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RESEARCH ARTICLES

MYO9B Gene Polymorphism (SNP rs2305767) Associated with Celiac Disease in Iraqi Patients

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Abstract

Introduction: The *MYO9B* gene is involved in maintaining the intestinal barrier and is linked with a higher risk of developing celiac disease (CD). The aim of this investigation was to link the MYO9B gene polymorphism to CD in a patient sample from Iraq.
Patients and methods: In Bagdad, Iraq, 30 CD patients and 20 control individuals participated in the study. The *MYO9B* gene polymorphism was analyzed by gene sequencing. Enzyme-linked immunosorbent serologic assay was used to determine the concentrations of anti-transglutaminase (tTG) Ab, anti-gliadin Ab, and anti-endomysial (EMA) Ab in CD patients' serum.
Results: Three genotypes (Homozygote CC, Heterozygote CT, and Mutant Homozygote TT) and two alleles (C and T) were found for single nucleotide polymorphism (SNP) rs2305767. CD patients had three genotypes (CC, CT, and TT) for SNP, but only CC genotype was discovered in controls. The Hardy–Weinberg equilibrium analysis showed a significant difference between the patients and controls. Patients with CD had greater serum levels of gliadin, EMA, and tTG than healthy controls ($P < 0.05$).
Conclusion: Our findings suggest that SNP rs2305767 was associated with CD.

1. Introduction

Small intestine mucosa damage is a consequence of gluten sensitivity, which is the hallmark of celiac disease (CD), a persistent immune-mediated disorder [1]. It affects 0.6–1.0% of people in the United States and the majority of European countries, making it the most prevalent immunological gastrointestinal disorder in the West [2]. Although CD can manifest at any age, the majority of adult cases happen in the fourth and fifth decades of life. Most patients initially exhibit the "classical" symptoms of malabsorption, which include failure to thrive, diarrhea, or weight loss [1]. Nonetheless, it is becoming clear that CD can present with a wide range of clinical symptoms, including "non-classical" ones such as anemia, gastrointestinal distress, glossitis, and symptoms similar to irritable bowel syndrome. The only proven cure for the condition at this time is to adhere to a rigorous gluten-free diet for the rest of patient's life [3]. A major contributing aspect to CD predisposition is genetics. The Human Leukocyte Antigen DQ (HLA-DQ) locus is the most potent and thoroughly studied genetic susceptibility factor in CD [4]. Toxic peptides produced from cereal are presented to intestinal immune cells by HLA-DQ. In Northern Europe, nearly 90% of CD patients have the HLA-DQ2 haplotype (DQA1*0501/DQB1*0201), while one-third of the general population also carries this haplotype. Arguing that although the disease's hereditary nature can be explained by the HLA connection, this is not enough.

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An estimated 35% of the heritable risk of CD is linked to the *HLA-DQ* gene [5, 6]. The development of CD has been associated with recent genome-wide association (GWA) and multilineage studies of several common non-HLA genes, including SH2B adaptor protein 3 (SH2B3), cytotoxic T-lymphocyte antigen 4 (CTLA-4), interleukin-2 (IL-2), and protein tyrosine phosphatase nonreceptor 22 (PTPN22) [6, 7]. Understanding the non-HLA genetic components of CD will help create new treatment strategies and get a better understanding of the pathophysiology of CD. Most recently, the 19p13.1 Myosin IXB (*MYO9B*) gene was found to be susceptible to both CD and Inflammatory Bowel Disease (IBD) [7]. They may share genetic risk factors for autoimmune diseases [8]. The *MYO9B* gene encodes the Ras homologous (Rho) family's guanosine-triphosphatase (GTPase) activating protein, which is involved in the cytoskeletal structure of epithelial cells and influences the formation of tight junctions [7]. Human intestinal epithelial cells are among the tissues and cell types that express *MYO9B* [9]. *In vivo* intestinal permeability is positively linked with the elevated *MYO9B* gene expression [10]. Immune cells also exhibit high expression of *MYO9B*, indicating a direct involvement in immune activity [11]. The *MYO9B* gene has several single nucleotide polymorphisms (SNPs) found in it. Of them, rs1545620 (exon 20), rs1457092 (intron 20), rs2305767 (intron 14), and rs2305764 (intron 28) are the four polymorphisms that are examined most frequently [9]. At about a 2.8:1 female–male ratio, a greater preference is displayed for females [12], and two peaks [13], the first occurring after the weaning of the newborn and the second wave occurring in the thirties or later years of life [14]. For those who are genetically vulnerable, gluten consumption triggers the autoimmune illness CD. Around 1% of people globally have it. Improved CD diagnosis during the past few years has led to a rise in case diagnoses, particularly in the subclinical form of the illness. Various forms of presentation are proposed, and the clinical presentation might vary greatly—extra-intestinal, refractory, prospective, and intestinal [15]. In children aged <3 years, digestive disorders, such as diarrhea, appetite loss, underweight, and bloating in the abdomen, are more common [16]. Constipation, diarrhea, abdominal colic, and distension are the digestive symptoms of adult CD. The extra-intestinal symptoms of CD can vary and show up in both adult and pediatric cases as follows: symptoms related to the skeletal system such as growth retardation and short stature, neurological symptoms, psychological disorders, ocular symptoms, dermatological symptoms, fatigue, musculoskeletal symptoms, cardiopulmonary symptoms, and reproductive system [17].

2. Purpose of the Study

To link the *MYO09B* polymorphism to CD in a sample of Iraqi patients.

3. Methods

3.1. Design of the study

This case-control study aims to identify *MYO9B* single nucleotide polymorphism (SNP) rs2305767 and compare it with non-Celiac subjects recruited from Baghdad's medical city between November 2022 and February 2023. A total of 30 patients, with the age ranging from 1 to 60 years, were included. Patients whose clinical symptoms were strongly suggestive of CD were diagnosed by specialists. In all, 20 controls were enrolled in the Biomarker analysis. Complete blood count (CBC) was performed by Abbott Hematology's CELL-DYN Ruby auto-analyzer. The serum levels of anti-gliadin immunoglobulin G (IgG), anti-gliadin IgA, anti-transglutaminase (tTG) IgG, tTG IgA kit (AESKU/Germany) and anti-endomysial IgG, anti-endomysial IgA kit (GENERIC ASSAYS GmbH/Germany) were measured using enzyme-linked immunosorbent serologic assay (ELISA) to quantify the concentration of each antibody in serum.

3.2. Molecular detection of *MYO9B* Gene Polymorphism (SNP rs2305767)

All samples (30 patients and 20 controls) were subjected to the molecular detection of the *MYO9B* gene polymorphism (SNP rs2305767). ReliaPrep™ Blood gDNA Miniprep System (Promega, Madison, WI, US) was used for complete genome extraction from blood samples.

3.3. Primers

The lyophilized form of these primers was supplied by Macrogen Company (Rockville, MD, USA). The lyophilized primers were dissolved in a stock solution that was free of nucleases, resulting in a final concentration of 100 pmol/µL. A workable primer solution of 10 pmol/µL was created by mixing 90 µL of nuclease-free water with 10 µL of primer stock solution (stored at -20°C in a freezer). (see Table 1).

3.4. Reaction set-up and thermal cycling protocol

The polymerase chain reaction (PCR) protocol used for the *MYO9B* gene analysis included an initial denaturation

Table (1): Forward and reverse primers for the *MYO9B* gene used in SNP genotyping.

Primer	Sequence 5'-3'	Annealing temp (°C)	Product size (bp)
rs2305767-F	TCTCTTGAGTAGCTGGGATTA	60	745
rs2305767-R	GTGTCGGGATTTGGGTTAG		

step at 95°C for 5 min, followed by 30 cycles of denaturation at 95°C, annealing at 60°C, and extension at 72°C, each for 30 s. This was completed with a final extension at 72°C for 7 min. The protocol concluded with a holding step at 10°C for 10 min to stabilize reaction products before further analysis.

3.5. Electrophoresis using agarose gel

The PCR amplification was followed by agarose gel electrophoresis to confirm the presence of amplification. Only particular requirements of the isolated DNA were necessary for PCR to be successful.

3.6. Standard sequencing

The ABI3730XL, an automated DNA sequencer, was used to singer sequence the PCR products that were sent to Macrogen Corporation, South Korea. Email was used to collect findings, which were then assessed using the Geneious software.

3.7. Statistical analysis

Data analysis was conducted using IBM SPSS-29 (IBM Statistical Packages for Social Sciences, version 29, Chicago, IL, USA). Student's *t*-test was used to compare two independent mean values, and ANOVA assessed differences across multiple groups, with statistical significance set at $P \leq 0.05$. Odds ratios (OR) quantified the likelihood of an outcome based on exposure, where OR > 1 indicates increased likelihood, and OR < 1 indicates decreased likelihood. 95% confidence interval (95% CI) provided insight into the precision of OR, with confidence interval excluding 1 indicates statistical significance.

4. Results

4.1. Single nucleotide polymorphism of the *myo9b* gene

The current study focused on the involvement of the *MYO9B* gene in the etiology of CD by examining SNPs found on the *myosin* gene, which was described in chromosome 19 (intron 28), region rs2305767.

4.2. Detection of rs2305767 SNP in study groups

SNP rs2305767 (C/T), chromosome 19 (780 bp) was presented with three genotypes (homozygote CC, heterozygote CT, and mutant homozygote TT) and two alleles (*C* and *T*) (Figure 1). Three genotypes (CC, CT, and TT) of SNP were recognized in CD patients, while in controls, 75% observed (CC), 20% (CT), and only 5% observed (TT) genotypes. Analysis of HWE revealed significant differences between the patient and control groups (Table 2).

Comparison between the patient and control groups showed significant differences. The homozygous TT genotype and heterozygous CT genotype in CD patients had higher frequency than controls ($P = 0.005$, $P = 0.002$, respectively). On the other hand, the genotype CC was the most common one in the control group (wild type). In the conditions of this study, rs2305767 polymorphism was associated to CD. The CC genotype of rs2305767 polymorphism acted as a protective factor (wild) and TT and CT genotypes as risk factors. This result supports the idea that polymorphism in the *MYO9B* gene is associated with CD (Figures 2–4).

4.3. Hematologic characteristics of the study groups

The results shown in Table 3 display a summarized comparison of hematology tests among the study

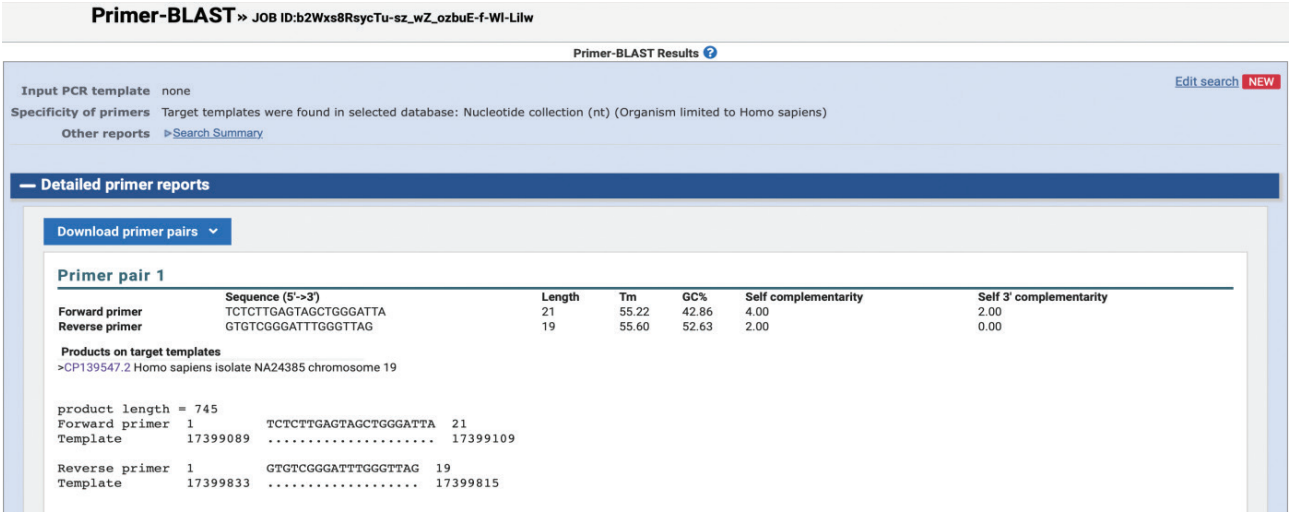

Figure (1): Primer-BLAST alignment results showing the binding sites of rs2305767-specific primers on Homo sapiens chromosome 19 (template CP1399547.2), producing a 745-bp amplicon. (Source: https://www.ncbi.nlm.nih.gov/tools/primer-blast/primertool.cgi?ctg_time=1753720803&job_key=b2Wxs8RsycTu-sz_wZ_ozbuE-f-Wl-Lilw)

Table (2): Genotype numbers and percentages of the *MYO9B* gene (SNP rs2305767) as well as the Hardy–Weinberg equilibrium (HWE) for controls and CD patients.

SNP rs2305767	Celiac disease		Control		*P*-value	OR (95% CI)
	No.	%	No.	%		
TT	12	40.0	1	5.00	0.002*	9.0 (1.82–44.59)
CT	14	46.7	4	20.0	0.002*	10.5 (2.15–51.28)
CC (wild)	4	13.3	15	75.0	**	Ref.

Notes. *Significant difference between percentages using Pearson Chi-square test at 0.05 level.
**Odds ratio and its 95% confidence interval (95% CI) in comparison to the Reference group (wild).

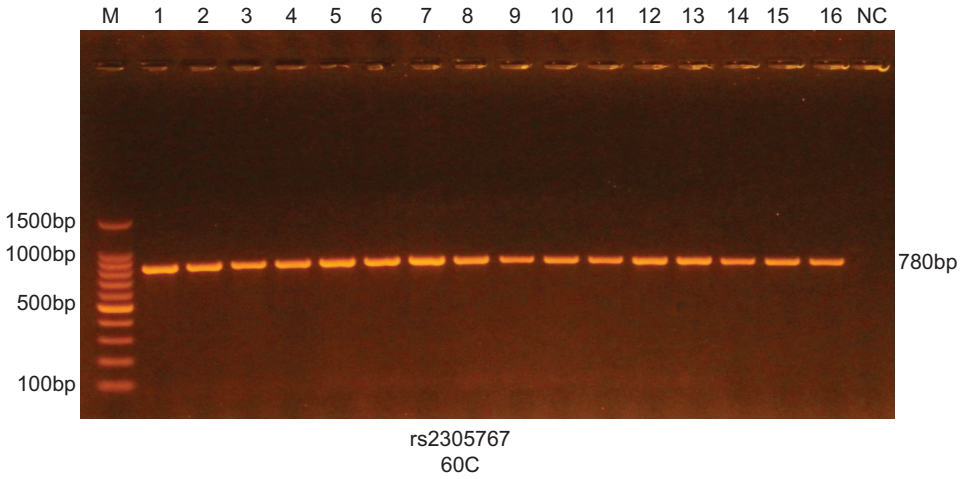

Figure (2): The *MYO9B* gene PCR products at 45 min of 1.5% agarose gel electrophoresis at 5 V/cm^2 shows bands with a molecular size of 780 bp (Lane M: 100-bp DNA ladder; CD patients and control).

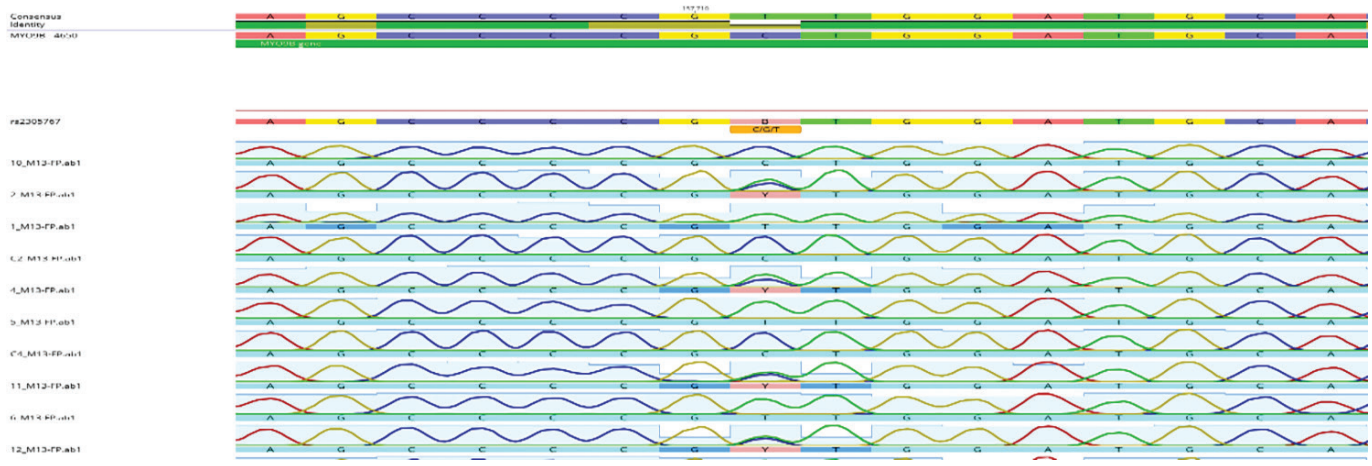

Figure (3): Three genotypes (CC, CT, and TT) are displayed in the DNA sequence chromatogram of the *MYO9B* gene SNP (rs2305767 A/G). Furthermore, the reference sequence (rs2305767) is provided.

groups (patient and control groups) within diversely signifying *P* values for each parameter, which involve the following: white blood corpuscles (WBC), 9.48±3.01 and 6.21±1.41 had significant differences (*P* = 0.003); hemoglobin (Hb), 10.56±3.51 and 14.10±1.39 with highly significant differences (*P* = 0.0001); platelets (PLT) 260±61 and 210±45 within nonsignificant differences (*P* = 0.09).

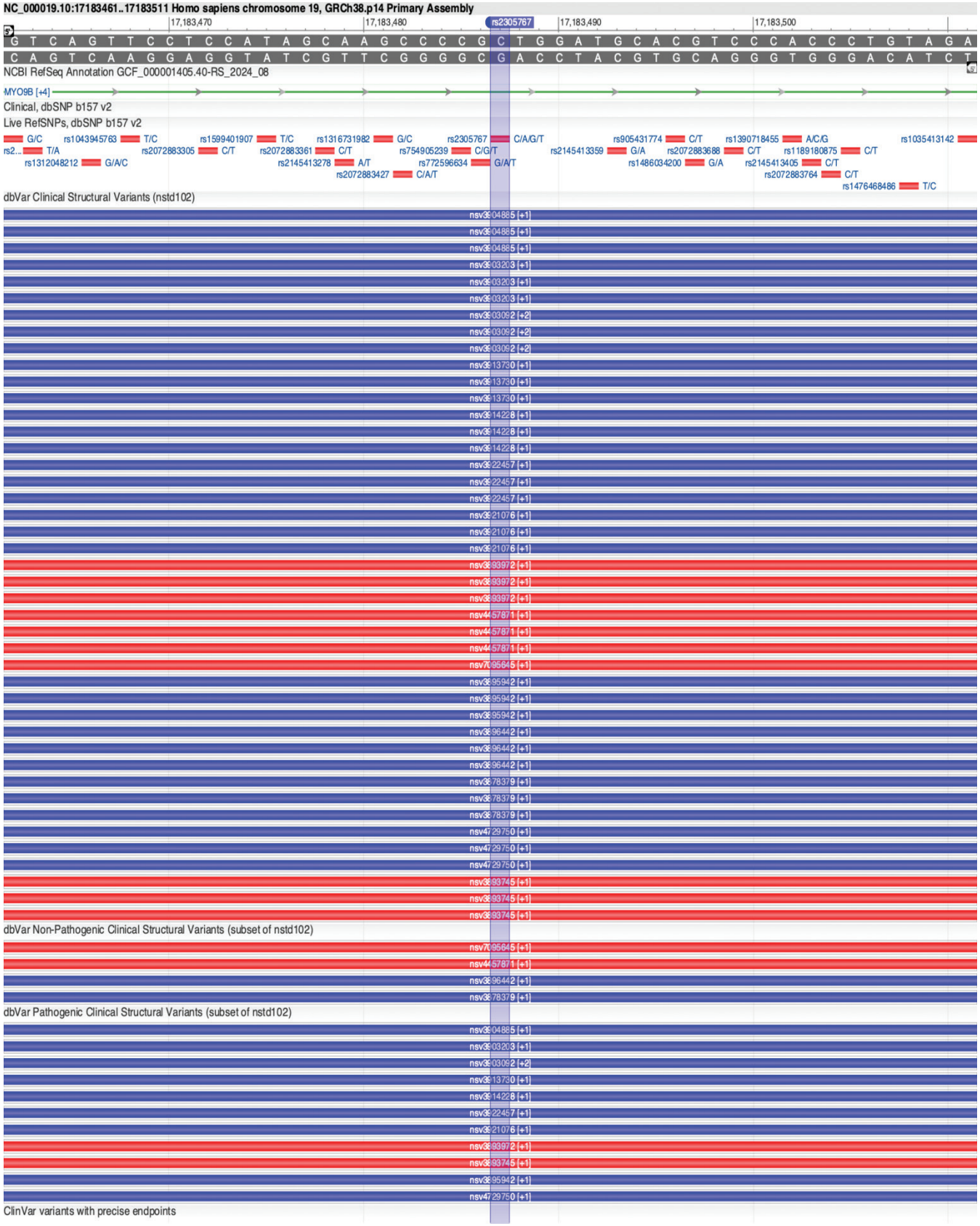

Figure (4): Visualization of rs2305767 variant and surrounding SNPs on chromosome 19 (GRCh38.p14). (Source: https://www.ncbi.nlm.nih.gov/variation/view/)

4.3. Detection of anti-gliadin, anti-transglutaminase, and anti-endomysial (IgG and IgA) in CD and control groups

Comparing the levels of anti-gliadin IgG and IgA serum in CD patients and the control group showed considerably higher levels of anti-gliadin IgG and IgA serum in CD patient group, compared to the control group. The concentration of anti-gliadin IgG and IgA increased significantly (P = 0.0001*) in CD patients, compared to healthy controls (Table 4).

The level of anti-tTG Ab was assessed in serum of both patient and control groups. Results (Table 4) showed a highly significant difference between the patient and control groups (P = 0.0001). The anti-tTG IgG and IgA median range for CD patients and healthy controls was 27.96, 33.52, and 2.88, 4.43 pg/mL, respectively (Table 4).

The general statistical parameters about anti-endomysial IgG and IgA as differentiated markers between patient and control groups showed highly significant differences (P = 0.0001). Likewise, the lowest mean ± SD values in the control group for anti-endomysial IgG and IgA were 3.47±0.79 and 4.02±0.64, respectively; the patient group showed the highest mean values of 26.17±4.38 and 31.40±6.69 for anti-endomysial IgG and IgA, respectively (Table 4).

5. Discussion

Demonstrating homozygosity for *MYO9B* and *HLA-DQ2* may be related to the prognosis of CD or the risk of developing CD [12,13]. The myosin gene *MYO9B*, which was recently discovered to be located on chromosome 19 (intron 28), encodes a single-headed molecular motor with a Rho-family GTPase activating protein (GAP) domain in the tail region that is a member of the class IX myosin family [7]. Being an essential component of the intestinal mucosa's structure, it is expressed in intestinal epithelial cells. *MYO9B* is a negative regulator that down-regulates Rho-dependent signaling pathways and promotes the inactivation of Rho GTPases [11]. Increased epithelial paracellular permeability is a result of tight junction construction and actin filament remodeling, both of which are significantly regulated by Rho GTPases [8,14]. The latter finding thus supports the theory that *MYO9B* variations could alter the gut's epithelial barrier function, making it easier for gluten peptides to penetrate the deeper mucosal layer and reach the immune system. RS2305767 agrees with the data of another study [9]. As shown in Table 2, which demonstrated the *MYO9B* rs2305767 polymorphism is connected to CD and Loeff *et al.*, Madrid; data [12] does not support this. Additionally, the meta-analysis of seven studies involving 4,894 controls and 1,965 CD patients yielded the most thorough evaluation of the significance of four frequent *MYO9B* polymorphisms to CD to date. The rs1545620 polymorphism is associated with an increased risk of CD in Europeans. However, no overall association was found between rs1457092, rs2305767, or rs2305764 and CD risk across the studied populations. Notably, in the Latin American subgroup, rs1457092 and rs2305767 showed a potential link to CD susceptibility.

First, the great general variability observed in global population is cleared by the similarities and differences that have been described. Second, environmental and genetic factors combine to determine the

Table (3): Hematologic characteristics of the study groups.

Hematology test	CD Mean ± SD (range)	Control Mean ± SD (range)	*P* value
Hb (g/dL)	10.56 ± 3.51 (6.16–16.0)	14.10 ± 1.39 (11.32–17.27)	0.0001*
WBC (10^9/L)	9.48 ± 3.01 (4.53–15.1)	6.21 ± 1.41 (4.0–9.10)	0.003*
Platelets (PLT) (10^9/L)	260 ± 61 (160–560)	210 ± 45 (171.1–364)	0.09 NS

Note. *Significant differences between two independent mean values using the Student's *t*-test or between two independent mean values using the ANOVA test at 0.05 level.

Table (4): Detection of anti-gliadin IgG and IgA in the patients with CD and the control group.

Groups	Mean ± SE					
	Anti-gliadin IgG (U/mL)	Anti-gliadin IgA (U/mL)	Anti-Transglutaminase IgG(U/mL)	Anti-Transglutaminase IgA(U/mL)	Anti-endomysial IgG (U/mL)	Anti-endomysial IgA (U/mL)
Patients	42.80 ± 8.96	34.95 ± 6.23	27.96 ± 5.82	33.52 ± 7.27	26.17 ± 4.38	31.40 ± 6.69
Controls	5.43 ± 1.09	5.51 ± 1.05	2.88 ± 0.73	4.43 ± 0.64	3.47 ± 0.79	4.02 ± 0.64
T-test	17.923*	12.556*	11.650*	14.488*	8.834*	13.344*
P value	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001

Note. *P < 0.01.

complex nature of CD. Various environmental influences within different ethnic groups may modify an individual's genetic vulnerability to CD. Third, clinical variation between various ethnic groups could contribute to the discrepancy's explanation. More information is needed to help understand how these polymorphisms and CD relate to one another. CD is a prevalent factor in the development of many hematologic abnormalities, with anemia being the most frequently observed anomaly.

It was observed in the present study that there was a significant decrease in hemoglobin level in CD patients, compared to healthy groups, and this result was in agreement with other studies conducted in Iraq [18,19,20]. It was also in agreement with the findings of Güngör and Acıpayam [21] in Turkey, who proved a significant relationship between CD and hemoglobin level. The total mean count of WBCs was significantly elevated in CD patients. CD is a small intestine mucosal illness that causes malabsorption because of an inflammatory reaction to gluten; neutrophils and lymphocytes are key players in inflammatory process [11]. In agreement with these findings, previous studies also demonstrated that WBC counts were elevated in CD patients [19,22].

CD patients revealed some significant variations in PLT counts; however, Table 3 shows that the difference was not significant ($P = 0.09$). Several studies about PLT count in CD patients agreed with the result of the current study [23,24].

Gluten exposure in the small intestine has the potential to cause aberrant immune responses and the production of various autoantibodies that can impact multiple organ systems. This causes an inflammatory reaction and may lead to villous atrophy, which is the shrinkage of the villi lining of the small intestine, which affects the absorption of nutrients in the intestine. Serological assays, such as anti-tTG Ab, anti-gliadin antibodies (AGA), and anti-endomysial antibodies (EMA), are typically used to diagnose CD; nevertheless, small bowel biopsy and histological analysis are required to confirm intestinal damage [2]. On the other hand, the measurements of anti-gliadin IgG and IgA (Table 5) indicate a substantial and statistically significant ($P < 0.0001$) increase in the levels of anti-gliadin IgG and IgA in the sera of CD patients. This result matches the results other studies conducted in Iraq [25] as well as globally [26].

Testing of sera for anti-gliadin IgG and IgA immunoreactivity to gliadin is typically an initial step in the complex process of identifying gluten intolerance. It has been demonstrated that an anti-tTG is the most suitable marker for mass screening of CD. It is also helpful in determining whether patients would benefit from regular follow-up and a gluten-free diet. The Triticeae class of grass, which includes wheat, barley, and rye, produces wheat gliadin and similar gluten proteins, which trigger the production of T-cells that lead to tTG autoimmunity. A characteristic of T-cells is their capacity to respond to Ags, such as gliadin, which are HLA-DQ8 and DQ2-restricted antigens. Consistent with these findings, earlier research reported considerably higher levels of serum anti-Ttg Ab in CD patients, compared to the controls. In Iraq [27], studies showed positive results as in Iran [28] as well as globally [29]. There was the lowest expression for each of the EMA and Ttg, which clearly appeared in healthy controls, while there was an elevated expression in patients with CD, as shown in Table 6 Anti-EMA is a laborious confirmatory test that is not feasible in all centers. Moreover, this test also needs expert observers and is expensive. We were able to determine antibodies to endomysium using ELISA instead of fluorescent immunoassay [30]. Serology testing is an effective diagnostic technique for CD screening and follow-up, according to the study's findings. These outcomes are in line with earlier research carried out in Iraq [31,32].

6. Conclusion

Our results provided the first evidence that rs2305767 alleles of the *MYO9B* gene are linked to an elevated risk of CD development, and could impact the etiology and pathophysiology of CD. Patients with CD usually have decreased Hb levels and increased WBC counts, compared to the general population. In addition, PLT counts revealed some significant differences, but these differences were not statistically significant. In this study, anti-tTG Ab and anti-gliadin and anti-endomysial Ab were used and all demonstrated a significant increase in the serum levels of CD patients, compared to the controls. This shows that the best serological tool for diagnosing CD and distinguishing it from other medical conditions with comparable symptoms is still its antibody profile.

Table (5): Detection of anti-Transglutaminase IgG & IgA in the CD and the control group.

Groups	**Mean ± SE**	
	Anti-Transglutaminase IgG	Anti-Transglutaminase IgA
Patients	27.96 ± 5.82	33.52 ± 7.27
Controls	2.88 ± 0.73	4.43 ± 0.64
T-test	11.650**	14.488**
P-value	0.0001	0.0001

Note.**(P < 0.01)

Table (6): Detection of anti-endomysial IgG & IgA in the celiac disease and the control group.

Groups	Mean ± SE	
	Anti-endomysial IgG	Anti-endomysial IgA
Patients	26.17 ± 4.38	31.40 ± 6.69
Controls	3.47 ± 0.79	4.02 ± 0.64
T-test	8.834**	13.344**
P-value	0.0001	0.0001

Note. **($P < 0.01$)

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Ethical Approval

This study was approved by the Ethics Committee of Cankiri Karatekin University's, Cankiri, Turkey. The research was conducted in accordance with the Declaration of Helsinki's principles (project No. 208184214). Prior to any intervention, written informed consent was obtained from all subjects. All ethical concerns, including plagiarism, data manipulation, and double publication, were fully addressed by the authors. This prospective clinical study neither intervened patients' therapy nor provided any physical risks to participants; it just summarized clinical data and samples provided by the participants. Furthermore, information about the participants was secluded.

Conflicts of Interest

No conflicting interests were disclosed by the authors.

Funding/Support and Limitation of the Study

There was no explicit financing obtained for this study from the public, private, or nonprofit sectors, which could limit the resources available for more extensive investigations. Additionally, the study was conducted on a specific human model. Further research is needed to confirm the findings and explore the mechanisms in diverse biological systems.

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