

Immunometabolic dysregulation in gastric ulcer: Association of pro- and anti-inflammatory cytokines, trace elements, and atherogenic lipid profile

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Abstract

Gastric ulcer remains a prevalent gastrointestinal disorder characterized by disruption of the gastric mucosal barrier due to an imbalance between injurious and protective factors. Recent attention has focused on the role of inflammatory mediators, micronutrient status, and metabolic alterations in the progression of this condition. Cytokines are known to promote inflammatory cascades and mucosal damage and modulates immune responses during gastric injury. Also, trace elements including zinc and copper are essential for maintaining antioxidant defense, immune function, and tissue repair. This case–control study included 60 patients diagnosed with gastric ulcer and 60 apparently healthy individuals as normal controls. All participants were recruited from the Gastroenterology and Hepatology Hospital in Al-Najaf. The two groups were matched in terms of age (20–40 years) and body mass index to minimize potential confounding effects. Serum levels of IL-1 β , IL-10, zinc, copper, and lipid profile parameters were assessed using standard laboratory methods. Statistical analysis was carried out using SPSS version 26. Due to non-normal distribution of variables, median values were used for data representation. Spearman's rank correlation test was applied to evaluate associations among variables, and the receiver operating characteristic curve analysis was used to determine the diagnostic performance of the studied parameters. The findings revealed a significant elevation in both IL-1 β and IL-10 levels among gastric ulcer patients compared to controls, indicating activation of inflammatory and regulatory immune pathways. Zinc levels were significantly reduced in patients, suggesting a possible link between micronutrient depletion and impaired mucosal defense. The present study demonstrated a combined pattern of inflammatory activation, zinc deficiency, and lipid profile disturbance in gastric ulcer patients. These alterations may collectively contribute to disease pathogenesis and could serve as useful indicators for disease assessment and monitoring.

Keywords: IL-10, IL-1 β , Dyslipidemia, Gastric mucosa, *H. pylori*.

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Introduction

Gastric ulcer is a chronic gastrointestinal disorder characterized by a disruption of the gastric mucosal integrity resulting from an imbalance between aggressive factors, such as gastric acid secretion, *Helicobacter pylori* infection, oxidative stress, non-steroidal anti-inflammatory drugs, and protective mechanisms including mucus-bicarbonate barrier, mucosal blood flow, and epithelial regeneration mechanisms.^{1,2}

In recent years, increasing evidence suggested that gastric ulcer is not merely a localized gastric lesion but a systemic inflammatory condition involving complex immune and metabolic interactions. Pro-inflammatory cytokines, particularly interleukin-1 beta (IL-1 β), plays a central role in gastric mucosal injury by promoting leukocyte recruitment, enhancing inflammatory signaling pathways, and suppressing gastric acid regulation, which further exacerbates mucosal damage.^{3,4} Conversely, interleukin-10 (IL-10) is a key anti-inflammatory cytokine that maintains immune homeostasis by inhibiting excessive inflammatory responses. A reduction in IL-10 was associated with uncontrolled inflammation and delayed ulcer healing.⁵

Trace elements such as zinc and copper are essential micronutrients involved in antioxidant defense systems, enzymatic activity, and tissue repair processes. Zinc is particularly important for epithelial regeneration, immune modulation, and protection against oxidative stress-induced damage. Several studies have reported that zinc deficiency is associated with increased susceptibility to gastrointestinal inflammation and impaired mucosal healing.^{6,7} Copper, on the other hand, participates in redox reactions and antioxidant enzyme systems; however, its role in gastric ulcer remains less consistent across studies.⁷

Furthermore, disturbances in lipid metabolism have been increasingly recognized in chronic inflammatory diseases. Dyslipidemia, characterized by elevated total cholesterol, triglycerides, and low-density lipoprotein (LDL) together with reduced high-density lipoprotein (HDL), may contribute to oxidative stress and

endothelial dysfunction, thereby enhancing inflammatory injury in gastrointestinal tissues.^{8,9} This metabolic-inflammatory interaction suggested a bidirectional relationship between lipid abnormalities and gastric mucosal damage.

Despite growing evidence on individual roles of cytokines, trace elements, and lipid metabolism in gastric pathology, limited studies have simultaneously evaluated these parameters in a single clinical model using non-parametric statistical approaches. Therefore, the present study was designed to investigate the alterations in IL-1 β , IL-10, zinc, copper, and lipid profile parameters in patients with gastric ulcer compared with healthy controls using median-based statistical analysis.

Subjects and Methods

This study was designed as a case-control clinical investigation conducted to evaluate inflammatory cytokines, trace elements, and lipid profile parameters in patients with gastric ulcer compared with normal control individuals.

The study enrolled 60 patients diagnosed with gastric ulcer and 60 apparently healthy normal controls. All participants were recruited from the Gastroenterology Unit of Al-Sadr Medical City Hospital in Najaf, Iraq. The control group was matched with patients in terms of age and body mass index (BMI) to reduce potential confounding effects. The age of participants ranged between 20–40 years, and BMI values were relatively comparable between both groups.

Venous blood samples were collected from all participants under aseptic conditions after an overnight fasting period. Serum was separated by centrifugation and stored appropriately until biochemical analysis. Serum levels of IL-1 β and IL-10 were measured using commercial sandwich enzyme-linked immunosorbent assay (ELISA) kits supplied by Elabscience Biotechnology Inc. (Houston, Texas, USA), according to the manufacturer's instructions. This method allows high sensitivity and specificity for cytokine quantification.

While serum concentrations of zinc and copper, in addition to lipid profile parameters

including triglycerides (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C), were determined using commercial enzymatic colorimetric assay kits (Spinreact, Girona, Spain) according to the manufacturer's instructions. All photometric absorbance measurements were performed using a spectrophotometer (BioSystems S.A., Barcelona, Spain). Low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) levels were calculated indirectly using established mathematical formulas based on the measured lipid parameters. VLDL-C was estimated using the formula: $VLDL-C = TG/5$; LDL-C was calculated using the Friedewald formula: $LDL-C = Total\ Cholesterol\ (TC) - HDL-C - VLDL-C$.

Statistical Analysis

This was performed using the Statistical Package for the Social Sciences (SPSS) version 26. Due to non-normal distribution of data, results were expressed as median and interquartile range (IQR). The Mann–Whitney U test was used to

compare differences between groups. Spearman's correlation coefficient was applied to assess relationships between studied parameters. In addition, Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the sensitivity and specificity of the investigated biomarkers in distinguishing patients from controls. A p-value of less than 0.05 was considered statistically significant.

Results

Table 1 show no significant change in either age or body mass index between patients and normal control individuals.

The present study demonstrated that there were no statistically significant differences in age or body mass index (BMI) between patients with gastric ulcer and healthy controls. This indicates appropriate matching between groups, which reduces the influence of confounding demographic variables on the biochemical outcomes.

Table 1. Demographic and clinical characteristics of the study participants.

Characteristic	Patients (n=60)	Controls (n=60)	p-value
	Mean $\pm$ SD	Mean $\pm$ SD	
Age (years)	30.42 $\pm$ 9.59	29.59 $\pm$ 9.59	NS
BMI (kg/m ²)	22.64 $\pm$ 3.60	22.70 $\pm$ 3.39	NS

p > 0.05 is not significant (NS).

The results in Table 2 demonstrated a significant elevation of both IL-1 β and IL-10 in patients with gastric ulcer compared with normal controls. This cytokine pattern suggested the coexistence of an active inflammatory response alongside a compensatory anti-inflammatory regulatory mechanism.

In addition, this study demonstrated a significant reduction in serum zinc levels in patients with gastric ulcer, whereas copper levels did not show a significant decrease compared with normal controls. In contrast, copper levels did not show a significant change,

suggesting that copper homeostasis is less affected in gastric ulcer or may be regulated through more stable physiological buffering mechanisms.

On the other hand, the present study revealed a distinct dyslipidemic pattern in patients with gastric ulcer characterized by significant elevation of triglycerides, total cholesterol, LDL, and very low-density lipoprotein (VLDL), accompanied by a significant reduction in high-density lipoprotein (HDL) compared with normal controls.

Table 2. Concentration of interleukin-1 and interleukin-10, zinc, copper, and lipid profile in patients with peptic ulcer compared to the control group.

Parameter	Patients	Controls	p-value
	Median	Median	
IL-10 pg/ml	58.83 (56.95-59.80)	23.83 (21.9-24.80)	<0.001
IL-1B pg/ml	45.90 (43.77-47.17)	20.99 (18.77-22.17)	<0.001
Zn µg/dl	109.50 (79.00-136.00)	159.5 (129.00-186.00)	<0.001
Cu µg/dl	63.50 (45.50-79.25)	58.50 (40.50-74.25)	NS
T.G mg/dl	76.50 (65.00-82.00)	65.00 (61.25-75.00)	<0.001
Cholesterol mg/dl	177.50 (167.00-187.62)	170.00 (158.32-184.72)	NS
LDL mg/dl	128.00 (119.0-139.30)	120.40 (107.55-130.85)	0.007
VLDL mg/dl	15.30 (13.00-16.40)	13.00 (12.25-15.00)	<0.001
HDL mg/dl	33.05 (29.02-37.00)	36.90 (33.02-41.125)	0.002

Zn: zinc, Cu: copper, T.G: triglycerides, LDL: Low-density lipoprotein, VLDL: very low-density lipoprotein, HDL: high-density lipoprotein. $p > 0.05$ is not significant (NS).

Data in Table 3 indicated a significant positive correlation between interleukin 1B and interleukin-10, a non-significant negative correlation between interleukin-10 and copper, and a non-significant negative correlation between copper and zinc.

In contrast, Copper plays a more stable role in redox homeostasis through ceruloplasmin-dependent pathways and may not exhibit strong direct correlation with cytokine fluctuations in acute inflammatory settings.

Table 3. Correlation coefficient between interleukins and trace elements.

		IL10	IL1B	Zn	Cu
Spearman's rho	IL-10	Correlation Coefficient	1.000	0.367**	0.105
		Sig. (2-tailed)	.	$p < 0.004$	NS
		N	60	60	60
	IL-1B	Correlation Coefficient	0.367**	1.000	0.028
		Sig. (2-tailed)	$p < 0.004$	.	NS
		N	60	60	60
	Zn ²	Correlation Coefficient	0.105	0.028	1.000
		Sig. (2-tailed)	NS	NS	.
		N	60	60	60
	Cu ²	Correlation Coefficient	-0.090-	0.009	-0.012-
		Sig. (2-tailed)	NS	NS	NS
		N	60	60	60

** Correlation is significant at the $p < 0.01$ level (2-tailed). not significant (NS).

The area under the curve (AUC) for IL-10 was 0.919, indicating an "excellent" diagnostic accuracy. At a cut-off value of 33.87 pg/ml, IL-10 achieved a balanced sensitivity and specificity of 90%. Similarly, IL-1 β exhibited diagnostic power with an AUC of 0.88. At a threshold of 30.04

pg/ml, it also yielded a sensitivity and specificity of 90%. Both parameters reached high statistical significance ($p < 0.001$), confirming their reliability as potential non-invasive diagnostic tools, (Table 4, figure 1).

Table 4. The ROC analysis parameters in patients with Gastric Ulcer.

Parameters	Cut off	Sensitivity %	Specificity%	AUC	95% CI of AUC	<i>p</i> value
IL10 pg/ml	33.87	90	90	0.919	0.864- 0.974	0.001
IL 1 β pg/ml	30.04	90	90	0.88	0.812- 0.948	0.001

$p \leq 0.05$ is significant.

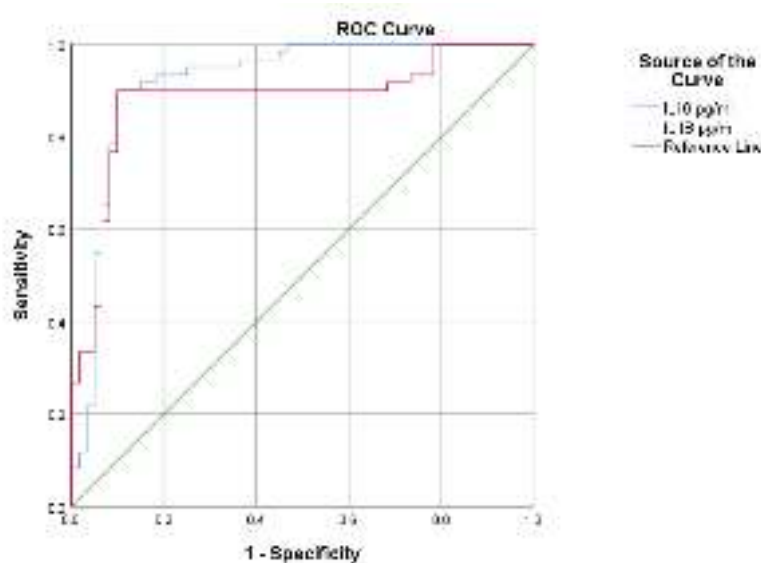


Figure 1. ROC curve comparing IL-10 and IL-1 β in patients with Gastric Ulcer.

Discussion

The age is a key biological factor associated with immune regulation, inflammatory activity, and gastrointestinal physiology. However, the lack of a significant age difference between the studied groups as showed in table 1 suggests that the observed biochemical alterations are not attributable to age-related changes such as immunosenescence or chronic low-grade inflammation (inflammaging).^{10, 11} Therefore, the differences detected in inflammatory and metabolic markers are more likely related to disease-specific mechanisms rather than age variability.

Similarly, BMI is strongly associated with systemic inflammation, metabolic dysregulation, and lipid abnormalities. Increased BMI has been linked to elevated inflammatory cytokines and altered lipid metabolism in various clinical conditions. Nevertheless, the comparable BMI between groups in this study suggests that obesity-related inflammatory activation did not influence the observed results. This supports the interpretation that biochemical differences are independent of body composition and reflect underlying disease pathology rather than metabolic status.^{12, 13} Overall, the absence of significant differences in age and BMI strengthens the validity of the study findings

and ensures that the observed biochemical and inflammatory changes are primarily disease-driven.

So, the significant increase in IL-1 β reflects activation of the innate immune response in gastric mucosal injury. IL-1 β is a key pro-inflammatory cytokine produced via inflammasome activation in macrophages and epithelial cells in response to *H. pylori* infection, oxidative stress, and mucosal damage. It amplifies inflammatory cascades through nuclear factor kappa-light-chain-enhancer of activated B cells (NF- κ B) signaling, leading to increased recruitment of inflammatory cells and further tissue injury. Elevated IL-1 β was consistently associated with gastric inflammation severity and ulcer progression.^{14,15}

Interestingly, the concurrent elevation of IL-10 observed in this study suggests activation of a counter-regulatory anti-inflammatory response. IL-10 is a crucial immunomodulatory cytokine that functions to limit excessive inflammation by inhibiting the synthesis of pro-inflammatory mediators such as IL-1 β , TNF- α , and IL-6. Its increase in gastric ulcer patients may represent a compensatory feedback mechanism aimed at controlling excessive inflammatory activity and preventing further mucosal damage.^{16, 17}

This dual elevation of IL-1 β and IL-10 indicated a dynamic immunological balance rather than a simple pro-inflammatory dominance. In chronic inflammatory conditions such as gastric ulcer, persistent antigenic stimulation can lead to simultaneous activation of inflammatory and regulatory pathways. While IL-1 β drives mucosal injury, IL-10 is up-regulated as a protective response to limit tissue destruction and promote resolution of inflammation. However, in many cases, this compensatory increase in IL-10 may be insufficient to fully counteract the strong pro-inflammatory signaling mediated by IL-1 β .^{15, 17} The interaction between IL-1 β and IL-10 suggests the presence of an active but dysregulated immune response in gastric ulcer, where both inflammatory activation and anti-inflammatory regulation occur simultaneously. This reflects the complexity of cytokine networks in gastric mucosal disease and

highlights that gastric ulcer is not solely a pro-inflammatory condition but also involves endogenous attempts to restore immune homeostasis. Overall, the findings support the concept that gastric ulcer involves a bidirectional immune response characterized by simultaneous up-regulation of inflammatory and regulatory cytokines, reflecting ongoing tissue injury and attempted mucosal protection.

In addition, these findings suggested a selective disruption in trace element homeostasis during gastric ulcer disease, with zinc being more sensitive to inflammatory and oxidative changes than copper. The significant decrease in zinc levels may be explained by its increased utilization during inflammatory stress and oxidative damage. Zinc is a critical trace element involved in maintaining epithelial integrity, antioxidant defense, and immune regulation. In inflammatory conditions, zinc is redistributed from plasma to the liver as part of the acute phase response, leading to reduced circulating levels.⁶ Additionally, zinc deficiency has been shown to enhance NF- κ B activation and promote the production of pro-inflammatory cytokines, including IL-1 β , thereby amplifying inflammatory responses in gastric mucosal injury.^{18, 19} Importantly, the observed elevation of IL-1 β in the present study may be mechanistically linked to zinc depletion. Reduced zinc availability is known to enhance inflammasome activation and increase IL-1 β maturation, which contributes to persistent mucosal inflammation and delayed healing. At the same time, zinc deficiency may suppress anti-inflammatory signaling pathways, indirectly contributing to reduced IL-10 activity and further imbalance of the cytokine network.^{18, 20} Unlike zinc, copper is more tightly bound to transport proteins such as ceruloplasmin, which may maintain serum copper levels within a narrower physiological range even under inflammatory conditions. This could explain the absence of a statistically significant change in copper levels despite ongoing inflammation. The differential behavior of zinc and copper in this study highlights the distinct biological roles of these trace elements in inflammatory diseases. Zinc appears to be more directly involved in immune modulation and

inflammatory regulation, whereas copper may play a secondary or more stable role in redox balance. The interaction between reduced zinc levels and elevated IL-1 β further supports the concept of a zinc–cytokine axis in gastric ulcer pathophysiology, where trace element deficiency contributes to immune dysregulation and sustained inflammatory injury.^{19, 20}

This lipid signature reflects a state of metabolic dysregulation closely linked to systemic inflammatory activation. Recent evidence increasingly supports the concept that lipid metabolism is not merely a passive metabolic process but an active regulator of inflammatory signaling pathways.²² Elevated triglycerides and LDL fractions have been shown to promote immune cell activation, particularly macrophage polarization toward a pro-inflammatory phenotype, leading to increased production of cytokines such as IL-1 β and TNF- α . These lipid-induced inflammatory responses are mediated through oxidative stress pathways and activation of NF- κ B signaling, thereby amplifying systemic and mucosal inflammation.^{21, 22}

In contrast, HDL exerts a protective anti-inflammatory and antioxidant role by inhibiting lipid peroxidation, neutralizing oxidative stress, and modulating immune cell activity. The observed reduction in HDL levels in gastric ulcer patients may therefore contribute to loss of this protective buffering capacity, facilitating uncontrolled inflammatory responses and enhancing susceptibility to mucosal injury.²¹ Importantly, recent studies have demonstrated a bidirectional relationship between lipid abnormalities and gastrointestinal inflammation, suggesting that dyslipidemia may not only be a consequence of chronic inflammatory states but also a contributing factor to disease progression.²² Elevated triglyceride-rich lipoproteins have been associated with increased expression of IL-1 β and other pro-inflammatory mediators in immune cells, indicating a direct lipid–cytokine interaction axis.^{22, 23}

Furthermore, LDL and VLDL particles may undergo oxidative modification under inflammatory conditions, resulting in the formation of oxidized lipoproteins that further

stimulate immune activation and tissue injury. This creates a self-perpetuating cycle in which inflammation alters lipid metabolism, and dyslipidemia in turn amplifies inflammatory signaling, thereby sustaining gastric mucosal damage.²³

This relationship indicates that elevated pro-inflammatory cytokines (such as IL-1 β) trigger a secondary, protective response characterized by an increase in anti-inflammatory cytokines (such as IL-10). Rather than functioning in isolation, the immune system actively up-regulates anti-inflammatory pathways in an attempt to counterbalance the ongoing systemic inflammation and limit tissue damage in these patients. From a mechanistic perspective, IL-1 β acts as an early driver of innate immune activation through inflammasome signaling, leading to amplification of downstream inflammatory cascades. However, sustained IL-1 β activity does not occur in isolation; it triggers feedback regulatory pathways aimed at limiting tissue damage. One of the key counter-regulatory responses is the induction of IL-10 production by monocytes and regulatory T cells. Therefore, the positive correlation between IL-1 β and IL-10 may indicate a proportional compensatory immune response, where stronger inflammatory stimulation is met with stronger anti-inflammatory regulation in an attempt to restore mucosal homeostasis.^{24, 25}

Recent immunological models support this concept of “parallel activation,” where IL-10 is not simply a suppressive endpoint but is actively induced in response to heightened inflammatory signaling. In chronic inflammatory conditions such as gastric mucosal injury, IL-10 elevation often reflects an adaptive response to ongoing IL-1 β -mediated tissue stress rather than a primary anti-inflammatory dominance. This explains why both cytokines may rise concurrently while still indicating active disease processes.²⁴

The correlation analysis in the present study revealed a significant positive association between IL-1 β and IL-10, suggesting a tightly coupled immunoregulatory response in gastric ulcer pathology. In relation to trace elements, zinc and copper showed differential interaction patterns within the inflammatory network. Zinc

is known to exert a modulatory effect on cytokine signaling, particularly by inhibiting NF- κ B activation and reducing IL-1 β transcriptional activity.²⁶ It also supports epithelial repair mechanisms and stabilizes immune responses. Therefore, alterations in zinc levels may indirectly influence the IL-1 β –IL-10 axis by modulating the intensity of inflammatory signaling.²⁶ Its weaker association in the present analysis suggests that copper is less dynamically involved in short-term immune modulation compared with zinc.^{26, 27} The positive correlation between IL-1 β and IL-10 reflects a tightly regulated but activated immune network in gastric ulcer, where inflammatory escalation is counterbalanced by compensatory regulatory signaling. The interaction between cytokines and trace elements further suggests that micronutrient status may modulate the intensity and balance of inflammatory responses in gastric mucosal disease.

The ROC curve analysis revealed that systemic levels of IL-10 and IL-1 β exhibit high discriminative power, suggesting that the clinical manifestations of peptic ulcer disease (PUD) are closely mirrored by a distinct peripheral cytokine signature. The result of IL-10 slightly outperforms IL-1 β is particularly noteworthy. While IL-1 β is a well-characterized pro-inflammatory driver of gastric mucosal damage, IL-10 acts as a critical immunomodulatory. The high AUC of IL-10 suggests that the systemic compensatory anti-inflammatory response is a highly sensitive indicator of active gastroduodenal ulceration. A recent research in 2025 has emphasized that *H. pylori* infection not only triggers inflammation but also exploits the IL-10 axis to modulate the host immune response, often leading to a state of chronic immune activation that is reflected in the serum.²⁸ IL-1 β is a potent inhibitor of gastric acid secretion and a primary mediator of the inflammatory cascade in the gastric niche. The high sensitivity and specificity at the 30.04 pg/ml threshold reinforce the concept that PUD is characterized by a "cytokine storm" at the local level that spills over into the systemic circulation. Recent high-impact studies have utilized ROC analysis to confirm that IL-1 β , along with other members of the IL-1 family, are

among the most reliable biomarkers for predicting gastric disease progression and the severity of *H. pylori*-induced lesions, the parallel high performance of both pro-inflammatory (IL-1 β) and anti-inflammatory (IL-10) markers reflects the complex "immunological tug-of-war" occurring in the gastric mucosa.²⁹ In PUD, the failure of IL-10 to adequately neutralize the damage caused by IL-1 β and other inflammatory mediators leads to ulcer formation. The high AUC values observed in our cohort suggest that the magnitude of this imbalance is substantial enough to be detected with high precision in the serum. This aligns with recent bioinformatics and clinical evidence suggesting that metabolic-inflammatory signatures are superior to traditional markers for the early detection of *H. pylori*-related complications. Furthermore, the strong positive correlation between these cytokines (as shown in Table 3 of the main study) suggests that a combined "cytokine score" might further enhance diagnostic accuracy, a trend currently being explored in cutting-edge gastroenterology research.³⁰

In conclusion, this study demonstrated that gastric ulcer is associated with significant immune, metabolic, and micronutrient alterations. A marked increase in IL-1 β together with an increase in IL-10 reflects an active inflammatory response accompanied by a compensatory anti-inflammatory mechanism. Zinc levels were significantly reduced, while copper showed no significant change, indicating selective trace element imbalance. Lipid profile abnormalities were evident, with increased TC, TG, LDL, and VLDL and decreased HDL, suggesting systemic metabolic dysregulation linked to inflammation. Correlation findings, particularly the positive association between IL-1 β and IL-10, indicated a coordinated immune response involving simultaneous inflammatory activation and regulatory control. Overall, cytokines (especially IL-1 β and IL-10) may serve as early biomarkers of gastric ulcer, reflecting early immune activation before advanced tissue damage occurs.

Author Contributions

FMA, conducted the practical part and recorded the results. BMA and HFSA, contributed to data analysis and manuscript editing. BMA, did the statistical analysis. FMA, manuscript writing, and concept development. All authors reviewed and approved the final draft of the article.

Declaration of Conflicting Interests

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Ethical approval

The protocol of the study was reviewed and approved by the Research Ethics Committee of the Faculty of Medicine, University of Kufa (Ref.: H K/114, dated October 30, 2025).

Informed consent

Before sample collection, an informed consent was obtained from each participant.

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