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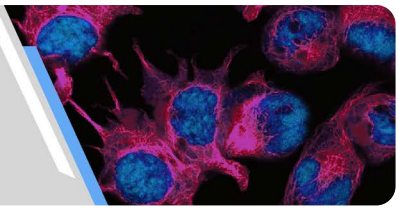

Clinicopathological Correlation of Stool Microscopy, Fecal Calprotectin, and *Helicobacter pylori* Status in Patients with Gastrointestinal Symptoms

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Abstract

Background: Gastrointestinal disorders are a major global health burden, particularly in low- and middle-income countries where diagnostic resources are limited. Stool microscopy remains a commonly used first-line investigation; however, it has limited ability to differentiate functional disorders from inflammatory conditions. Fecal calprotectin is a reliable non-invasive marker of intestinal inflammation, but its clinicopathological correlation with stool microscopy findings and *Helicobacter pylori* infection has not been well studied in Bangladesh.

Objective: To assess the clinicopathological correlation between stool microscopy findings, fecal calprotectin levels, and *Helicobacter pylori* status among patients presenting with gastrointestinal symptoms.

Methods: This cross-sectional study was conducted at Dhaka General Hospital, Bangladesh, from October to December 2024. A total of 328 symptomatic patients were enrolled. Stool samples underwent macroscopic and microscopic examination, including assessment of pus cells, red blood cells (RBCs), mucus, yeast, and occult blood. Fecal calprotectin was measured using immunoassay and categorized as normal (≤50 µg/g), mild (>50–200 µg/g), or severe (>200 µg/g). *H. pylori* status, stool rotavirus antigen, and systemic biochemical markers were evaluated. Statistical analyses included Mann–Whitney U tests, correlation analysis, and multinomial logistic regression. **Results:** The study population comprised 179 males (54.57%) and 149 females (45.43%), with a mean age of 34.30 ± 21.82 years. *H. pylori* infection was detected in 32.62% of patients. Median fecal calprotectin levels were significantly higher in *H. pylori*–positive patients compared with negatives (165.2 vs. 46.0 µg/g; $p < 0.001$). Elevated FC levels were also associated with positive stool occult blood tests (315 vs. 48.8 µg/g; $p < 0.001$), presence of RBCs on microscopy (233 vs. 55 µg/g; $p < 0.001$), and stool rotavirus positivity (146.5 vs. 40.1 µg/g; $p < 0.001$). In multinomial logistic regression, *H. pylori* positivity independently increased the odds of mild FC elevation by 2.40-fold (95% CI: 1.17–4.91; $p = 0.017$) and severe elevation by 5.94-fold (95% CI: 2.73–12.93; $p < 0.001$). Pancreatic enzymes, particularly lipase ($r = 0.400$; $p < 0.001$), showed the strongest positive correlation with fecal calprotectin.

Conclusion: This study demonstrates a strong, dose-dependent association between *Helicobacter pylori* infection and fecal calprotectin elevation, suggesting that *H. pylori* contributes to intestinal inflammation beyond its gastric effects. Biochemical and pathogen-specific markers were more reliable indicators of inflammatory severity than stool morphology alone.

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Keywords: Fecal calprotectin; *Helicobacter pylori*; Stool microscopy; Gastrointestinal inflammation; Bangladesh

Introduction

Gastointestinal (GI) disorders constitute a major global public health burden and are among the most common causes of healthcare utilization worldwide. Patients frequently present with nonspecific symptoms such as abdominal pain, diarrhea, constipation, bloating, dyspepsia, and gastrointestinal bleeding, which often overlap between infectious, inflammatory, and functional gastrointestinal conditions. This clinical overlap complicates diagnosis, particularly in low- and middle-income countries (LMICs), where access to advanced diagnostic modalities remains limited (1). Stool microscopy continues to be a widely used first-line investigation for gastrointestinal symptoms in LMICs due to its low cost and availability. It enables detection of intestinal parasites, ova, cysts, inflammatory cells, and occult bleeding, providing preliminary information on infectious and inflammatory processes. However, stool microscopy is limited by operator dependence, variable sensitivity, and its inability to reliably distinguish functional disorders from early or low-grade intestinal inflammation, which may result in diagnostic uncertainty (2). Fecal calprotectin (FC), a neutrophil-derived calcium-binding protein, has emerged as a reliable non-invasive biomarker of intestinal inflammation. Elevated fecal calprotectin levels are strongly associated with organic inflammatory conditions such as inflammatory bowel disease, infectious colitis, and colorectal pathology, whereas functional gastrointestinal disorders typically demonstrate normal values. The use of fecal calprotectin has been well established in high-income countries to reduce unnecessary endoscopic evaluations; however, its application and clinical correlation remain underexplored in South Asian populations, including Bangladesh (3).

Helicobacter pylori infection remains highly prevalent worldwide, particularly in developing regions. It is a well-recognized cause of chronic gastritis, peptic ulcer disease, mucosa-associated lymphoid tissue lymphoma, and gastric malignancy. Emerging evidence suggests that *H. pylori* may also influence intestinal immune responses and gut microbial balance, potentially contributing to lower gastrointestinal symptoms and subclinical inflammation, although its relationship with fecal inflammatory markers and stool microscopic findings remains inconsistent (4). South Asia, including Bangladesh, faces a dual burden of persistent enteric infections and a rising prevalence of inflammatory and functional gastrointestinal disorders. In Bangladesh, high rates of parasitic infestations and *H. pylori* infection coexist with limited diagnostic resources, leading to continued reliance on stool microscopy without routine integration of inflammatory biomarkers. Data examining the combined clinicopathological relevance of stool microscopy, fecal calprotectin, and *H. pylori* status in this setting remain scarce. This study aimed to assess the clinicopathological correlation between stool microscopy findings, fecal calprotectin levels, and *Helicobacter pylori* status among patients presenting with gastrointestinal symptoms.

Materials and Methods

Study Design, Setting, and Study Population

This cross-sectional clinicopathological study was conducted at Dhaka General Hospital, Bangladesh, over a three-month period from October 2024 to December 2024. A total of 328 patients presenting to the outpatient and inpatient departments with gastrointestinal symptoms, including abdominal pain, diarrhea, constipation, bloating, dyspepsia, or gastrointestinal bleeding, were consecutively enrolled. Patient data were collected prospectively as part of routine clinical evaluation. Demographic information, including age and gender, along with relevant clinical details, were recorded at the time of enrollment. All laboratory investigations were performed using patient-derived biological samples.

Stool Morphological and Microscopic Analysis

Fresh stool samples were collected from all participants in sterile, leak-proof containers and processed immediately in accordance with standard laboratory protocols. Each sample underwent macroscopic examination to assess physical characteristics such as color, consistency (formed, semi-formed, or liquid), and the presence of visible mucus or blood. Microscopic evaluation was performed using saline and iodine wet mount preparations to identify and quantify cellular components and inflammatory markers, including pus cells, red blood cells, and macrophages, reported per high-power field. Additionally, stool samples were examined for parasitological and dietary components, including ova, cysts, yeast cells, fat globules, vegetable cells, and muscle fibers, to assess intestinal infection, inflammation, and malabsorption.

Biochemical and Immunological Assays

Fecal calprotectin was measured as a marker of intestinal inflammation using standardized immunoassay techniques. Stool occult blood testing was performed to detect gastrointestinal bleeding, and stool rotavirus antigen testing was conducted when clinically indicated. The presence of *Helicobacter pylori* was determined using routine diagnostic methods available at the study center. To evaluate extra-intestinal and systemic involvement, venous blood samples were collected for biochemical analysis. Serum pancreatic enzymes, including amylase and lipase, as well as liver function parameters such as bilirubin, alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase, were measured using automated analyzers following standard laboratory procedures.

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Fecal Calprotectin Categorization and Predictor Variables

Fecal calprotectin levels, expressed in micrograms per gram, were treated as the primary outcome variable. For clinical interpretation and statistical modeling, calprotectin values were categorized into three severity levels based on established thresholds: normal (≤ 50 µg/g), mild elevation (> 50 to ≤ 200 µg/g), and severe elevation (> 200 µg/g). Potential predictor variables included demographic factors such as gender, macroscopic stool findings including the presence of mucus and visible blood, microscopic parameters such as red blood cells, yeast, ova, and cysts, and laboratory markers including stool occult blood test results and *Helicobacter pylori* status. All predictor variables were coded as binary indicators for regression analyses.

Statistical Analysis

Statistical analyses were performed using **R software (RStudio version 2024.12.0)**. Descriptive statistics summarized demographic and stool morphological data. Associations between stool microscopic findings and fecal calprotectin (FC) levels were assessed using correlation analysis, while group differences in FC concentrations were evaluated with the Mann–Whitney U test. A multinomial logistic regression model was used to identify factors associated with FC severity, with Normal FC as the reference category. Predictor variables included *Helicobacter pylori* status, stool microscopic findings, and biochemical markers. Results were expressed as adjusted odds ratios with 95% confidence intervals, and statistical significance was defined as $p < 0.05$.

Result

Demographic Characteristics

A total of 328 patients were included in the analysis, comprising 179 males (55%) and 149 females (45%) (Figure 1). The overall mean age of the participants was 34.30 ± 21.82 years. The mean age of male participants was 32.83 ± 22.50 years, while that of female participants was 36.06 ± 20.91 years, with no statistically significant age difference observed between genders ($p > 0.05$). Age distribution across predefined age groups was comparable between male and female participants, as summarized in Table 1.

Stool Morphology

Table 2 summarizes the stool morphological characteristics of the study population. The majority of stool samples were brown in color (77.74%), followed by smaller proportions of whitish, greenish, and reddish stools. Regarding consistency, semi-solid stools were most common (35.67%), followed by soft (26.22%) and solid (13.11%) stools, while watery, loose, and liquid stools together accounted for a smaller proportion of samples.

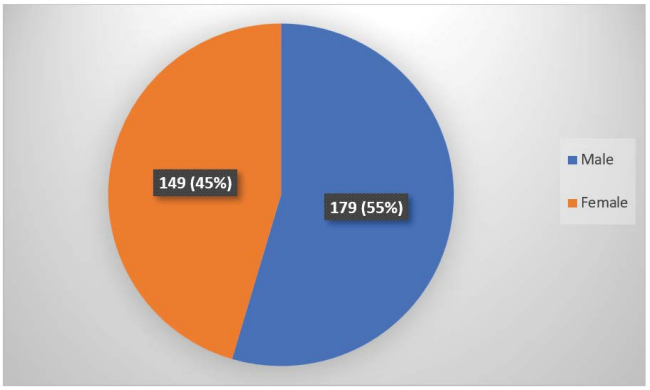

Figure 1: Gender Distribution

Table 1. Demographic Characteristics of the Patients

Age group	Male (55%)	Female (45%)
01-10	44	24
11-20	11	10
21-30	24	31
31-40	21	22
41-50	37	22
51-60	15	19
61-70	21	12
71-80	6	9
Total	**179**	**149**

Table 2: Stool morphology Profile

Stool morphology	Type	Number (n)	Percentages (%)
Color	Brown	255	77.74
	Whitish	19	5.79
	Straw	2	0.61
	Greenish	17	5.18
	Yellowish	6	1.83
	Reddish	16	4.88
	Dark Brown	8	2.44
	Blackish	5	1.52
Consistency	Soft	86	26.22
	Semi Solid	117	35.67
	watery	29	8.84
	Loose	28	8.54
	Solid	43	13.11
	Liquid	25	7.62

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Table 3 presents the stool microscopy findings of the study population. Visible blood was observed in a small proportion of samples (5.18%), while mucus was present in the majority, predominantly at low to moderate levels. No parasitic cysts were detected in any sample. Most stools showed no excess fat or macrophages, although mild to moderate fat globules and macrophages were observed in a subset of patients. Vegetable cells and muscle fibers were frequently identified, suggesting varying degrees of dietary residue and malabsorption. Stool occult blood test positivity was observed in 17.07% of cases. Yeast cells were rarely detected. Approximately one-third of patients tested positive for stool rotavirus and *Helicobacter pylori*, indicating a notable burden of infectious etiology among symptomatic patients.

Table 3: Stool microscopy observation profile

Stool microscopy	Types	Number (n)	Percentage (%)
Blood	Not Visible	311	94.82
	Visible	17	5.18
Mucus	No	0	0
	Present (+)	282	85.98
	Present (++)	46	14.02
Cyst	No	328	100
	present	0	0
Fat	Nil	291	88.72
	(+)	11	3.34
	(++)	26	7.93
Vegetable Cell	Nil	224	68.29
	(+)	88	26.28
	(++)	13	3.96
	(+++)	3	0.91
Stool OBT	Negative	272	82.93
	Positive	56	17.07
Macrophage	Nil	267	81.4
	Present	61	18.6
Muscle fiber	(+)	142	43.29
	Nil	166	50.61
	(++)	19	5.79
Yeast	(++)	2	0.61
	Not Found	323	98.48
	(+)	3	0.91
Stool Rota Virus	Negative	221	67.38
	Positive	107	32.62
H. Pylori	Negative	221	67.38
	Positive	107	32.62

Table 4 summarizes the distribution of pus cells and red blood cells (RBCs) observed per high-power field (HPF) in stool microscopy. Most samples showed low to moderate pus cell counts, with the highest proportion observed in the 0–5/HPF category, indicating mild inflammatory activity in a large subset of patients. A smaller proportion exhibited higher pus cell counts, suggesting more pronounced intestinal inflammation. The majority of stool samples had no detectable RBCs, while a minority demonstrated varying degrees of RBC presence, ranging from occasional to plentiful, reflecting localized mucosal irritation or bleeding in a subset of cases.

Table 4: Stool observation for pus cell and RBC categories percentages

Stool observations	Categories	Number (n)	Percentage (%)
Pus Cell /HPF	Nil	2	0.61
	Plenty	80	24.4
	(0-5)	153	46.64
	(1-3)	10	3.05
	(2-4)	17	5.18
	(4-6)	23	7.01
	(6-8)	25	7.62
	(8-10)	8	2.43
	(10-12)	4	1.22
	(15-20)	5	1.52
RBC / HPF	Nil	265	80.79
	(0-2)	13	3.96
	(2-4)	7	2.13
	(4-6)	9	2.74
	(6-8)	6	1.83
	(8-10)	9	2.74
	Plenty	14	4.27
	Occasional	1	0.3
	(10-12)	2	0.6
	<12	2	0.6

Table 5 presents the comparative analysis of fecal calprotectin levels across different stool microscopy findings. Significantly higher fecal calprotectin concentrations were observed among patients with positive stool occult blood tests, presence of red blood cells in stool, *Helicobacter pylori* infection, and stool rotavirus positivity (all $p < 0.001$), indicating a strong association with intestinal inflammation. Patients with detectable yeast also showed a modest but statistically significant difference in fecal calprotectin levels. In contrast, the presence of visible blood and cysts in stool samples was not significantly associated with fecal calprotectin concentrations.

Age showed no significant correlation with stool calprotectin (Spearman's $r = 0.065$, $p = 0.241$). In contrast, pancreatic and hepatic enzymes demonstrated significant positive correlations. Amylase ($r = 0.367$, $p < 0.001$) and lipase ($r = 0.400$, $p < 0.001$) exhibited moderate correlations, with

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Table 5: Comparative Analysis of Fecal Calprotectin by Stool Microscopy Findings

Stool microscopy finding	Category	Fecal calprotectin, median (IQR) µg/g	p-value
Blood	Absent	93.8 (31.1–195.5)	
	Present	130.2 (130.2–130.2)	0.59
Stool OBT	Negative	48.8 (27.2–145.8)	
	Positive	315 (129.1–410.2)	< 0.001*
Cyst	Absent	95 (33–195.4)	
	Present	28.9 (17–208.2)	0.206
RBC / HPF	Absent	55 (30–155.1)	
	Present	233 (92.4–390.2)	< 0.001*
Yeast	Absent	107.8 (35–220.5)	
	Present	88.6 (23.7–162)	0.0081
H.Pylori	Absent	46 (27.5–142.5)	
	Present	165.2 (64.7–385)	< 0.001
Stool Rota Virus	Absent	40.1 (23.7–152.6)	
	Present	146.5 (118.4–393.8)	< 0.001

Table 6: Correlation between stool calprotectin and clinical variables

Variables	Correlation coefficient (r)	P-value
Age	0.065	0.241
Amylase	0.36698261	< 0.001*
ALT	0.37020343	< 0.001*
AST	0.21313417	< 0.001*
Lipase	0.40002822	< 0.001*

Table 7: Multinomial Logistic Regression Analysis of Factors Associated with Fecal Calprotectin Severity

Predictor	Level	Adjusted Odds Ratio	95% CI	p-value
Gender	Mild	0.51	0.05, 5.23	0.571
	Severe	2.63	0.21, 32.50	0.451
Mucus	Mild	0.83	0.36, 1.94	0.669
	Severe	0.67	0.20, 2.23	0.516
RBC	Mild	0.75	0.24, 2.36	0.624
	Severe	,2.30	0.74, 7.18	0.15
Yeast	Mild	0.50,"[	0.05, 5.01	0.554
	Severe	0.76	0.07, 8.52	0.821
H. pylori	Mild	2.4	1.17, 4.91	0.017*
	Severe	5.94	2.73, 12.93	< 0.001*

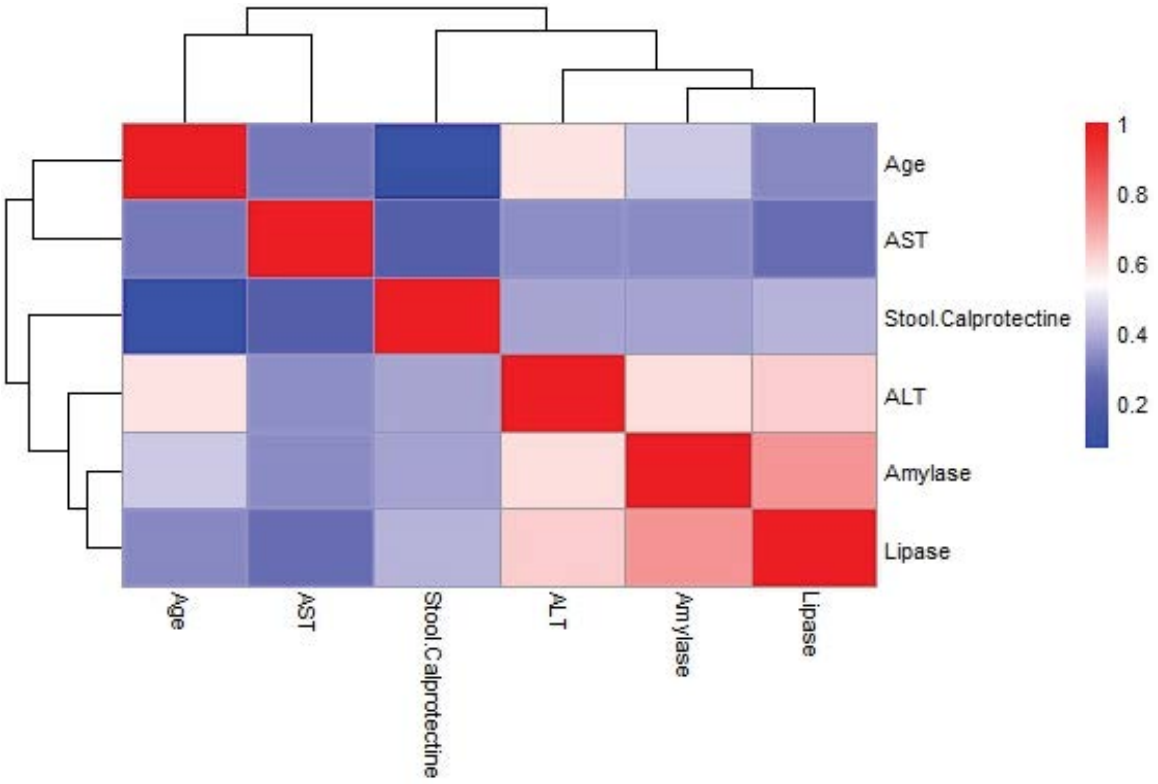

Figure 2: Hierarchical cluster correlation heatmap showing relationships among biochemical parameters with stool calprotectine. Color intensity represents correlation strength (red = strong positive correlation; blue = weak or negative correlation). (white = strong positive correlation; grey = weak or negative correlation). Dendrograms indicate clustering based on similarity patterns

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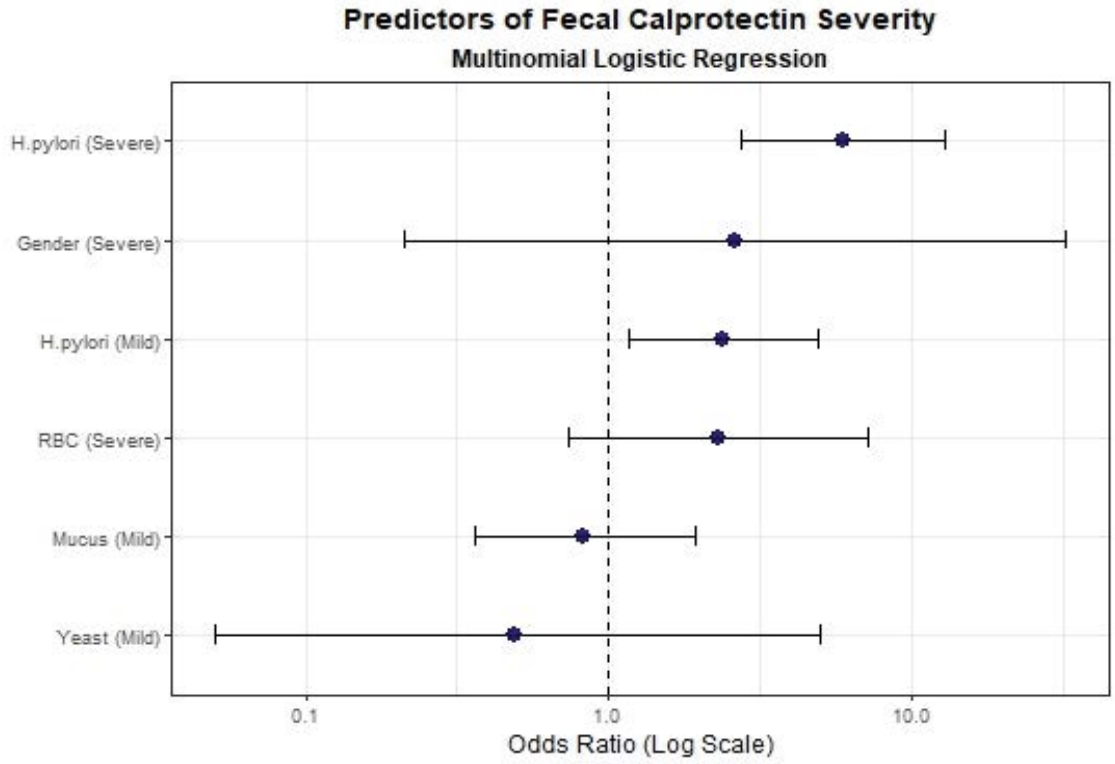

Figure 3: Predictors of Fecal Calprotectin Severity. Forest plot displaying adjusted odds ratios (aOR) and 95% confidence intervals for Mild and Severe calprotectin elevation (Reference: Normal). *H. pylori* positivity demonstrates a significant, dose-dependent association with increasing inflammatory severity ($p < 0.05$).

lipase being the strongest predictor among the tested variables (Table 6). Similarly, liver enzymes ALT ($r = 0.370$, $p < 0.001$) and AST ($r = 0.213$, $p < 0.001$) were significantly correlated with stool calprotectin, though the strength of association was weaker compared to pancreatic enzymes shown in figure 2. These findings suggest that elevated stool calprotectin is significantly associated with increased levels of pancreatic (amylase, lipase) and hepatic (ALT, AST) enzymes, whereas age does not appear to influence calprotectin levels. The strongest correlation was observed with lipase, highlighting a potential link between pancreatic enzyme activity and intestinal inflammation.

A multinomial logistic regression was conducted to assess the association between various clinical and microbiological markers and the severity of fecal calprotectin elevation. The presence of ***H. pylori*** emerged as the most significant predictor of intestinal inflammation. Compared to the "Normal" reference group ≤ 50 µg/g, ***H. pylori*** positivity was associated with a **2.40-fold increase** in the odds of having "Mild" calprotectin elevation ($p = 0.017$) and a **5.94-fold increase** in the odds of "Severe" elevation ($p < 0.001$*). While the presence of **RBCs** showed a positive trend toward severe inflammation (AOR = 2.30), this association did not reach statistical significance ($p = 0.150$). Other parameters, including Gender, Mucus, and Yeast, were not significantly associated with calprotectin levels ($p > 0.05$) (Table 7). Variables such as Blood and OBT exhibited quasi-complete separation due to high correlation with the "Severe" category; while they were controlled for in the model, their coefficients were non-estimable and were omitted from the graphical forest plot (Figure 3) to maintain scale integrity.

Discussion

In this cross-sectional clinicopathological study of 328 patients presenting with gastrointestinal symptoms, we evaluated the relationship between stool microscopy findings, fecal calprotectin levels, infectious markers, and systemic biochemical parameters. The most important finding was the strong, dose-dependent association between *Helicobacter pylori* infection and fecal calprotectin severity. Patients positive for *H. pylori* had significantly higher median fecal calprotectin levels (165.2 µg/g) compared with *H. pylori*–negative patients (46.0 µg/g; $p < 0.001$). In multivariate analysis, *H. pylori* positivity independently increased the odds of mild fecal calprotectin elevation by 2.40-fold and severe elevation by 5.94-fold, highlighting its dominant role in intestinal inflammatory activity. Fecal calprotectin is a well-established surrogate marker of neutrophil-mediated intestinal inflammation and is widely used to differentiate organic gastrointestinal disease from functional disorders

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(5,6). In the present study, elevated fecal calprotectin levels were significantly associated with markers of mucosal injury and infection. Patients with positive stool occult blood tests demonstrated markedly higher calprotectin levels (median 315 µg/g) compared with OBT-negative individuals (48.8 µg/g; $p < 0.001$). Similarly, the presence of red blood cells on stool microscopy was associated with a more than fourfold increase in median fecal calprotectin (233 vs. 55 µg/g; $p < 0.001$). Stool rotavirus positivity, observed in 32.62% of patients, was also associated with significantly higher calprotectin levels (146.5 vs. 40.1 µg/g; $p < 0.001$), supporting the link between acute enteric infection and intestinal neutrophilic inflammation (6,7).

A key contribution of this study is the demonstration that *H. pylori* infection is not merely associated with upper gastrointestinal pathology but is also linked to downstream intestinal inflammation. While 32.62% of patients were *H. pylori* positive, this subgroup accounted for a disproportionately high burden of elevated fecal calprotectin. Previous studies have suggested that *H. pylori* can modulate systemic immune responses, disrupt gut microbial balance, and promote inflammatory signaling beyond the stomach (8,9). Our findings are consistent with earlier reports by Manolakis et al. and Ruggiero et al., who observed increased fecal inflammatory markers in *H. pylori*–infected individuals, supporting an immunologically mediated gut inflammatory response (10,11). In contrast, macroscopic stool characteristics such as mucus and yeast presence were not independently associated with fecal calprotectin severity in multivariate analysis. Although mucus was present in nearly all samples and yeast was detected in a small subset, neither variable showed a statistically significant association after adjustment. These findings indicate that gross stool morphology lacks specificity for identifying inflammatory severity and should not be relied upon as a surrogate for mucosal inflammation. Similar limitations of stool microscopy alone have been highlighted in previous studies (12), reinforcing the superior diagnostic value of biochemical and pathogen-specific markers.

Correlation analysis further demonstrated that fecal calprotectin levels were significantly associated with systemic biochemical markers. Moderate positive correlations were observed with pancreatic enzymes, particularly lipase ($r = 0.400$, $p < 0.001$) and amylase ($r = 0.367$, $p < 0.001$), as well as with hepatic enzymes ALT ($r = 0.370$, $p < 0.001$) and AST ($r = 0.213$, $p < 0.001$). These findings suggest a potential interaction between intestinal inflammation and pancreatic–hepatic involvement, possibly mediated through increased intestinal permeability or shared inflammatory pathways, as proposed in previous studies (13,14). Age, however, showed no significant correlation with fecal calprotectin, indicating that inflammation rather than demographic factors drives calprotectin elevation in this population. In multinomial regression analysis, *H. pylori* remained the strongest independent predictor of fecal calprotectin severity, whereas gender, mucus, yeast, and RBC presence did not reach statistical significance after adjustment. Stool blood and occult blood test results exhibited quasi-complete separation due to their near-universal presence in patients with severe calprotectin elevation, a known limitation in regression modeling of highly predictive variables (15). Although these variables were excluded from graphical modeling, their strong univariate associations underscore their continued clinical relevance. Overall, our findings indicate that fecal calprotectin reflects underlying inflammatory and infectious processes rather than macroscopic stool characteristics. The strong and consistent association between *H. pylori* infection and fecal calprotectin elevation has important clinical implications, particularly in resource-limited settings such as Bangladesh, where diagnostic options are constrained. Incorporating fecal calprotectin testing alongside *H. pylori* screening may enhance diagnostic accuracy, facilitate early identification of inflammatory pathology, and improve patient stratification and management.

Conclusion

In summary, this study demonstrates a strong, dose-dependent association between *Helicobacter pylori* infection and fecal calprotectin elevation, with *H. pylori*–positive patients showing nearly sixfold higher odds of severe intestinal inflammation. These findings support a broader inflammatory role of *H. pylori* beyond the stomach and highlight fecal calprotectin as a reliable marker of clinically meaningful gastrointestinal inflammation. Routine *H. pylori* screening should be considered in patients presenting with elevated fecal calprotectin to identify potential inflammatory drivers and guide targeted management.

Strengths and Limitations

This study benefits from a relatively large sample size, detailed stool microscopy profiling, integration of infectious and biochemical markers, and multivariate modeling to identify independent predictors of inflammatory severity. However, its cross-sectional design limits causal inference, and the absence of endoscopic or histopathological confirmation restricts direct assessment of mucosal pathology. Additionally, *H. pylori* strain virulence factors were not evaluated, which may influence inflammatory responses.

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