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Research Article

Evaluation of Systemic Inflammation Associated with Celiac Disease

Çölyak hastalığı ile ilişkili sistemik enflamasyonun değerlendirilmesi

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Abstract

Aim: The ultimate goal of the study is to measure systemic inflammation in celiac disease and identify associated parameters, thus preventing systemic side effects by combating systemic inflammation in Celiac Disease (CD).

Material and Methods: The Systemic Inflammatory Index (SII) was calculated as "platelet count × neutrophil count/ lymphocyte count" in patients with CD and healthy individuals without a diagnosis of CD who were evaluated for other reasons, who applied to the Internal Medicine Clinics of our centers between 2015-2025. Demographic characteristics, Anti-tissue transglutaminase (Anti-TTG), anti-gliadin, Human leucocyte antigenes HLADQB102, HLADQA103, HLADQA105, hemogram, ESR, CRP, and routine biochemical markers were extracted from medical records.

Results: A total of 200 participants were included: 102 CD patients and 98 controls. Females constituted 60.1% of the cohort. The mean SII was 636.06 in CD patients and 466.48 in controls, with no statistically significant difference ($p = 0.037$). No correlation was observed between SII and autoantibody or genetic markers. Hemoglobin and platelet levels differed significantly between groups ($p = 0.011$ and $p = 0.001$). SII showed a strong positive correlation with ESR and hemoglobin, and a weak correlation with CRP.

Conclusion: Although SII was higher in CD patients, it was not significantly associated with serological or genetic markers. SII may reflect systemic inflammation in CD beyond traditional markers. Further prospective studies are needed to clarify its role in diagnosis and monitoring.

Keywords: Celiac Disease, inflammation, systemic inflammatory index

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Öz

Amaç: Çalışmanın nihai amacı, çölyak hastalığında sistemik inflamasyonu ölçmek ve ilişkili parametreleri belirlemek, böylece Çölyak Hastalığı'nda (ÇH) sistemik inflamasyonla mücadele ederek sistemik yan etkileri önlemektir.

Gereç ve Yöntemler: 2015-2025 yılları arasında merkezlerimizin Dahiliye Kliniklerine başvuran ÇH tanılı hastalar ve başka nedenlerle değerlendirilen ÇH tanısı olmayan, sağlıklı bireylerde Sistemik İnflamatuar İndeks (Sİİ) "trombosit sayısı × nötrofil sayısı/lenfosit sayısı" olarak hesaplandı. Demografik özellikler, Anti-doku transglutaminaz (Anti-TTG), anti-gliadin, insan lökosit antijenleri olan HLADQB102, HLADQA103, HLADQA105, hemogram, eritrosit sedimentasyon hızı (ESR), C-reaktif protein (CRP) değerleri hastane kayıt sisteminden alınmıştır

Bulgular: 102 CD hastası ve 98 kontrol grubu olarak toplam 200 katılımcı çalışmaya dahil edilmiştir. Kohortun %60,1'ini kadınlar oluşturmuştur. Ortalama Sİİ değeri ÇH hastalarında 636,06 ve kontrol grubunda 466,48 olup istatistiksel olarak anlamlı bir fark bulunmamıştır (p = 0,037). Sİİ ile otoantikor veya genetik belirteçler arasında korelasyon gözlenmemiştir. Hemoglobin ve trombosit düzeyleri gruplar arasında anlamlı farklılık göstermiştir (p = 0,011 ve p = 0,001). Sİİ, ESR ve hemoglobin ile güçlü bir pozitif korelasyon ve CRP ile zayıf bir korelasyon gösterdi.

Sonuçlar: Sİİ, CD hastalarında daha yüksek bulunmuş olmasına rağmen, serolojik veya genetik belirteçlerle anlamlı bir ilişki göstermemiştir. Sİİ, geleneksel belirteçlerin ötesinde ÇH'deki sistemik inflamasyonu yansıtıyor olabilir. Tanı ve izlemdeki rolünü netleştirmek için daha ileri prospektif çalışmalara ihtiyaç vardır.

Anahtar Kelimeler: Çölyak hastalığı, inflamasyon, sistemik inflamatuvar indeks

Introduction

Celiac disease (CD) is an intestinal disease that is triggered by gluten intake, can occur at any time in life and has highly variable clinical manifestations. The disease is characterized by histopathological changes such as mucosal inflammation, villus atrophy, and crypt hyperplasia, which regress with removing gluten from the diet. CD is a systemic disease not limited to the gastrointestinal system but can also cause various clinical findings in different organs and systems [1]. Although there is evidence of autoimmune or genetic transmission, its etiology is unclear [2]. Autoimmune markers, genetic variation analyses, and biopsy material findings are diagnostic tools in symptomatic patients [3]. CD can present a serious disease picture with systemic symptoms and findings in some patients, while in some cases, it is milder. Typical gastrointestinal symptoms include diarrhea, abdominal discomfort, bloating and constipation. However, CD can also present with extraintestinal systemic symptoms and signs such as fatigue, weight loss, skin rashes, anemia, and osteoporosis. The mainstay of treatment is a lifelong gluten-free diet. A gluten-free diet provides clinical and histopathologic improvement. However, complete histologic normalization may take years, and some patients may have persistent symptoms that require further evaluation and treatment [4]. CD is associated with many autoimmune diseases. For this reason, the evaluation of systemic inflammation is important for this patient group [1,5]. Since systemic findings are associated with systemic inflammation, many indices have been developed to evaluate inflammation. For example, studies examine whether there is a relationship between platelet indices and the histopathological Marsh score used in CD [6]. Another tool is the systemic inflammatory index (SII) [7], which evaluates diseases with multisystemic effects. SII may be a valuable biomarker to evaluate systemic inflammation in CD patients [8].

It is essential to evaluate the factors that increase systemic inflammation in CD patients and to take the necessary precautions by raising patient awareness on this issue. We aimed to evaluate the SII value in individuals followed up with a diagnosis of CD and to show whether there is a difference in SII value depending on autoantibody or genetic allele positivity. In this way, we aim to determine the priority of patients expected to have systemic effects during the follow-up of these patients and to pave the way for new approaches that can benefit patients in clinical follow-ups.

Material and Methods

We retrospectively evaluated SI levels in patients diagnosed with CD and healthy individuals admitted to the Internal Medicine Clinics of Sivas Cumhuriyet University and Yozgat Bozok University between 2015 and 2025. The study was conducted following the ethical standards specified in the Declaration of Helsinki, and approval was obtained from the Ethics Committee of Yozgat Bozok University (2025-GOKAEK-257-2025.04.09/456).

SII is a parameter calculated with the formula "platelet count × neutrophil count/lymphocyte count". Demographic data such as age and gender, medical history, anti-tissue transglutaminase (anti-TTG) and anti-gliadin, human leucocyte antigenes HLADQB102, HLADQA103, HLADQA105, and hemogram values of individuals who were followed up with the diagnosis of CD were retrospectively obtained from the hospital record system. The study included individuals aged 18 years and over, those being followed up with a diagnosis of CD, those with no history of systemic disease other than CD (malignancy, rheumatological disease, etc.), and those not receiving anti-inflammatory treatment for any reason. Patients diagnosed with immune deficiency, those with known malabsorption/absorption diagnoses other than CD, and those with a history of previous gastrointestinal surgery were excluded from the study. The control group, which was made up of outpatients over the age of 18 who were admitted to the internal medicine outpatient clinic for other reasons and had no acute infections or chronic illnesses, was chosen based on the age and gender distribution of the patient group.

Statistical Analysis

Data analyses were performed using the SPSS 20.00 (Statistical Package for the Social Sciences, SPSS Inc., Chicago, IL) program. Descriptive statistics were given as mean ± standard deviation for continuous variables, median and quartile distribution for data not conforming to normal distribution, and as percentages for categorical variables. Whether the groups were normally distributed was determined using the Kolmogorov-Smirnov test. The independent group t-test was used to measure differences between groups, and the Chi-square test was used to measure categorical variables. However, the Mann-Whitney U test was used if the groups were not normally distributed. The p values below 0.01 and 0.05 were considered statistically significant. When the data were normally distributed, the Pearson correlation method was used, and when they were not normally distributed, the Spearman correlation method was used.

Results

After screening the records, 144 patients diagnosed with CD were identified. After excluding patients with no autoantibody and genetic test results, 102 patients with CD were included in the study. The study included 98 healthy volunteers. 119 (60.1%) of the individuals included in the study were female and 73 (36.9%) were male. The mean age was 41.50 ± 13.04 in the patient group and 28.74 ± 6.62 in the healthy group. In the study in which systemic inflammation was evaluated, ESR among the inflammation indicators was calculated as median 8 (Interquartile range: 12) min-max: 2-48; CRP median 1.94 (Interquartile range: 2.46) min-max: 0.09-34. While the mean ESR in the patient group was 13.02 ± 12.20, it was calculated as 7.67 ± 4.94 in the healthy group. A statistically significant difference was found between the patient and healthy groups in ESR (p = 0.000). While the mean CRP in the patient group was 3.43 ± 4.86, it was calculated as 2.32 ± 2.97 in the healthy group. The difference between the groups in terms of CRP levels was not found to be statistically significant (p=0.067). SII was calculated as 632.38 ± 489.32; median 519.44 (Interquartile range: 432.10); min-max: 133.52-3260.37. The mean SII was calculated as 636.06 in the patient group and 466.48 in the healthy group. SII showed no statistically significant difference between the groups (p = 0.037).

When the overall distribution of genetic markers among patients with CD was evaluated, HLA-DQA1*03 demonstrated the highest positivity rate (73.5%), followed by HLA-DQA1*05 (56.9%) and HLA-DQB1*02 (36.3%). In the analysis of CD-specific serological markers, anti-TTG positivity was more prevalent than anti-gliadin antibody positivity (19.6%, 34.3%; 18.6%, 24.5%) (Table 1).

Table 1. Genetic and autoimmune parameters and distribution in Celiac Disease patients.

HLADQB1-02	
Positive	58 (56.9%)
Negative	44 (43.1%)
HLADQA1-03	
Positive	37 (36.3%)
Negative	65 (63.7%)
HLADQA1-05	
Positive	75 (73.5%)
Negative	27 (26.5%)
Anti-TTG IgG	
Positive	20 (19.6%)
Negative	82 (80.4%)
Anti-TTG IgA	
Positive	35 (34.3%)
Negative	67 (65.7%)
Anti_Gliadin IgG	
Positive	19 (18.6%)
Negative	83 (81.4%)
Anti_Gliadin IgA	
Positive	25 (24.5%)
Negative	77 (75.5%)
Abbrev.: Anti-TTG IgG: anti-tissue transglutaminase IgG, Anti-TTG IgA: anti-tissue transglutaminase IgA)	

In this study, the relationship between autoantibody levels, genetic variants, and the SII was investigated in patients with

CD. A strong negative correlation was identified between HLA-DQB102 and HLA-DQA103, as well as between HLA-DQA103 and HLA-DQA105. Conversely, HLA-DQB102 exhibited a positive correlation with serological autoantibody markers. However, no statistically significant association was observed between autoantibody levels, HLA genetic variants, and SII values (Table 2).

Table 2. Association of autoantibody and genetic variation results with SII in Celiac Disease.

	HLADQB102	HLADQA103	HLADQA105	SII
Anti-TTG IgG	.330**	-0.167	0.128	0.116
Anti-TTG IgA	.379**	-0.159	.293**	0.112
Anti_Gliadin IgG	.264**	-0.151	0.116	-0.014
Anti_Gliadin IgA	.220*	-0.003	0.135	0.086
SII	0.052	-0.072	0.090	1

Abbrev.: Anti-TTG IgG: anti-tissue transglutaminase IgG, Anti-TTG IgA: anti-tissue transglutaminase IgA, SII: Systemic Inflammatory Index, ** Correlation is significant at the 0.01 level, * Correlation is significant at the 0.05 level

Upon analysis of the biochemical parameters of patients with CD compared to the healthy control group, hemoglobin and platelet levels were found to differ significantly between the groups ($p = 0.011$ and $p = 0.001$, respectively). In contrast, no statistically significant differences were observed in liver function tests, uric acid, or vitamin D levels (Table 3).

In the evaluation of the association between biochemical parameters and systemic inflammation, the SII demonstrated a strong positive correlation with ESR and a weak positive correlation with CRP. Additionally, a significant positive correlation was observed between hemoglobin levels and SII. While liver function tests exhibited occasional correlations with ESR and CRP levels, no meaningful association was identified between these parameters and SII. Moreover, no correlation was found between SII and vitamin D or uric acid levels (Table 4).

Table 3. Evaluation of biochemical data between the CD group and the healthy group.

Parameter	Group	n	mean	std. deviation	p
Hemoglobin	patient	102	13.1719	2.11397	0.011
	healthy	90	14.0400	2.49646	
Platelet count	patient	102	276.7716	85.29189	0.001
	healthy	90	244.3333	48.55392	
Protein	patient	100	77.6200	64.74390	0.285
	healthy	90	70.6333	5.87225	
Albumin	patient	100	43.9176	8.76164	0.781
	healthy	90	44.2467	7.54352	
Uric acid	patient	97	5.0258	4.52636	0.145
	healthy	90	7.0100	12.04868	
Vitamin D	patient	87	16.5398	8.83112	0.136
	healthy	87	18.5117	8.51086	
ALT	patient	102	19.0784	9.97987	0.202
	healthy	90	17.4000	8.16790	
AST	patient	102	20.3529	11.49313	0.024
	healthy	90	17.5333	4.59897	
GGT	patient	102	22.0392	28.83546	0.930
	healthy	90	21.7000	24.72657	
T. Bilirubin	patient	100	1.1395	6.26215	0.347
	healthy	90	0.5470	0.21353	
D. Bilirubin	patient	101	0.3367	1.18523	0.275
	healthy	90	0.2070	0.07006	

Abbrev.: std. deviation: standard deviation, ALT: Alanine transaminase, AST: Aspartate transaminase, GGT: Gamma-glutamyltransferase, T. bilirubin: Total bilirubin, D. bilirubin: Direct bilirubin

Table 4. Association of biochemical parameters with SII.

	SII	ESR	CRP	Protein	Albumin	Vitamin D
SII	1	.244**	,161*	-0,060	0,091	-0.071
Hemoglobin	-.207**	-.316**	-0.085	-0.055	0.084	0.096
Uric acid	-0.063	-0.042	0.076	-0.085	0.001	0.006
ALT	0.046	0.138	0.207**	0.115	-0.043	0.085
AST	-0.060	0.056	0.072	0.056	-0.002	-0.060
GGT	0.044	0.186*	0.453**	-0.022	0.062	0.057
Total bilirubin	0.073	.266**	0.102	-0.003	0.025	0.057

Abbrev.: SII: Systemic inflammatory index, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, ALT: Alanine transaminase, AST: Aspartate transaminase, GGT: Gamma-glutamyltransferase, ** Correlation is significant at the 0.01 level, * Correlation is significant at the 0.05 level

Discussion

CD is a chronic inflammation-related autoimmune enteropathy triggered by dietary gluten in genetically predisposed individuals [9]. Inflammation has an important effect in CD. It is shown that the disease starts as low-grade inflammation long before it becomes clinically manifest [10]. In CD patients, it has been observed that systemic inflammation markers return to normal when a gluten-free diet is followed [11]. Currently, there is no widely accepted measure of disease activity for clinical trials. However, patient-reported outcomes such as the CD Symptom Diary and morphometric histology can be used to assess treatment efficacy [12]. Interleukin-1β (IL-1β) and Interleukin-6 (IL-6) are markers of the proinflammatory process in these enterocytes [10]. Monitoring CD patients with antibody or cytokine titers and mucosal biopsy is difficult in daily clinical practice.

In a study comparing the ESR and CRP parameters and leukocyte count, which are known to increase in inflammatory diseases, in active CD patients and controls, no significant difference was found between the two groups. It was observed that there was no significant difference in the same parameters between the subgroups of CD patients who adhered to and did not adhere to a gluten-free diet [13]. These results suggest that classical acute-phase reactants and ESR may not provide definitive information specifically about disease severity. On the other hand, none of the markers are reliable indicators of dietary compliance and disease severity in CD patients. Some CD patients may present with vague clinical and nonspecific routine laboratory test results, regardless of the severity of systemic inflammation. This nonspecific manifestation leads to delayed diagnosis of CD in these patients [13]. Early diagnosis of CD is crucial in preventing complications such as anemia, vitamin deficiencies, osteopenia, osteoporosis.

To reveal the level of systemic inflammation in CD, various indices and ratios that are more practical and easily accessible have been tried. Neutrophil-to-lymphocyte ratio (NLR) is sensitive in diagnosing CD, and 80% sensitivity and 41% specificity were obtained with a cut-off value of 2.32 [14]. In addition, the delta neutrophil index (DNI) has been investigated as a potential marker for dietary compliance in CD patients, but no significant association was found with serological tests [15]. These findings indicate that searching for reliable inflammatory markers for managing CD continues.

SII, as a specific term and formula, was first presented in the literature in 2014 as an independent predictor of recurrence and survival after surgery in hepatocellular carcinoma patients. It has recently gained traction as a broader prognostic marker in various inflammatory and chronic diseases. SII is an index immune-inflammation-based prognostic score using lymphocyte, platelet and neutrophil counts [16]. SII has been found valuable in predicting the need for intubation in COVID-19 patients and in shedding light on patient management during the severe disease course [17]. A significant relationship has also been found between disease severity and SII in coronary artery disease [18]. SII is a versatile marker that may be significant regarding the prognosis and severity of all chronic and systemic diseases with underlying inflammation.

In this study, SII was found to be higher in CD patients compared to the control group. However, this difference was not statistically significant. SII showed significant correlation with ESR and statistically insignificant correlation with CRP, which are classical inflammation parameters. One plausible explanation may relate to the characteristics of the control group, which comprised individuals without a formal CD diagnosis but who might have other gluten-related disorders such as non-celiac gluten sensitivity (NCGS), which also involve inflammatory components. The presence of underlying inflammation in the control group could have minimized the difference in SII, thereby reducing the statistical power [19].

In a study of 161 CD patients and a control group of 75 patients, significantly higher SII rates were found in the CD group. In that study, SII was considered a superior diagnostic marker to other inflammation markers [8]. In another study using blood tests obtained from CD patients at the time of diagnosis and 6 months after starting a gluten-free diet, SII was significantly lower than baseline at 6 months [20].

A meta-analysis demonstrating the potential utility of SII in diagnosing the presence of active disease in immunologic diseases found no significant correlation between disease duration and the traditional markers of inflammation, ESR and CRP. This study demonstrates that SII is also useful in the early stage of immunologic diseases [21].

In our study, genetic variation positivity was correlated with ESR, but no significant correlation was found with SII. The direct association of CD-related genetic variation positivity with disease severity is unknown [22]. Moreover, the absence of a relationship between genetic markers and SII aligns with the understanding that genetic predisposition confers susceptibility but does not determine the degree of inflammatory response or disease severity.

Another potential explanation for the lack of statistical significance is the timing of the laboratory data. Most blood samples in this study were collected during follow-up visits, not at the time of diagnosis when the inflammatory burden might be highest.

In conclusion, SII can be used as a parameter indicating inflammation in CD. It is satisfactorily informative as a biomarker, mainly when evaluated with the acute-phase reactants. Although the mechanism underlying systemic inflammation in CD and its relationship with disease manifestations is not yet evident, it is an important disease pathogenesis. Data on its measurement are essential for managing, following up and treating the disease. Therefore, further studies on larger scales and with different methodologies are needed.

Conflict of Interest

None of the authors report any conflicts of interest relevant to this study.

Financial Disclosure

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Ethical Approval

This study is approved by Yozgat Bozok University Non-Interventional Clinical Research Ethics Committee with decision number 2025-GOKAEK-257_2025.04.09_456.

Authors' Contributions

ŞT developed the study concept and design, conducted data collection, performed statistical analyses, and drafted the manuscript. DF contributed to methodological validation, data interpretation, literature synthesis, and critical revision of the manuscript. Both authors approved the final version and take full responsibility for the scientific content.

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