of serological testing and is recommended, with a specificity of 97% and a sensitivity of >95% [3]. Anti-deamidated gliadin peptide IgG (anti-DGP IgG) provides additional diagnostic value especially in patients with IgA deficiency. The indirect immunofluorescence detection of endomysial antibody IgA (EMA-IgA) is an extremely useful tool in confirmatory settings given its almost perfect specificity (>99%) [4].According to Rubio-Tapia et al. , a strong serological panel constituted of these markers reflects the immune activation underlying the pathogenesis of celiac disease.One of the most serious consequences of celiac disease-induced damage to the mucosal surface is the inability to absorb important micronutrients such as iron, folate, vitamin B12, calcium and vitamin D and the impact of this on nutritional status.

Clinical symptoms of nutritional deficiencies were enumerated by Zanini et al. and include iron-deficiency anemia, megaloblastic anemia, osteoporosis, growth retardation, and peripheral neuropathy. In active, untreated celiac disease, the systematic lowering of Body Mass Index (BMI) reflects the cumulative influence of malabsorption on somatic growth and body composition. Deterioration of mucosal lining and nutritional deficit are inversely related to the rate of reduction in body mass index (BMI).The risk for celiac disease is higher in families with a family history of the disorder. Lifetime risk for first-degree relatives is significantly higher at 10–15% than the general population risk of 1–2% [5]. This is due to the largely hereditary nature of HLA-mediated susceptibility. In Iraq, the reported consanguinity rates of 33% to 46% [6] may lead to high probability of HLA risk allele homozygosity within families, which could result in the transmission of disease from one generation to the next. Clinically critical parameters such as HLA status, comprehensive serological biomarker panel, dietary deficiencies, body mass index (BMI), and family history have

never been systematically and concurrently evaluated in an Iraqi research of celiac disease patients. This research fills a critical need in the available evidence.

Patients and Methods

Study Design and Setting

Scientists from Baghdad, Iraq's Gastroenterology and Clinical Immunology Outpatient Clinic at [Baghdad teaching hospital] conducted a prospective case-control study from January to December 2024. Following the principles established down in the Declaration of Helsinki, the study could not begin until all participants or their legal guardians had given written informed consent

Participants

Forty celiac patients from Iraq aged 5–45 years were studied. A positive serology result (anti-tTG IgA ≥10× upper limit of normal and/or positive anti-DGP IgG), together with associated gastrointestinal symptoms and/or HLA risk allele positivity is required. The control group consisted of 40 healthy, age- and sex-matched adults with negative serology results and no autoimmune disorders or gastrointestinal symptoms. Other autoimmune diseases, immunosuppressive medication, inflammatory bowel disease, and >3 months without gluten were exclusion criteria. Patients' presenting symptoms (e.g., persistent diarrhea, abdominal distension, fatigue, weight loss, and anemia) were recorded with a standardized clinical questionnaire, including symptom duration, medication history, eating habits, and gluten exposure. The WHO classifies individuals by body mass index (BMI) as underweight (<18.5), normal (18.5-24.9), overweight (25-29.9), or obese (≥30). Symptom intensity was assessed using the validated Gastrointestinal Symptom Rating Scale (GSRS, range 0-100). Three generation family history of celiac disease was well preserved. As it was of importance to Iraqis we documented the consanguinity of the parents. Venous blood was taken for serological testing in fasting volunteers (10–12 h). To rule out selective IgA deficiency, total serum IgA was measured by nephelometry before interpreting anti-tTG IgA results. ELISA (Euroimmun AG, Lübeck, Germany) measured anti-tTG IgA; values over 10 U/mL were positive. Anti-DGP IgG was measured by ELISA (Inova Diagnostics, San Diego, USA). Indirect immunofluorescence detected EMA-IgA on monkey oesophagus slices from the Birmingham Binding Site. We measured CRP with immunoturbidimetry. Laboratory personnel were blinded to assay results and performed according to the manufacturer's instructions . Serum folate, vitamin B12, ferritin, TIBC, serum iron and CBC were determined by Roche Cobas c501 and Beckman Coulter DxH 900 analyzers. The WHO defined anemia as hemoglobin levels below 11.0 g/dL for children aged 5-11, 11.5 for 12–14, 12.0 for adult women, and 13.0 for adult men.

Statistical Analysis

Analyses were performed using the Statistical Package for the Social Sciences, Version 26.0 (IBM Corp, Armonk, NY). Continuous variables are expressed as mean ± SD. Comparisons across groups were carried out using Student's t-test or Mann-Whitney U test. For categorical variables, chi-square tests with odds ratios (OR) and 95% confidence intervals (CI) were used. The link between continuous variables were assessed by Pearson and Spearman correlation coefficients. Multivariate

logistic regression analysis was used to find independent variables of rise in anti-tTG IgA and decrease in body mass index. A p-value of <0.05 was judged statistically significant.

Results

Demographic and Clinical Characteristics

The celiac patients were predominantly female (52.5%) and had an average age of 18.6 ± 10.4 years, ranging from 5 to 45. The most common initial symptoms were chronic diarrhea (82.5% of cases), stomach distension (77.5% of cases), fatigue (72.5%), weight loss (67.5% of cases), and anemia (62.5%). The control group had a body mass index (BMI) of 22.4 ± 3.6 kg/m², but the sick group had an average BMI of 18.6 ± 3.2 kg/m{ (p<0.001). The percentage of underweight patients was determined to be nearly half (47.5%). The majority of the fifteen people who tested positive for celiac disease were first-degree relatives; the percentage of those people who tested positive was 42.5%. Table 1 shows that 37.5% of patient families reported consanguinity, which is higher than the control group's 15.0% (p=0.018), reflecting the high endogamy rates typical of the Iraqi community [7].

Table 1. Demographic and Clinical Characteristics of Study Participants.

Variable	Celiac Disease (n=40)	Controls (n=40)	p-value
Age (years), mean ± SD	18.6 ± 10.4	18.2 ± 9.8	0.861
Female, n (%)	21 (52.5%)	20 (50.0%)	0.819
BMI (kg/m²), mean ± SD	18.6 ± 3.2	22.4 ± 3.6	<0.001
Underweight (BMI<18.5), n (%)	19 (47.5%)	4 (10.0%)	<0.001
Chronic Diarrhoea, n (%)	33 (82.5%)	1 (2.5%)	<0.001
Abdominal Distension, n (%)	31 (77.5%)	2 (5.0%)	<0.001
Anaemia, n (%)	25 (62.5%)	3 (7.5%)	<0.001
Family History (+), n (%)	17 (42.5%)	0 (0%)	<0.001
Consanguinity, n (%)	15 (37.5%)	6 (15.0%)	0.018
Disease Duration (months)	28.4 ± 18.6	—	—

HLA Status

The number of healthy controls who tested positive was only 25 percent, compared to 85 percent of celiac patients who tested positive for the HLA risk allele (OR = 16.3, 95% CI: 5.28-50.3; p<0.001). Fifteen percent of celiac patients who tested negative for HLA had lower antibody titres and milder symptom scores. The levels of anti-tTG IgA, GSRS scores, and body mass index (BMI) were all substantially higher in HLA-positive patients compared to HLA-negative individuals (p<0.05 for all). The likelihood of a family history of celiac disease was higher among patients who tested positive for the HLA gene (p=0.038; see Table 2 and Figures 1, 2).

Table 2. HLA Risk Allele Positivity in Celiac Patients vs. Controls.

HLA Status	Celiac n (%)	Controls n (%)	OR (95% CI)	p-value
HLA Positive (any risk allele)	34 (85.0%)	10 (25.0%)	16.3 (5.28–50.3)	<0.001
HLA Negative	6 (15.0%)	30 (75.0%)	0.06 (0.02–0.19)	<0.001
HLA Positive with Family History	14 (41.2% of HLA+)	0 (0%)	—	<0.001

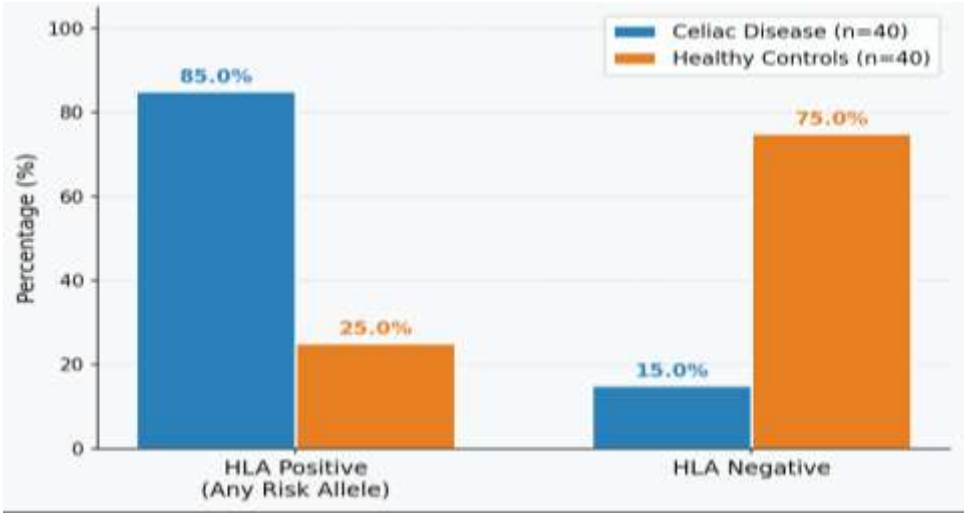


Figure 1. HLA Risk Allele Positivity Rate in Celiac Disease Patients vs. Healthy Controls (p<0.001).

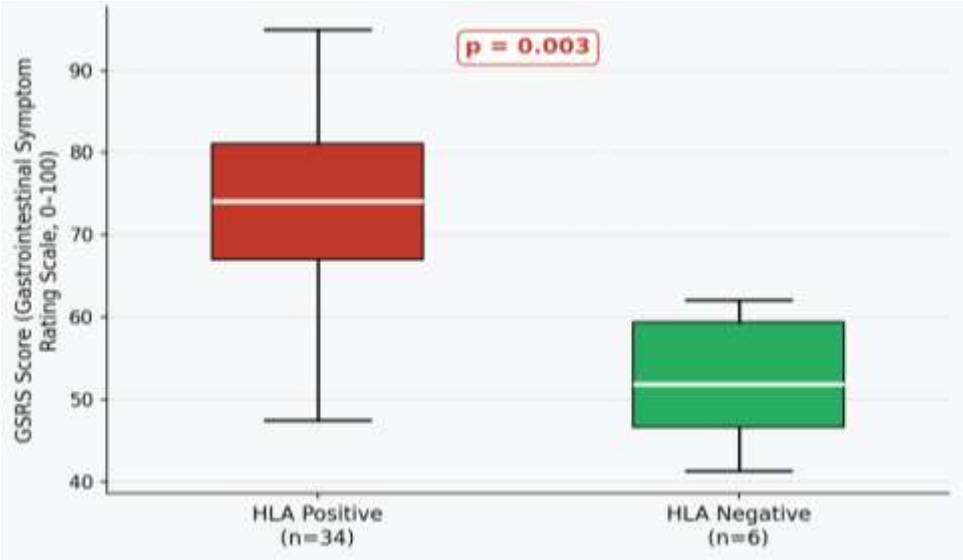


Figure 2. Gastrointestinal Symptom Severity (GSRS Score) by HLA Positivity Status (p=0.003).

Serological Biomarkers

Celiac patients had significantly higher levels of Anti-tTG IgA (142.6 ± 38.4 U/mL) than controls (4.8 ± 1.6 U/mL; p<0.001). The levels of Anti-DGP IgG were significantly greater (98.4 ± 28.6 vs. 6.2 ±

2.1 U/mL; p<0.001). In the case of EMA-IgA, 80% of patients were positive, but just 5% of controls were. The overall serum IgA level in humans was noticeably greater than in animals (182.4 ± 42.6 mg/dL vs. 248.6 ± 58.4 mg/dL) (p<0.001). Two patients, or 5% of the total, were shown to have a particular IgA deficit by the use of anti-DGP IgG testing. There is a substantial correlation between anti-tTG IgA and anti-DGP IgG (r = 0.84, p<0.001), as shown in Table 3 and Figures 3, and an inverse correlation with BMI (r = −0.62, p<0.001) [8].

Table 3. Serological Biomarker Levels in Celiac Disease Patients vs. Controls.

Serological Parameter	Celiac (n=40)	Controls (n=40)	p-value
Anti-tTG IgA (U/mL)	142.6 ± 38.4	4.8 ± 1.6	<0.001
Anti-DGP IgG (U/mL)	98.4 ± 28.6	6.2 ± 2.1	<0.001
EMA-IgA Positive, n (%)	32 (80.0%)	2 (5.0%)	<0.001
Total Serum IgA (mg/dL)	248.6 ± 58.4	182.4 ± 42.6	<0.001

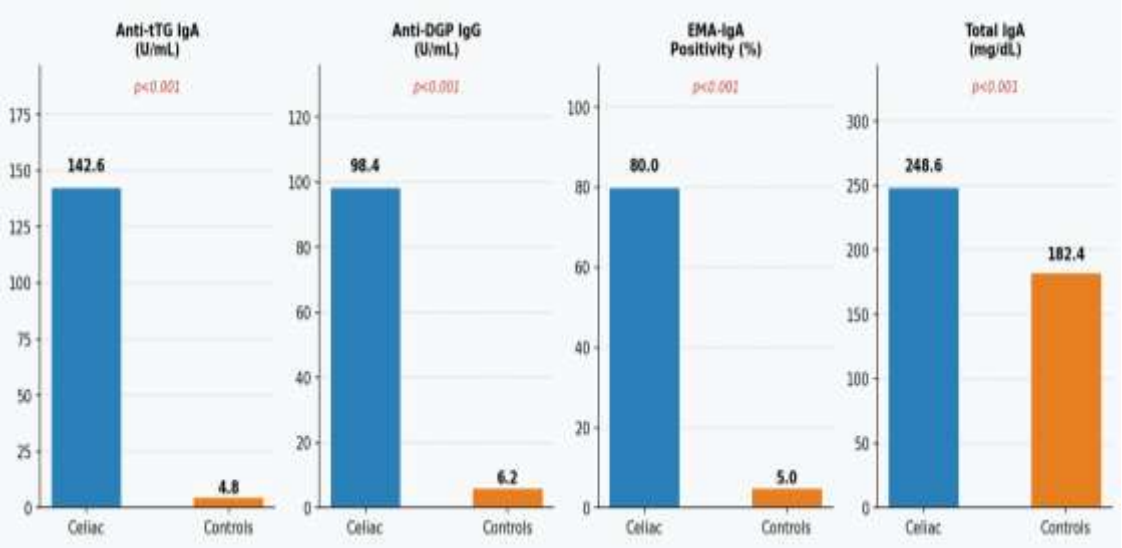


Figure 3. Serological Biomarker Levels in Celiac Disease Patients vs. Healthy Controls (p<0.001 for all).

BMI and Nutritional Deficiencies

The body mass index of celiac patients was significantly lower (18.6±3.2 kg/m² vs. 22.4±3.6 kg/m², p<0.001) and 47.5% were considered underweight (BMI<18.5 kg/m²). The HLA-positive group (17.8±2.8 kg/m²) had the lowest body mass index among the three groups, compared to the controls group (22.4±3.6 kg/m²; p<0.001) and the HLA-negative group (21.4±2.4 kg/m²). The following variables showed the inverse connection between BMI and anti-tTG IgA (r=−0.62, p<0.001), anti-DGP IgG (r=−0.54, p<0.001), and CRP (r=−0.53, p<0.001). Tables 4 and Figures 4-6show that there were consistently low levels of the following nutrients: haemoglobin (9.8 ± 1.6 vs. 13.6 ± 1.2 g/dL), serum iron (48.2 ± 14.6 vs. 98.4 ± 22.4 μg/dL), ferritin (12.4 ± 4.8 vs. 68.2 ± 18.6 ng/mL), folate (4.2 ± 1.4 vs. 9.8 ± 2.2 ng/mL), and vitamin B12 (186.4 ± 48.2 vs. 424.2 ± 88.6 pg/mL). All these levels were significantly lower (p < 0.001) [9].

Table 4. BMI and Nutritional/Haematological Parameters in Celiac Patients vs. Controls.

Nutritional Parameter	Celiac (n=40)	Controls (n=40)	p-value
BMI (kg/m²)	18.6 ± 3.2	22.4 ± 3.6	<0.001
Haemoglobin (g/dL)	9.8 ± 1.6	13.6 ± 1.2	<0.001
Serum Iron (μg/dL)	48.2 ± 14.6	98.4 ± 22.4	<0.001
Ferritin (ng/mL)	12.4 ± 4.8	68.2 ± 18.6	<0.001
Serum Folate (ng/mL)	4.2 ± 1.4	9.8 ± 2.2	<0.001
Vitamin B12 (pg/mL)	186.4 ± 48.2	424.2 ± 88.6	<0.001
Anaemia, n (%)	25 (62.5%)	3 (7.5%)	<0.001
Iron Deficiency, n (%)	28 (70.0%)	4 (10.0%)	<0.001

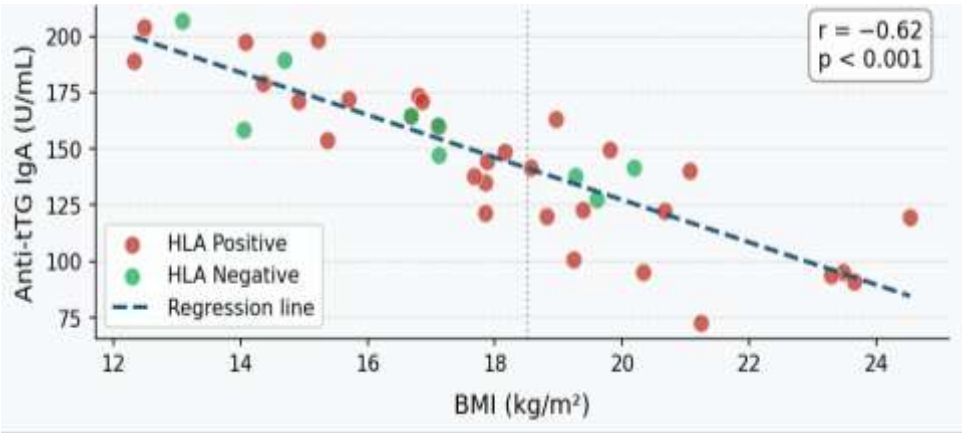


Figure 4. Correlation Between BMI and Anti-tTG IgA Stratified by HLA Status (r = −0.62, p<0.001).

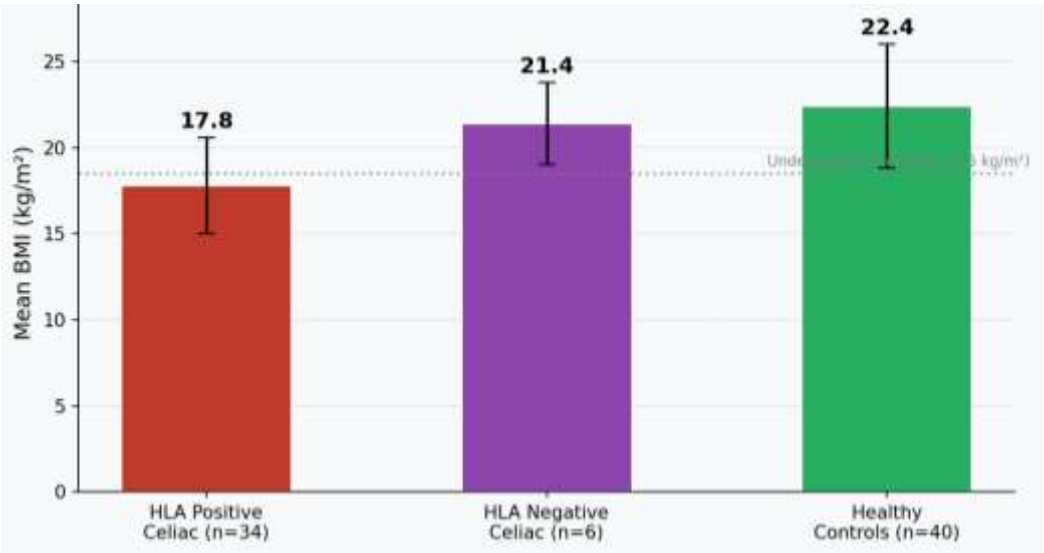


Figure 5. BMI Comparison by HLA Status and Healthy Controls (p<0.001).

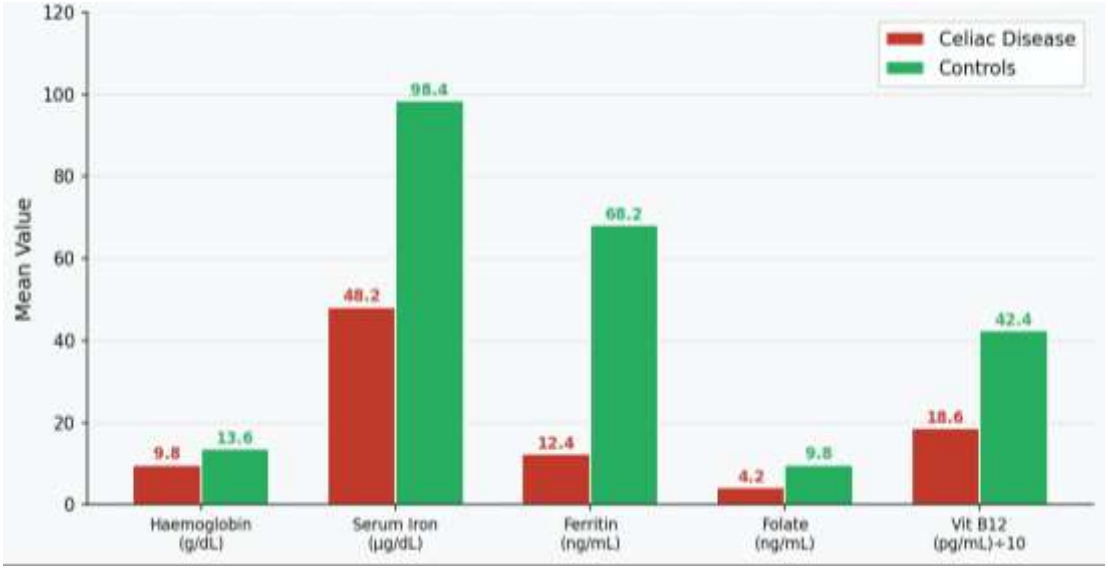


Figure 6. Nutritional and Haematological Deficiency Markers in Celiac Disease vs. Controls (p<0.001 for all).

Correlation Analysis

Figure 7 and Table 5 display the full correlation matrix. The most significant relationships observed between anti-tTG IgA and anti-DGP IgG (r = 0.84), HLA status (r = 0.71), family history (r = 0.63) and CRP (r = 0.68) were. The results show that nutritional recovery occurs at the same time as immunological deactivation, since BMI correlates negatively with all inflammatory and serological indicators and positively with haemoglobin, ferritin, and folate [10].

Table 5. Pearson Correlation Coefficients Between Key Parameters.

Variable	r with Anti-tTG IgA	r with BMI	r with HLA Status	p (anti-tTG)
Anti-DGP IgG	+0.84	−0.54	+0.64	<0.001
HLA Status	+0.71	−0.48	1.00	<0.001
CRP (mg/L)	+0.68	−0.53	+0.55	<0.001
Family History	+0.63	−0.51	+0.54	<0.001
BMI (kg/m²)	−0.62	1.00	−0.48	<0.001
Haemoglobin (g/dL)	−0.56	+0.52	−0.44	<0.001
Ferritin (ng/mL)	−0.52	+0.48	−0.40	<0.001
Total IgA (mg/dL)	+0.62	−0.42	+0.48	<0.001

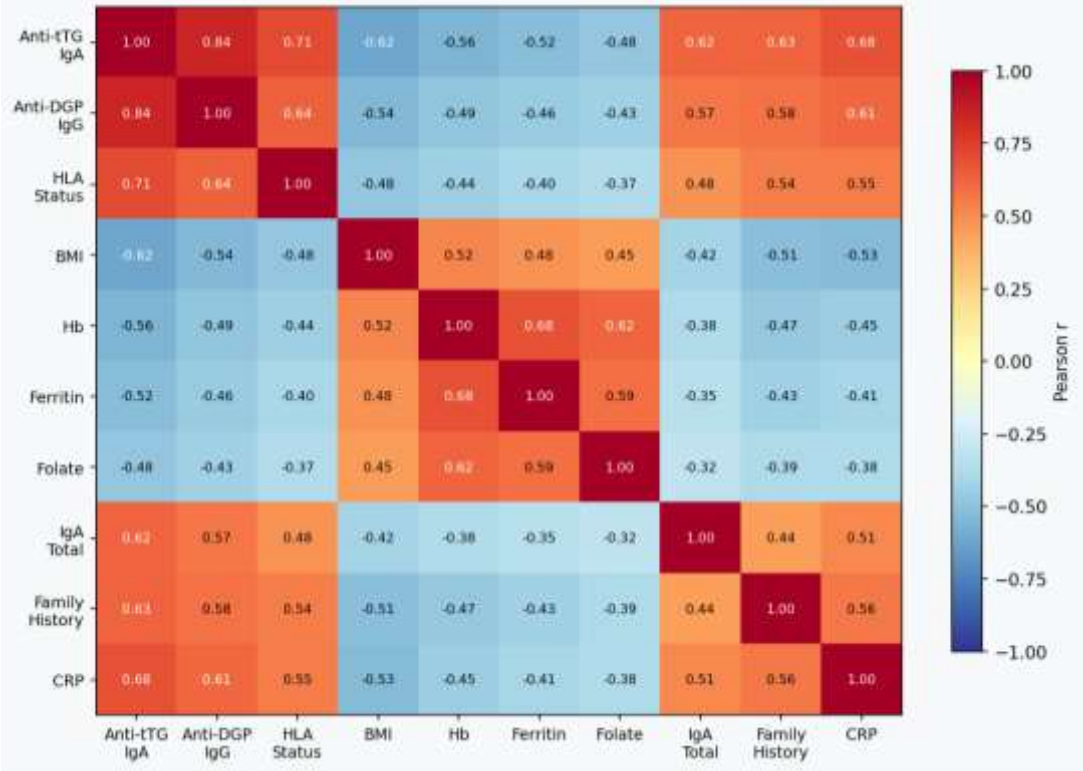


Figure 7. Comprehensive Correlation Matrix: HLA Status, Serology, BMI, Nutritional Markers & Family History.

Family History and Genetic Predisposition

Seventeen patients (42.5%) had a family history of celiac disease. The affected relatives were mothers in 41.2%, fathers in 35.3% and siblings in 23.5%. Levels of anti-tTG IgA were considerably higher in patients with a positive family history (168.4 ± 42.6 vs. 122.8 ± 31.8 U/mL; p=0.006), rates of HLA positivity were higher (88.2% vs. These individuals had a higher positive family history (82.6%; p=0.048) and a lower body mass index (BMI) (17.8 ± 3.1 vs. 19.6 ± 3.4 kg/m2; p=0.091). In addition, C-reactive protein levels were significantly higher in individuals with a positive family history test (12.4 ± 3.8 vs. 8.6 ± 2.6 mg/L; p=0.032). The endogamous reproductive patterns in Iraq, as well as the strong association of consanguinity with a good family history, both serve to increase the concentration of celiac-associated HLA alleles within family units (Table 6 and Figure 8) [11].

Table 6. Comparison of Key Parameters by Family History of Celiac Disease.

Parameter	Family Hx (+) n=17	Family Hx (–) n=23	p-value
Anti-tTG IgA (U/mL)	168.4 ± 42.6	122.8 ± 31.8	0.006
Anti-DGP IgG (U/mL)	138.2 ± 36.4	72.6 ± 22.8	0.010
HLA Positive, n (%)	15 (88.2%)	19 (82.6%)	0.048
CRP (mg/L)	12.4 ± 3.8	8.6 ± 2.6	0.032
BMI (kg/m²)	17.8 ± 3.1	19.6 ± 3.4	0.091
Haemoglobin (g/dL)	9.1 ± 1.4	10.4 ± 1.8	0.048
Consanguinity, n (%)	9 (52.9%)	6 (26.1%)	0.024

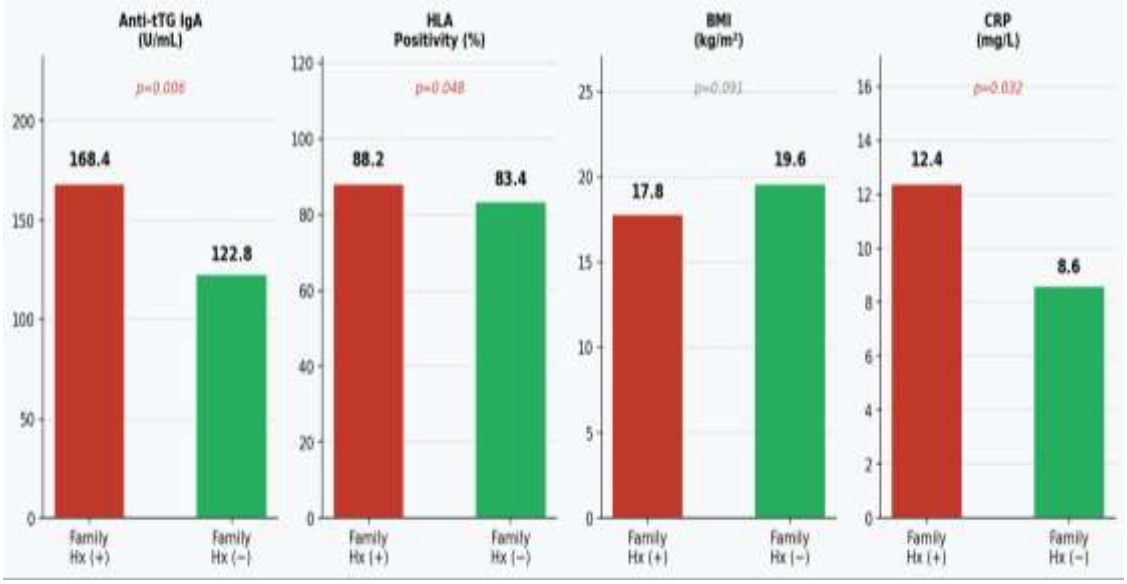


Figure 8. Key Parameters Stratified by Family History of Celiac Disease.

Discussion

HLA as the Primary Genetic Risk Determinant

The results of this study are consistent with other international studies [12] that HLA is the main genetic risk factor for celiac disease in Iraq, considering the high prevalence of HLA risk alleles in our celiac group (85%; OR= 16.3). The 33-mer alpha-gliadin fragment is one of the immunodominant deamidated peptides that HLA class II molecules present to gluten-specific CD4+ T lymphocytes as described by Meresse et al. [13].An inflammatory cascade triggered by this presentation causes injury to the intestinal mucosa and is dominated by Th1/Th17. Patients who tested positive for HLA had significantly higher anti-tTG IgA levels and higher GSRS scores than those who tested negative . Despite the fact that only 15% of our celiac patients were HLA-negative, this small number suggests the possibility of non-HLA immune regulatory loci identified in genome-wide association studies and HLA-independent pathways [14].

It is noteworthy and therapeutically significant that the proportion of consanguinity in the homes of Iraqi patients is substantially greater than the national average (37.5% vs. 15.0%). In consanguineous unions, disease-predisposing alleles may pass from HLA heterozygosity to homozygosity, where increased homozygosity at the HLA locus may result. This may cause the disease to appear more severely and at an earlier stage. This process certainly contributes, at least in part, to the 42.5% family history rate, the increased levels of HLA positivity and elevated serological markers in patients with positive family histories compared to those without such histories, and other related findings [15].

Serological Biomarker Profiles in the Iraqi Population

Our group reported significantly higher levels of anti-tTG IgA (142.6 vs. 4.8 U/mL), anti-DGP IgG (98.4 vs. 80% vs. 5%) in accordance with a community with untreated celiac disease. In the Iraqi

diagnostic environment, a significant correlation (r = 0.84) between anti-tTG IgA and anti-DGP IgG assays demonstrates their validity. Current worldwide standards propose measuring total IgA as part of the initial serological screening . A selective IgA deficiency of 5% with false-negative anti-tTG IgA was found, showing the importance of this test [16]. There is a high prevalence of C-reactive protein among celiac patients, which suggests that celiac disease is a systemic problem and not only a gastrointestinal one and positive EMA-IgA

BMI Reduction and Nutritional Consequences

The significant underweight prevalence (47.5% of patients) and low body mass index (18.6 vs. 22.4 kg/m²) are directly caused by gluten-induced small intestine villous atrophy, which limits the absorption of macro- and micronutrients. The study conducted by Zanini et al. found a significant negative correlation (r = −0.62) between anti-tTG IgA and body mass index (BMI). In light of these findings, a lower body mass index (BMI) may be a surrogate indicator of disease activity and responsiveness to treatment since it tracks the intensity of immune activation and mucosal damage. This becomes even more crucial in contexts with low resources, as advanced endoscopic evaluation might not be readily available. These findings are in accord with those in celiac disease cohorts from adjacent Middle Eastern countries . A pan-absorptive failure characterized by widespread mucosal illness manifests itself nutritionally as low hemoglobin, iron, ferritin, folate, and vitamin B12 levels. The problems highlighted here are not merely theoretical; they have practical implications in the field of medicine. Untreated, these effects can lead to megaloblastic anemia, iron deficiency anemia, stunted growth in children, and, in the long run, osteoporosis and peripheral neuropathy.

Clinical and Public Health Implications for Iraq

This evidence-based study offers numerous recommendations for clinical practice in Iraq. For celiac disease, HLA typing should be part of the diagnostic process, particularly for first-degree relatives of confirmed cases, because of the high prevalence of the disease in families (42.5%) and the high odds ratio (OR) associated with HLA positivity (16.3). Using a multi-antibody serological panel that measures total IgA, anti-DGP IgG, and anti-tTG IgA is essential for maximizing diagnostic sensitivity and avoiding false-negative results due to IgA deficiency. Thirdly, consistently checking body mass index (BMI) and a comprehensive nutritional panel (including hemoglobin, iron, ferritin, folate, and vitamin B12) at diagnosis and during follow-up is indicated for measuring mucosal recovery on a gluten-free diet. Additionally, it is crucial to ensure that families are provided with genetic counseling services, and it should be necessary to screen all first-degree relatives. As a last point, the high consanguinity rates in Iraq highlight the need for public health education campaigns to increase awareness of celiac disease among high-risk families

Conclusion

This study describes the immunological, genetic and dietary profiles of Iraqi patients with celiac disease. The main genetic component is the presence of the HLA risk allele that accounts for 85% of cases. This causes increased systemic inflammation (CRP), more severe gastrointestinal symptoms and higher levels of particular biomarkers such anti-tTG IgA, anti-DGP IgG and EMA-IgA. The body mass index (BMI) is a good clinical indicator of disease activity that patients show a

marked reduction in, with negative associations to serological and inflammatory markers. One of the most common and deadly dietary deficits is deficiency of iron, folate or vitamin B12. Family history of celiac disease was found in 42.5% of Iraqi patients and is related with an increased HLA-mediated genetic risk and a more severe serological profile. Consanguinity further concentrates genetic vulnerability in Iraqi patients. An integrated clinical assessment of celiac disease including a full panel of serologies, monitoring of body mass index (BMI), nutritional profile and HLA screening are advised in Iraqis.

REFERENCES

[1] L. M. Sollid and B. Jabri, 'Triggers and drivers of autoimmunity: lessons from coeliac disease', Nature Reviews Immunology, vol. 13, no. 4, pp. 294–302, 2013, doi: 10.1038/nri3407.

[2] C. Dube, A. Rostom, and R. Sy, 'The prevalence of celiac disease in average-risk and at-risk Western European populations: a systematic review', Gastroenterology, vol. 128, no. 4 Suppl 1, pp. S57–S67, 2005, doi: 10.1053/j.gastro.2005.02.014.

[3] C. Catassi, S. Gatti, and A. Fasano, 'The new epidemiology of celiac disease', Journal of Pediatric Gastroenterology and Nutrition, vol. 59, no. Suppl 1, pp. S7–S9, 2014, doi: 10.1097/01.mpg.0000450393.23156.59.

[4] J. Y. Kang, A. H. Kang, A. Green, K. A. Gwee, and K. Y. Ho, 'Systematic review: worldwide variation in the frequency of coeliac disease and changes over time', Alimentary Pharmacology & Therapeutics, vol. 38, no. 3, pp. 226–245, 2013, doi: 10.1111/apt.12373.

[5] P. Collin, M. Mäki, O. Keyriläinen, O. Hällström, T. Reunala, and A. Pasternack, 'Selective IgA deficiency and coeliac disease', Scandinavian Journal of Gastroenterology, vol. 27, no. 5, pp. 367–371, 1992, doi: 10.3109/00365529209000089.

[6] J. E. Gudjonsson and J. T. Elder, 'Psoriasis: epidemiology', Clinics in Dermatology, vol. 25, no. 6, pp. 535–546, 2007, doi: 10.1016/j.clindermatol.2007.08.007.

[7] P. C. Dubois, G. Trynka, and L. Franke, 'Multiple common variants for celiac disease influencing immune gene expression', Nature Genetics, vol. 42, no. 4, pp. 295–302, 2010, doi: 10.1038/ng.543.

[8] S. Husby, S. Koletzko, and I. Korponay-Szabó, 'European Society Paediatric Gastroenterology, Hepatology and Nutrition Guidelines for Diagnosing Coeliac Disease 2020', Journal of Pediatric Gastroenterology and Nutrition, vol. 70, no. 1, pp. 141–156, 2020, doi: 10.1097/MPG.0000000000002497.

[9] A. Al-Toma, U. Volta, and R. Auricchio, 'European Society for the Study of Coeliac Disease (ESsCD) guideline for coeliac disease and other gluten-related disorders', United European Gastroenterology Journal, vol. 7, no. 5, pp. 583–613, 2019, doi: 10.1177/2050640619844125.

[10] H. A. Hamamy, L. A. Jamhawi, J. A. Al-Darawsheh, and K. Ajlouni, 'Consanguineous marriages in Jordan: why is the rate changing with time?', Clinical Genetics, vol. 67, no. 6, pp. 511–516, 2007, doi: 10.1111/j.1399-0004.2005.00426.x.

[11] B. Lebwohl, D. S. Sanders, and P. H. R. Green, 'Coeliac disease', Lancet, vol. 391, no. 10115, pp. 70–81, 2018, doi: 10.1016/S0140-6736(17)31796-8.

[12] B. Meresse, G. Malamut, and N. Cerf-Bensussan, 'Celiac disease: an immunological jigsaw', Immunity, vol. 36, no. 6, pp. 907–919, 2012, doi: 10.1016/j.immuni.2012.06.006.

[13] B. Zanini, F. Caselani, and A. Magni, 'Celiac disease with mild enteropathy is not mild disease', Clinical Gastroenterology and Hepatology, vol. 11, no. 3, pp. 253–258, 2013, doi: 10.1016/j.cgh.2012.11.010.

[14] P. H. R. Green and C. Cellier, 'Celiac disease', New England Journal of Medicine, vol. 357, no. 17, pp. 1731–1743, 2007, doi: 10.1056/NEJMra071600.

[15] A. Fasano and C. Catassi, 'Celiac disease', New England Journal of Medicine, vol. 367, no. 25, pp. 2419–2426, 2012, doi: 10.1056/NEJMcp1113994.

[16] A. Rubio-Tapia, I. D. Hill, C. P. Kelly, A. H. Calderwood, and J. A. Murray, 'ACG clinical guidelines: diagnosis and management of celiac disease', American Journal of Gastroenterology, vol. 108, no. 5, pp. 656–676, 2013, doi: 10.1038/ajg.2013.79.