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Original Research Article

Detection of Genetic Variants in the CD24 and DLG5 Genes and Their Connection with Inflammatory Bowel Disease in an Iraqi Population.

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Abstract: Inflammatory bowel disease (IBD) encompasses various hereditary variables, including CD24 and DLG5 polymorphisms. This study aims at assessing the impact of CD24 and DLG5 gene variants on IBD risk in Iraqi patients. Our sequencing revealed mutations in the CD24 and DLG5 genes. variant at NC_000006.12: g.106971888C>T (rs3805401). For DLG5, a polymorphism at NC_000010.11:g.77856847 T>A/C/G was corresponded to the SNP rs1248696. Allele frequency of CD24 rs38305401 did not show considerable significance between Crohn's disease and ulcerative colitis TC genotype: OR = 1.09, 95% CI 0.34–3.54; CC genotype: OR = 2.50, 95% CI 0.55–11.45. The results of this study indicate that genetic variations in genes regulating cell adhesion and signaling may contribute to modulating the clinical manifestations of inflammatory bowel disease. Integrating traditional genetic association research with genome sequencing technology is critical for identifying and defining the complete range of disease-associated genetic variations.

Keywords: Inflammatory bowel disease, Crohn's disease, ulcerative colitis, CD24, DLG5.

INTRODUCTION

Inflammatory bowel disease (IBD) is classified into two types: Crohn's disease (CD) and ulcerative colitis (UC). which are basically chronic inflammatory disorders of the gastrointestinal system. These illnesses are consistent and characterized by alternating periods of exacerbation and remission [1]. Inflicting any section of the digestive tract [2]. This disease's influence has mostly extended to the areas of the colon and terminal ileum [3]. Despite sophisticated investigations, the cause of CD still remains not specified. various population studies have indicated that CD is a complex disease influenced by both environmental and genetic factors [4]. Studies suggest that genetic factors play an important part in the development of CD [5]. The original genome-wide association studies (GWAS) have come up with their findings in 2005, using genome-wide single nucleotide polymorphism (SNP) chips [6,7]. Many common genetic variants are the driving forces constitute the majority of complex (i.e., non-single-gene Mendelian) hereditary illnesses, which serve as the conceptual ground for GWAS [8]. Genome-wide association studies have identified some 320 loci related to a higher risk of inflammatory bowel disease (IBD) [9, 10, 11]. Inflammatory bowel disease is tied up to an increasing number of genetic variables. The current number of loci linked to IBD is thought of as representing only a small fraction of the genetic risk [12]. Extensive research was conducted when a link was discerned between DLG5 and IBD. Numerous lines of inquiry have pointed to DLG5's role in growth, cancer, and sex-related differences in addition to IBD [13]. DLG5 is a gene that encodes a protein. It endorses signal transduction complexes and assembles receptors and transducers as

a membrane-associated guanylate kinase MAGUK family member. This protein helps maintain epithelial cell polarity and adhesion in many organs and tissues [14]. It has been pointed out that mutations in DLG5 contribute to human disease in two ways. It is downregulated in cancers of the lung, liver, breast, prostate, and bladder but overexpressed in pancreatic adenocarcinoma [13]. Population-based works have discovered an increase in IBD cases in recently industrialized nations in South America, Asia, and Africa [1]. In the Iraqi community, most patients were coming descending from urban areas or the city center of Mosul. There is an increasingly long list of genetic factors associated with IBD [15]. Accordingly, there exists an need urgent to find out additional genetic contributions. Numerous cell types show the human CD24 molecule gene inherent in chromosome 6, which codes for a tiny, highly glycosylated protein of 20–70 amino acids that is connected to the cell membrane by a glyco-phosphotidyl-inistol (GPI) anchor [16] CD24 plays a physiological role in the maturation of T and B cells in the activation of CD4 and CD8 T cells by providing a CD28-independent co-stimulatory signal [17, 18], neuronal development [19], alongside tissue renewal homeostasis under physiologic conditions [20]. Accumulating evidence implicates genetic variants in the CD24 gene as playing a role in the pathogenesis of gastric cancer [21], inflammatory bowel disease IBD [22, 23], and multiple autoimmune disorders [24].

The aims of the study

This study aims at delineating the allelic and genotype frequencies of the SNPs rs3805401 and rs1248696 in the CD24 and DLG5 genes, respectively, and evaluating their correlation with the risk of IBD in a sample from the Iraqi population.

METHODOLOGY

Sampling

This case-control study was conducted on 90 participants: 30 of whom are diagnosed with Crohn's disease and other 30 with ulcerative colitis, and still other are 30 healthy participants as a control group. The sampling period, which spanned from November 2023 until May 2024, informed consent from each patient involved in the study. Blood samples were collected from Nasiriyah General Hospital and Shatra Hospital in Dhi Qar Province. Neither cases that failed to meet the study criteria nor those which were not disease-associated are included in this work. This study protocol is consistent with the ethical and moral values proposed in 1975 Declaration of Helsinki. The study's implementation in the health department institutions was approved by the research ethics committees of the Thi-Qar Health Department (Document 119/2025, dated 2025/3/23).

Genomic DNA Extraction, Sequencing

The gSYNC™ Genomic DNA Kit is applied to DNA extraction/genomic DNA purification [Geneaid, Taiwan], following the manufacturer's guidelines, which is followed by purity and concentration assessment. The extracted DNA was then stored at 25°C until analysis for genotyping. Electrophoresis was used in conformity with the standard method of using agarose gel to detect DNA [25].

Genomic DNA Extraction, PCR and Sequencing

The study involved the PCR amplification and sequencing of both CD24 and DLG5. The first fragment, 373 bp, contains the CD24 gene, while the second fragment, 522 bp, has the DLG5 gene, using the primers designed from the National Center for Biotechnology Information (NCBI) GenBank website using Primer3Plus. Bioneer prepares the primers F-5-ATGCAGAAGAGAGAGTGAGAC-3 and R-5-ATTTTTGAGGCATCTCTAGG-3 for the CD24 gene, as well as F-5-GACATGAGGTGGGGGAAAGG-3 and R-5-TGAGTGCTTGGATGCTGGAG-3 for the DLG5 gene, as a dried product. The PCR reaction was performed in a volume of 20 µl, comprising 1 µl each of forward (F) and reverse [R] at a concentration of 10 pmol/µL, as well as 1 µl of each primer. PCR products were then separated on 2% agarose gel electrophoresis at 5 v/cm² for 1 hour to confirm amplification in the existence of a DNA ladder marker (1 kb) (Promega, USA) with 1 × Tris-borate-EDTA buffer and visualized with UV light (302 nm) subsequent to staining by ethidium bromide [26]. The gel presented distinct bands of the 373-bp product for CD24 and the 522-bp product for DLG5. The two products of PCR were purified and sequenced using the BigDye terminator (V.3.1) and the right primers. Macrogen Inc. (South Korea) supplied DNA sequencing services.

Variants Identification

The CD24 gene sequence results were aligned to the reference sequence NC_000006.12 and the DLG5 gene sequence results were aligned to the reference sequence NC 000010.11 using the NCBI-BLAST website.

Statistical Analysis

Statistical analysis, using the Chi-square test at a significance level of $P<0.05$, was conducted for all of the samples under scruting. The odds ratio (OR) test was also employed to study the genotype frequency of two genes using the SPSS program. The DNAsp program was resorted to so as to analyze the genetic diversity of the- genes under discussion [27].

RESULTS

IBDs can be defined as complex hereditary diseases involving multiple genes. Linkage studies show that some genomic areas linked to IBD susceptibility genes are unique to CD or UC, while others are shared by both. Agarose gel electrophoresis confirmed the purity and integrity of the gDNA before proceeding with downstream analyses. The PCR products were confirmed by gel electrophoresis, and final PCR bands were run out on 2% agarose gel, and bands were identified with the expected amplicon size. Amplified clear single band at approximately 373 bp, depicting the CD24 region in (Figure 1). At 522 bp, there is a distinct single band that represents the DLG5-targeted region (Figure 2).

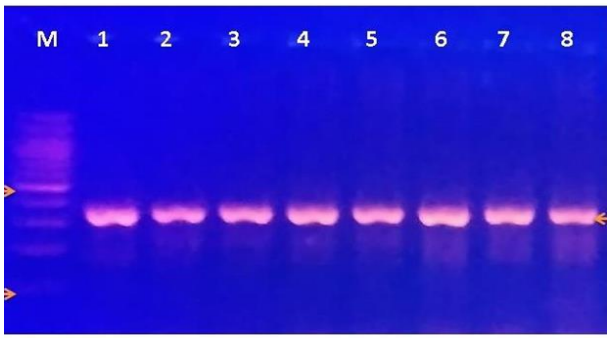

Figure 1: Amplification of CD24 gene fragment, fractionated samples on 2% agarose gel electrophoresis stained with Ethidium Bromide M: 1Kb ladder marker, Lanes [1-8] PCR products of IBD patients with samples band size 373bp.

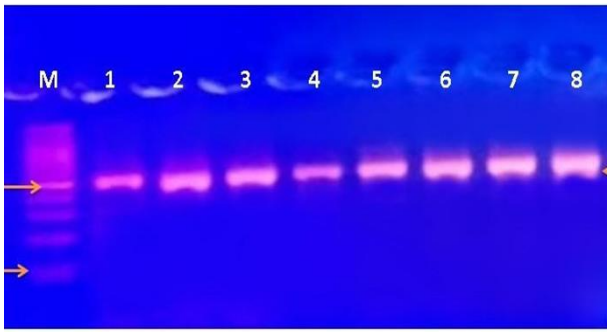

Figure 2: Amplification of DLG5 gene fragment, fractionated samples on 2% agarose gel electrophoresis stained with Ethidium Bromide M: 1Kb ladder marker, Lanes [1-8] PCR products of IBD patients with samples band size 522bp.

Using the CD24 reference gene sequence (NC_000006.12), sequence alignment analysis specified a genetic variant in a group of 60 IBD patients.This variation was found to be (T>C) substitution in the CD24 gene at genomic location g.106971888. Under the identifier rs3805405, this variant is associated with a single nucleotide polymorphism (SNP) that is listed in the global database of gene variants (dbSNPs).

Utilizing the DLG5 gene's reference sequence (NC_000010.11), sequence alignment analysis revealed the presence of a genomic variant in the patient cohort under study. This variant was found to be a (T>C) substitution at the genomic location NC_000010.11:g.77856847 in the DLG5 gene. This variant corresponds to an SNP identified as rs1248696 T>A,C,G in the worldwide gene variant database dbSNP. The researchers employed the Hardy-Weinberg equilibrium (HWE) equation to find the allelic frequency.

Genotype Distribution and Association Analysis

It has been pointed out that the CD24 gene's genotype analysis revealed one single nucleotide polymorphism, rs3805401 (T>C). What is more, the wild-type TT genotype is identified as not associated with the increased risk of CD odds ratio (OR = 1.0). Its percentage was distributed in the CD group (33.33%) and the control group (26.66%). Furthermore, comparative analysis of the distribution of the TC genotype did not reveal a statistically significant association, as it was distributed in the Crohn's disease group (53.33%) and the control group (46.66%) (OR = 1.09, 95% CI = 0.34–3.54), Similarly, the CC genotype did not reveal a statistically significant association since it was less common in Crohn's patients (13.33%) than in controls (26.66%) with an odds ratio (OR) of 2.50 and a 95% confidence interval (CI = 0.55–11.45).

Table 1 below presents the distribution of CD24 rs3805401 genotypes among patients with CD and controls.

Table (1) Genotypes and allele frequencies of the rs3805401 for the CD patients and control groups.

Genotypes	CD group N=30)%(	control group N=30)%(	OR	CI 95%
TT	%33.33)(10	8 (26.66%)	1.0	
TC	16 (53.33%)	14 (46.66%)	1.09	0.34 - 3.54
CC	4 (13.33%)	8 (26.66%)	2.50	0.55-11.45
Allele Frequency				
T	0.60	0.50	1.0	
C	0.40	0.50	1.50	0.72-3.09

It has been observed that CD24 genetic polymorphism showed no association with the risk of UC. The TT genotype were dissociated with UC, with an odds ratio of (1.0), The genotype percentage did not exhibit a significant difference between patients and controls (33.33% vs 26.66%). Similarly, the TC and CC genotypes showed OR = 1.60 (95% CI = 0.48-5.29) and OR = 1.57 (95% CI = 0.40-6.14), respectively. In both cases, the confidence intervals were very wide, indicating that there were no statistically significant associations. Table 2, on the other hand, presents the genotype distribution of the CD24 gene and its association with the risk of UC. as shown in (Table 2).

Table (2) Genotypes and allele frequencies of rs3805401 for the UC patients and control groups.

Genotypes	UC group N=30 (%)	Control group N=30(%)	OR	CI 95%
TT	11 (36.67%)	8 (26.66%)	1.0	
TC	12 (40.00%)	14 (46.66%)	1.60	0.48 - 5.29
CC	7 (23.33%)	8 (26.66%)	1.57	0.40-6.14
Allele Frequency				
T	0.60	0.50	1.0	
C	0.40	0.50	1.30	0.63 -2.68

In the UC and CD groups, as shown in Tables 1 and 2, the allele frequencies are as follows: major T allele = 0.60 and minor C allele = 0.40; in the control group, TT = 0.50 and C = 0.50. However, the frequency noted in the present study population did not have any statistical significance.

To examine the significance of DLG5 variants in IBD susceptibility, analysis of the rs1248696 genotype in the DLG5 gene revealed the absence of the TT genotype of the DLG5 gene in CD cases and the control, and the TC genotype were observed in (16.67%) of patients and were not found in the control, with an OR = 0.09 and a (95% CI = 0.007 - 11.88). However, due to a wide confidence interval, this association is not statistically significant. The CC genotypes were the most common one in the CD group (83.33%) and the control group (100%), with an OR of 1.19 and (95% CI of 0.02-62.43); but there were no statistically significant correlation. Table 3 shows the distribution of the DLG5 rs1248696 genotype among CD patients and the control group.

Table (3) Genotypes and allele frequencies of the rs1248696 for the CD patient groups and the control group

Genotypes	CD group N=30(%)	control group N=30(%)	OR	CI 95%
TT	0 (0%)	0 (0%)	1.0	
TC	5 (16.67%)	0 (0.0%)	0.09	0.007 - 11.88
CC	25 (83.33%)	30 (100.00%)	1.19	0.02-62.43
Allele Frequency				
T	0.083	0.00	1.0	
C	0.917	0.60	11.99	0.64 -220

Comparative analysis revealed that the TC genotype were exclusively present among UC patients (10.00%) compared to the control group, which resulted in an OR of 0.16 (95% CI of 0.001–19.45). The CC genotype showed high prevalence in the ulcerative colitis patient group (90.00%) and the control group (100%), odds ratio (OR = 1.11), and (95% CI of 0.02-57.81). These findings were no significant association. The TT genotypes were very rare in the examined groups (0.0%). The distribution of genotypes for UC patients and healthy controls were presented in Table 4.

Table 4: Genetic patterns and allele frequencies of the rs1248696 for the two groups of UC patients and the control group.

Genotypes	UC group N=30(%)	control group N=30(%)	OR	CI 95%
TT	0 (0%)	0 (0%)	1.0	
TC	3(10.00%)	0 (0.0%)	0.16	0.001-19.45
CC	27(90.00%)	30 (100.00%)	1.11	0.02-57.81
Allele Frequency				
T	0.05	0.00	1.0	
C	0.95	0.60	7.37	0.37 -145

Analysis of allelic frequency for the rs1248696 variant in the DLG5 gene demonstrated no statistically significant difference between patients with IBD (CD and UC) and control groups. As seen in Tables 3 and 4, the T allele was infrequent to completely absent among controls, its presence were detected among both patient groups at frequencies of 0.05 in CD and 0.083 in UC. Whereas the C allele was found to be present more frequently in cases, 0.917 in CD patients (OR = 11.99, 95% CI 0.64-220.00) and 0.95 in UC patients (OR = 7.37, 95% CI 0.37-145.00) than in controls in both groups, where its frequency reached 0.6.

DISCUSSION

Inflammatory bowel diseases IBD are a group of complicated hereditary illnesses that involve more than one gene. Some of the genomic areas linked in IBD susceptibility genes have been found only in CD or UC, whereas others are shared between the two diseases, according to linkage studies [28]. To establish the clinical significance and genetic predisposition to IBD, the CD24 gene in our sequencing revealed the presence of SNP rs3805405 T>C. The rs3805401 SNP has been associated with susceptibility to and progression of multiple sclerosis [29]. Conversely, no positive association has been observed between the MS subtype and the CD24v/v genotype, nor has it been demonstrated to reflect susceptibility to disease progression. [30]. In this

study, sequencing alignment data did show that a single nucleotide variant (SNV), rs1248696 T>C, was present among the local population. Although the results are still debatable, there is compelling evidence from recent studies linking polymorphisms of DLG5 (rs1248696) to an increased risk of IBD [31]. By contrast, the variant (rs1248696) in the DLG5 gene has been related to an increased risk of developing IBD, as this variant has a dominant genetic pattern in the disease pathways [32]. The results of allele frequency analysis of the CD24 gene in this study indicated that the TC genotype was predominant, while the CC genotype was less common in the CD and UC patients and healthy controls. The T allele frequency was high (0.6), while the C allele was less prevalent (0.4). The results of the allelic frequency analysis for the genetic variant under study revealed a clear pattern indicating that the C allele is the dominant allele across diverse global populations, as seen in the GnomAD and TOPMED databases, with a frequency of approximately 0.57. Analysis of the rs1248696 variant in the DLG5 gene reveals several important findings with potential clinical implications. In this study, we found two genotypes in the allelic frequencies of the DLG5 gene, similarly distributed in the CD and UC patient groups. This marks bias towards an association between the CC/TC genotypes and disease incidence but is not statistically confirmed with respect to the complete absence of the T genotype in the study population, which may be attributed to the small sample size. Indeed, allele frequencies in the DLG5 gene are more common among IBD patients [33]. Genetic frequencies for the rs1248696 variant did not significantly differ between groups ($P > 0.80$), and the meta-analysis using the Dutch data did not show any association [34]. The analysis showed no statistically considerable association between the rs1248696 genetic variation in the DLG5 gene and an increased risk of CD in the study sample ($P = 0.27$, $OR = 1.14$). Conversely, a non-synonymous single nucleotide polymorphism (SNP), R30Q rs1248696, has been associated with an increased risk of IBD, and a common genotype (called the "A" genotype) has been associated with a decreased risk [35]. All in all, the present study methodology and participant count (30 per group) yielded valuable data for the first analysis and care is warranted in generalizing the findings. This study underscores the necessity for more comprehensive future research, involving bigger participant cohorts to validate and endorse the findings.

CONCLUSION

This study shows that there is no statistically significant correlation between the examined genetic polymorphisms in the CD24 and DLG5 genes and IBD in the Iraqi cohort. Scientific concepts indicate that changes in genes governing cell adhesion and communication may modulate the clinical symptoms of the disease in some populations. Our findings enhance comprehension of the intricate and multifaceted character of IBD, which may obscure the influence of an individual genetic variant in limited research.

Study Limitations

The most significant limitation of this study is its relatively small cohort size (30 patients per group).

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