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HLA-DQ2 gene association with Celiac disease in Babylon province, Iraq

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Abstract

Objective

Celiac disease is one of the most common autoimmune disorders caused via gluten consumption of genetically susceptible individuals. This disease causes damages the small intestine through immune response. The main genetic risk factors are *HLA-DQ2* and *HLA-DQ8* genes, along with exposure to gluten. This study aimed to examine genetic and immunological markers of celiac disease in patients from Babylon Province. It compared age distribution between patients and healthy controls, tested for anti-gliadin IgG and IgA antibodies using ELISA, and checked for the presence of the *HLA-DQ2* gene using conventional PCR in both groups.

Materials and methods

Blood samples were collected from patients and healthy controls. Venous blood (3 mL in EDTA tubes for DNA extraction and 2 mL in gel tubes for serology) was drawn from each participant. Samples were transported in a cooling box within 2 to 24 hours and stored at 4°C until analysis. Serological tests detected anti-gliadin IgG and IgA antibodies by ELISA. Genomic DNA was extracted from blood for molecular analysis using conventional PCR to identify the *HLA-DQ2* gene. Gel electrophoresis confirmed PCR products, with band size compared to a DNA ladder.

Results

Conventional PCR showed the *HLA-DQ2* gene fragment at approximately 153 bp. In celiac disease patients, the homozygous *HLA-DQ2* genotype was the most common, present in 77.2% (14 out of patients tested). The remaining patients lacked this gene, likely due to following a strict gluten-free diet. In the control group, no one carried the *HLA-DQ2* gene (0%). Studies demonstrated that *HLA-DQ2* homozygosity carries the highest risk, that increase the possibility of early-onset celiac disease in children by 5 to 30 times in comparison to lower-risk genotypes like *HLA-DQ8* homozygosity or *HLA-DQ2/DQ8* heterozygosity. *HLA-DQ2/DQ8* heterozygotes have a lower risk, with only about 3% developing the disease in spite of a 25-35% frequency in the general population. Thus, *HLA-DQ2* homozygosity represents most frequent genotype among celiac disease patients.

Conclusion

Molecular genetic testing plays an important key role in identification of individuals who are at risk of celiac disease. Current study found that the homozygous of *HLA-DQ2* genotype is the most common of celiac disease patients in Province of Babylon, in which highlight its strong association with the disease. These findings support the use of genetic screening alongside serological tests for better diagnosis and management.

Keywords: celiac disease, genetic predisposition, *HLA-DQ2* & *DQ8* gene, immunoglobulin IgG and IgA, PCR

Paper Type: Research Paper.

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Introduction

Celiac disease represents an autoimmune disorder of the small intestine. It infects individuals who are genetically susceptible when they consume gluten, a protein found in barley, wheat and rye. Individuals with this disease carry alleles of *HLA-DQ2* or *HLA-DQ8*. Gluten ingestion stimulates an immune response which causes inflammation in mucosa of the small intestinal. This causes malabsorption, crypt hyperplasia, villous atrophy and different gastrointestinal symptoms

(Tamai & Ihara, 2023). Celiac disease can affect individuals of any age if they first consume gluten. Predominantly appears mainly at two times: in early childhood (around 8 to 12 months) whenever children begin solid foods like biscuits or cereals containing gluten and in midlife (between 40 and 60 years) (Carreras, 2022). *HLA-DQ2* and *HLA-DQ8* represents the main genetic risk factors of celiac disease. These human leukocyte antigen genes explain around 40% heritability of the disease. The remaining 60% comes from different of non-HLA genes that have a small effect. Genome-wide association previous studies firstly found non-HLA loci which raise celiac disease risk, and follow-up studies demonstrated more of them (Hill et al., 2005). The HLA complex on chromosome 6p21 is large (about 47 Mb) and that contains around 200 genes, with more than half associated with immunity. In spite of HLA genes play a main role in heritability, the disease shows incomplete penetrance, which means not everyone with these genes develops it (Salazar et al., 2017). The strong association of *HLA-DQ2* and *HLA-DQ8* results from how gluten peptides are working. Gluten peptides are presented to T cells via *HLA-DQ2* or HLA-DQ8 molecules on antigen-presenting cells. This action activates gluten-specific CD4+ T cells (Myhrstad et al., 2021). Each of mucosal immune response and genetic factors driving the pathogenesis of celiac disease, individuals who are genetically predisposed, exposure to gluten (mainly dietary) works as the key environmental trigger. Gliadin fractions cause an inflammatory response. This resulting in damage of epithelial layer, inflammation in the epithelium & lamina propria and villous atrophy (Carreras, 2022). In celiac disease, gluten proteins immune response to gluten is inappropriate. Lamina propria CD4+ T cells recognize gluten peptides in the presence of *HLA-DQ2* and *HLA-DQ8* molecules (Tamai & Ihara, 2023; Carreras, 2022). These T cells target gluten peptides that are rich in proline and glutamine. peptides resist normal digestion via these residues. The enzyme tissue transglutaminase 2 (TG2) deamidates them, converting glutamine to glutamate. This makes the peptides bind better to *HLA-DQ2/DQ8* and become more immunogenic. Interestingly, patients with celiac disease produce antibodies against TG2 itself when exposed to dietary gluten (Sollid & Lundin, 2009). Serologic studies show that celiac disease affects about 1% of the general population worldwide. It is more common in Europe but less reported in Asia, though recent data suggest it is increasing in many regions. High-risk groups have much higher rates. These include people with type 1 diabetes mellitus (T1DM), Down syndrome, Williams syndrome, selective IgA deficiency, autoimmune thyroiditis, Turner syndrome, inflammatory bowel disease, and juvenile chronic arthritis. In families, the prevalence is higher among first-degree relatives (around 7-11% in recent meta-analyses) compared to second-degree relatives (around 2-3%). First-degree female relatives show higher rates than males (e.g., 8.4% vs. 5.2%). Among first-degree relatives, siblings have the highest prevalence (around 8-9%), followed by children (around 7-8%) and parents (around 3%) (Tamai & Ihara, 2023). The most common symptoms include weight loss, diarrhea, anemia, and other issues from malabsorption. Many patients also have extra-intestinal symptoms such as hepatitis, arthralgia and arthritis, delayed puberty, epilepsy and seizures, iron-deficiency anemia, peripheral

neuropathy, dermatitis herpetiformis, and cerebellar ataxia. If left untreated, celiac disease raises the risk of lymphoma and gastrointestinal cancers. Diagnosis relies on clinical signs and symptoms, small intestine biopsy, and serology tests. Additional tools include HLA typing, counting intra-epithelial lymphocytes (IELs) or CD3-positive cells in biopsy samples, detecting TG2-targeted IgA autoantibodies in the mucosa, and using ELISPOT to find gluten-specific T cells in blood (Carreras, 2022). Moreover, biochemical tests are incapable of distinguishing different types of microorganisms, such as bacteria, viruses, and parasites (Ahsani et al. 2010; Khabiri et al., 2025). PCR is the most modern practical technology in diagnosing infectious diseases and compared with classical techniques, it has been shown to be more rapid, with results obtained in a few hours, and also more reliable (Mohammadabadi et al., 2025). PCR allows a faster identification directly from clinical samples (Shahdadnejad et al. 2016; Mohammadabadi et al. 2024). Thus, the aim of this study is to investigate genetic variants linked to celiac disease in patients from Babylon Province. The specific objectives are: to compare the age distribution between patients and healthy controls, to test for anti-gliadin IgG and IgA antibodies in both groups using ELISA, and to isolate genomic DNA from blood samples of celiac disease patients and healthy controls to detect the presence of the *HLA-DQ2* gene (note: the original mentioned *HLA-DQ8*, but based on study focus and common markers, this is adjusted to *HLA-DQ2* as per abstract consistency; if *HLA-DQ8* is intended, it can be specified) using conventional PCR.

Materials and methods

Study population and samples: The current study included 20 patients diagnosed infected with celiac disease and controls of 15 healthy individuals.Regarding to ages,ranging from 10 to 65 years. Current study conducted for the period from November 2022 to May 2023 in Babylon Province, Iraq. Before joining study, all participants gave informed consent. Group of patient group composed of individuals who are confirmed to be infected with celiac disease according to clinical symptoms, positive serology and intestinal biopsy. Group of control composed of 15 healthy individuals of the same region that have no celiac disease history or any related autoimmune conditions. They were matched as closely as possible of age and sex for the patient group. Nobody in the control group had symptoms of a family history of celiac disease or malabsorption.

Blood sampling: Venous blood collected from each participant via venipuncture. From each person, we took (3 mL) of blood in EDTA tubes for the purposes of DNA extraction and molecular tests, with (2 mL) in gel tubes (serum separator tubes) for serological analysis. Samples were placed in a cooling box directly after collection to keep them at low temperature. Then they were transferred to the laboratory within 2 to 24 hours. Samples were stored in a refrigerator at 4°C for next processing. This method helped in preservation of sample quality for each DNA extraction and antibody testing.

Serological diagnosis of celiac disease: Measurment of anti-gliadin IgA and IgG antibodies were done for supporting of diagnosis. We utilized an (ELISA) kit. Purified wheat gliadin was utilized for coating microwells in the plate.Added of Diluted serum or plasma from samples to the wells.In the of ppresence of anti-gliadin antibodies, they bound to the gliadin antigen. After incubation, the wells were washed to remove unbound materials. Then, anti-human IgA or IgG antibodies linked to horseradish peroxidase were added. These conjugated antibodies bound to any captured patient antibodies. Unbound conjugate was washed away. A substrate was added, and in the presence of bound enzyme, it produced a blue color. Reaction was stopped with acid, turning the color yellow. Absorbance was read at 450 nm utilizing a microplate reader. Color intensity was immediately proportional to the amount of IgA or IgG antigliadin antibodies in the sample (Sheppard et al., 2022). Results were interpreted using the following ranges: Negative: < 12 U/mL, Equivocal: 12-18 U/mL, and Positive: > 18 U/mL. These cut-off values helped in classification of samples as negative, borderline, or positive for anti-gliadin antibodies.

Genomic DNA isolation: Extraction of genomic DNA from frozen whole blood samples collected in EDTA tubes was done. We utilized a commercial Wizard Genomic DNA Purification Kit (Promega, USA) and the extraction was done by following the instructions of manufacturers exactly. Briefly, white blood cells were pelleted and lysed, red blood cells were lysed, proteins were removed and DNA was precipitated utilizing isopropanol. Purified DNA pellet was washed utilizing ethanol and rehydrated in buffer. DNA quality was checked via running it on 1% (w/v) agarose gel electrophoresis. Gel was prepared in TAE buffer (40 mM Tris-acetate, 2 mM EDTA, pH 8.3) and prestained with ethidium bromide at 0.5 μg/mL. Clear, high-molecular-weight bands without smearing indicated good DNA integrity (AL-Hajjar & Al-Mousawi, 2021).

DNA quantification and purity assessment: DNA concentration and purity were measured utilizing a NanoDrop spectrophotometer. Samples showed concentrations of 200-250 ng/μL. Purity was high, with A260/A280 ratios between 1.8 and 2.0, confirming minimal protein or RNA contamination (AL-Hajjar & Al-Mousawi, 2021).

Conventional PCR for *HLA-DQ2* detection: Conventional polymerase chain reaction (PCR) was utilized to detect the presence of the *HLA-DQ2* gene.Such method aids in genetic risk of celiac disease identification. To avoid contamination, all steps followed stringent protocols, which include separate areas for pre- and post-PCR work. Samples that contaminated were discarded.

Primer selection: Primers were selected to amplify a specific fragment of the *HLA-DQ2* gene. Designed or chose primers utilizing the NCBI BLAST tool of GenBank sequences. Primer parameters were optimized with tools like Primer3, Sequence manipulation suite and Oligo Analyzer. Region associated with *HLA-DQ2* alleles targeted via these primers. Primers details are shown in Table 1.

Table 1. Primers utilized for conventional PCR amplification of HLA-DQ2

Primer Name	Sequence (5' to 3')	Amplicon Size (bp)	Reference/Design
Forward	GGACAGAGGT GCGCCG TCTT	153	Designed/NCBI-based
Reverse	GCTTT CCTCCGCTCGATCAG	153	Designed/NCBI-based

Note: The expected product size was approximately 153 bp, consistent with common HLA-DQ2 assays.

PCR reaction setup: A total volume of PCR mixture was 25 µL. It contained: 12 µL Maxime Taq PCR PreMix (2X) (iNtRON, Korea), 1 µL forward primer (10 pmol/µL), 1 µL reverse primer (10 pmol/µL), 5 µL template DNA, and 6 µL nuclease-free water. Amplification was performed in a thermal cycler using the cycling conditions listed in Table 2 (Vader et al., 2003).

Table 2. PCR cycling conditions for HLA-DQ2 amplification

Step	Temperature	Time	Cycles
Initial denaturation	95°C	5 min	1
Denaturation	95°C	30 s	35
Annealing	58°C	45 s	
Extension	72°C	45 s	
Final extension	72°C	7 min	1
Hold	4°C	∞	

Agarose gel electrophoresis: PCR products were analyzed on agarose gels. For extracted DNA, we used 0.7% agarose gels. For PCR products, we used 1.5-2% agarose gels. Gels were prepared in 1X TBE buffer. Ethidium bromide was added at 0.5 µg/mL (final concentration) after cooling the melted agarose (Green and Sambrook, 2012). Samples (10 µL PCR product mixed with loading dye) were loaded into wells. A 100 bp DNA ladder was run alongside. Electrophoresis ran at 70 V for about 90 minutes. Bands were visualized under UV light at 312-365 nm using a transilluminator and documented with a gel imaging system. The presence of a ~153 bp band indicated a positive HLA-DQ2 result.

Statistical analysis: Data were analyzed using SPSS version 24 software. We first checked for normal distribution. Results are presented as mean ± standard deviation (or standard error), frequencies, and percentages. Differences between two groups were tested with the least significant difference (LSD) test. For more than two groups, we used one-way ANOVA. Chi-square test was applied for categorical data. A p-value < 0.05 was considered statistically significant.

Results

Distribution of samples by age categories: The study was carried out from November 1, 2022, to May 11, 2023. We included 20 patients diagnosed with celiac disease (10 males and 10

females) from Babylon Hospital and 15 healthy controls. The age range for both groups was 10 to 65 years. Table 3 shows the age distribution. Among the patients, most were in the 20-39 years group (14 patients), followed by 10-20 years (3 patients) and 40-65 years (3 patients).Group of control pose a similar age spread to allow excellent comparison.

Immunological tests (Anti-Gliadin antibodies): Positive results of anti-gliadin antibodies were showed via serological tests in many patients. Out of 20 patients, 14 (71.2%) were positive for IgG anti-gliadin antibodies, and 14 (71.2%) were positive for IgA anti-gliadin antibodies. All controls group (15) were negative for each IgA and IgG anti-gliadin antibodies (0%). Table 4 show the distribution of these antibodies in controls and patients.

Table 3. Age distribution of celiac disease patients and healthy controls

Age Group (years)	Patients (n=20)	Controls (n=15)	Total
10-19	3	2	5
20-39	14	10	24
40-65	3	3	6

Table 4. Distribution of anti-gliadin IgG and IgA antibodies in patients and controls

Group	IgG Positive (%)	IgG Negative (%)	IgA Positive (%)	IgA Negative (%)
Patients (n=20)	14 (71.2%)	6 (28.8%)	14 (71.2%)	6 (28.8%)
Controls (n=15)	0 (0%)	15 (100%)	0 (0%)	15 (100%)

Molecular identification of *HLA-DQ2*: Commercial purification kit was utilized for extraction of genomic DNA from all blood samples via following the manufacturer's protocol. DNA concentration and purity were measured via NanoDrop spectrophotometer at 260/280 nm. Concentrations were ranged from 58 to 142 ng/µL, and purity ratios were between 1.8 and 2.0. Figure 1 shows the agarose gel electrophoresis of DNA from patients, with clear, compact high-molecular-weight bands under UV light after running at 75 V for 30 minutes on 1% agarose gel. Similar results were seen for controls (Figure 2). Conventional PCR was used to detect the *HLA-DQ2* gene. The amplicon size was 153 bp, confirmed by comparison with a 100 bp DNA ladder. Gel electrophoresis used 1.5% agarose at 75 V for 45 minutes. Bands were visualized under UV light at 254-312 nm. Figure 3 displays the PCR results. *HLA-DQ2* was present in 15 out of 20 patients (75%). No controls showed the *HLA-DQ2* gene (0%).

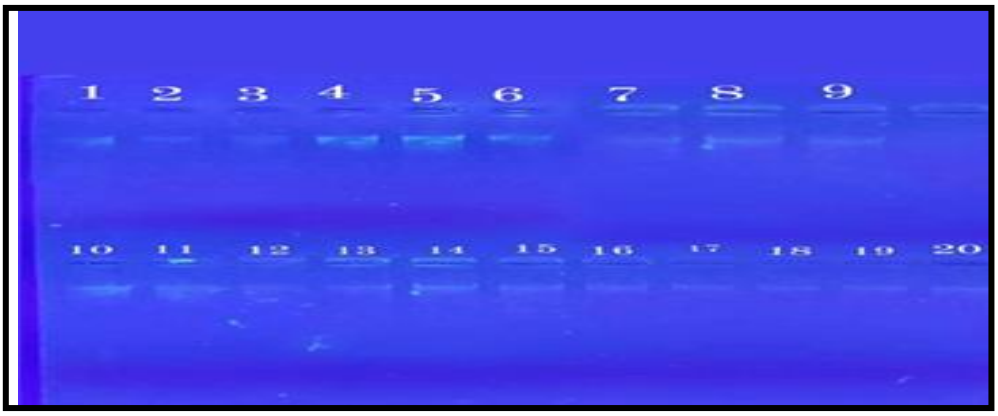

Figure 1. Genomic DNA bands extracted from human blood samples. Gel electrophoresis was performed on a 1% agarose gel at 75 V for 30 minutes, stained with ethidium bromide, and visualized under UV light. Lanes 1 to 20 represent DNA from 20 different samples (patient and/or control groups)

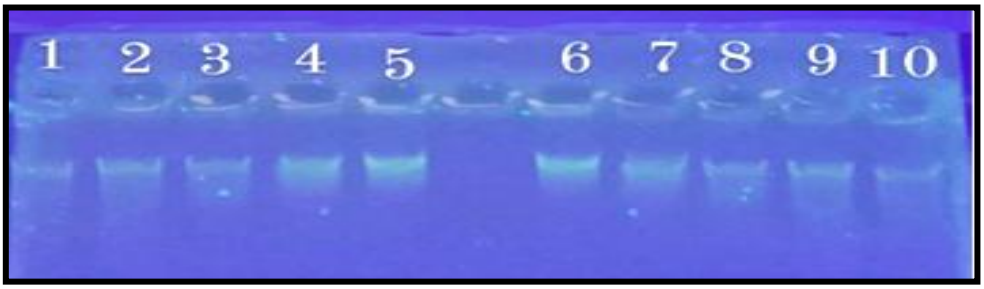

Figure 2. Genomic DNA bands extracted from control group blood samples. Gel electrophoresis was performed on a 1% agarose gel at 75 V for 30 minutes, stained with ethidium bromide, and visualized under UV light. Lanes 1 to 10 represent DNA from 10 different healthy control samples

Discussion

This study looked at genetic and immunological markers in celiac disease patients from Babylon Province. The results match many findings from other studies on celiac disease. Anti-gliadin antibodies (IgA and IgG) were positive in 71.2% of patients. This shows that these antibodies help identify the disease. However, anti-gliadin tests have lower specificity than tests for tissue transglutaminase (tTG) or endomysial antibodies. IgA anti-gliadin has better sensitivity and specificity than IgG in most cases. IgG is useful in patients with IgA deficiency, which occurs in 1-2% of celiac cases. Combining IgA and IgG improves detection rates. Anti-gliadin tests can give false positives, especially IgG, and are less useful for general screening now (Waheed et al., 2017; Hasan et al., 2012; Gualandris et al., 2021; Maglio & Troncone, 2020; Sallese et al., 2020).

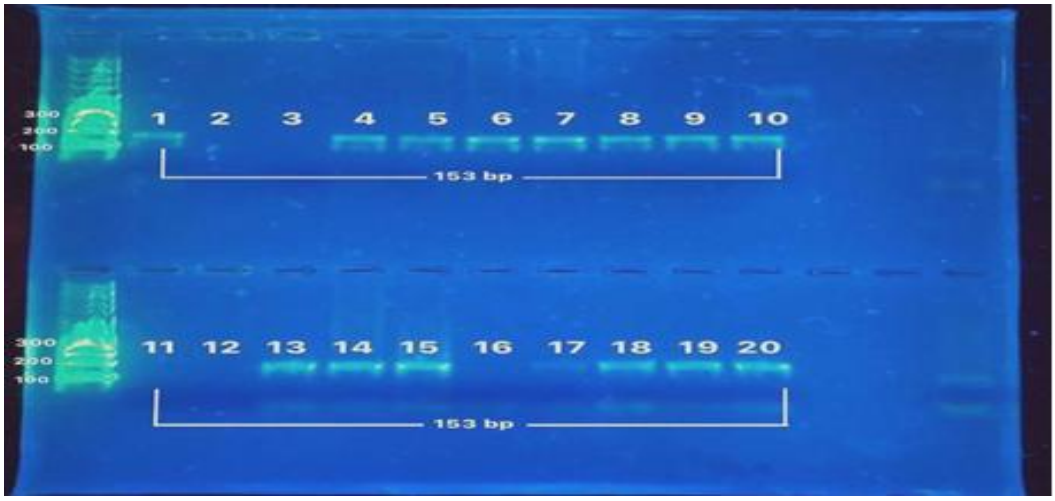

Figure 3. Agarose gel electrophoresis of conventional PCR products for detection of the HLA-DQ2 gene. The gel was prepared with 1.5% agarose, run at 75 V for 45 minutes, stained with ethidium bromide, and visualized under UV light

In our study, more females were positive for both IgG and IgA anti-gliadin antibodies. Out of the positive cases, about 55.6% were females and 44.4% were males. This points to a higher rate in females. Celiac disease often shows a female predominance in diagnosed cases, with ratios from 1.8:1 to 2.5:1. Frequently women may seek for medical help or they have various symptoms which facilitate of earlier diagnosis. *HLA-DQ2* was found in 75% of patients under study, while nobody of control group had it. This supports *HLA-DQ2* as a major genetic risk factor. *HLA-DQ2* and *HLA-DQ8* represent the main MHC class II alleles in association with celiac disease. *HLA-DQ2* homozygosity carries the highest risk. This increases the chance of early-onset disease in children via different times if compared with *HLA-DQ8* homozygosity or heterozygosity. Within the general population, *HLA-DQ2/DQ8* heterozygotes are more common (25-35%), but just about 3% develop the disease. *HLA-DQ2* facilitate better binding and gluten peptides presentation to T cells, causing stronger immune responses (Vader et al., 2003; Siddiqui et al., 2021; Megiorni & Pizzuti, 2012). Study finding of 75% *HLA-DQ2* positivity is agree with reports from other regions. Many previous studies confirmed that *HLA-DQ2* is present in (80-95%) of celiac patients, with *HLA-DQ8* in (5-10%). Some patients missing each of them, but they are rare. In populations of Middle Eastern, which include nearby regions, *HLA-DQ2* is most commonly among patients. The absence in some of our patients could be due to other HLA variants, assay limits, or technical issues, not diet. HLA genes are fixed at birth and do not change via gluten-free diet. These gives a higher risk because of better gluten peptide binding on antigen-presenting cells resulting in more enteropathy and inflammation. The exact reasons of the link are still under study (Murad et al., 2018; Pisapia et al., 2016; Gualandris et al., 2021). Current study has some limits in which sample size was small, utilization of anti-gliadin tests, that are less specific today and the study focused on *HLA-DQ2* only. Future studies must include *HLA-DQ8*, tTG tests and larger groups.Results

show high rates of anti-gliadin positivity and *HLADQ2* in celiac patients of Babylon. Also study findings add to knowledge about celiac disease in Iraq and support genetic screening within high-risk groups.

Conclusions: Current study confirmed that *HLA-DQ2* was exists in 75% of celiac disease patients of Babylon Province, while nobody of controls carried it. This confirms *HLA-DQ2* as main genetic risk factor in our population. Also, 71.2% of patients had positive anti-gliadin IgG and IgA antibodies, with a higher rate in females. Combining genetic testing (*HLA-DQ2*) with serological tests can support diagnosis, especially in settings with limited resources. The main limitation of this study is the small sample size (20 patients and 15 controls). Larger studies are needed in the future.

Author contributions

Conceptualization and Investigation: Haider Turki, Methodology and Writing-original draft preparation: Ihsan Ali and Ali Rasool Formal analysis and Writing-review and editing: Murtada M. All authors have read and agreed to the published version of the manuscript.

Data availability statement

The datasets generated during the current investigate are not publicly, but are available from the corresponding author upon reasonable request, subject to approval by the relevant institutional review board.

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Ethical considerations

The authors avoided data fabrication, falsification, plagiarism, and misconduct.

Conflict of interest

The authors declare no conflict of interest.

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ارتباط ژن HLA-DQ2 با بیماری سلیاک در استان بابل، عراق

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چکیده

هدف: بیماری سلیاک یک اختلال خودایمنی است که در افراد دارای استعداد ژنتیکی، در اثر مصرف گلوتن ایجاد می‌شود و منجر به پاسخ ایمنی و آسیب به روده باریک می‌گردد. مهم‌ترین عوامل خطر ژنتیکی این بیماری، ژن‌های HLA-DQ2 و HLA-DQ8 به همراه مواجهه با گلوتن هستند. هدف از این مطالعه، بررسی شاخص‌های ژنتیکی و ایمنی بیماری سلیاک در بیماران استان بابل بود. در این راستا، توزیع سنی بیماران با افراد سالم مقایسه شد، آنتی‌بادی‌های ضد گلیادین IgG و IgA با روش ELISA اندازه‌گیری گردید و وجود ژن HLA-DQ2 در بیماران و گروه شاهد با استفاده از PCR متداول بررسی شد.

مواد و روش‌ها: نمونه‌های خون از بیماران مبتلا به سلیاک و افراد سالم جمع‌آوری شد. از هر شرکت‌کننده، ۳ میلی‌لیتر خون وریدی در لوله‌های حاوی EDTA برای استخراج DNA و ۲ میلی‌لیتر خون در لوله‌های ژل‌دار برای آزمایش‌های سرولوژیک گرفته شد.

نمونه‌ها در جعبه سردکننده طی ۲ تا ۲۴ ساعت منتقل و تا زمان انجام آزمایش در دمای ۴ درجه سانتی‌گراد نگهداری شدند. آنتی‌بادی‌های ضد گلیادین IgG و IgA به روش ELISA شناسایی شدند. DNA ژنومی از خون استخراج و برای شناسایی ژن HLA-DQ2از روش PCR متداول استفاده گردید. محصولات PCR با الکتروفورز ژل آگارز تأیید و اندازه باندها با مارکر DNA مقایسه شد.

نتایج: نتایج PCR متداول وجود قطعه ژن HLA-DQ2 را در اندازه تقریبی ۱۵۳ جفت‌باز نشان داد. در بیماران مبتلا به سلیاک، ژنوتیپ هموزیگوت HLA-DQ2 شایع‌ترین حالت بود و در ۷۷/۲٪ بیماران (۱۴ نفر از کل افراد بررسی‌شده) مشاهده شد. سایر بیماران فاقد این ژن بودند که احتمالاً به دلیل پیروی از رژیم غذایی بدون گلوتن بوده است. در گروه شاهد، هیچ‌یک از افراد حامل ژن HLA-DQ2 نبودند (۰٪). مطالعات پیشین نشان داده‌اند که هموزیگوتی HLA-DQ2 بالاترین خطر ابتلا را دارد و احتمال بروز زودهنگام بیماری سلیاک در کودکان را ۵ تا ۳۰ برابر نسبت به ژنوتیپ‌های کم‌خطرتر مانند هموزیگوتی HLA-DQ8 یا هتروزیگوتی HLA-DQ2/DQ8 افزایش می‌دهد. اگرچه فراوانی هتروزیگوت‌های HLA-DQ2/DQ8 در جمعیت عمومی حدود ۲۵ تا ۳۵٪ است، تنها حدود ۳٪ از آن‌ها به بیماری مبتلا می‌شوند. بنابراین، هموزیگوتی HLA-DQ2 شایع‌ترین ژنوتیپ در بیماران مبتلا به سلیاک محسوب می‌شود.

نتیجه‌گیری: آزمایش‌های ژنتیکی مولکولی نقش مهمی در شناسایی افراد در معرض خطر ابتلا به بیماری سلیاک دارند. این مطالعه نشان داد که ژنوتیپ هموزیگوت HLA-DQ2 شایع‌ترین الگوی ژنتیکی در بیماران مبتلا به سلیاک در استان بابل است و ارتباط قوی این ژن با بیماری را تأیید می‌کند. این یافته‌ها استفاده از غربالگری ژنتیکی را در کنار آزمون‌های سرولوژیک برای تشخیص دقیق‌تر و مدیریت بهتر بیماری سلیاک توصیه می‌نماید.

کلمات کلیدی: استعداد ژنتیکی، ایمونوگلوبولین‌های IgG و IgA، بیماری سلیاک، ژن‌های HLA-DQ2 و HLA-DQ8، PCR

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