

The role of the PLR–NLR combination in the prediction of the presence of *Helicobacter pylori* and its associated complications

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Abstract

Background/Aim: The aim of this study was to investigate the role of the platelet-to-lymphocyte ratio (PLR)–neutrophil-to-lymphocyte ratio (NLR) combination, in the prediction of the presence of *Helicobacter pylori* (HP) and its associated complications in the gastrointestinal system.

Patients and Methods: In all, 1289 patients who underwent esophagogastroduodenoscopy and biopsy for HP were included in the study.

Results: The ratio of patients with moderate and severe chronic gastritis was higher in HP (+) group than HP (–) group. The ratio of patients with levels 1–3 atrophy and intestinal metaplasia was higher in HP (+) group. Compared with HP (–) group, HP (+) had higher PLR and NLR levels. The ratio of HP (+) patients was higher in high-risk group compared with low- and medium-risk groups. HP invasion stage, the intestinal metaplasia level, and the ratio of patients with atrophy level “3” were higher in high-risk group compared with low- and medium-risk groups. Regression analysis showed that the PLR–NLR combination was an independent risk factor for both HP presence and moderate and severe chronic gastritis.

Conclusion: We found the PLR–NLR combination to be a good predictor of HP presence and gastrointestinal complications associated with HP.

Keywords: Chronic gastritis, *Helicobacter pylori*, intestinal metaplasia, lymphocyte, neutrophil

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INTRODUCTION

Helicobacter pylori (HP) is a Gram-negative, spiral-shaped, flagellar, and microaerophilic bacillus found in the gastric mucosa of more than 50% of individuals worldwide.^[1] HP has been shown to be a predisposition for gastric ulcers and gastric atrophy, as well as certain gastric cancers.^[2] Although a non-invasive bacterial species, it increases T-cell

activation by generating antigenic substances such as heat shock protein, urease, and lipopolysaccharide. The increase in T-cell activation leads to an increase in the secretion of cytokines such as interleukin-1, interleukin-6, interleukin-8, and tumor necrosis factor alpha (TNF α). The changes in the gastrointestinal system because of this increased inflammatory response may also lead to damage

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in non-gastrointestinal tissues.^[3] The relationship between HP and vascular disorders has been confirmed in many studies.^[4]

The damage caused by HP on tissues is believed to be associated with increased inflammatory markers resulting from immune response and platelet activation.^[5,6] Platelet activation, on the other hand, is believed to occur as a result of the induction of 8-iso-prostaglandin F_2^α and other biologically active isoeicosanoids together with lipid peroxidation because of mild inflammation associated with HP.^[7-11] Increased mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and platelet count are indicators of platelet activation in laboratory examinations.^[12]

Many studies highlight that chronic inflammation and platelet activation increase with the presence of HP. In light of this information, we believe that host tissue damage as a result of HP invasion is correlated with chronic inflammation and platelet activation.

Thus, in this study we aim to explore the role of platelet, MPV, PDW, PCT, neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) in the prediction of the presence of HP and its correlating complications such as chronic gastritis, gastric atrophy, and intestinal metaplasia (IM).

PATIENTS AND METHODS

Study population, design, and setting

This retrospective study was performed in the Gastroenterology Clinic of the Turkish High Specialty Training and Research Hospital and the Internal Medicine Clinic at the Ankara Numune Training and Research Hospital.

Patients presenting to our clinics between January 2014 and January 2016 with complaints of pain, dyspepsia, heartburn, and postprandial bloating and who underwent esophagogastroduodenoscopy and biopsy for HP were included in the study. The patients were included in the study in order of application without regard to age, sex, or race.

Patients who were found to have active and chronic infection or hematological disease, or who did not undergo adequate pathological evaluation, did not have complete blood count results available, had thrombocytopenia (platelet count $<150 \times 10^9 \mu\text{L}^{-1}$) and thrombocytosis (platelet count $>450 \times 10^9 \mu\text{L}^{-1}$), and used antiagregant, anticoagulant, and immunosuppressive drugs were excluded from the study.

A receiver-operating characteristic (ROC) curve with Youden index was used to determine the best PLR and NLR cut-off values for HP (+) prediction. For HP (+), the threshold value of PLR was found to be 116.2 with 77.1% sensitivity and 65.1% specificity [area under the curve (AUC) \pm standard error (SE) = 0.795 ± 0.013 ; $P < 0.001$], whereas the threshold value of NLR was found to be 2.43 with 59.6% sensitivity and 87.0% specificity (AUC \pm SE = 0.766 ± 0.013 ; $P < 0.001$). In cases where the patient had NLR and PLR values over the threshold values, he or she was classified as being high-risk (PLR > 116.2 and NLR > 2.43). In cases where the patient had an NLR or PLR value over the respective threshold value, he or she was classified as being medium-risk (PLR > 116.2 or NLR > 2.43). In all other cases, the patient was classified as being low-risk (PLR ≤ 116.2 and NLR ≤ 2.43).

The white blood cell (WBC), neutrophil, lymphocyte, hemoglobin, platelet, MPV, PDW, and PCT values of the patients were recorded from patient files.

Endoscopic examination

Esophagogastroduodenoscopy was carried out by the same endoscopist using FUJINON XL-4450 (Fujifilm Medical Co., Tokyo, Japan). Biopsies were taken from the antrum, incisura angularis, and corpus for HP, the IM, and the atrophic gastritis.

Pathological evaluation

Biopsies taken from corpus and antrum were stained with hematoxylin–eosin and modified giemsa—which is used for *Helicobacter* organisms—and separately assessed according to the Sydney grading system.^[13] According to the Sydney grading system, chronic inflammation was graded as being mild, moderate, or severe depending on the increase rate of lymphocytes and plasmocytes in lamina propria. Activity was graded as being mild, moderate, or severe depending on the presence of neutrophils on lamina propria, epithelium, or crypt lumen. Atrophy was graded as mild, moderate, or severe depending on the degree of gland loss. IM was graded depending on the area covered by intestinalized epithelium, with less than one-third of mucosa being mild, one-third to two-third being moderate, and more than two-third of the mucosa being severe. HP density was graded as being mild in cases where the microorganism was scarce and difficult to find, as moderate in cases where the microorganism was more generalized and formed small groups, and as severe in cases where the microorganism formed large colonies. Cases were reported as being mild (stage 1), moderate (stage 2), and severe (stage 3).

Statistical analysis

Statistical Package for Social Sciences (SPSS) for Windows 20 (IBM SPSS Inc., Chicago, IL, USA) and MedCalc11.4.2 (MedCalc Software, Mariakerke, Belgium) software were used for statistical assessments. The normal distribution of the data was evaluated with Shapiro–Wilk test. Numeric variables were presented as mean \pm standard deviation. In comparison between two groups, either the Student's *T*-test or the Mann–Whitney *U*-test was used. In comparison between three groups, analysis of variance (ANOVA) test was used for parametrically distributed data, and Kruskal–Wallis *H*-test was used for non-parametrically distributed data. Bonferroni correction was applied to paired comparisons. Categorical variables were presented as numbers and percentages. Chi-square test and Fisher's exact test were used for comparison of categorical data obtained from the combination of these levels. Independent predictors of HP positivity and gastritis severity with the presence of HP were examined using stepwise logistic regression analysis. Of the parameters that were found to be invariably associated with the outcome, but also in a strong relationship with certain other parameters, only the variables showing the strongest invariable association with the outcome ($P < 0.25$) were included in stepwise multivariable logistic regression analysis.^[14] The ROC curve analysis comparing the HP presence values against diagnostic discrimination was examined using AUC. In statistical analysis, $P < 0.05$ with 95% confidence interval and 5% margin of error was considered to be statistically significant.

RESULTS

Entire population findings

The study population included a total of 1289 patients, 438 HP (–) (34%) and 851 HP (+) (66%). The mean age of the patients was 52.0 ± 14.7 years. About 53.1% ($n = 684$) of the population was female. Around 35.8% ($n = 461$) of the patients had mild chronic gastritis, 44% ($n = 567$) had moderate chronic gastritis, and 20.2% ($n = 261$) had severe chronic gastritis. The stomach wall invasion stage of HP was “0” in 34% ($n = 438$) of the patients, “1” in 23% ($n = 296$) of the patients, “2” in 22.5% ($n = 290$) of the patients, and “3” in 20.6% ($n = 265$) of the patients. The atrophy level was “0” in 63.5% ($n = 818$) of the patients, “1” in 25.8% ($n = 332$) of the patients, “2” in 5.8% ($n = 75$) of the patients, and “3” in 5% ($n = 64$) of the patients. The level of IM was “0” in 71.6% ($n = 923$) of the patients, “1” in 13.4% ($n = 173$) of the patients, “2” in 9.4% ($n = 121$) of the patients, and “3” in 5.6% ($n = 72$) of the patients. The majority of the patients (20.2%, $n = 260$) had been diagnosed with pangastritis.

Comparison between HP groups

The mean age of HP (+) patients was higher compared with HP (–) patients (55.1 ± 15.2 vs 50.4 ± 15.8 ; $P < 0.001$). There were more female patients in HP (+) patient group (47.9% vs 55.7%; $P = 0.009$). All the patients with severe chronic gastritis were in HP (+) group, and the ratio of patients with moderate chronic gastritis was higher in HP (+) group (32% vs 50.2%; $P < 0.001$). In terms of HP invasion stage in HP (+) group, it was stage 1 in 34.8% ($n = 296$) of the patients, stage 2 in 34.1% ($n = 290$) of the patients, and stage 3 in 31.1% ($n = 265$) of the patients. The ratio of patients with an atrophy level of “0” was lower in HP (+) group compared with HP (–) group, whereas the ratio of patients with atrophy levels “1”–“3” was higher ($P < 0.001$). In terms of IM, the ratio of patients with IM level “0” was higher in HP (–) group, whereas the ratio of patients with IM level “1”–“3” was higher in HP (+) group.

Compared with HP (–) group, HP (+) group had a higher mean platelet ($240,719 \pm 57,554$ vs $269,700 \pm 77,804$; $P < 0.001$), MPV (10 ± 0.6 vs $9.1 \pm 0.99.1 \pm 0.9$; $P < 0.001$), mean neutrophil (4123 ± 1183 vs 4907 ± 1825 ; $P < 0.001$) levels, and a lower mean lymphocyte level (2365 ± 696 vs 1808 ± 577 ; $P < 0.001$). The mean PLR (107.4 ± 29.5 vs 168 ± 52.8 ; $P < 0.001$) and mean NLR (1.8 ± 0.6 vs 3.2 ± 1.4 ; $P < 0.001$) levels were higher in HP (+) group compared with HP (–) group. No significant difference was observed between the groups in terms of other laboratory findings [Table 1].

Combination groups

The ratio of HP (+) patients was higher in high-risk group compared with low- and medium-risk groups (91.4% vs 69.9% vs 35%; $P < 0.001$). HP invasion stage, the IM level, and the ratio of patients with atrophy level “3” were higher in high-risk group compared with low- and medium-risk groups ($P < 0.05$). The hemoglobin level was lower in high-risk group compared with low- and medium-risk groups (13 ± 2 vs 14 ± 2 vs 14 ± 2 ; $P < 0.001$). The PCT level was similar in medium- and high-risk groups, and lower in low-risk group (0.24 ± 0.08 vs 0.23 ± 0.06 vs 0.21 ± 0.04 ; $P < 0.001$). The WBC level was higher in high-risk group compared with the other two risk groups (8016 ± 2234 vs 6756 ± 1910 vs 7592 ± 1866 ; $P < 0.001$) [Table 2].

Regression analysis

The stepwise multivariable logistic regression analysis including variables found to be associated with HP (+) and $P < 0.25$ showed that being male [odds ratio (OR)=0.714; $P = 0.014$], having a high IM stage (stage 2: OR = 1.599; $P = 0.048$; stage 3: OR = 2.243; $P = 0.033$), and being

Table 1: Distribution of demographic and clinical findings according to HP presence

Variables	HP (-) n=438	HP (+) n=851	P
Age (years)	55.1±15.2	50.4±15.8	<0.001*
Gender, n (%)			
Female	210 (47.9)	474 (55.7)	0.009*
Male	228 (52.1)	377 (44.3)	
Pathological diagnosis, n (%)			
Mild chronic gastritis	298 (68)	163 (19.2)	<0.001*
Moderate chronic gastritis	140 (32)	427 (50.2)	
Severe chronic gastritis	0 (0)	261 (30.7)	
HP invasion grade, n (%)			
0	438 (100)	-	<0.001*
1	-	296 (34.8)	
2	-	290 (34.1)	
3	-	265 (31.1)	
Atrophy level, n (%)			
0	306 (69.9)	512 (60.2)	<0.001*
1	105 (24.0)	227 (26.7)	
2	14 (3.2)	61 (7.2)	
3	13 (3.0)	51 (6.0)	
Intestinal metaplasia, n (%)			
0	333 (76.0)	590 (69.3)	0.002*
1	58 (13.2)	115 (13.5)	
2	36 (8.2)	85 (10.0)	
3	11 (2.5)	61 (7.2)	
WBC (μL)	7371.5±2006.2	7475.5±2114.4	0.395
Neutrophil (μL)	4123±1183	4907±1825	<0.001*
Lymphocyte (μL)	2365±696	1808±577	<0.001*
Hemoglobin (g/dL)	14±2	14±2	0.135
Platelet (μL)	240719±57554	269700±77804	<0.001*
MPV (fl)	9.1±0.9	10±0.6	<0.001*
PDW (%)	16.6±0.7	16.7±1.2	0.193
PCT (%)	0.22±0.06	0.23±0.07	0.018*
PLR	107.4±29.5	168±52.8	<0.001*
NLR	1.8±0.6	3.2±1.4	<0.001*

Categorical variables are shown in numbers and percentage, and numerical variables are shown as mean±standard deviation. * $P < 0.05$ statistically significant. HP: *Helicobacter pylori*; WBC: White blood cell; MPV: Mean platelet volume; PDW: Platelet distribution width; PCT: Plateletcrit; PLR: Platelet-to-lymphocyte ratio; NLR: Neutrophil-to-lymphocyte ratio

in medium- (OR = 4.166; $P < 0.001$) and high-risk groups (OR = 19.312; $P < 0.001$) were independent predictors of the presence of HP [Table 3]. HP (+) patients had a higher AUC value according to their PLR–NLR combination components [Figure 1].

The regression model, which was created based on mild chronic gastritis group, showed that HP invasion stage, IM level, and PLR–NLR combination were predictors of a moderate risk of chronic gastritis. According to the model, when compared against HP invasion stage “0” patients, a moderate chronic gastritis risk was 2319 times higher for HP invasion stage “1” patients, 15,363 times higher for HP invasion stage “2” patients, and 40,798 times higher for HP invasion stage “3” patients. Compared with IM stage “0”

Table 2: Distribution of demographic and clinical findings according to risk groups

Variables	Low risk n=408	Intermediate risk n=452	High risk n=429	P
Age (years)	53±15 [‡]	50±14 ^{†*}	52±15 [‡]	0.011*
Gender, n (%)				
Female	190 (46.6)	281 (62.2)	213 (49.7)	<0.001*
Male	218 (53.4)	171 (37.8)	216 (50.3)	
Diagnosis, n (%)				
HP (-)	265 (65.0)	136 (30.1)	37 (8.6)	<0.001*
HP (+)	143 (35.0)	316 (69.9)	392 (91.4)	
Pathological diagnosis, n (%)				
Mild chronic gastritis	217 (53.2)	149 (33)	95 (22.1)	<0.001*
Moderate chronic gastritis	152 (37.3)	215 (47.6)	200 (46.6)	
Severe chronic gastritis	39 (9.6)	88 (19.5)	134 (31.2)	
HP invasion grade, n (%)				
0	265 (65.0)	136 (30.1)	37 (8.6)	<0.001*
1	54 (13.2)	103 (22.8)	139 (32.4)	
2	44 (10.8)	115 (25.4)	131 (30.5)	
3	45 (11.0)	98 (21.7)	122 (28.4)	
Atrophy level, n (%)				
0	271 (66.4)	301 (66.6)	246 (57.3)	0.002*
1	105 (25.7)	111 (24.6)	116 (27.0)	
2	16 (3.9)	26 (5.8)	33 (7.7)	
3	16 (3.9)	14 (3.1)	34 (7.9)	
Intestinal metaplasia, n (%)				
0	300 (73.5)	334 (73.9)	289 (67.4)	<0.001*
1	54 (13.2)	58 (12.8)	61 (14.2)	
2	41 (10.0)	44 (9.7)	36 (8.4)	
3	13 (3.2)	16 (3.5)	43 (10.0)	
WBC (μL)	7592±1866 ^{‡*}	6756±1910 ^{†*}	8016±2234 ^{†*}	<0.001*
Hemoglobin (g/dL)	14±2 [‡]	14±2 [‡]	13±2 ^{†*}	<0.001*
MPV (fl)	9.2±0.9	10.7±39.6	26.9±376.2	0.418
PDW (%)	16.6±0.7	16.6±1.5	16.5±0.6	0.285
PCT (%)	0.21±0.04 ^{‡*}	0.23±0.06 [†]	0.24±0.08 [†]	<0.001*

Categorical variables are shown in numbers and percentage, and numerical variables are shown as mean ± standard deviation. * $P < 0.05$ statistically significant. [†] $P < 0.05$ vs low risk (benferroni correction). [‡] $P < 0.05$ vs intermediate risk (benferroni correction). ^{‡*} $P < 0.05$ vs high risk (benferroni correction). HP: *Helicobacter pylori*; WBC: White blood cell; MPV: Mean platelet volume; PDW: Platelet distribution width; PCT: Plateletcrit

patients, a moderate chronic gastritis risk was 2029 times higher for IM stage “1” patients, 2476 times higher for IM stage “2” patients, and 4632 times higher for IM stage “3” patients. Compared with low-risk group according to PLR–NLR combination, the moderate chronic gastritis risk was 2061 times higher for medium-risk group and 3006 times higher for high-risk group [Table 3].

The regression model, which was created based on moderate chronic gastritis group, showed that HP invasion stage, IM level, and PLR–NLR combination were predictors of a severe risk for chronic gastritis. According to the model; when compared with HP invasion stage “0” patients, a severe chronic gastritis risk was 2372 times higher for HP

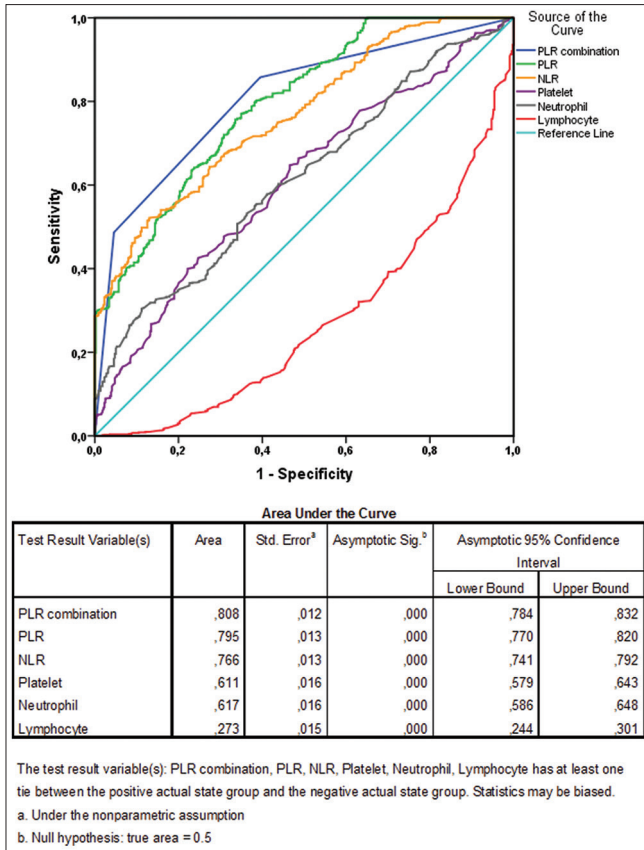


Figure 1: ROC curve analysis comparing the *Helicobacter pylori* presence values against diagnostic discrimination

invasion stage “1” patients, 17,151 times higher for HP invasion stage “2” patients, and 45,089 times higher for HP invasion stage “3” patients. Compared with IM stage “0” patients, a severe chronic gastritis risk was 11,704 times higher for IM stage “3” patients. Compared with low-risk group according to PLR–NLR combination, a severe chronic gastritis risk was 1585 times higher for medium-risk group and 2260 times higher for high-risk group [Table 3].

DISCUSSION

The role of PLR–NLR combination, a non-invasive marker, in prediction of the presence of HP and gastrointestinal system complications associated with HP was examined for the first time in this study. The ratio of patients with moderate and severe chronic gastritis was higher in HP (+) group than HP (–) group. The ratio of patients with levels 1–3 atrophy and IM was higher in HP (+) group. Compared with HP (–) group, HP (+) group had a higher mean platelet, neutrophil, PLR, and NLR levels, as well as a lower mean lymphocyte level. The ratio of HP (+) patients was higher in high-risk group compared with low- and medium-risk groups. HP invasion stage, IM level, and the ratio of patients with atrophy level “3” were higher

Table 3: Factors associated with HP presence and gastritis severity

Variables	OR	95% CI		P
		Lower	Upper	
HP (+) (ref: negative)				
Male (ref: female)	0.714	0.545	0.935	0.014*
Intestinal metaplasia (ref: 0)				
1	1.139	0.766	1.694	0.52
2	1.599	1.010	2.555	0.048*
3	2.243	1.067	4.715	0.033*
PLR–NLR combination (ref: low risk)				
Intermediate risk	4.166	3.119	5.564	<0.001*
High risk	19.312	12.993	28.704	<0.001*
Nagelkerke R=0.496; P<0.001*				
Moderate chronic gastritis (ref: mild gastritis)				
HP invasion grade (ref: 0)				
1	2.319	1.703	3.158	<0.001*
2	15.363	9.505	24.83	<0.001*
3	40.798	17.525	94.973	<0.001*
Intestinal metaplasia (ref: 0)				
1	2.029	1.348	3.053	0.001*
2	2.476	1.866	5.211	<0.001*
3	4.632	2.61	8.221	<0.001*
PLR–NLR combination (ref: low risk)				
Intermediate risk	2.061	1.535	2.765	<0.001*
High risk	3.006	2.182	4.14	<0.001*
Nagelkerke R = 0.367; P<0.001*				
Severe chronic gastritis (ref: moderate gastritis)				
HP invasion grade (ref: 0)				
1	2.372	1.726	3.261	<0.001*
2	17.151	10.526	27.947	<0.001*
3	45.089	19.262	105.542	<0.001*
Intestinal metaplasia (ref: 0)				
1	0.615	0.371	1.018	0.058
2	1.536	0.975	2.42	0.064
3	11.704	5.922	23.13	<0.001*
PLR–NLR combination (ref: low risk)				
Intermediate risk	1.585	1.012	2.482	0.044*
High risk	2.26	1.46	3.498	<0.001*
Nagelkerke R=0.329; P<0.001*				

* P<0.05 statistically significant. OR: Odds ratio; 95% CI: 95% confidence interval; HP: *Helicobacter pylori*; PLR: Platelet-to-lymphocyte ratio; NLR: Neutrophil-to-lymphocyte ratio

in high-risk group compared with low- and medium-risk groups. Regression analysis showed that PLR–NLR combination was an independent risk factor for both HP presence and moderate and severe chronic gastritis.

Our extensive literature review showed that HP (+) patients also had higher rates of severe gastritis, chronic atrophy, and IM.^[15-21] In one study, the antral atrophy rate was found to be considerably higher in HP (+) patients compared with HP (–) patients.^[21] Fatema *et al.* found the rates for chronic gastritis and pangastritis to be higher in HP (+) compared to HP (–) group.^[19] In a study conducted in Mexico, patients with IM were given HP

eradication treatment. One year following the treatment, it was observed that IM regressed in 54.3% of the patients.^[22] This shows that HP is closely related with IM. Similar to the literature, we too had found higher rates of severe gastritis, chronic atrophy, and IM in HP (+) cases. Moreover, it was observed that gastritis severity, atrophy level, and IM level increased considerably as HP invasion increased. This shows that all these three gastrointestinal complications are directly related to HP.

We previously mentioned that gastrointestinal or non-gastrointestinal complications associated with HP occur through the activation of immune system through various pathways. The vascular endothelial growth factor and TNF α level, which are both inflammatory markers, were found to be higher in HP (+) patients compared with HP (–) patients in a study conducted by Siregar *et al.*, which supports this idea.^[23] Farah *et al.* found the NLR level to be higher in HP (+) patients compared with HP (–) patients.^[24] Also, the NLR level was observed to increase as the severity of gastritis associated with HP increased. The results of our study are similar to the study mentioned above. Also, we found that NLR level was related to chronic atrophy and IM. Moreover, we found that as a result of our risk classification, an NLR value above 2.42—whether on its own (medium risk) or in combination with PLR (high risk)—was a predictor of both HP presence and severe chronic gastritis. These results show that NLR and PLR–NLR combination are good non-invasive markers that can be used to determine HP presence and chronic gastritis associated with HP.

It was shown in previous studies that platelet activation was involved in most gastrointestinal or non-gastrointestinal complications that occur because of altered immunity secondary to HP.^[25] Platelet count, MPV, PDW, and PCT levels are expected to change because of platelet activation. Matsukawa *et al.* found platelet level to be higher in HP (+) compared with HP (–) patients.^[26] In a different study conducted by the same team, HP (+) patients were given eradication treatment, and a significant decrease was observed in platelet counts of the patients 8 weeks after the treatment.^[27] Similar to the studies mentioned above, the platelet count was found to be higher in HP (+) group in our study as well. Umit and Umit found MPV to be higher in HP (+) compared with HP (–) group.^[3] The results of our study are similar to the study mentioned above. We were not able to find any study involving other platelet indices. In our study, PDW and PCT values were higher in HP (+) group compared with HP (–) group, albeit not statistically significant.

Farah *et al.* found PLR level to be higher in HP (+) compared to HP (–) group.^[24] In the same study, PLR level was found to be correlated with symptoms associated with HP. Similarly, PLR level was found to be higher in HP (+) group in our study as well. Also, we found that PLR level was related to chronic atrophy and IM. Moreover, we found as a result of our risk classification that a PLR value of above 116.2—whether on its own (medium risk) or in combination with NLR (high risk)—was a predictor of both HP presence and moderate and severe chronic gastritis. These results show that PLR and PLR–NLR combination are good non-invasive markers that can be used to determine the presence of HP and chronic gastritis associated with it.

The main limitations of our study include its retrospective design and the fact that the role of PLR–NLR combination, a non-invasive marker, in prediction of HP presence and gastrointestinal system complications associated with HP was not tested in clinical practice. Another limitation of our study was that PLR–NLR combination was not compared with other non-invasive markers used to determine HP presence.

In conclusion, we found PLR–NLR combination to be a good predictor of HP presence and gastrointestinal complications associated with HP (moderate and severe gastritis). We believe that PLR–NLR combination will be a great convenience for clinicians as an easy-to-use, easily accessible, and inexpensive index.

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Conflicts of interest

There are no conflicts of interest.

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