

Helicobacter pylori infection and acute pancreatitis severity: findings from a retrospective cohort study

 Yunus Halil Polat*,  Başak Çakal

Department of Gastroenterology, Ankara Training and Research Hospital, University of Health Sciences, Ankara, Türkiye

Cite this article as: Polat YH, Çakal B. *Helicobacter pylori* infection and acute pancreatitis severity: findings from a retrospective cohort study. *J Compr Surg*. 2026;4(4):88-93. doi:10.51271/JOCS-0083

Received: 24.09.2026

Accepted: 24.10.2026

Published: 28.10.2026

ABSTRACT

Aims: While *Helicobacter pylori* (HP) is a well-known pathogen in gastroduodenal diseases, its potential role in acute pancreatitis remains unclear. This study aimed to investigate the association between HP infection and disease severity in patients with acute pancreatitis.

Methods: In this retrospective cohort study, 143 adult patients diagnosed with acute pancreatitis between January 2014 and October 2024 were analyzed. HP status was determined via histopathological examination of gastric biopsy specimens. Patients were stratified as HP-positive or HP-negative. Clinical severity was evaluated using the Ranson score and BISAP score. Radiologic severity was assessed using the Computed Tomography Severity Index (CTSI) and the revised Atlanta classification. Laboratory parameters, intensive care unit (ICU) admission, and length of hospital stay were also compared between groups.

Results: Of the 143 patients, 55 (38.5%) were HP-positive and 88 (61.5%) were HP-negative. No statistically significant differences were observed between the two groups in terms of Atlanta classification subtypes ($p=0.256$), clinical severity scores, or key laboratory values. HP positivity was not associated with ICU admission or longer hospitalization. However, in subgroup analyses, HP-positive patients with biliary pancreatitis demonstrated higher clinical severity scores (Ranson and BISAP), while no differences were observed in non-biliary cases.

Conclusion: HP infection was not significantly associated with the overall clinical or radiological severity of acute pancreatitis. However, exploratory subgroup analyses suggested higher clinical severity scores in patients with biliary pancreatitis, which should be interpreted cautiously. These findings indicate that the relationship between HP infection and acute pancreatitis severity may vary depending on etiology and requires further investigation.

Keywords: *Helicobacter pylori*, acute pancreatitis, disease severity, Atlanta classification

INTRODUCTION

Acute pancreatitis is an inflammatory disorder of the pancreas characterized by sudden onset of abdominal pain, elevated pancreatic enzymes, and a variable clinical course ranging from mild, self-limiting inflammation to severe necrotizing disease with systemic complications and organ failure.¹ Acute pancreatitis has a multifactorial etiology, with gallstones (20-70%), alcohol use (up to 30%), and hypertriglyceridemia (approximately 9%) being the most common causes, while less frequent etiologies include drug-induced, autoimmune, and genetic forms of the disease.² *Helicobacter pylori* (HP) is a gram-negative, microaerophilic bacterium that colonizes the gastric mucosa and is well known for its association with chronic gastritis, peptic ulcer disease, and gastric malignancies.³ In recent years, HP has also been implicated in several extragastric conditions, including cardiovascular diseases, metabolic syndrome, and autoimmune disorders.⁴ Recent studies have suggested that HP infection may exert systemic effects beyond the stomach, contributing to extra-gastric inflammatory responses through mechanisms such

as extracellular vesicle release and potential direct effects on pancreatic tissue.^{5,6} These findings support a possible link between HP and pancreatic inflammation, although its clinical relevance remains unclear.

Several pathophysiological mechanisms have been proposed to explain a potential link between HP infection and pancreatic inflammation. These include bacterial translocation, immune-mediated injury, molecular mimicry, and alterations in the gut-pancreas axis.⁴⁻⁹ Recent studies have demonstrated that HP infection can significantly influence gut microbial composition, leading to dysbiosis and altered systemic immune responses, which may contribute to systemic inflammatory amplification.^{10,11} Some studies have demonstrated an increased prevalence of HP infection in patients with chronic or acute pancreatitis, suggesting a possible role in pancreatic inflammation.^{12,13} However, other studies failed to show a significant association, and current evidence remains inconclusive.

*Corresponding Author: Yunus Halil Polat, yunushpolat@gmail.com



Despite these conflicting findings, the role of HP infection in acute pancreatitis severity remains poorly defined. Previous studies have often relied on serological tests, had heterogeneous patient populations, or did not systematically apply validated severity indices. Therefore, the present study aimed to specifically investigate whether histologically confirmed HP infection is associated with the clinical and radiological severity of acute pancreatitis. By employing both clinical (Ranson, BISAP) and radiological (CTSI, revised Atlanta classification) scoring systems, we sought to provide a more robust evaluation and address gaps in the existing literature.

METHODS

Ethics

This study was approved by the Ankara Training and Research Hospital Ethics Committee (Date: 09.10.2024, Decision No: 258/2024). The requirement for informed consent was waived by the ethics committee due to the retrospective nature of the study. All procedures were performed in accordance with the ethical standards of the institutional and/or national research committee and with the Declaration of Helsinki.

Study Design and Patient Selection

This retrospective cohort study was conducted at the Gastroenterology Department of Ankara Training and Research Hospital between January 2014 and October 2024. This study was conducted retrospectively by reviewing existing medical records, and no additional interventions were applied to the patients. Adult patients (≥ 18 years) who were diagnosed with acute pancreatitis and had undergone HP testing were included. The diagnosis of acute pancreatitis was established based on at least two of the following criteria: characteristic abdominal pain, elevated serum amylase and/or lipase levels (≥ 3 times the upper limit of normal), or radiologic findings consistent with pancreatitis. Patients with a history of chronic pancreatitis, active or previous gastrointestinal infections other than HP, or incomplete clinical records were excluded. A total of 160 patients with a diagnosis of acute pancreatitis were initially screened. After excluding 17 patients (7 with missing biopsy data, 6 with incomplete records, and 4 younger than 18 years), 143 patients were included in the final analysis (88 HP-negative and 55 HP-positive). Patient selection and exclusions are summarized in Figure 1 (flow diagram). HP status was determined by histopathological examination of gastric mucosal biopsies obtained during upper gastrointestinal endoscopy. Histopathological evaluation was performed using routine hematoxylin-eosin (H&E) staining, and the presence of HP was determined by experienced pathologists based on characteristic morphological features. No additional special staining methods or immunohistochemical analyses were performed. However, quantitative assessment of bacterial load or grading of colonization density was not performed in this study. In all included patients, gastric biopsies for HP testing were obtained contemporaneously during hospitalization for the index episode of acute pancreatitis. Patients were categorized into HP-positive and HP-negative groups based on the biopsy results. This retrospective design reflects real-world clinical practice and provides insight into hospitalized patients with acute pancreatitis. Upper gastrointestinal endoscopy was performed based on specific clinical indications rather than as a routine procedure in all patients with acute pancreatitis.

These indications included evaluation of suspected upper gastrointestinal pathology (such as dyspepsia, anemia, or gastrointestinal bleeding), assessment prior to endoscopic retrograde cholangiopancreatography (ERCP) in selected biliary cases, and persistent or unexplained abdominal symptoms not fully attributable to pancreatitis.

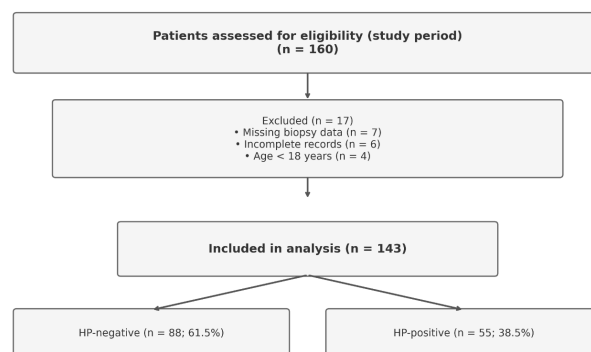


Figure 1. Flowchart of study population selection

Accordingly, the study population consisted of patients who underwent clinically indicated endoscopic evaluation during hospitalization. For subgroup analyses, patients were categorized according to pancreatitis etiology as biliary or non-biliary. The non-biliary group included alcohol-related, hyperlipidemia-associated, post-ERCP, and other less common etiologies.

Clinical and Laboratory Parameters

Demographic characteristics such as age and gender were recorded. Comorbidity burden was assessed using the Charlson Comorbidity Index (CCI). The clinical severity of pancreatitis was assessed using both the Ranson criteria and the BISAP score. Systemic inflammatory response syndrome (SIRS) was evaluated based on heart rate, respiratory rate, body temperature, and leukocyte count. The Computed Tomography Severity Index (CTSI) was used to assess morphologic severity based on contrast-enhanced abdominal CT scans. This scoring system evaluates the presence of pancreatic edema, necrosis, and peripancreatic fluid collections. Additionally, all patients were classified according to the revised Atlanta classification into morphologic subtypes (normal, edematous, necrotizing) and clinical severity categories (mild, moderate, severe). The final severity classification was determined retrospectively using clinical and radiological findings documented throughout the hospitalization period, including the development of local complications and organ failure when present. Clinical outcomes such as length of hospital stay, need for intensive care unit (ICU) admission, and pancreatitis-related complications were documented. Laboratory parameters included serum amylase, lipase, glucose, white blood cell count, hemoglobin, platelet count, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), urea, creatinine, lactate dehydrogenase (LDH), total bilirubin, liver function tests, and triglyceride levels. Inflammatory status was assessed using serum CRP, erythrocyte sedimentation rate (ESR), white blood cell count, and the presence of SIRS.

Statistical Analysis

All data analyses were performed using IBM SPSS Statistics (version 28.0, IBM Corp., Armonk, NY, USA). Normality of continuous variables was evaluated using the Kolmogorov-Smirnov test. Variables with normal distribution are reported

as mean±standard deviation, whereas those not normally distributed are described using median values along with interquartile ranges (IQR: 25th to 75th percentile). Categorical variables were expressed as counts and percentages, and compared using the chi-square test or Fisher's exact test, as appropriate. Categorical data were summarized as frequencies and percentages. For comparisons between two groups (HP positive and HP negative), the independent samples t-test was employed for continuous variables with normal distribution, while the Mann-Whitney U test was used for those not following a normal distribution. Categorical variables were compared using either the Chi-square test or Fisher's exact test, based on the expected cell counts. Specifically, the association between HP status and acute pancreatitis severity was assessed using Chi-square or Fisher's exact tests for categorical severity classifications (revised Atlanta criteria), and Mann-Whitney U tests for continuous severity scores (CTSI, BISAP, Ranson). Subgroup analyses according to pancreatitis etiology (biliary vs. non-biliary) were performed to explore potential confounding effects. In addition, exploratory multivariate logistic regression analysis was considered to adjust for potential confounders such as age, gender, comorbidity burden, and etiology. However, due to the limited number of moderate and severe cases, inclusion of multiple variables in the regression model was not statistically feasible. Therefore, the analysis was restricted to univariate and subgroup comparisons, and this limitation was acknowledged when interpreting the findings. A p-value of <0.05 was considered statistically significant. As this was a retrospective study based on an available dataset, an a priori power analysis was not performed. However, the sample size was determined by the number of eligible patients within the study period. The potential limitation related to statistical power has been acknowledged in the discussion section.

Missing data were handled using available-case analysis, meaning that patients were not excluded from the study due to missing laboratory or imaging values. Analyses were performed using the maximum number of available observations for each variable. The overall proportion of missing data was low (<5% for all variables), and no variable exceeded 10% missingness. Sensitivity analysis comparing complete-case versus available-case results showed no material differences in statistical significance. Therefore, multiple imputation was not deemed necessary. Percentages were calculated using available denominators, and totals may not always equal 100% due to rounding.

RESULTS

A total of 143 patients diagnosed with acute pancreatitis were included in the analysis. Among these, 88 (61.5%) were HP negative and 55 (38.5%) were HP positive. In the present cohort, the most common etiology was biliary origin, accounting for 69.2% (n=99) of cases. Alcohol-related pancreatitis was the second most frequent cause, observed in 17.5% (n=25) of the patients. Hyperlipidemia was identified in 9.8% (n=14) of cases. Additionally, pancreatitis following endoscopic retrograde cholangiopancreatography (ERCP) was reported in 0.7% (n=1), and other rare etiologies constituted 2.8% (n=4).

The demographic and clinical characteristics of the study population are presented in [Table 1](#). There were no statistically significant differences between HP-positive and HP-negative groups in terms of age (p=0.904), gender distribution

(p=0.420), length of hospital stay (p=0.120), CTSI (p=0.773), Ranson score (p=0.333), BISAP score (p=0.120), or ICU admission rate (p=0.414). The CCI tended to be lower in the HP-positive group but did not reach statistical significance (p=0.069). The frequency of pleural effusion and fever also did not differ significantly between groups (p=0.806 and p=0.060, respectively).

There were no statistically significant differences between HP groups in terms of CRP (p=0.132), ESR (p=0.175), white blood cell count (p=0.463), hemoglobin (p=0.330), platelet count (p=0.510), glucose (p=0.367), urea (p=0.947), creatinine (p=0.355), amylase (p=0.818), LDH (p=0.788), AST (p=0.847), ALT (p=0.947), total bilirubin (p=0.853), or triglyceride levels (p=0.381). These parameters were used as markers of systemic inflammation.

The distribution of Atlanta classification subtypes in the study population is presented in [Figure 2](#). In the morphologic categorization, 18.2% of HP-negative patients were classified as normal, 71.6% as edematous, and 10.2% as necrotizing. Among HP-positive patients, 10.9% were classified as normal, 83.6% as edematous, and 5.5% as necrotizing. Regarding clinical severity, HP-negative patients were classified as normal (18.2%), mild (43.2%), moderate (38.6%), and severe (0%), while HP-positive patients were classified as normal (10.9%), mild (52.7%), moderate (34.5%), and severe (1.8%). No statistically significant association was observed between HP status and Atlanta morphologic classification (p=0.256). Similarly, no significant association was found between HP status and the Atlanta Clinical Severity Index (p=0.311). As summarized in [Table 2](#), the distribution of clinical severity according to the revised Atlanta classification (mild, moderately severe, severe) was comparable between HP-positive and HP-negative groups. Similarly, median CTSI, BISAP, and Ranson scores did not differ significantly between the two groups ([Table 3](#)).

Subgroup analyses according to pancreatitis etiology (biliary vs. non-biliary) were performed to explore potential confounding effects. The results are presented in [Table 4](#). In the biliary pancreatitis subgroup, HP-positive patients demonstrated significantly higher clinical severity scores (Ranson and BISAP) compared to HP-negative patients. However, no significant difference was observed in radiological severity (CTSI). In contrast, no statistically significant differences were observed between HP-positive and HP-negative patients in the non-biliary subgroup (all p>0.05). These findings should be interpreted cautiously due to subgroup sample size.

DISCUSSION

The current study investigated whether HP infection has any clinical relevance in the context of acute pancreatitis, specifically in relation to disease severity and radiologic classification. Although HP has been implicated in several extra-gastric inflammatory conditions, clinical studies evaluating its association with acute pancreatitis have yielded inconsistent results.^{14,15} The theoretical basis for HP's involvement in pancreatic disease lies in its capacity to trigger systemic inflammation, alter gut microbiota composition, and modulate immune responses. Animal models have suggested that HP infection may influence pancreatic injury through cytokine-mediated cross-talk or via bacterial translocation

Table 1. Demographic, clinical, and laboratory features of acute pancreatitis patients stratified by HP status

Parameters	Study population (n=143)	HP negative group (n=88)	HP positive group (n=55)	p-value
Age (years)	56.59±15.95	56.72±17.02	56.38±14.22	0.904
Gender (F) n (%)	59 (41.3%)	34 (38.6%)	25 (45.5%)	0.420
Length of hospital stay (days)	7 (4-11)	7 (4-13)	6 (3-10)	0.120
CTSI	2 (2-3)	2 (1-3)	2 (2-3)	0.773
CCI	2 (1-4)	2.5 (1-4)	2 (0-4)	0.069
Ranson score	1 (0-2)	1 (0-2)	1 (0-2)	0.333
BISAP score	1 (0-2)	1 (0-2)	0 (0-1)	0.120
Pleural effusion n (%)	17 (11.9%)	10 (11.4%)	7 (12.7%)	0.806
Fever n (%)	74 (51.7%)	51 (58.0%)	23 (41.8%)	0.060
ICU admission rate n (%)	17 (11.9%)	12 (13.6%)	5 (9.1%)	0.414
ESR (mm/h)	23 (11.8-40)	25.5 (13-39.8)	20 (8.8-40.5)	0.175
CRP (mg/L)	125 (40-252.3)	140 (50-243)	84 (26-249)	0.132
White blood cell count (10 ³ /μL)	12169.65±5032.12	12414.77±4355.33	11777.45±5981.00	0.463
Hemoglobin (gr/dl)	12.88±4.24	12.61±4.64	13.32±3.50	0.330
Platelet count (10 ³ /μL)	244811.19±86639.77	248602.27±97688.76	238745.45±65599.58	0.510
Glucose (mg/dl)	146.20±68.94	150.33±70.23	139.60±66.93	0.367
Urea (mg/dL)	30.43±12.38	30.49±11.07	30.35±14.34	0.947
Serum creatinine (mg/dl)	0.84 (0.65-0.98)	0.82 (0.65-0.96)	0.89 (0.61-1.01)	0.355
Amylase	678 (313-1684)	738 (371.8-1545.5)	593 (238-2211)	0.818
LDH	306.56±152.84	309.30±162.15	302.18±137.96	0.788
AST (U/L)	43 (18-110)	36 (22-98.5)	47 (16-153)	0.847
ALT (U/L)	38 (17-116)	36 (17.3-89.8)	38 (17-183)	0.947
Total bilirubin (mg/dl)	0.82 (0.39-1.53)	0.81 (0.38-1.47)	0.83 (0.39-1.81)	0.853
Triglycerides (mg/dl)	117 (85-166)	121 (82.3-177)	113 (87-156)	0.381

Data are shown as mean±SD, median (IQR), or n (%). Percentages are based on available-case denominators. Totals may not equal 100% due to rounding. No statistically significant differences were observed between HP-positive and HP-negative groups. HP: *Helicobacter pylori*, F: Female, CTSI: Computed Tomography Severity Index, CCI: Charlson Comorbidity Index, BISAP: Bedside Index for Severity in Acute Pancreatitis, ICU: Intensive care unit, SIRS: systemic inflammatory response syndrome, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, LDH: Lactate dehydrogenase, AST: aspartate aminotransferase, ALT: alanine aminotransferase, SD: Standard deviation, IQR: Interquartile range

Atlanta Classification

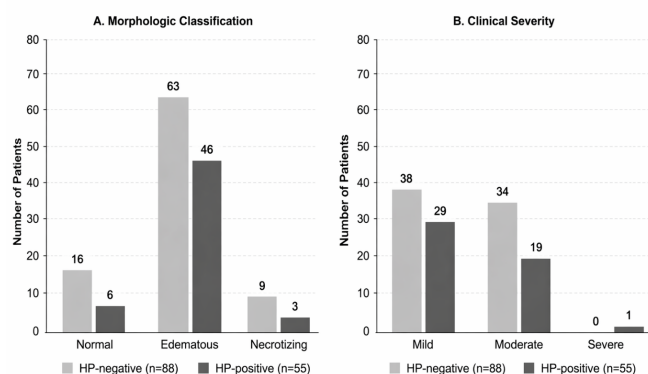


Figure 2. Distribution of revised Atlanta classification subtypes according to HP status (HP-positive vs. HP-negative)

HP: *Helicobacter pylori*

Table 2. Distribution of acute pancreatitis severity according to revised Atlanta classification by HP status

Revised Atlanta severity	HP-positive (n=55)	HP-negative (n=88)	p-value
Mild	29 (52.7%)	38 (43.2%)	
Moderately severe	19 (34.5%)	34 (38.6%)	
Severe	1 (1.8%)	0 (0%)	
Normal*	6 (10.9%)	16 (18.2%)	
Total	55 (100%)	88 (100%)	0.311

*The "normal" category was used as a descriptive designation for patients without radiologically detectable pancreatic abnormalities (CTSI=0). It does not represent an additional clinical severity category within the revised Atlanta classification. Data are presented as n (%). p-value calculated by chi-square test (Fisher's exact when applicable). No statistically significant differences were observed between HP-positive and HP-negative groups. HP: *Helicobacter pylori*

Table 3. Severity scores in HP-positive vs HP-negative patients

Severity score	HP-negative [median (IQR)]	HP-positive [median (IQR)]	p-value
CTSI	2 (1-3)	2 (2-3)	0.773
BISAP	1 (0-2)	0 (0-1)	0.120
Ranson	1 (0-2)	1 (0-2)	0.333

Data are presented as median (interquartile range). p-values calculated by Mann-Whitney U test. HP: *Helicobacter pylori*, IQR: Interquartile range, CTSI: Computed Tomography Severity Index, BISAP: Bedside Index for Severity in Acute Pancreatitis

from the upper gastrointestinal tract to the pancreatic microenvironment.^{4,8-12,16} In addition, HP is known to upregulate pro-inflammatory cytokines such as TNF- α , IL-6, and IL-8, which have also been implicated in the progression of severe acute pancreatitis.¹⁷⁻¹⁹ However, the clinical evidence supporting such interactions remains weak. A case-control study by Khan et al.¹⁴ examined the potential association between HP infection and alcoholic acute pancreatitis by comparing 50 patients with first-episode alcoholic pancreatitis to 50 alcohol-matched controls without pancreatitis. The seroprevalence of HP infection did not significantly differ between the two groups, suggesting no causal link with the onset of pancreatitis. Our results indicate that HP infection was not associated with overall clinical or radiologic severity of acute pancreatitis. This suggests that the presence of HP may represent an incidental finding rather than a definitive contributing factor in disease progression.

Table 4. Subgroup analysis of clinical and radiological severity according to pancreatitis etiology and HP status

Parameter	Biliary HP-negative (n=61)	Biliary HP-positive (n=38)	p-value	Non-biliary HP-negative (n=27)	Non-biliary HP-positive (n=17)	p-value
Ranson score	1 (0-2)	2 (1-3)	0.004	1 (0-2)	1 (0-2)	0.812
BISAP score	1 (0-1)	2 (1-2)	<0.001	1 (0-2)	1 (0-1)	0.734
CTSI	2 (1-3)	2 (2-3)	0.218	2 (1-3)	2 (1-3)	0.911

Data are presented as median [interquartile range]. P-values correspond to comparisons between HP-positive and HP-negative groups within each etiology subgroup and were calculated using the Mann-Whitney U test. HP: *Helicobacter pylori*, BISAP: Bedside Index for Severity in Acute Pancreatitis, CTSI: Computed Tomography Severity Index

One of the key strengths of this study lies in its well-defined cohort, composed of patients with a confirmed diagnosis of acute pancreatitis and systematically assessed HP status based on histopathological examination of gastric biopsies. Unlike studies relying solely on serology or antigen tests, our diagnostic method reflects active infection, thereby improving the validity of group stratification. Additionally, the use of established clinical and radiologic severity indices (Ranson, BISAP, CTSI, and Atlanta classification) allowed for comprehensive assessment of disease burden across multiple dimensions. The inclusion of detailed laboratory and clinical data, combined with objective outcomes such as ICU admission and hospital length of stay, further strengthens the internal consistency and clinical relevance of our findings. Moreover, our study is among the few to investigate not only the presence of HP infection but also its potential correlation with inflammatory indices and multi-dimensional severity markers in acute pancreatitis. Importantly, the use of histopathological examination of gastric biopsies ensured accurate identification of active HP infection, reducing the risk of misclassification inherent in serology-based studies. Interestingly, exploratory subgroup analysis revealed higher clinical severity scores among HP-positive patients in the biliary pancreatitis group. However, this finding was not observed in non-biliary cases and was not supported by radiological severity indices. Given the relatively small subgroup sizes and multiple comparisons, the possibility of a type I error (false-positive finding) cannot be excluded. Therefore, these findings should be considered hypothesis-generating and require confirmation in larger prospective studies.

The retrospective design, selected study population, and absence of quantitative HP assessment should be considered when interpreting these findings, as discussed in the Limitations section.

Although our study did not demonstrate a significant overall association, HP infection may theoretically influence pancreatic inflammation through alterations in gut microbiota, immune dysregulation, epithelial barrier disruption, and activation of pro-inflammatory signaling pathways.^{5,6,8,16} Increased expression of inflammatory mediators, including IL-6 and TNF- α , has also been reported in association with HP infection;¹⁷⁻¹⁹ however, the clinical relevance of these mechanisms to acute pancreatitis severity remains uncertain.

Limitations

Several limitations should be acknowledged. First, the retrospective design and relatively small sample size may limit statistical power and the ability to control for potential confounding factors. Second, HP testing was restricted to patients who underwent clinically indicated upper gastrointestinal endoscopy and gastric biopsy, introducing

potential selection bias and limiting the generalizability of the findings. Although baseline characteristics were compared between included and excluded patients, residual selection bias cannot be excluded. Therefore, these findings may not be directly generalizable to the broader population of patients with acute pancreatitis who do not undergo upper gastrointestinal endoscopy.

Multivariate analysis was not feasible due to the small number of severe cases. Another limitation of our study is that potential confounders such as pancreatitis etiology (e.g., gallstones, alcohol use) could not be fully adjusted for in the analysis, which may have influenced the observed associations. Third, the study population was drawn from a single tertiary care center, which may limit the generalizability of findings to broader or more diverse populations. Missing data were minimal and addressed using available-case analysis, with sensitivity checks confirming consistency of results. Furthermore, due to the observational design, causality cannot be inferred. While our findings suggest no clear association between HP positivity and acute pancreatitis severity, prospective longitudinal studies are required to determine whether HP plays any modulatory or predisposing role in pancreatic inflammation, particularly in idiopathic or recurrent cases.

CONCLUSION

As a result, HP infection was not significantly associated with the overall clinical or radiological severity of acute pancreatitis. However, subgroup findings suggest that this relationship may vary depending on pancreatitis etiology, particularly in biliary cases. These findings should be interpreted cautiously and require confirmation in larger prospective studies. Although its biological plausibility in pancreatic inflammation has been suggested, our findings do not provide sufficient evidence to support a clinically meaningful association. From a clinical perspective, the present findings do not currently provide sufficient evidence to support routine HP screening or eradication specifically for patients with acute pancreatitis. Future prospective multicenter studies are warranted to further explore potential immunologic or microbiome-mediated links between HP and pancreatic pathology.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Ankara Training and Research Hospital Ethics Committee (Date: 09.10.2024, Decision No: 258/2024).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and

there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: YHP, BÇ; Design: YHP, BÇ; Control: YHP, BÇ; Resources: YHP, BÇ; Materials: YHP, BÇ; Data Collection and/or Processing: YHP, BÇ; Analysis and/or Interpretation: YHP, BÇ; Literature Review: YHP, BÇ; Writing the Article: YHP, BÇ; Critical Review: YHP, BÇ.

REFERENCES

1. Szatmary P, Grammatikopoulos T, Cai W, et al. Acute pancreatitis: diagnosis and treatment. *Drugs*. 2022;82(12):1251-1276. doi:10.1007/s40265-022-01766-4
2. Mihoc T, Latcu SC, Secasan CC, et al. Pancreatic morphology, immunology, and the pathogenesis of acute pancreatitis. *Biomedicines*. 2024;12(11):2627. doi:10.3390/biomedicines12112627
3. Ali A, AlHussaini KI. *Helicobacter pylori*: a contemporary perspective on pathogenesis, diagnosis and treatment strategies. *Microorganisms*. 2024;12(1):222. doi:10.3390/microorganisms12010222
4. Gravina AG, Zagari RM, De Musis C, Romano L, Loguercio C, Romano M. *Helicobacter pylori* and extragastric diseases: a review. *World J Gastroenterol*. 2018;24(29):3204-3221. doi:10.3748/wjg.v24.i29.3204
5. Bawali P, Brahma A, Rana SR, Pal A, Bhattacharyya A. *Helicobacter pylori* infection and inflammatory events: the extracellular vesicle-connect in driving gastrointestinal tract cancers. *Front Med (Lausanne)*. 2024;11:1444242. doi:10.3389/fmed.2024.1444242
6. Hurtado-Monzón EG, Valencia-Mayoral P, Silva-Olivares A, Bañuelos C, Velázquez-Guadarrama N, Betanzos A. The *Helicobacter pylori* infection alters the intercellular junctions on the pancreas of gerbils (*Meriones unguiculatus*). *World J Microbiol Biotechnol*. 2024;40(9):273. doi:10.1007/s11274-024-04081-0
7. Leal-Lopes C, Velloso FJ, Campopiano JC, Sogayar MC, Correa RG. Roles of commensal microbiota in pancreas homeostasis and pancreatic pathologies. *J Diabetes Res*. 2015;2015:284680. doi:10.1155/2015/284680
8. Testerman TL, Morris J. Beyond the stomach: an updated view of *Helicobacter pylori* pathogenesis, diagnosis, and treatment. *World J Gastroenterol*. 2014;20(36):12781-12808. doi:10.3748/wjg.v20.i36.12781
9. Manes G, Balzano A, Vaira D. *Helicobacter pylori* and pancreatic disease. *JOP*. 2003;4(3):111-116.
10. Fakharian F, Asgari B, Nabavi-Rad A, et al. The interplay between *Helicobacter pylori* and the gut microbiota: an emerging driver influencing the immune system homeostasis and gastric carcinogenesis. *Front Cell Infect Microbiol*. 2022;12:953718. doi:10.3389/fcimb.2022.953718
11. Hua Z, Xu L, Zhu J, et al. *Helicobacter pylori* infection altered gastric microbiota in patients with chronic gastritis. *Front Cell Infect Microbiol*. 2023;13:1221433. doi:10.3389/fcimb.2023.1221433
12. Rabelo-Gonçalves EM, Roesler BM, Zeitune JM. Extragastric manifestations of *Helicobacter pylori* infection: possible role of bacterium in liver and pancreas diseases. *World J Hepatol*. 2015;7(30):2968-2979. doi:10.4254/wjh.v7.i30.2968
13. Warzecha Z, Dembiński A, Ceranowicz P, et al. Deleterious effect of *Helicobacter pylori* infection on the course of acute pancreatitis in rats. *Pancreatol*. 2002;2(4):386-395. doi:10.1159/000065086
14. Khan J, Pelli H, Lappalainen-Lehto R, Järvinen S, Sand J, Nordback I. *Helicobacter pylori* in alcohol induced acute pancreatitis. *Scand J Surg*. 2009;98(4):221-224. doi:10.1177/145749690909800405
15. Bulajic M, Panic N, Löhr JM. *Helicobacter pylori* and pancreatic diseases. *World J Gastrointest Pathophysiol*. 2014;5(4):380-383. doi:10.4291/wjgp.v5.i4.380
16. Franceschi F, Zuccalà G, Roccarina D, Gasbarrini A. Clinical effects of *Helicobacter pylori* outside the stomach. *Nat Rev Gastroenterol Hepatol*. 2014;11(4):234-242. doi:10.1038/nrgastro.2013.243
17. Saviano A, Morabito Loprete MR, Pignataro G, et al. *Helicobacter pylori*, atherosclerosis, and coronary artery disease: a narrative review. *Medicina (Kaunas)*. 2025;61(2):346. doi:10.3390/medicina61020346
18. Chen M, Huang X, Gao M, et al. *Helicobacter pylori* promotes inflammatory factor secretion and lung injury through VacA exotoxin-mediated activation of NF-κB signaling. *Bioengineered*. 2022;13(5):12760-12771. doi:10.1080/21655979.2022.2071011
19. Fei X, Chen S, Li L, et al. *Helicobacter pylori* infection promotes M1 macrophage polarization and gastric inflammation by activation of NLRP3 inflammasome via TNF/TNFR1 axis. *Cell Commun Signal*. 2025;23(1):6. doi:10.1186/s12964-024-02017-7