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ORIGINAL ARTICLE

Clinical and laboratory characteristics of *Helicobacter pylori* serology in patients with diabetic foot infection: A retrospective case-control study

Diyabetik ayak enfeksiyonu olan hastalarda *Helicobacter pylori* serolojisinin klinik ve laboratuvar özellikleri: Retrospektif vaka-kontrol çalışması

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ABSTRACT • **Background and Aims:** *Helicobacter pylori* infection is not limited to gastrointestinal diseases and has been associated with systemic inflammation, endothelial dysfunction, and metabolic disturbances. Emerging evidence suggests that *Helicobacter pylori* may influence glycemic control and vascular complications in patients with diabetes mellitus. This study aimed to investigate the association between *Helicobacter pylori* immunoglobulin A and immunoblobulin G seropositivity and diabetic foot infection in patients with diabetes mellitus presenting with dyspeptic symptoms. **Materials and Method:** This retrospective study was conducted between 2023 and 2024 and included 34 patients followed for complicated diabetic foot infection with documented *Helicobacter pylori* serological data and 34 age- and sex-matched controls without diabetic foot infection. Demographic characteristics, laboratory parameters, and *Helicobacter pylori* immunoglobulin A and immunoglobulin G seropositivity rates were compared between groups. **Results:** *Helicobacter pylori* immunoglobulin A positivity was significantly higher in the patient group than in controls (73.5% vs. 29.4%; p = 0.0006), whereas *Helicobacter pylori* immunoglobulin G positivity did not differ between groups. Patients with diabetic foot infection showed significantly higher inflammatory markers (C-reactive protein, erythrocyte sedimentation rate) and neutrophil counts compared with controls. **Conclusion:** *Helicobacter pylori* immunoglobulin A seropositivity was significantly more frequent in patients with diabetic foot infection compared with controls and may reflect increased systemic inflammatory burden in this population. However, because serological tests have limited specificity for detecting active *Helicobacter pylori* infection, these findings should be interpreted cautiously and considered hypothesis-generating. Larger prospective studies using more accurate diagnostic methods are required to clarify the clinical relevance of this association and to determine whether *Helicobacter pylori* eradication may influence clinical outcomes in diabetic foot infection.

Key words: Diabetic foot infection, *Helicobacter pylori*, *Helicobacter pylori* seropositivity, systemic inflammation

ÖZET • **Giriş ve Amaç:** *Helicobacter pylori* enfeksiyonu yalnızca gastrointestinal hastalıklarla sınırlı olmayıp sistemik inflamasyon, endotelyal disfonksiyon ve metabolik bozukluklarla da ilişkilendirilmiştir. Giderek artan kanıtlar, *Helicobacter pylori*'nin diyabetes mellituslu hastalarda glisemik kontrolü ve vasküler komplikasyonları etkileyebileceğini düşündürmektedir. Bu çalışma, dispeptik semptomlar gösteren diyabetes mellituslu hastalarda *Helicobacter pylori* immünglobulin A ve immünglobulin G seropozitifliği ile diyabetik ayak enfeksiyonu arasındaki ilişkiyi araştırmayı amaçlamıştır. **Gereç ve Yöntem:** 2023-2024 yılları arasında gerçekleştirilen bu retrospektif çalışmaya, komplike diyabetik ayak enfeksiyonu tanısı ile izlenen ve *Helicobacter pylori* serolojik verileri bulunan 34 hasta ile yaş ve cinsiyet açısından eşleştirilmiş, diyabetik ayak enfeksiyonu olmayan 34 kontrol olgu dahil edildi. Demografik özellikler, laboratuvar parametreleri ve *Helicobacter pylori* immünglobulin A ve immünglobulin G seropozitiflik oranları gruplar arasında karşılaştırıldı. **Bulgular:** *Helicobacter pylori* immünglobulin A pozitifliği hasta grubunda kontrol grubuna göre anlamlı derecede yüksekti (%73.5 vs. %29.4; p = 0.0006), *Helicobacter pylori* immünglobulin G pozitifliği açısından ise anlamlı fark saptanmadı. Hasta grubunda inflamasyon belirteçleri (C-reaktif protein, sedimantasyon), nötrofil sayısında artış gözlendi. **Sonuç:** Diyabetik ayak enfeksiyonu olan hastalarda *Helicobacter pylori* immünglobulin A seropozitifliği kontrollere göre anlamlı derecede daha sık görülmüştür ve bu durum bu popülasyondaki sistemik inflamatuvar yükün artışını yansıtabilir. Bununla birlikte, serolojik testlerin aktif *Helicobacter pylori* enfeksiyonunu tespit etmede sınırlı özgüllüğe sahip olması nedeniyle, bu bulgular dikkatli bir şekilde yorumlanmalı ve hipotez oluşturucu olarak değerlendirilmelidir. Bu ilişkinin klinik önemini açıklığa kavuşturmak ve *Helicobacter pylori* eradikasyonunun diyabetik ayak enfeksiyonunda klinik sonuçları etkileyip etkilemediğini belirlemek için daha doğru tanı yöntemleri kullanan daha büyük prospektif çalışmalara ihtiyaç vardır.

Anahtar kelimeler: Diyabetik ayak enfeksiyonu, *Helicobacter pylori*, *Helicobacter pylori* seropozitifliği, sistemik inflamasyon

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INTRODUCTION

Diabetes mellitus (DM) is a multisystemic metabolic disease characterized by impaired immune compensation, which predisposes patients to various classical and opportunistic infectious agents that can negatively affect clinical outcomes. Poorly controlled DM leads to numerous complications, among which diabetic foot infection (DFI) is one of the most significant. Multiple infectious agents can contribute to the development of DFI. While several studies have investigated the prevalence of *Helicobacter pylori (H. pylori)* infection in type 2 diabetes and its metabolic effects (1), no studies have specifically assessed its impact on the occurrence or progression of diabetic foot infection (DFI).

In this study, we aimed for the first time to evaluate patients with DFI and known *H. pylori* serological status, to investigate the potential impact of *H. pylori* positivity on the course of diabetic foot infections. If *H. pylori* positivity is shown to increase susceptibility to DFI or influence its progression, eradication therapy might offer clinical benefit by reducing the burden and cost associated with the treatment of this serious complication of type 2 diabetes.

DM is a chronic disease characterized by hyperglycemia due to relative or absolute insulin deficiency or peripheral insulin resistance, and it can lead to multisystem involvement. Poor glycemic control, in combination with peripheral neuropathy, ischemia, and trauma, facilitates the development of DFI (2,3). A wide range of pathogens may be implicated. *Staphylococcus* aureus is the most commonly identified microorganism, but Streptococcus, Enterococcus, Enterobacteriaceae, and Pseudomonas species can also be isolated. DFI may begin as a superficial infection and progress to osteomyelitis or require amputation in severe cases (4). Treatment involves wound care, antibiotic therapy, and amputation when necessary (5).

H. pylori is one of the most prevalent infections worldwide. It is well known for its association with chronic gastritis, peptic ulcers, MALT lymphoma, and gastric cancer, but it has also been identified as a contributing factor to endothelial dysfunction and atherosclerosis (6,8-10). Treatment typically includes combinations of antibiotics such as amoxicillin, clarithromycin, metronidazole, or levofloxacin, together with proton pump inhibitors (7).

The aim of this study was to investigate the association between *H. pylori* seropositivity and diabetic foot infection by evaluating biochemical parameters in patients with confirmed foot infections. If a significant association is found, *H. pylori* eradication could potentially contribute to reducing the morbidity and mortality related to diabetic foot infections.

MATERIALS and METHOD

This retrospective study included a total of 68 participants, consisting of 34 patients diagnosed with diabetic foot infection (DFI) and 34 age- and sex-matched control subjects. The patient group comprised individuals who were hospitalized at Giresun University Training and Research Hospital between 2023 and 2024, had a diagnosis of diabetes mellitus for at least 10 years, presented with dyspeptic symptoms, and had available medical records including previously performed *Helicobacter pylori (H. pylori)* serological testing. Patients were retrospectively identified via random screening of the hospital database (HIMS).

Diabetic foot infection severity was assessed according to the Wagner classification system, and patients with Wagner grade ≥ 2 were included in the study, reflecting moderate to severe infections requiring multidisciplinary management. These patients had received comprehensive clinical care,

including wound care, debridement, intravenous antibiotic therapy, and, when indicated, surgical interventions such as amputation.

The control group consisted of age- and sex-matched individuals who presented with dyspeptic symptoms, had available *H. pylori* serology results in their medical records, and had no history of diabetic foot infection.

Demographic characteristics and laboratory parameters were obtained retrospectively from patient records. The evaluated variables included age, sex, hemoglobin (HGB), hematocrit (HCT), mean corpuscular volume (MCV), platelet count (PLT), neutrophil count (NEU), lymphocyte count (LYM), eosinophil count, blood urea nitrogen (BUN), creatinine, glucose, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total protein, albumin, gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), calcium, phosphorus, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), thyroid-stimulating hormone (TSH), and *H. pylori* immunoglobulin G (Hp-IgG) and immunoglobulin A (Hp-IgA) seropositivity.

The study protocol was approved by the Clinical Research Ethics Committee of Giresun University Faculty of Medicine Training and Research Hospital (Approval number: E-53593568-771-244441999, dated 10/06/2024; Decision number: 22/05/2024/01). Due to the retrospective nature of the study, informed consent was not obtained. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Statistical Analysis

Statistical analyses were performed using the SPSS software version 23.0 for Windows (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median (minimum-maximum), while categorical variables were presented as counts and percentages.

The distribution of the data was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests for normality. Comparisons between groups were made using the Student's t-test for normally distributed variables and the Mann-Whitney U test for variables that did not show normal distribution. Categorical variables were compared using the Chi-square test. Correlation analysis was performed to assess relationships between numerical variables.

Data are presented as frequencies (n, %) and means with 95% confidence intervals (CI). A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 34 patients with diabetic foot infection and 34 age- and gender-matched healthy controls were included in the study. The mean age and gender distribution were comparable between the two groups ($p = 0.458$ and $p = 1.000$, respectively).

Hematological analysis revealed a pronounced inflammatory profile in the patient group. White blood cell, neutrophil, and platelet counts were significantly higher in patients than in controls, whereas hemoglobin and hematocrit levels were significantly lower (all $p < 0.05$). Mean corpuscular volume did not differ between groups (Table 1).

Renal function parameters were markedly impaired in the patient group, with significantly higher serum creatinine and urea levels compared to controls (both $p < 0.001$). Fasting glucose levels were also significantly elevated in patients ($p < 0.001$) (Table 1).

Regarding liver-related biochemical parameters, ALT and AST levels were significantly higher in the patient group, accompanied by modest but significant increases in GGT and ALP levels (all $p < 0.05$). Total protein and albumin levels were similar between the two groups (Table 1).

Inflammatory markers were markedly elevated in patients with diabetic foot infection, with significantly higher CRP and ESR levels compared to controls (both $p < 0.001$) (Table 1).

Evaluation of *H. pylori* serology demonstrated a significantly higher rate of Hp-IgA positivity in the patient group compared to controls (73.5% vs. 29.4%, $p = 0.0006$). In contrast, no statistically significant difference was observed between groups in terms of Hp-IgG positivity (55.9% vs. 47.1%, $p = 0.467$) (Table 2).

Tablo 1 Comparison of demographic and laboratory parameters between patient and control groups

Parameter	Patient Group (n = 34) Mean (95% CI)	Control Group (n = 34) Mean (95% CI)	p-value*
Age (years)	69.1 (65.2-73)	69.5 (66.9-72.1)	0.458
WBC (10^3/μL)	8.9 (7.6-10.1)	6.6 (6.1-7.2)	**0.004**
HGB (g/dL)	10.5 (9.9-11.1)	12.9 (12.4-13.5)	**< 0.001**
HCT (%)	33.1 (31.5-34.8)	38.7 (37.3-40.1)	**< 0.001**
MCV (fL)	86.6 (84.3-88.9)	86.2 (83.7-88.7)	0.873
PLT (10^3/μL)	306 (262-349)	250 (223-278)	**0.039**
NEUT (10^3/μL)	6.42 (5.24-7.61)	4.03 (3.56-4.50)	**< 0.001**
LYM (10^3/μL)	1.68 (1.41-1.94)	1.91 (1.67-2.14)	0.068
EOS (10^3/μL)	0.28 (0.19-0.36)	0.29 (0.16-0.42)	0.727
CRE (mg/dL)	1.70 (1.45-1.95)	0.81 (0.74-0.89)	**< 0.001**
UREA (mg/dL)	76.7 (58.6-94.8)	35.3 (31.3-39.3)	**< 0.001**
GLU (mg/dL)	173 (144-201)	110 (99-120)	**< 0.001**
ALT (U/L)	76.0 (36.7-188.8)	16.5 (13.6-19.4)	**< 0.001**
AST (U/L)	53.8 (4.6-102.9)	18.7 (16.3-21.0)	**< 0.001**
Total Protein (g/L)	63.1 (57.8-68.2)	61.6 (58.2-67.0)	0.322
Albumin (g/L)	34.9 (31.8-36.4)	35.2 (33.2-37.0)	0.522
GGT (U/L)	63.9 (36.1-91.6)	35.9 (10.6-61.2)	**0.023**
ALP (U/L)	116.2 (93.2-139.2)	89.2 (73.8-104.5)	**0.041**
CA (mg/dL)	8.87 (8.69-9.05)	9.50 (9.32-9.68)	**< 0.001**
P (mg/dL)	3.74 (3.36-4.13)	3.49 (3.31-3.67)	0.444
CRP (mg/L)	62.7 (36.2-89.3)	5.1 (1.7-8.6)	**< 0.001**
ESR (mm/h)	78.3 (65.1-91.5)	29.7 (21.1-38.2)	**< 0.001**
TSH (μIU/mL)	2.22 (1.24-3.20)	3.35 (0.47-6.23)	0.520

*Bold p-values indicate statistical significance ($p < 0.05$).

WBC: White blood cell; HGB: Hemoglobin; HCT: Hematocrit; MCV: Mean corpuscular volume; PLT: Platelet; NEUT: Neutrophil; LYM: Lymphocyte; EOS: Eosinophil; CRE: Creatinine; GLU: Glucose; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; GGT: Gamma-glutamyl transferase; ALP: Alkaline phosphatase; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate.

Tablo 2 Gender distribution and *Helicobacter pylori* serology results in patient and control groups

Parameter		Patient	Control	p-value
Gender	Male Female	20 (58.8) 14 (41.2)	20 (58.8) 14 (41.2)	1.000
Hp-IgA	Negative Positive	9 (26.5) 25 (73.5)	24 (70.6) 10 (29.4)	**0.0006**
Hp-IgG	Negative Positive	15 (44.1) 19 (55.9)	18 (52.9) 16 (47.1)	0.467

Hp-IgA; *Helicobacter pylori*-immunoglobulin A; Hp-IgG; *Helicobacter pylori*-immunoglobulin G.

DISCUSSION

Diabetic foot infection (DFI) represents one of the most severe and costly complications of diabetes mellitus (DM), frequently leading to prolonged hospitalization, limb amputation, and increased mortality (2,3). The pathogenesis of DFI is multifactorial and involves poor glycemic control, peripheral neuropathy, peripheral arterial disease, impaired immune response, and chronic systemic inflammation. Management of DFI requires a complex, multidisciplinary approach including wound care, antibiotic therapy, metabolic control, and, in selected cases, surgical intervention (5). Identifying additional factors that may contribute to the inflammatory and vascular burden of DFI therefore remains clinically relevant.

Helicobacter pylori (H. pylori) infection is a highly prevalent chronic infection that primarily affects the gastrointestinal tract but has also been implicated in various extra-gastrointestinal conditions (6,7). Previous studies have reported a higher prevalence of *H. pylori* infection in patients with type 2 DM and suggested its potential role in promoting insulin resistance, chronic inflammation, and impaired glycemic control (8). Moreover, accumulating evidence indicates that *H. pylori* infection may contribute to endothelial dysfunction, atherosclerosis, and vascular complications through persistent immune activation and inflammatory mediator release (10-13). However, the relationship between *H. pylori* infection and diabetic foot infection—a specific and severe complication of diabetes—has not been previously investigated.

In the present study, Hp-IgA seropositivity was significantly higher in patients with diabetic foot infection than in controls, whereas Hp-IgG seropositivity did not differ between groups. Although IgA antibodies may reflect mucosal immune exposure, serological tests alone cannot reliably distinguish active from past infection. Current international guidelines, including the Maastricht VI/Florence Consensus Report, do not recommend serology as a primary diagnostic method for detecting active *H. pylori* infection because of its limited specificity (9). Therefore, the observed association should be interpreted cautiously; however, the significantly higher Hp-IgA seropositivity in patients with diabetic foot infection suggests a possible link that warrants further investigation.

Consistent with this hypothesis, patients with diabetic foot infection demonstrated a pronounced inflammatory profile, including significantly elevated C-reactive protein, erythrocyte sedimentation rate, and neutrophil counts. Chronic systemic inflammation plays a central role in endothelial dysfunction, impaired microcirculation, delayed wound healing, and susceptibility to infection, all

of which are key mechanisms in the development and progression of DFI. *H. pylori* infection has been associated with systemic inflammatory responses that may contribute to vascular injury and atherosclerotic processes (10-13), and may further increase the inflammatory burden in susceptible diabetic patients.

Renal function parameters, including creatinine and urea levels, were significantly higher in the diabetic foot group. This finding is most likely related to long-standing diabetes, diabetic nephropathy, and the systemic inflammatory state associated with severe infection rather than a direct effect of *H. pylori* infection. Similarly, elevated glucose levels reflect poor glycemic control, which is a well-established risk factor for infection susceptibility, impaired wound healing, and adverse outcomes in DFI.

Liver enzyme abnormalities observed in the patient group, including elevated alanine aminotransferase and aspartate aminotransferase levels accompanied by modest increases in gamma-glutamyl transferase and alkaline phosphatase, may reflect a combination of metabolic dysfunction-associated fatty liver disease, diabetes-related hepatocellular injury, cholestatic effects, and medication use during hospitalization. Although albumin and total protein levels were not significantly different between groups, relatively lower albumin levels with preserved total protein may suggest mild globulin elevation, consistent with chronic inflammation and acute-phase response rather than overt hepatic synthetic dysfunction.

Hematological findings further support the presence of chronic inflammatory stress in patients with DFI. Reduced hemoglobin and hematocrit levels in the presence of normal mean corpuscular volume are compatible with anemia of chronic disease, a common finding in patients with persistent infection and systemic inflammation. Elevated platelet counts may also reflect inflammatory activation, as reactive thrombocytosis is frequently observed in chronic inflammatory states.

Taken together, these findings suggest a potential association between Hp-IgA seropositivity, systemic inflammation, and diabetic foot infection. Future studies with larger sample sizes and multivariate analyses are needed to determine whether Hp-IgA seropositivity is independently associated with diabetic foot infection. This study provides novel evidence suggesting a potential link between Hp-IgA seropositivity, systemic inflammation, and DFI, thereby opening a new perspective on the immunopathogenesis of diabetic foot disease.

The main limitations of this study include its retrospective design and relatively small sample size, which restrict causal inference and limit generalizability. Because of the relatively small sample size, multivariate analysis to adjust for potential confounding factors could not be performed. In addition, *H. pylori* infection was assessed using serological methods rather than invasive diagnostic techniques such as upper gastrointestinal endoscopy or histopathology; therefore, active infection, atrophic gastritis, and intestinal metaplasia could not be evaluated. Furthermore, the effects of in-hospital management, including strict glycemic control and additional treatments related to diabetes mellitus beyond antibiotics and proton pump inhibitors, could not be fully assessed due to the retrospective nature of the study.

Despite these limitations, the study has several important strengths. To our knowledge, this is the first study to investigate the association between *H. pylori* seropositivity and diabetic foot infection. Blood samples were obtained prior to antibiotic therapy, reducing the risk of false-negative serological results. Moreover, patients receiving proton pump inhibitors were excluded, minimizing potential confounding effects on *H. pylori* serology. These factors enhance the reliability of the ob-

served association between Hp-IgA positivity and diabetic foot infection.

Hp-IgA seropositivity was significantly more frequent in patients with diabetic foot infection compared with controls. However, because serological tests have limited specificity for detecting active H. pylori infection, these findings should be interpreted cautiously and considered hypothesis-generating. Larger prospective studies using more accurate diagnostic methods are needed to clarify the clinical relevance of this association and to determine whether *H. pylori* eradication may influence clinical outcomes in diabetic foot infection.

Data Availability Statement: *The datasets generated and/or analyzed during the current study are not publicly available due to institutional regulations and patient confidentiality but are available from the corresponding author upon reasonable request.*

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