Egyptian Journal of Medical Microbiology Volume 34 / No.4 / October 2025 51-59 Online ISSN: 2537-0979

ORIGINAL ARTICLE

Correlation Between Adhesion Molecules (ICAM-1 & E-Selectin) and Diagnostic Antibodies (TTG & AGA) in Celiac Disease

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Key words:
Celiac disease, Endothelial adhesion molecules, ICAM-1, E-selectin, Autoantibodies, Inflammation

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ABSTRACT

Background: *Celiac disease (CD) is a chronic autoimmune disorder triggered by gluten ingestion in genetically susceptible individuals, characterized by intestinal inflammation, villous atrophy, and the production of specific autoantibodies, including anti-tissue transglutaminase (TTG) and anti-deamidated gliadin peptides (AGA). Endothelial adhesion molecules, such as intercellular adhesion molecule-1 (ICAM-1) and E-selectin, play a pivotal role in immune cell recruitment and inflammation.* ***Objective:*** *This study aimed to evaluate the correlation between the gene expression of ICAM-1 and E-selectin and serum levels of diagnostic antibodies (TTG-IgA, TTG-IgG, AGA-IgA, and AGA-IgG) in CD patients.* ***Methodology:*** *A case-control study was conducted involving 124 CD patients and 60 healthy controls. Gene expression of ICAM-1 and E-selectin was quantified using reverse transcription-polymerase chain reaction (RT-PCR), while serum antibody levels were measured via enzyme-linked immunosorbent assay (ELISA).* ***Results:*** *A significant positive correlation was observed between TTG-IgA and TTG-IgG levels ($r = 0.85$, $P < 0.001$), whereas AGA-IgA and AGA-IgG exhibited a weak correlation ($r = 0.12$, $P=0.187$). ICAM-1 and E-selectin expression was significantly downregulated in CD patients compared to controls ($P < 0.001$), with a strong positive correlation between the two molecules ($r=0.86$, $P<0.001$). ICAM-1 expression demonstrated a moderate positive correlation with antibody levels ($P<0.05$), suggesting its involvement in immune activation and inflammation in CD. In contrast, E-selectin expression did not significantly correlate with antibody levels, indicating a more complex or indirect role in CD pathogenesis.* ***Conclusion:*** *These findings highlight the potential of ICAM-1 as a biomarker for immune activation in CD and suggest that endothelial adhesion molecules may contribute to disease progression. Further research is warranted to elucidate their precise mechanisms in CD pathogenesis.*

INTRODUCTION

Celiac disease (CD) is a chronic autoimmune disorder triggered by gluten ingestion in genetically predisposed individuals, characterized by intestinal inflammation, villous atrophy, and the production of specific autoantibodies such as anti-tissue transglutaminase (TTG) and anti-deamidated gliadin peptides (AGA) [1;2] . While serological testing for TTG-IgA, TTG-IgG, AGA-IgA, and AGA-IgG antibodies via enzyme-linked immunosorbent assay (ELISA) remains the cornerstone of CD diagnosis, emerging evidence highlights the role of endothelial activation and adhesion molecules in disease pathogenesis [3]Among these, intercellular adhesion molecule-1 (ICAM-1) and E-selectin, which mediate leukocyte migration to inflamed tissues, are proposed as key mediators of intestinal mucosal damage in CD [4; 5]

Recent studies suggest a potential interplay between systemic inflammation and autoantibody production in CD. Elevated levels of ICAM-1 and E-selectin have been observed in autoimmune conditions, reflecting endothelial dysfunction and enhanced leukocyte recruitment [6;7]However, the relationship between these adhesion molecules and CD-specific antibodies (TTG/AGA) remains underexplored. Reverse transcription-polymerase chain reaction (RT-PCR) enables precise quantification of ICAM-1 and E-selectin mRNA expression in peripheral blood, offering insights into their transcriptional regulation during active disease [8] Concurrently, ELISA-based measurement of TTG and AGA antibodies (IgA/IgG subclasses) provides a reliable assessment of humoral immune responses [9].

This study aims to investigate the correlation between the gene expression of endothelial adhesion molecules (ICAM-1 and E-selectin), measured via RT-PCR, and the serum levels of diagnostic antibodies (TTG-IgA, TTG-IgG, AGA-IgA, and AGA-IgG), quantified by ELISA, in CD patients. By integrating molecular and serological approaches, this work seeks to unravel potential links between endothelial activation, systemic inflammation, and autoimmune responses in CD, which may inform novel biomarkers or therapeutic targets.

METHODOLOGY

Study Design and Population:

This is a case-control study that included 124 patients clinically diagnosed with celiac disease (CD) and a control group of 60 healthy individuals matched by age and gender. The patient cohort consisted of 83 females (66.9%) and 41 males (33.1%), with a mean age of approximately 18.7 years. Blood samples were collected from patients at Al-Najaf Educational Hospital, Al-Sadr Medical City, and the Gastroenterology and Hepatology Specialized Center, as well as central laboratories in Najaf province, Iraq, between August 1, 2024, and November 1, 2024.

Ethical Consideration:

The study was approved by the relevant institutional review boards. Informed consent was obtained from all participants or their legal guardians before sample collection. All study procedures adhered to the ethical guidelines outlined in the Declaration of Helsinki.

Blood Sampling and Serum Preparation:

Under aseptic conditions, 5 mL of venous blood was collected from each participant. Blood samples were collected in EDTA tubes for RNA extraction and in serum separator tubes for antibody analysis. Serum was separated by centrifugation at 3000 rpm for 10 minutes at 4°C and stored at -80°C until further analysis.

Measurement of Anti-TTG and Anti-AGA Antibody Levels

Principle:

An enzyme-linked immunosorbent assay (ELISA) was used to quantify serum levels of anti-tissue transglutaminase (TTG) and anti-deamidated gliadin peptides (AGA) antibodies, including both IgA and IgG subclasses. This method is based on antigen-antibody interactions and is recognized for its high sensitivity and specificity.

Procedure:

Serum antibody levels were measured using commercial ELISA kits in accordance with the manufacturer's instructions.

- AGA-IgA and AGA-IgG were determined using the Scanlisa Anti-Gliadin-IgA Antibody and Anti-Gliadin-IgG Antibody ELISA kits (Scimedx Corporation, Denville, NJ).
- TTG-IgA and TTG-IgG levels were measured using the BINDAZYME human IgA and IgG Anti-Tissue Transglutaminase EIA Kit (The Binding Site, Ltd, Birmingham, UK).

All assays were validated using appropriate quality control criteria. Antibody levels were expressed as mean ± standard deviation (SD), and statistical correlations were analyzed.

Quantitative Real-Time PCR (qPCR) for ICAM-1 and E-Selectin Gene Expression

RNA Extraction:

Total RNA was extracted from peripheral blood mononuclear cells (PBMCs) using TRIzol reagent (TransGen Biotech, China) following the manufacturer's instructions. RNA purity and concentration were assessed using a NanoDrop spectrophotometer, with an acceptable A260/A280 ratio of 1.8-2.0.

cDNA Synthesis: Complementary DNA (cDNA) was synthesized from 1 µg of total RNA using a reverse transcription kit (Solarbio, China) with random hexamers. The reaction conditions were as follows:

- 70°C for 5 min
- 42°C for 60 min
- 25°C for 10 min
- 95°C for 5 min

Quantitative Real-Time PCR (qPCR):

Primer Sequences: Primers for ICAM-1, E-selectin, and the housekeeping gene (GAPDH) were designed using Primer-BLAST (NCBI):

Gene	Forward Primer	Reverse Primer
ICAM-1	5'-CTTCCTCACCGTGTACTGGAC-3'	5'-GGCAGCGTAGGGTAAGGTTC-3'
E-selectin	5'-GATGGACGCTCAATGGCTCT-3'	5'-TGGACTCAGTGGGAGCTTCA-3'
GAPDH	5'-GGAGCGAGATCCCTCCAAAAT-3'	5'-GGCTGTTGTCATACTTCTCATGG-3'

Reaction Setup: qPCR was performed using SYBR Green Master Mix (Solarbio, China) in a 20 µL reaction volume. The thermal cycling conditions were as follows:

- 95°C for 10 minutes
- 40 cycles of 95°C for 15 seconds and 60°C for 1 minute

Data Analysis:

Gene expression levels were normalized to GAPDH and calculated using the 2^(-ΔΔCt) method. Fold changes in gene expression were determined by comparing patient samples to healthy controls.

Statistical Analysis:

Pearson's correlation coefficient (r) was used to assess relationships between ICAM-1/E-selectin expression and antibody levels. Data were presented as mean ± SD, and correlation plots were generated using GraphPad Prism (version 10.4.1). A p-value < 0.05 was considered statistically significant.

RESULTS

Sample Collection and Patient Demographics

A total of 124 blood samples were collected from patients clinically diagnosed with celiac disease (CD). The cohort consisted of 83 females (66.9%) and 41 males (33.1%). The mean age of the patients in this study is approximately 18.7 years Samples were obtained from Al-Najaf Educational Hospital, Al-Sadr Medical City, and the Gastroenterology and Hepatology Specialized Center, as well as central laboratories in Najaf province during the period from August 1, 2024, to November 1, 2024.

1. Serological Analysis of Autoantibodies

The detection of specific autoantibodies in CD patients is a crucial diagnostic and monitoring tool. The analysis of serum samples revealed the following mean antibody levels (± SD) as in table (1). A statistical analysis indicated a significant difference in AGA-IgA levels **(p = 0.0259**), suggesting its potential relevance in CD diagnosis.

Table 1: The mean of autoantibodies level in the serum of CD patients

Antibodies	Mean ± SD	P-Value (p < 0.05)
AGA-lgA	10.78 ±21.83	F = 3.12
AGA-lgG	10.37±30.13	p = 0.0259
TTG-lgA	23.36±63.11	
TTG-lgG	15.64± 36.73	

The correlation coefficient analysis revealed significant associations among certain antibodies (table 2):

- A strong positive correlation was observed between **TTG-IgA and TTG-IgG (r = 0.85)**, indicating their concurrent elevation in CD patients.
- AGA-IgA and AGA-IgG exhibited a weak correlation (**r=0.12**), suggesting that their diagnostic utility may differ in individual cases.
- TTG-IgA showed negligible correlation with AGA antibodies, reinforcing findings that tissue transglutaminase antibodies are more specific to CD pathogenesis.

Table 2: The correlation coefficient between auto-antibodies in CD patients

Parameter	AGA-IgA	AGA-IgG	TTG-IgA	TTG-IgG
AGA-IgA	1.00			
AGA-IgG	0.12	1.00		
TTG-IgA	-0.07	-0.05	1.00	
TTG-IgG	0.03	0.10	0.85	1.00

2-The gene expression of Adhesion molecules in celiac disease patients

The results in table (3) presents the mean gene expression levels of two adhesion molecules, E-selectin and ICAM-1, in both celiac disease (CD) patients and control groups. The data is expressed as folding changes relative to the control group, which is set to a baseline value of 1. The correlation coefficient between the two molecules was 0.86, suggesting a strong positive relationship in their expression patterns in CD patients.

For E-selectin, the mean gene expression in CD patients was 0.748±1.21, indicating a reduction in expression compared to the control group. Similarly, ICAM-1 indicated a downregulation of gene expression compared to the control group, with a mean fold change of 0.511 ± 0.86. This suggests a reduced expression of this intercellular adhesion molecule in CD patients.

The melting curves for GAPDH, E-selectin, and ICAM-1 genes, as depicted in Figure 1, provide further insight into the specificity and integrity of the PCR amplification. The distinct and sharp peaks in the melting curves for each gene indicate high specificity of the primers used and the absence of non-specific products or primer-dimers in contrast to GAPDH. This supports the reliability of the gene expression data presented in figure (1).

Table 3: The mean of gene expression of E-selectin and ICAM-1 in control and CDpatients

Parameters	Folding change: Mean ± SD	
	Patients	Control
E-selectin	0.748±1.21	1
ICAM-1	0.511±0.86	1
correlation coefficient	0.86	

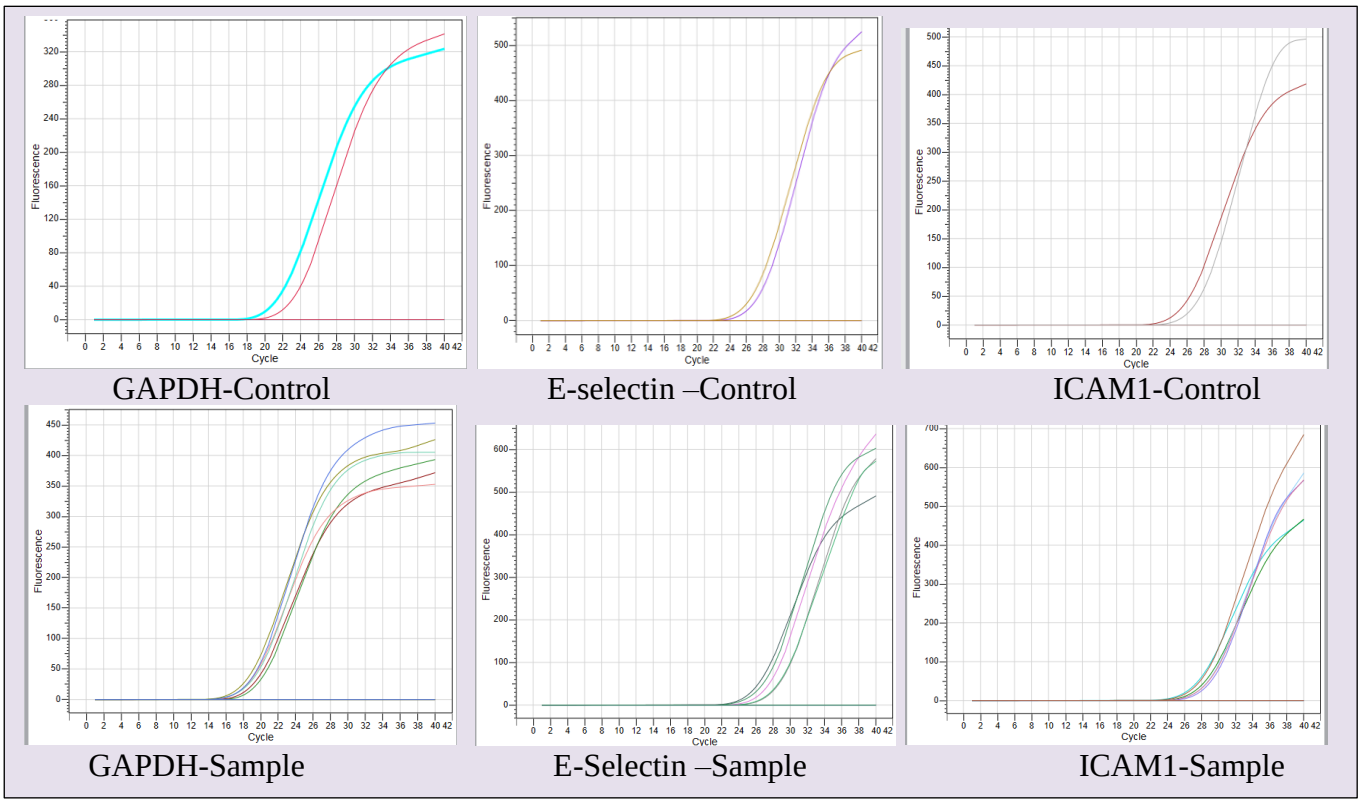

Fig. 1: The melting curves of GAPDH, E-selectin and ICAM-1 genes in control and CD patients

3. Correlation between Adhesion molecules expression levels and antibody

This study examined the correlation between ICAM-1 and E-selectin expression levels and antibody (GTT & AGA) levels in 124 patients, including 83 females and 41 males. The statistical analysis was conducted using correlation coefficients to assess the strength and significance of the relationship between these biomarkers.

3.1. Correlation Between ICAM-1 Expression and Antibody Levels

Figure (2) illustrates the correlation between ICAM-1 expression fold change and GTT & AGA antibody levels in CD patients.Pearson's correlation coefficient (r) indicates a moderate positive correlation, suggesting that as ICAM-1expression increases, antibody levels also tend to rise. The p-value is statistically significant ($p < 0.05$), confirming that the observed correlation is unlikely due to chance. This finding suggests that ICAM-1 plays a role in immune activation and inflammation, potentially contributing to disease progression in CD patients.

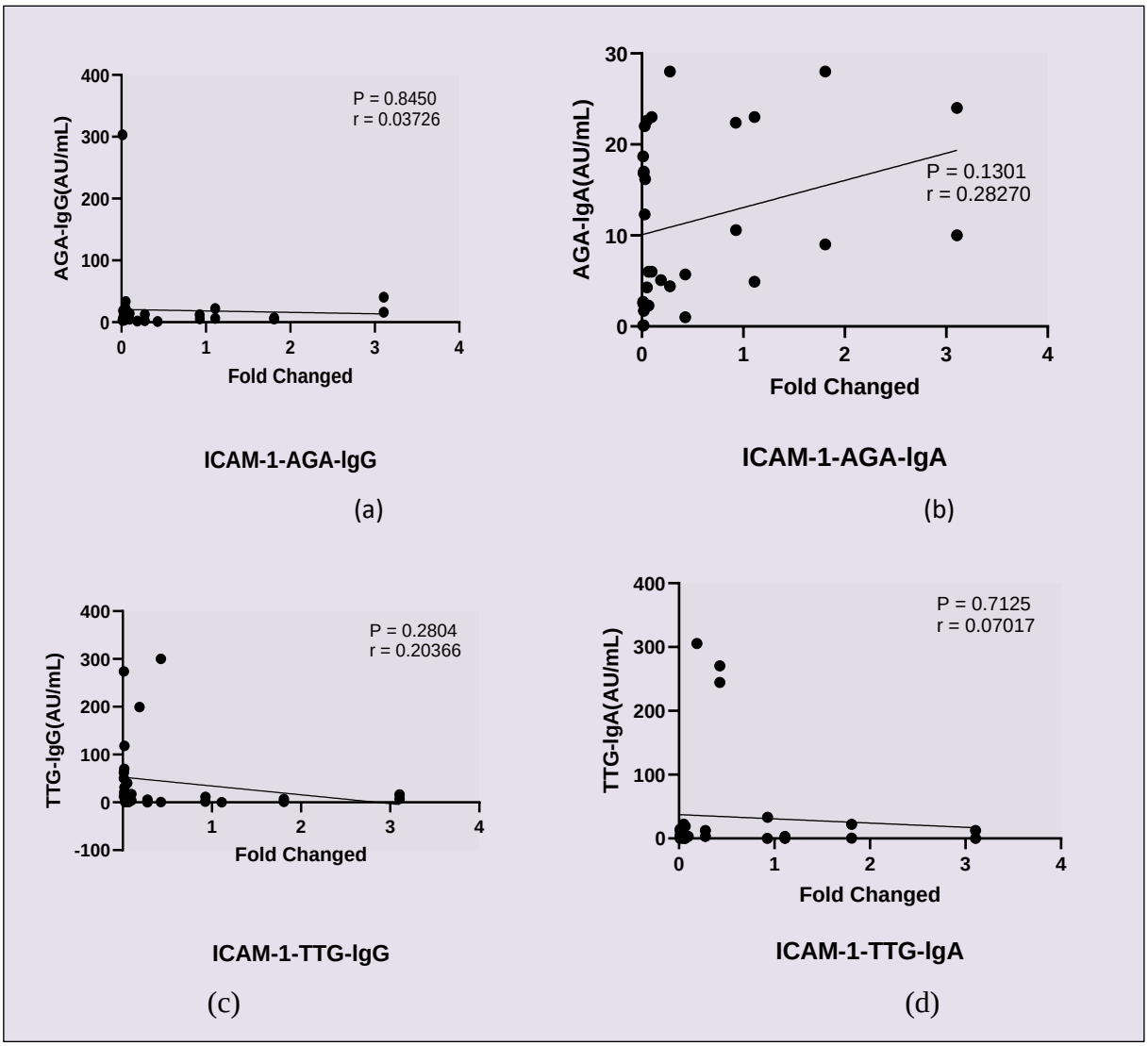

Fig. 2: The correlation of ICAM-1 folding change with the levels Antibodies (GTT & AGA)

3.2. Correlation Between E-selectin Expression and Antibody Levels

Figure (3) presents the correlation between E-selectin fold change and GTT & AGA antibody levels in CD patients. The correlation coefficient (r) suggests a weaker relationship compared to ICAM-1, indicating that E-selectin expression changes do not strongly correlate with antibody levels. The p-value is not statistically significant ($p > 0.05$), implying that E-selectin variations may not directly influence antibody production in these patients.

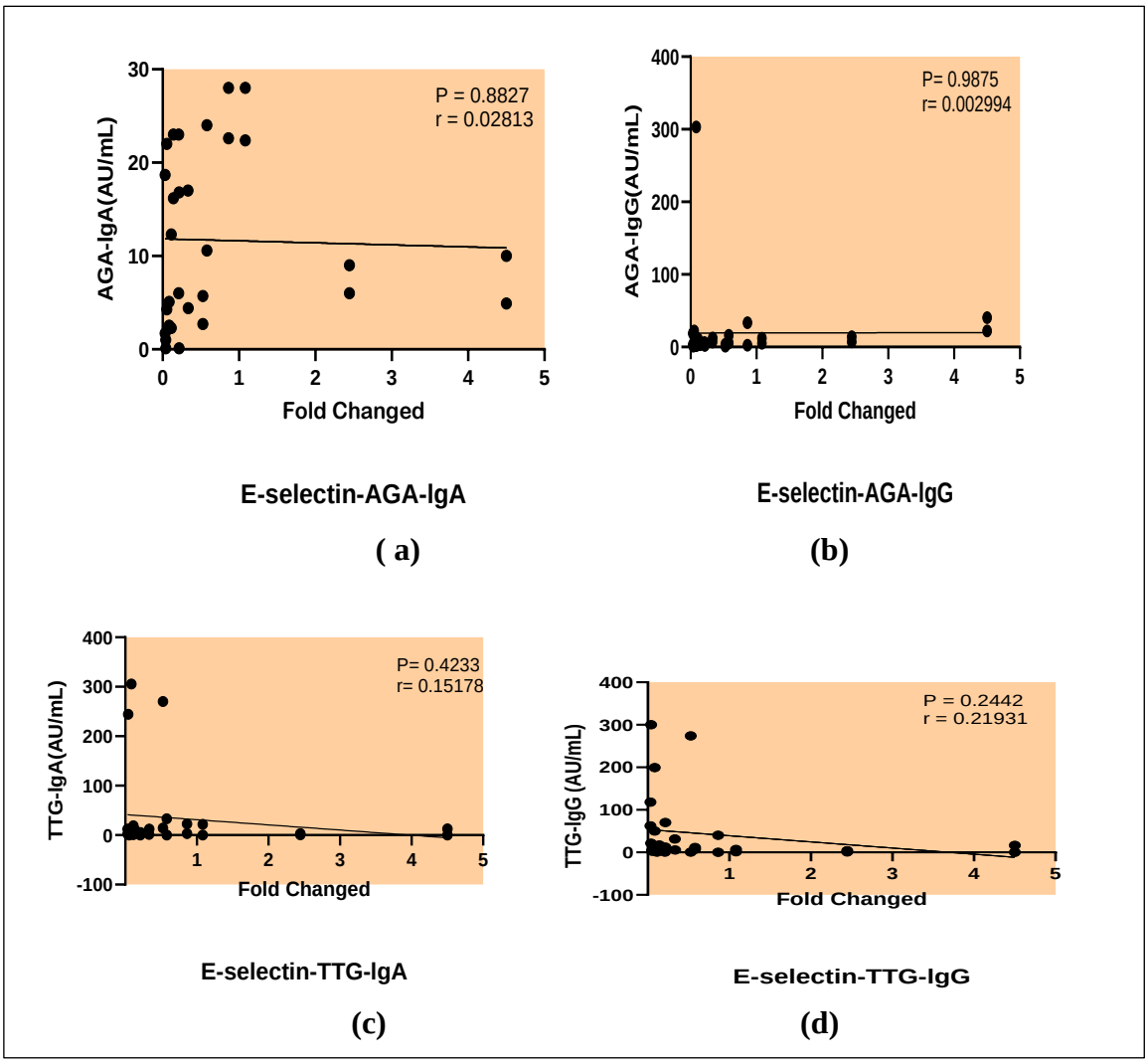

Fig. 3: The correlation of E-selectin folding change with the levels Antibodies (GTT & AGA) in CD

DISCUSSION

The findings of this study align with established literature regarding the diagnostic significance of autoantibodies in celiac disease (CD). Tissue transglutaminase antibodies (TTG-IgA and TTG-IgG) are recognized as the most reliable serological markers due to their high specificity and sensitivity[10]. The strong correlation observed between TTG-IgA and TTG-IgG in this study further reinforces their diagnostic robustness. Conversely, anti-gliadin antibodies (AGA-IgA and AGA-IgG) demonstrated lower specificity, consistent with previous research that has deemed them less reliable for CD diagnosis, particularly in adult populations[10]. Nevertheless, AGA may still be relevant for pediatric screenings and cases of non-celiac gluten sensitivity[11].

While TTG-IgA is widely utilized as the primary serological test, studies suggest that patients with IgA deficiency may yield false-negative TTG-IgA results, necessitating the use of IgG-based markers such as TTG-IgG or deamidated gliadin peptide (DGP) antibodies[12;13]. Furthermore, previous studies indicate that serological markers alone are insufficient for a definitive CD diagnosis, especially in cases of seronegative CD or early-stage disease. Small intestinal biopsy remains the gold standard, particularly when antibody levels are inconclusive.

The gene expression analysis of adhesion molecules in CD patients revealed a downregulation of E-selectin and ICAM-1 compared to controls, with a strong correlation between the two molecules. The accuracy and specificity of the gene expression analysis were confirmed by melting curve assessments, reinforcing the validity of these findings. Interestingly, these results contrast with certain previous studies [8] that reported increased ICAM-1 expression in jejunal biopsies of untreated CD patients. This study identified strong

ICAM-1 staining in the lamina propria, particularly among immune cells, but not on enterocytes or intraepithelial lymphocytes (IELs). Additionally, gluten challenge tests demonstrated a rapid increase in ICAM-1 expression within two hours, indicating its immediate immune response to gluten exposure. More recent findings [14] reported significantly elevated serum soluble ICAM-1 levels in newly diagnosed CD patients compared to controls (336 ± 99 ng/mL vs. 263 ± 67 ng/mL, $p = 0.025$). However, unlike E-selectin, ICAM-1 levels did not significantly decrease following adherence to a gluten-free diet (GFD), suggesting a more stable expression pattern compared to other adhesion molecules[15;16].

A strong correlation ($r=0.86$) was observed between ICAM-1 and E-selectin expression, suggesting co-regulation of these molecules in CD patients[14]. Additionally, higher ICAM-1 levels were associated with short stature, as patients with lower height-for-age z-scores exhibited increased ICAM-1 concentrations. This supports the notion that chronic inflammation and endothelial dysfunction in CD may contribute to growth disorders in children[17;18].

ICAM-1 is a critical protein mediating interactions between immune cells and endothelial cells. It is typically upregulated during inflammatory responses, driven by pro-inflammatory cytokines such as TNF-α and IL-1β[19]. In CD, the immune system exhibits an abnormal response to gluten, leading to the activation of adhesion molecules like ICAM-1, which facilitates leukocyte infiltration into intestinal tissue[8;20]. However, the observed decrease in ICAM-1 expression in this study may be attributed to several factors. First, different disease stages may influence ICAM-1 levels, as patients in this study may be at more advanced stages of CD, where chronic endothelial damage results in decreased ICAM-1 expression. Second, adherence to a strict GFD is known to reduce immune activation in the intestine, potentially leading to lower ICAM-1 expression[21]. Third, individual and genetic variations may account for differences in ICAM-1 expression due to genetic predispositions and environmental interactions[22].

The positive correlation between ICAM-1 and antibody levels aligns with previous studies indicating that ICAM-1 facilitates immune cell adhesion and activation. Increased ICAM-1 expression has been associated with autoimmune diseases, further supporting its role in CD pathogenesis[9;23]. This suggests that ICAM-1 may serve as a potential biomarker for immune activation in CD patients, aiding in disease monitoring and therapeutic targeting.

The findings of this study also suggest that E-selectin may play a more complex or indirect role in immune responses, potentially through interactions with other adhesion molecules or inflammatory mediators. Unlike ICAM-1, E-selectin exhibited a weak and non-significant correlation with antibody levels. This observation is consistent with research suggesting that E-selectin is primarily involved in endothelial activation rather than direct immune cell interactions[24;25]. Its role may be more prominent in vascular inflammation rather than direct antibody-mediated immune responses. The lack of a strong correlation between E-selectin and antibody levels suggests that its role in CD may be independent of humoral immune responses, warranting further investigation.

CONCLUSION

Our findings delineate a complex interplay between humoral immunity and endothelial activation in CD. ICAM-1 emerges as a promising biomarker for immune activity, while E-selectin appears more relevant to vascular inflammation. These results underscore the need for multidimensional assessment of CD pathogenesis, integrating serologic, endothelial, and histologic parameters for comprehensive disease characterization.

Recommendation

Based on the findings of this study, it is recommended that future research focuses on validating the correlation between adhesion molecule expression and autoantibody levels in celiac disease using larger, more diverse cohorts. Longitudinal studies assessing changes in ICAM-1 and E-selectin expression before and after gluten-free diet initiation may offer insights into their roles in disease progression and remission. Further investigation into tissue-level expression and the involvement of pro-inflammatory cytokines could help clarify the underlying mechanisms. Additionally, evaluating soluble forms of these molecules in serum may aid in developing non-invasive biomarkers for disease monitoring.

Declarations:
Consent for publication: Not applicable
Availability of data and material: Data are available upon request.
Competing interests: The author(s) declare no potential conflicts of interest with respect to the research, authorship and/or publication of this article.
This manuscript has not been previously published and is not under consideration in another journal.
Funding: Authors did not receive any grants from funding agencies.

Ethical Approval

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and was approved by the Institutional Review Board (IRB) of the University of Kufa, Faculty of Science, Pathological Analysis Department. Written informed

consent was obtained from all adult participants prior to their inclusion in the study. For participants under 18 years of age, written informed consent was obtained from their parents or legal guardians.

Acknowledgement

We are grateful to the staff and administration of Al-Najaf Educational Hospital, Al-Sadr Medical City, and the Gastroenterology and Hepatology Specialized Center for their support in sample collection and data acquisition. Special thanks are extended to the central laboratories in Najaf province for their technical assistance.

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