Iran J Pathol. 2027;22(1):57–61 | ISSN 2345-3656 | MAIN SUBJECTS GI, Liver & Pancreas Pathology

Correlation of Serum Interleukin-6 and Interleukin-10 Levels with Helicobacter pylori Infection Burden in Gastric cancer patients

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RECEIVED 16 Feb 2026 ACCEPTED 09 Aug 2026 PUBLISHED ONLINE 11 Oct 2026 DOI 10.22034/ijp.2026.2085584.3627

ABSTRACT

Background & Objective: Gastric cancer (GC) as the fourth most common cancer and the second most common cause of cancer death worldwide, is a major global health burden. *Helicobacter pylori* infection is a key driver of gastric carcinogenesis. This study has evaluated the serum levels of interleukin-6 (IL-6) and interleukin-10 (IL-10) in GC patients before and after surgery and assess their correlation with *H. pylori* infection burden.

Methods: This case-control study was conducted between June 2020 and June 2021 at Shahid Beheshti and Besat hospitals in Hamadan, Iran, involving 35 patients with confirmed GC and 23 healthy controls. Serum levels of IL-6 and IL-10 were measured by ELISA preoperatively and post-gastrectomy. *H. pylori* infection was detected via histopathological examination of gastric biopsies.

Results: Preoperative serum levels of IL-10 were significantly higher in GC patients compared to controls ($p < 0.001$), while IL-6 levels showed no significant difference. A significant decrease was observed in both cytokine levels after tumor resection ($p < 0.001$). Furthermore, a significant positive correlation was found between *H. pylori* infection burden and levels of both IL-6 ($r = 0.445$, $p = 0.007$) and IL-10 ($r= 0.379$, $p = 0.025$).

Conclusion: The significant elevation of IL-10 in GC patients and its notable decrease post-surgery highlights its potential role as a diagnostic biomarker. The strong correlation between *H. pylori* burden and cytokine levels underscores the interplay between bacterial infection and host inflammatory response in GC.

KEYWORDS Gastric cancer (GC), interleukin-10, interleukin 6, H. pylori

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INTRODUCTION

Cancer pathogenesis often arises from the complex interplay between environmental exposures and genetic susceptibility. Among environmental factors, chronic inflammation and infections are well-established risk factors for Gastric Cancer (GC) (1–3). Despite advances in oncology, GC remains a significant global health challenge, largely due to the fact that a majority of cases are diagnosed at advanced stages, resulting in high mortality rates (4–7). Despite a worldwide decline in incidence, this cancer is the fourth most common cancer and the second most frequent cause of death from cancer worldwide (6). Infection of the gastric mucosa with Helicobacter pylori is one of the main etiological factors of distal GC (8). The variation in clinical outcomes among *H. pylori* infected individuals may be partly attributed to differences in the host inflammatory responses. Consequently, the early identification of infection-induced molecular alterations is critical for discovering potential biomarkers for early GC detection, which would enable timely intervention and improve patient survival (9).

Inflammation is a hallmark of cancer that plays a central role in tumor progression by promoting cellular proliferation, angiogenesis, metastasis, and resistance to therapy (10). Key features of cancer-associated inflammation include the infiltration of leukocytes, sustained cytokine production, tissue remodeling, and angiogenesis. The interaction between *H. pylori* and the gastric epithelium directly induces the production of pro-inflammatory interleukins, such as IL-8, IL-6, and IL-1β (11). Cytokines, which are glycosylated polypeptides secreted by various immune cells, are pivotal regulators of the immune response and are broadly categorized as either pro-inflammatory or anti-inflammatory (12). In the context of cancer, different cytokines contribute variably to tumorigenesis and progression, while some promote tumor development and metastasis, others exert anti-tumor effects that may inhibit disease progression (13).

Interleukin-6 (IL-6) is an important cytokine involved in regulating immune responses, hematopoiesis, and acute-phase reactions, playing a key role in host defense mechanisms (9, 14). The human IL-6 gene is located on chromosome 7p21, and its expression is inducible by various stimuli, including cytokines, lipopolysaccharides, and viral infections, rather than being constitutively expressed by normal cells. In contrast, IL-10 is a potent anti-inflammatory cytokine that modulates immune responses by inhibiting the synthesis of pro-inflammatory cytokines and suppressing antigen-presenting functions in macrophages and T cells (15).

Due to their central roles in immune regulation, both IL-6 and IL-10 have been implicated as diagnostic or prognostic biomarkers in several solid tumors (16, 17). *H. pylori* infection and genetic predisposition are recognized as major risk factors for gastric carcinogenesis; beyond their independent effects, they may also interact synergistically in common pathways that drive the carcinogenic process (18, 19).

Therefore, this study aimed to compare serum levels of interleukins 6 and 10 in patients with GC before and after surgical resection and to evaluate their correlation with *H. pylori* infection status.

MATERIALS AND METHODS

2.1. Study Design and Population

This case-control study was conducted between June 2020 and June 2021 at Shahid Beheshti and Besat hospitals in Hamadan, Iran. The study population included 35 patients with histopathology confirmed GC and 23 healthy controls. Controls were matched to cases based on relevant demographic factors and confirmed to be GC-free through clinical and pathological evaluation. Exclusion criteria for all participants included; intraoperative mortality, diagnosis of autoimmune diseases or diabetes, recent use (within two weeks) of anti-secretory agents, antimicrobials (including antibiotics or bismuth compounds), or nonsteroidal anti-inflammatory drugs (NSAIDs). Additionally, patients who failed to return for postoperative follow-up testing were excluded from final analysis. Individuals with a history of anti-Helicobacter pylori treatment were specifically excluded from the control group.

Sample collection and laboratory analysis

Peripheral blood samples were obtained from GC patients preoperatively and again three weeks post-gastrectomy. Control subjects provided a single blood sample at enrollment. All samples were collected using standard venipuncture techniques and processed by centrifugation to isolate serum. Serum aliquots were stored at -21°C until subsequent analysis.

Serum concentrations of IL-6 and IL-10 were quantified using commercial sandwich ELISA kits (BT Lab Company, INDIA) according to the manufacturer's protocol. Briefly, standards were prepared by serial dilution to generate a standard curve. Each well received 40 µL of serum sample, 10 µL of biotin-conjugated detection antibody, and 50 µL of streptavidin-HRP solution. After 60-minute incubation at 37°C and washing, 50 µL each of chromogen substrates A and B were added. The plate was incubated for 10 minutes at 37°C in darkness before terminating the reaction with 50 µL stop solution. Optical density was measured at 450 nm using a TECAN microplate reader.

C-reactive protein (CRP) levels in serum of patients and controls were measured using a qualitative/semiquantitative kit (Anisan Company, Iran) by latex agglutination method according to a standard protocol. Briefly, one drop (50 µL) of serum samples, positive control, and negative control were mixed with one drop (50 µL) of latex reagent on three separate parts of a glass slide, shaken manually for exactly two minutes, and examined for visible agglutination, which indicated a positive result (CRP > 6 mg/L). Results were recorded as; coarse agglutinated particles with a clear background (positive; 3+), medium agglutinated particles with a semi-opaque background (positive; 2+), small agglutinated particles with a opaque background (positive; 1+), and uniform suspension and absence of agglutination (negative). To reduce the risk of false negative results due to the prozone effect, all non-reactive samples were retested reflexively at a 1:10 dilution in 0.9% saline.

Helicobacter pylori detection

Helicobacter pylori infection status was determined through histopathological examination of gastric biopsy specimens. During the initial diagnostic workup, multiple biopsy samples were obtained from standard locations via gastroscopy prior to any surgical intervention. For patients undergoing gastrectomy, follow-up biopsy specimens were collected three weeks post-operatively during surveillance endoscopy to assess eradication status.

All tissue specimens were fixed in 10% neutral buffered formalin, processed through standard histological protocols, and embedded in paraffin. Sections of 4-5 µm thickness were cut and stained with modified Giemsa stain according to established laboratory protocols. The stained sections were examined independently by two experienced gastrointestinal pathologists who were blinded to the clinical data. The presence of characteristic curved or spiral-shaped bacteria along the gastric epithelial surface or within the gastric pits was considered positive for H. pylori infection. In cases of discrepant readings between pathologists, the slides were reviewed jointly to reach a consensus diagnosis. This method provided both diagnostic confirmation and information regarding bacterial density and distribution within the gastric mucosa.

Statistical Analysis

Categorical data are presented as frequencies and percentages. The normality of continuous variables was determined using the Kolmogorov-Smirnov test. As cytokine levels were not normally distributed, the non-parametric Wilcoxon signed-rank test was employed to compare pre- and post-operative IL-6 and IL-10 levels within the patient group. The Mann-Whitney U test was used for comparisons of continuous variables between the patient and control groups. Categorical variables, including *H. pylori* infection status, were compared using the Chi-square test or Fisher's exact test, as appropriate. Correlations between interleukin levels and *H. pylori* infection were assessed using Spearman's rank correlation coefficient. A two-tailed p-value of less than 0.05 was considered statistically significant for all analyses, which were performed with a 95% confidence level.

RESULTS

3.1. Demographic of participant

A total of 58 participants were enrolled in this study, comprising 35 patients with histologically confirmed GC and 23 healthy controls. The GC group consisted of 26 males (74.3%) and 9 females (25.7%), while the control group included 16 males (69.6%) and 7 females (30.4%). No significant difference was observed in gender distribution between the two groups (P = 0.694).

Cytokine levels, CRP and *Helicobacter pylori* infection in GC patients and healthy controls

Analysis of serum cytokine levels in GC patients revealed significant changes following surgical intervention. The mean serum IL-6 level decreased from 21.15 ± 24.35 pg/mL preoperatively to 16.04 ± 29.72 pg/mL postoperatively (P < 0.001). Similarly, the mean serum IL-10 level declined from 22.76 ± 51.93 pg/mL to 18.64 ± 44.00 pg/mL after surgery (P <

Table 1. Serum levels of IL-6 and IL-10 in GC patients before and after surgery

Items		Before surgery	After surgery	P-value
IL-6		21.15±24.35	16.04±29.72	<0.001
IL-10		22.76±51.93	18.64±44.00	<0.001
IL-6	Normal	16	26	<0.001
	Abnormal	19	9	
IL-10	Normal	2	12	0.044
	Abnormal	33	23	

Table 2. Distribution of interleukin levels, H. pylori infection, and CRP status in GC patients and controls

Items		Cancer	Control	P-value
IL-6	Normal	16 (45.7%)	12 (52.2%)	0.630
	Abnormal	19 (54.3%)	11(47.8%)	
IL-10	Normal	2 (5.7%)	16 (69.6%)	<0.001
	Abnormal	33 (94.3%)	7 (30.4%)	
HP		28.83±35.57	17.96±27.03	0.488
CRP	Negative	26 (74.3%)	20 (87.0)	0.238
	+	2 (5.7%)	1 (4.3%)	
	++	4 (11.4%)	1 (4.3%)	
	+++	2 (5.7%)	1 (4.3%)	
	++++	1 (2.9%)	0 (0.0%)	

0.001). The frequency of abnormal IL levels also showed significant normalization postoperatively for both IL-6 and IL-10 ($P < 0.05$), with the number of patients exhibiting normal IL-6 levels increasing from 16 to 26, and normal IL-10 levels increasing from 2 to 12 (Table 1).

Comparative analysis between preoperative GC patients and healthy controls demonstrated no significant difference in the frequency of abnormal IL-6 levels (54.3% vs. 47.8%, $P = 0.630$). However, abnormal IL-10 levels were significantly more prevalent in the GC group compared to controls (94.3% vs. 30.4%, $P < 0.001$) (Table 2).

The mean histopathological burden of H. pylori infection was higher in the GC group (28.83 ± 35.57) compared to controls (17.96 ± 27.03), though this difference did not reach statistical significance ($P = 0.488$). Similarly, CRP positivity rates showed no significant difference between GC patients (25.7%) and controls (13.0%) ($P = 0.238$). Notably, significant positive correlations were observed between H. pylori infection burden and both serum IL-10 ($r = 0.379$, $P = 0.025$) and IL-6 levels ($r = 0.445$, $P = 0.007$), suggesting a potential role for H. pylori in driving the inflammatory response in GC patients (Table 2).

DISCUSSION

The present study provides compelling evidence for the involvement of specific inflammatory cytokines in the pathogenesis of GC, with particular emphasis on IL-10 as a potential biomarker. Our findings demonstrate a significant elevation of serum IL-10 levels in GC patients compared to healthy controls, along with a substantial decrease in both IL-6 and IL-10 levels following surgical resection. These observations align with the growing body of literature supporting the crucial role of chronic inflammation in gastric carcinogenesis (3, 20).

The marked reduction in cytokine levels post-gastrectomy suggests that the tumor microenvironment serves as a significant source of these inflammatory mediators. This finding is consistent with studies by Ikeguchi M et al (16), who reported similar decreases in pro-inflammatory cytokines following tumor resection. The more pronounced association of IL-10 with GC, compared to IL-6, highlights the potential diagnostic utility of this anti-inflammatory cytokine in GC detection and monitoring. This observation supports the work of Szaflarska et al. (21), who identified IL-10 as a promising biomarker in gastrointestinal malignancies.

Notably, our study revealed significant positive correlations between H. pylori infection burden and both IL-6 and IL-10 levels, reinforcing the established link between H. pylori infection and GC development through inflammatory pathways (22, 23). The correlation between H. pylori and IL-10 is particularly intriguing, as it suggests a potential mechanism by which H. pylori may modulate the host immune response to facilitate persistent infection and promote carcinogenesis (24). This finding extends previous research by Pachathundikandi et al. (25), who demonstrated H. pylori-induced cytokine production in gastric epithelial cells.

While our study did not find statistically significant differences in H. pylori burden between cases and controls, the observed positive correlations with cytokine levels nevertheless support the concept that H. pylori contribute to gastric carcinogenesis through inflammation-mediated mechanisms (3). This aligns with the comprehensive model proposed by Polk and Peek (26), wherein H. pylori-induced chronic inflammation creates a microenvironment conducive to neoplastic transformation.

The lack of significant difference in IL-6 levels between GC patients and controls, despite the observed postoperative decrease, suggests that IL-6 may play a more complex role in GC progression. This finding partially contradicts some previous studies (27, 28) but may reflect the heterogeneous nature of GC or variations in disease stage among study participants.

In conclusion, our findings strengthen the evidence for IL-10 as a particularly relevant biomarker in GC, while also supporting the role of H. pylori infection in modulating the inflammatory

microenvironment. The significant postoperative decrease in both IL-6 and IL-10 levels underscores the contribution of the tumor microenvironment to systemic inflammation. These results contribute to the growing understanding of inflammation-mediated gastric carcinogenesis and highlight potential targets for future diagnostic and therapeutic strategies.

Limitations and Future Directions

Several limitations of our study warrant consideration. The relatively small sample size may have limited our ability to detect subtle differences in H. pylori burden and CRP levels between groups. Additionally, the single-center design and specific population characteristics may affect the generalizability of our findings. Future research should focus on larger, multi-center prospective studies incorporating genetic analyses of cytokine polymorphisms. Longitudinal monitoring of cytokine levels throughout disease progression and treatment could provide valuable insights into their dynamic changes and potential utility in treatment response assessment.

Funding

The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

Conflict of interest

The authors have no relevant financial or non-financial interests to disclose. The authors declare no conflict of interests.

Data Availability

The datasets generated during and/or analyzed during the current study are not publicly available due to privacy but are available from the corresponding author on reasonable request.

Ethics Approval

The study protocol was approved by the Ethics Committee of Hamadan University of Medical Sciences (Ethical Code: IR.UMSHA.REC.1398.812). Written informed consent was obtained from all participants prior to their enrollment in the study. A standardized checklist was used to systematically record clinical and pathological data for all study subjects.

Authors' Contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Yasser Hosseinzade and Hamidreza Ghadimipour. The first draft of the manuscript was written by Yasser Hosseinzade and Mohammadjavad Babaei and Arash Dehghan commented on previous versions of the manuscript. Arash Dehghan read and approved the final manuscript.

Acknowledgments

The authors would like to thank the staff of Be'sat Hospital and the Department of Pathology at Hamadan University of Medical Sciences for their valuable support and assistance during data collection and laboratory procedures.

Declaration of AI Use

The authors declare that no generative AI or AI-assisted technologies were used in the writing process of this manuscript.

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