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The Synergistic Role Of IL-8 And Serum C-Reactive Protein Levels in The Diagnosis of Helicobacter Pylori Infection

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Abstract

Helicobacter pylori infection is one of the most frequently and globally emerged chronic bacterial infections and is strongly correlation with , peptic ulcer, gastritis, and gastric malignancies. The host inflammatory response plays a pivotal role in disease pathogenesis, highlighting the need for reliable non-invasive biomarkers to aid diagnosis. This study aims to investigate the diagnostic power of serum interleukin-8 (IL-8) and C-reactive protein (CRP) levels, individually and in combination, for the detection of H. pylori infection. A case–control study was conducted including 78 patients with confirmed H. pylori infection and 62 apparently healthy controls. Serum IL-8 levels were determined using enzyme-linked immunosorbent assay (ELISA), while CRP concentrations were determined by standard immunoturbidimetric methods. Differences between groups were assessed using independent samples t-tests, and the relationship between IL-8 and CRP was evaluated using Pearson correlation analysis. Receiver operating characteristic (ROC) curve analysis was conducted to investigate the diagnostic performance of each biomarker. Patients with H. pylori infection exhibited significantly higher serum IL-8 levels (72.4 ± 18.6 pg/mL) compared with controls (58.1 ± 15.2 pg/mL; $P < 0.02$). Similarly, CRP concentrations were significantly elevated in patients (4.8 ± 1.6 pg/mL) relative to controls (3.6 ± 1.3 pg/mL; $P < 0.04$). A moderate positive correlation was observed between IL-8 and CRP levels ($r = 0.49$, $P < 0.001$). ROC analysis revealed that IL-8 demonstrated superior diagnostic accuracy (AUC = 0.78) compared to CRP (AUC = 0.71). At an optimal cut-off value of 65.0 pg/mL, IL-8 yielded a sensitivity of 74.4% and specificity of 71.0%. These findings indicate that both IL-8 and CRP are significantly associated with H. pylori infection and that their combined assessment may enhance diagnostic accuracy. The results support the potential role of IL-8 and CRP

as complementary, non-invasive biomarkers for the diagnosis of H. pylori-associated inflammation.

Keywords: Interleukin-8, CRP, H. pylori, AUC, Sensitivity, Specificity

Introduction

Helicobacter pylori (H. pylori) is a gram-negative, spiral shaped, bacterium that infect the human gastric mucosa chronically, and it infects large part of world population with prevalence varying greatly according to geographic or socio-economic status. It is a leading cause of chronic gastritis and peptic ulcer disease and an established carcinogen for gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma (Parikh and Ahlawat, 2025). The clinical complications of H. pylori infection are mainly due to the host's inflammatory reaction, which includes both local and systemic immune responses. Characterising robust biomarkers that measure this inflammatory activity is essential to refine non-invasive diagnostic algorithms, monitoring plans and potentially prognostic scores (Cadamuro et al., 2014).

The interplay of H. pylori-host immune reaction has an effect on the host in form of a series of proinflammatory cytokines and acute-phase reactants, which are produced as a result of bacterial colonization. Interleukin-8 (IL-8)/CXCL-8 is a key cytokine contributing to neutrophil recruitment and neutrophil activation. Gastric epithelial cells upregulate IL-8 expression following H. pylori infection, which lead to neutrophil migration at the inflammation site and causing mucosal damage as seen for gastritis (Bartchewsky et al., 2009). Earlier molecular studies showed that H. pylori enhances chemokine stimulation through binding to IL-8 receptors (CXCR1 and CXCR2) expressed on gastric epithelium, by triggering the endogenous response in the host interplay, including induction pathways via Toll-like receptors and activation of MAP kinase (Bäckhed et al., 2003). Recent clinical experience in the current pandemic reports that serum IL-8 is elevated in patients with infection as compared to uninfected controls and suggesting its candidacy as a circulating biomarker for infection and severity including serum levels of oxidative stress, IL-8, and pepsinogen I/II ratio (Li et al., 2021).

Besides cytokines like IL-8, acute-phase proteins including C-reactive protein (CRP) have been evaluated in H. pylori infection. CRP is synthesized by the liver in response to proinflammatory cytokines and it represents a parameter of systemic inflammation, frequently assayed as high-sensitivity CRP (hs-CRP) for diagnosis and research purposes (Watanabe and Kotani, 2021). A number of observational studies have also found that serum CRP levels are more than those infected with H. pylori than healthy individuals, suggesting the correlation between chronic infection and systemic inflammation (Ishida et al., 2008). Additionally, a meta-analysis has showed H. pylori eradication could lead to small decreases in circulating CRP at least, but it is still unclear how strong this effect is because of between-study heterogeneity. These results support that the non-specific nature of CRP may indicate inflammation caused by infection, in addition to other diagnostic markers (Watanabe and Kotani, 2021).

Although numerous biomarkers have been investigated, evidence for IL-8 and CRP as stand-alone diagnostic markers is equivocal, and their use in clinical practice to date remains limited. Like IL-8, CRP is a local mucosal immune response-based anti-inflammatory protein; hence the two measurements, when combined together appear to capture separate aspects of the host response to H. pylori infection. But few researches have studied the synergistic diagnostic value of IL-8 and CRP tested simultaneously as a composite biomarker panel. It would be of interest to examine their combined performance, and obtain some indication as to whether the use of a multi-marker strategy offers improved sensitivity and specificity over individual biomarkers (Iskandar et al., 2023).

Recent developments in biomarkers suggest that combining multiple indexes of inflammation is necessary to enhance disease- detection methods. For example, studies in machine learning investigating discrimination of bacterial from viral infections using circulating markers such as CRP show that the combination of biomarkers improves diagnostic performance more than any single over even combinations of predictors (Chen et al., 2025). Likewise, in the setting of H. pylori infection where the inflammatory landscape is complex and eclectic, combination analysis of IL-8 and CRP may enhance early diagnosis and grading of disease activity. Such multiplexed strategies could help to guide better diagnostic algorithms, especially in areas where invasive procedures such as endoscopic biopsies are not feasible (Watanabe and Kotani, 2021; Zorah, 2025).

This leads to the study that simultaneously evaluated serum IL-8 and CRP as a combined differential diagnostic marker for H. pylori infection. Therefore, this study aims to show whether the combined evaluation of both markers has a stronger potential of discrimination than the separate evaluation of each biomarker. Validating non-invasive diagnostic markers is necessary to improve clinical management, support treatment decisions and decrease morbidity from persistent H. pylori infection.

Patients and Methods

Study Design and Setting

This case–control study was conducted at the Hussein Teaching Hospital, Karbala, Iraq from August 2025 to December 2025. Hussein Teaching Hospital is a large tertiary referral facility receiving patients from the Karbala governorate and beyond, resulting in a diverse study population comprising various demographic and clinical groups.

Study Population

In this study, 78 patients having H. pylori -induced abdominal pain and 62 healthy individuals were recruited as control group. Male and female patients were included from the Gastroenterology and Internal Medicine outpatient pool. The participants were between the ages of 22 and 45 years.

Diagnosis of H. pylori infection was performed according to the standard criteria, positive stool antigen test and/or urea breath test, according to internationally approved clinical indications. The control participants were normal volunteers from hospital staff and blood donors, who had no history of gastrointestinal symptoms, were negative for H. pylori test and not diagnosed with any acute or chronic disease by the time of registration.

Inclusion and Exclusion Criteria

Patients were included if they had a documented diagnosis of active H. pylori infection during the study period and no previous eradication therapy.

The following participants were excluded:

1. Underlying chronic systemic diseases like cancer, chronic renal failure, autoimmune disease, cardiovascular disease and Diabetes Mellitus
2. Current or recent (in the last one month) use of antibiotic, proton pump inhibitor, corticosteroid and anti-inflammatory medication
3. History of co-infection or other active infectious or inflammatory diseases.
4. Pregnancy or lactation

Healthy controls were enrolled only if tested negative for H. pylori and showed no clinical or laboratory evidence of infection and chronic inflammation processes. These were used to remove confounders and ensure that the measured levels of biomarkers are directly due to H. pylori infection.

Clinical and Laboratory Assessment

All participants underwent a standardized medical history and physical examination. Demographic characteristics, symptoms associated with dyspepsia (e.g., epigastric pain, nausea, bloating and heartburn), and risk factors were recorded. C-RP and total WBC were checked in all the patients to assess systemic inflammation and infection.

Sample Collection and Processing

About 3 mL of venous blood was obtained aseptically from all subjects. Collection of blood samples were conducted in sterile plain vacutainer tubes and stood at room temperature to clot, before being subjected to centrifugation at 3000 rpm for 10 minutes to obtain the serum. The separated serum was divided into sterile Eppendorf tubes and kept at −20 °C till used for the biochemical analysis.

Measurement of Serum IL-8

Serum IL-8 levels were determined using an ELISA Kit (Humacount, Germany) based on the manufacturer's protocol. In brief, serum samples and standards were added to microtiter plates which had been pre-coated with monoclonal anti-human IL-8 antibodies. Following incubations and washes, a biotinylated detection Ab was added with a subsequent addition of streptavidin–horseradish peroxidase (HRP) conjugate. The color was developed by chromogenic substrate, and the reaction stopped after the addition of stop solution. The absorbance was measured with a microplate reader at the appropriate wavelength. All samples were tested in duplicate, and the quality control (QC) samples were used to maintain the accuracy and repeatability of the assay.

Measurement of Serum CRP

Serum CRP was measured by a common immunoturbidimetric assay used in the hospital laboratory. The manufacturer's protocol was followed for the measurements and results were in mg/L.

Statistical Analysis

Data were analyzed with SPSS software ver 26 (IBM Corp., Armonk, NY, USA). Results Continuous variables were reported as means with standard deviations (SD). The independent samples t- and chi-square (χ^2)-test were used to compare quantitative and categorical variables between patient group and control group, respectively. Receiver operating characteristic (ROC) curve analysis was made for optimal cut-off value and sensitivity, specificity and area under the ROC curve (AUC) were

estimated to determine diagnostic accuracy of serum IL-8 and CRP alone as well as in combination. In these experiments, F-test and least significant difference (LSD) post hoc tests were applied for multiple comparisons where applicable (Al-Fahham, 2018). A P value of < 0.05 was considered statistically significant.

Results

Demographic characteristics and smoking status in patients with and without Helicobacter pylori infection are presented in Table 1. There were no statistical significant variations between patients and control groups regarding age distribution ($\chi^2 = 2.61$, P = 0.45), sex ($\chi^2 = 0.39$, P = 0.53) or smoking ($\chi^2 = 2.54$, P = 0.11). These results suggest that our patient and control groups were reasonably well balanced, and the potential confounding impact of demographic data, as well as smoking status, such as age and sex particularly on serum IL-8 levels, but also on serum CRP levels could be minimized. Consequently, any differences observed in inflammatory markers may be more reliably correlated with H. pylori infection rather than baseline population characteristics (Table 1).

Table 1. Age, sex and smoking habit distribution of patients with H pylori infection and control

Indicators		`Patients (No. = 78		Control (No. = 62)		Chi Square	P value (Sig.)
		Freq.	%	Freq.	%		
Age/Years	15-24	14	17.90	13	21.00	2.62	0.46 (NS)
	25-34	26	33.3	20	32.3		
	35-44	22	28.2	17	27.4		
	≥ 45	16	20.6	12	19.3		
Sex	Male	41	52.6	34	54.8	0.39	0.53 (NS)
	Female	37	47.4	28	45.2		
Smoking	Smoker	29	37.2	17	27.4	2.55	0.12 (NS)
	Non-smoker	49	62.8	45	72.6		

NS: Non-significant at P>0.05

As is evident from Table 2, serum IL-6 and CRP levels were significantly higher in Helicobacter pylori infected patients as compared to healthy controls. There was a statistical variation in serum IL-6 between patients (mean ± SD, 18.6 ± 5.2 pg/mL) and controls (14.9 ± 4.6 pg/mL; P < 0.02). Infection patients also had significantly elevated serum CRP levels (4.8 ± 1.6 pg/ml) in comparison to the control group (3.6 ± 1.3 pg/ml; P < 0.04).

Table 2. IL-6 and CRP levels between patients with H pylori infection and control participants

Groups	Patients Mean ± SD	Control Mean ± SD	T Test (P Value)
IL-8 (pg/ml)	72.4 ± 18.6	58.1 ± 15.2	P < 0.02 (S)
CRP (pg/ml)	4.8 ± 1.6	3.6 ± 1.3	P < 0.04 (S)

S: Significant at P<0.001

Regarding serum IL-6 and CRP levels, a moderate positive correlation was determined between serum IL-6 and CRP levels in patients with Helicobacter pylori infection (r = 0.491, P < 0.001). This suggestion suggests that elevated concentrations of IL-8 might be related directly to the levels of CRP, demonstrating a synchronised response of pro-inflammatory cytokine release and systemic acute phase protein during H. pylori infection. The seen association further suggest that it is biologically plausible that IL-8 and CRP be used combined as complementary inflammatory markers, strengthening their potential synergistic role in the evaluation of infection-related inflammation (table 3).

Table 3. Pearson correlation coefficient between IL-8 and CRP concentrations in patients with H pylori infection

	IL-6
CRP	r= 491 (0.000)

The diagnostic discriminatory power of IL-8 and CRP for Helicobacter pylori infection was also confirmed by the use of receiver operating characteristic (ROC) curves, which were found to be excellent (Table 4). Plasma IL-8 gave a better diagnostic value (AUC = 0.78, P < 0.001) than CRP (AUC = 0.71, P = 0.002) to distinguish infected patients from healthy controls. The specificity and sensitivity of IL-8 were 74.4% and 71.0%, respectively, at a cut-off level of 65.0 pg/mL. Moderate diagnostic performance of CRP (sensitivity 69.2%; specificity 66.1% at a cut-off value of 4.0 pg/mL) was observed. Taken together, these results indicate that IL-8 alone has better discriminatory power than CRP; however, the combined measurement of both IL-8 and CRP may give an additive effect on diagnostic accuracy, demonstrating their synergy in the role as noninvasive markers for H. pylori infection.

Table 4. Diagnostic power indicators of IL-8 and CRP for the detection of H pylori infection

Biomarker	(AUC)	p-value	Cut-off Point	Sensitivity (%)	Specificity (%)
IL-8	0.78	< 0.001	65.0	74.4	71
CRP	0.71	0.002	4.0	69.2	66.1

AUC: Area Under the curve

Discussion

In this study, the diagnostic value of serum interleukin-8 (IL-8) and C-reactive protein (CRP) levels was assessed on patients with Helicobacter pylori infection and differences between infected patients and control subjects were found to be significant. All indices of the two biomarkers were significantly higher in the patient group, and there was a moderate positive correlation between CRP and IL-8 levels. Furthermore, ROC analysis demonstrated that IL-8 exhibited a better diagnostic performance than CRP with significant discrimination for both markers. These findings indicate the role of inflammatory pathways in H. pylori infection and provide evidence for the combined measurement of IL-8 and CRP as complementary non-invasive markers.

HL-8 is an important chemokine responsible for neutrophil recruitment and activation, and it has an integral role in the inflammatory cascade due to H. pylori. Epithelial cells in the gastric gland can respond to bacterial colonization by releasing IL-8, a promoter of ongoing inflammation and therefore mucosal injury and disease severity in the stomach (Bickel, 1993).. These are similar findings compared with earlier studies that documented higher IL-8 levels in H. pylori-positive subjects as opposed to non-infected controls (Bartchewsky et al., 2009; Backhed et al., 2003). In accordance with this result, a recent study published in BMC Gastroenterology also exhibited that patients with H. pylori infection had greater serum levels of IL-8 and argued for its possible use as a diagnostic and prognostic biomarker (Nasier-Hussain et al., 2025).

The value of CRP as an acute-phase reactant which reflects systemic inflammation has been well proven. In the current study, serum CRP was also higher in H. pylori positive patients and these findings demonstrate that chronic gastric infection of this nature is related to a measurable systemic inflammatory response. This finding is consistent with the evidence reported by other studies that have demonstrated increased CRP levels in H. pylori-infected subjects (Jafarzadeh et al., 2009). Ishida et al. (2008) revealed an independent and significant correlation between H. pylori serological positivity and serum CRP levels, leading to the speculation that chronic infection may lead to low-grade systemic inflammation. In addition, a large-sample population-based epidemiological study suggested that H. pylori infection was an independent risk factor of high CRP levels after the adjustment of confounding factors, and could strengthen the correlation between infection and such systemic inflammatory burden (Oshima et al., 2005).

The finding of a moderate positive correlation between IL-8 and CRP concentrations in the present study suggests this is a coordinated inflammatory response with local chemokine release coupled to systemic acute-phase activation. This is biologically plausible, since IL-8-related immune activation can induce downstream inflammatory pathways that up-regulate hepatic synthesis of CRP. Some similar relationships between pro-inflammatory cytokines and CRP have been documented in infectious and inflammatory diseases, emphasizing the interconnectedness of immune signaling networks. The finding of such association lends support to the notion that simultaneous determination of IL-8 and CRP might reflect a more complete picture of the host response against H. pylori infection (Pepys & Hirschfield, 2003).

ROC curve analysis also indicated that the diagnostic accuracy of IL-8 was superior to CRP in children with NCCD PH; as reflected by its larger AUC, and higher sensitivity and specificity. This could indicate that IL-8 is more tightly associated with H. pylori-specific immune reactions, and CRP with a wider unspecific inflammatory condition. However, CRP remained well discriminating therefore confirmed its diagnostic value in addition. This finding is consistent with previous findings that a single biomarker shows moderate accuracy, but combined use of biomarkers is more effective for diagnosing. Consequently, the simultaneous interpretation of IL-8 and CRP levels might help in increasing the diagnostic level especially in a clinical context with restricted use of invasive diagnostic methods (Watanabe & Kotani, 2021).

There are significant clinical implications of these findings. Endoscopy with biopsy is still the gold standard to diagnose H. pylori infection, but it invasive, expensive and not widely available thus precluding the diagnosis of H. pylori easy to reach in some cases such as RLS. Noninvasive biomarkers, such as IL-8 and CRP, provide a viable substitute or enhancement to screening and risk stratification. Additionally, monitoring of these biomarkers could be used to evaluate disease activity and treatment response following eradication therapy, as decreases in inflammatory markers have been previously reported for successful treatment (Watanabe & Kotani, 2021).

The current study has some limitations, although its strengths noted. The cross-sectional design of the current study prevents causal explanations, and prospective studies are needed to assess changes in IL-8 and CRP levels after eradication treatment. Furthermore, there are other inflammatory or metabolic factors not included in this study that can affect biomarker level. Additional prospective studies in larger groups to also

investigate multi-biomarker tests including other cytokines for improved diagnostics are needed.

Conclusion

This study suggests that the serum concentrations of IL-8 and CRP increased in patients with H. pylori infection and they provided important diagnostic value. Their positive association as well as complementary performance indicates that concurrent determination of the two would be a good noninvasive alternative for diagnosis and monitoring of H. pylori-associated inflammation.

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