

Clinicopathological Features of Biopsy-Proven Foveolar Hyperplasia in the Absence of Hypertrophic Gastropathy

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ABSTRACT

Foveolar hyperplasia is a common histopathological finding in gastric biopsies; however, its clinical relevance remains unclear, particularly in the absence of overt hypertrophic gastropathy or Ménétrier's disease. This retrospective study aimed to evaluate the clinical, laboratory, histopathological, and radiological characteristics of patients with foveolar hyperplasia detected in gastric corpus biopsies obtained from endoscopically normal or mildly edematous mucosa. Patients who underwent upper gastrointestinal endoscopy between 2022 and 2025 and had corpus biopsies were included. Those with histologically confirmed foveolar hyperplasia were compared with age- and sex-matched controls without foveolar hyperplasia. Clinical data, laboratory parameters, endoscopic findings, and computed tomography results were reviewed and analyzed. A total of 352 patients were included, comprising 178 patients with foveolar hyperplasia and 174 controls. Platelet counts were significantly higher in the foveolar hyperplasia group, whereas hemoglobin, C-reactive protein, ferritin, and albumin levels did not differ significantly between groups. *Helicobacter pylori* positivity and glandular atrophy were significantly less frequent in patients with foveolar hyperplasia. Multivariate logistic regression analysis identified higher platelet count and *H. pylori* negativity as independent factors associated with foveolar hyperplasia, although receiver operating characteristic analysis demonstrated limited diagnostic performance. These findings suggest that foveolar hyperplasia may be encountered incidentally in gastric biopsies without clinical or endoscopic evidence of hypertrophic gastropathy and does not appear to be associated with systemic inflammation or nutritional impairment. The clinical significance of its association with platelet elevation and *H. pylori* negativity remains uncertain.

Key words: Foveolar Hyperplasia. Hypertrophic Gastropathy. Endoscopy. Clinicopathology.

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ÖZET

Foveolar hiperplazi, gastrik biyopsilerde sık karşılaşılan bir histopatolojik bulgu olmasına rağmen, özellikle belirgin hipertrofik gastropati veya Ménétrier hastalığı yokluğunda klinik önemi net değildir. Bu retrospektif çalışmada, endoskopik olarak normal veya hafif ödemli korpus mukozasından alınan biyopsilerde saptanan foveolar hiperplazisi olan hastaların klinik, laboratuvar, histopatolojik ve radyolojik özelliklerinin değerlendirilmesi amaçlandı. 2022–2025 yılları arasında üst gastrointestinal endoskopi yapılan ve gastrik korpus biyopsisi alınan hastalar çalışmaya dahil edildi. Histolojik olarak foveolar hiperplazi saptanan hastalar, yaş ve cinsiyet açısından benzer foveolar hiperplazisi olmayan kontrol grubu ile karşılaştırıldı. Klinik veriler, laboratuvar parametreleri, endoskopik bulgular ve bilgisayarlı tomografi sonuçları analiz edildi. Toplam 352 hasta çalışmaya alındı; bunların 178'inde foveolar hiperplazi mevcuttu. Foveolar hiperplazi grubunda trombosit sayısı anlamlı olarak daha yüksekken, hemoglobin, C-reaktif protein, ferritin ve albümin düzeyleri açısından gruplar arasında anlamlı fark saptanmadı. *Helicobacter pylori* pozitifliği ve bez atrofisi foveolar hiperplazisi olan hastalarda daha düşük orandaydı. Çok değişkenli analizde trombosit sayısı ve *H. pylori* negatifliği foveolar hiperplazi ile bağımsız olarak ilişkili bulundu; ancak tanılabilir performans sınırlıydı. Bu bulgular, foveolar hiperplazinin hipertrofik gastropati bulguları olmaksızın gastrik biyopsilerde tesadüfen saptanabilen, sistemik inflamasyon veya beslenme bozukluğu ile ilişkili olmayan benign bir değişiklik olabileceğini düşündürmektedir.

Anahtar Kelimeler: Foveolar Hiperplazi. Hipertrofik Gastropati. Endoskopi. Klinikpatoloji.

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Foveolar hyperplasia is a histopathological finding characterized by elongation and branching of the gastric pits due to an increased proliferation of surface mucous (foveolar) epithelial cells within the gastric mucosa¹. Microscopically, it manifests as an expansion of the superficial mucosal compartment, accompanied by columnar epithelial hyperplasia and occasional stromal compaction². Clinically, foveolar hyperplasia is often associated with hypertrophic gastropathies, most notably Ménétrier's disease, where it coexists with features such as glandular atrophy, mucosal thickening, protein-losing

gastropathy, and hypoalbuminemia³⁻⁵. However, the presence of foveolar hyperplasia is not specific to Ménétrier's disease and can also occur in a variety of benign and reactive gastric conditions. These include chronic active gastritis, chemical (reactive) gastropathy, *H. pylori*-related surface inflammation, portal hypertensive gastropathy, and other hyperplastic or reparative states^{6,7}. In such cases, foveolar proliferation may be localized or diffuse, and does not necessarily imply a neoplastic process⁸. From a molecular standpoint, foveolar hyperplasia is considered a non-neoplastic lesion. Experimental models have shown that activation of the KRAS pathway in isthmal progenitor cells can induce foveolar expansion without triggering metaplasia or neoplastic transformation^{9,10}. Thus, foveolar hyperplasia may reflect a proliferative, yet biologically indolent, remodeling process within the gastric epithelium¹¹.

Despite its frequent occurrence in gastric biopsies, the clinical relevance of foveolar hyperplasia in patients without a formal diagnosis of Ménétrier's disease remains unclear¹². There is limited data on whether this histological finding correlates with laboratory abnormalities (e.g., anemia, hypoalbuminemia, elevated inflammatory markers), endoscopic appearance, gastric wall thickening on imaging, or the presence of accompanying mucosal inflammation. Clarifying the clinical, biochemical, and endoscopic correlates of non-Ménétrier foveolar hyperplasia may help determine its significance in routine practice.

Materials and Methods

This retrospective descriptive study was conducted at the Department of Gastroenterology, Ondokuz Mayıs University Faculty of Medicine Hospital. A total of 9011 upper gastrointestinal endoscopies were performed between January 2022 and December 2025. Among these, patients with available corpus biopsy specimens were identified, and pathology records were retrospectively reviewed.

Patients with clinical, endoscopic, or histopathological findings suggestive of Ménétrier's disease were excluded. Specifically, individuals with giant gastric folds, marked hypoalbuminemia, documented protein-losing gastropathy, or histological features consistent with Ménétrier's disease were not included in the study population.

The diagnosis of foveolar hyperplasia was established based on characteristic histopathological features, including elongation and branching of gastric pits, expansion of the superficial mucosal compartment, and increased proliferation of foveolar epithelium without evidence of dysplasia. All biopsy specimens were evaluated by experienced gastrointestinal pathologists according to standard diagnostic criteria.

Cases in which the diagnosis of foveolar hyperplasia was documented in corpus biopsy specimens were included in the study group (n=178). The control group consisted of patients from the same period who had normal-appearing corpus mucosa on endoscopy, underwent biopsy from the same region, and showed no evidence of foveolar hyperplasia on histopathological evaluation (n=174). Control patients were retrospectively selected to be comparable in terms of age, sex, and comorbidities, without strict one-to-one matching (Figure 1).

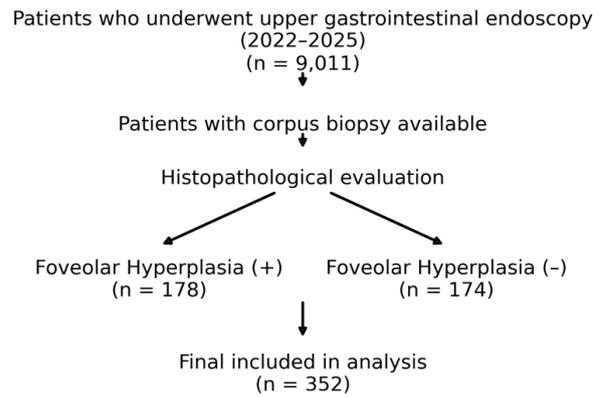


Figure 1.
Flow Diagram

For both groups, clinical presentation, demographics (age, sex, smoking status), and laboratory findings (hemoglobin, albumin, C-reactive protein (CRP), ferritin, iron, neutrophil and platelet counts) were extracted from the hospital information system. In addition, endoscopic findings, histopathological features, and abdominal imaging results (especially the presence of gastric wall thickening on computed tomography (CT)) were reviewed and analyzed.

This study was approved by the Ethics Committee of Ondokuz Mayıs University Faculty of Medicine (approval number: 671/2025).

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Kolmogorov–Smirnov and Shapiro–Wilk tests along with histogram and Q–Q plots. Data were presented as mean ± standard deviation (SD) for normally distributed variables or as median (min–max) for non-normally distributed variables. Categorical variables were expressed as numbers and percentages. For comparisons between groups, the independent samples t-test or the Mann–Whitney U test was used for continuous variables, and the chi-square or Fisher's exact test was applied for categorical variables, as appropriate. Receiver

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operating characteristic (ROC) curve analysis was conducted to assess the diagnostic performance of selected laboratory markers. Multivariate logistic regression analysis was performed to identify independent factors associated with foveolar hyperplasia. Variables with $p < 0.10$ in univariate analyses or with known clinical relevance were included in the model. A two-sided p -value < 0.05 was considered statistically significant.

Results

A total of 352 patients who underwent endoscopy, of whom 178 (50.6%) had histologically confirmed foveolar hyperplasia, were included in the analysis. The mean age of patients with foveolar hyperplasia was 60.1 ± 14.4 years, compared to 57.8 ± 14.0 years in those without. Female sex was slightly more common in the foveolar group (58.4% vs. 57.8%). Current smoking was significantly less frequent among patients with foveolar hyperplasia (5.6% vs. 13.9%), while the proportion of never and former smokers did not differ significantly. The most common presenting complaint in both groups was dyspeptic symptoms, followed by reflux symptoms, weight loss, screening endoscopy, nausea/vomiting, and anemia. No statistically significant differences were observed in the distribution of presenting symptoms between the groups (Table I).

Table I. Demographic and Clinical Characteristics

Characteristic	Foveolar Hyperplasia (+)	Foveolar Hyperplasia (-)	p-value
Demographics			
Age (mean \pm SD)	60.1 \pm 14.4	57.8 \pm 14.0	0.1124
Female, n (%)	104 (58.4%)	96 (57.8%)	0.9980
Smoking Status			
Current smoker, n (%)	10 (5.6%)	23 (13.9%)	0.0160
Never smoked, n (%)	123 (69.1%)	105 (63.3%)	0.2479
Former smoker, n (%)	45 (25.3%)	38 (22.9%)	0.6281
Presenting Complaint			
Dyspeptic symptoms, n (%)	40 (22.5%)	38 (22.9%)	0.9312
Reflux symptoms, n (%)	22 (12.4%)	18 (10.8%)	0.6924
Weight loss, n (%)	18 (10.1%)	17 (10.2%)	0.9782
Screening endoscopy, n (%)	16 (9.0%)	12 (7.2%)	0.5532
Nausea/vomiting, n (%)	14 (7.9%)	9 (5.4%)	0.3921
Anemia, n (%)	12 (6.7%)	10 (6.0%)	0.7789

Comparison between patients with and without Foveolar Hyperplasia

Glandular atrophy and *H. pylori* positivity were both significantly less frequent in the foveolar hyperplasia

group (84.3% vs. 92.8% and 15.7% vs. 33.1%, respectively). Inflammation was observed in nearly all patients in both groups. Gastropathy was more frequently reported in the non-foveolar group (97.6% vs. 83.1%). Gastric wall thickening on CT was significantly less common in patients with foveolar hyperplasia (2.4% vs. 10.3%) (Table II).

Platelet count ($\times 10^3/\mu\text{L}$) was significantly higher in patients with foveolar hyperplasia (295.2 ± 406.4 vs. 236.9 ± 91.3). Other laboratory parameters including white blood cell (WBC), neutrophils, hemoglobin, CRP, albumin, ferritin, iron, sedimentation rate, and neutrophil-to-platelet ratio showed no statistically significant differences between groups (Table III).

Diagnostic performance analysis showed that platelet count had the highest area under the curve (AUC=0.571, 95% CI: 0.510–0.632, $p=0.022$). In contrast, CRP (AUC=0.563, $p=0.170$), albumin (AUC=0.458, $p=0.253$), and the neutrophil-to-platelet ratio (AUC=0.455, $p=0.149$) did not demonstrate statistically significant discriminatory ability. Overall, the diagnostic performance of laboratory markers remained limited (Figure 2, Table IV).

Table II. Histopathological and Imaging Findings

Feature	Foveolar Hyperplasia (+)	Foveolar Hyperplasia (-)	p-value
Histopathological Findings			
Glandular atrophy, n (%)	150 (84.3%)	154 (92.8%)	0.022
Inflammation, n (%)	176 (98.9%)	166 (100.0%)	0.5092
<i>H. pylori</i> positive, n (%)	28 (15.7%)	55 (33.1%)	<0.001
Gastropathy reported, n (%)	148 (83.1%)	162 (97.6%)	<0.001
Imaging Findings			
Gastric wall thickening (CT), n (%)	2 (2.4%)	8 (10.3%)	0.0416

Abbreviations: CT, computed tomography; *H. pylori*, *Helicobacter pylori*.

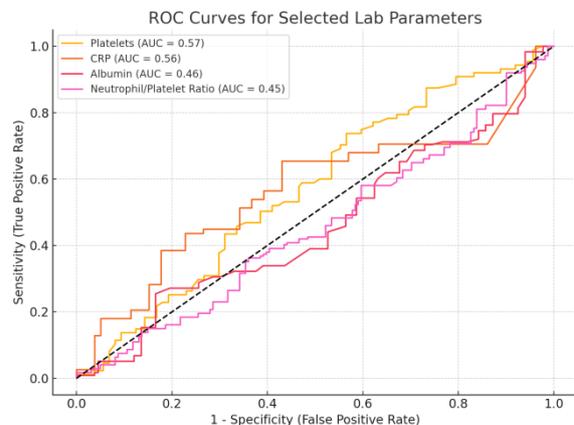


Figure 2. ROC Curves for Selected Lab Parameters

Table III. Laboratory Parameters

Parameter	Foveolar Hyperplasia (+)	Foveolar Hyperplasia (-)	p-value
Laboratory Parameters			
WBC ($\times 10^3/\mu\text{L}$)	7597.20 \pm 6261.65 (n=174)	7048.14 \pm 4002.64 (n=163)	0.3153
Neutrophils ($\times 10^3/\mu\text{L}$)	4764.51 \pm 4904.25 (n=174)	4380.78 \pm 2789.61 (n=164)	0.7074
Hemoglobin (g/dL)	12.66 \pm 2.43 (n=174)	12.19 \pm 2.08 (n=163)	0.1279
Platelets ($\times 10^3/\mu\text{L}$)	295.2 \pm 406.3 (n=175)	236.9 \pm 91.30 (n=161)	0.0243
Iron ($\mu\text{g/dL}$)	52.28 \pm 20.42 (n=26)	65.72 \pm 30.53 (n=19)	0.1505
Ferritin (ng/mL)	1635.08 \pm 4192.87 (n=22)	766.69 \pm 1364.60 (n=19)	0.5213
Sedimentation (mm/h)	38.30 \pm 14.99 (n=24)	36.88 \pm 15.32 (n=19)	0.7462
CRP (mg/L)	50.60 \pm 92.81 (n=63)	31.75 \pm 67.88 (n=56)	0.3636
Albumin (g/dL)	4.08 \pm 0.70 (n=32)	3.73 \pm 1.05 (n=10)	0.2427
Neutrophil/Platelet ratio	0.015 \pm 0.017 (n=173)	0.018 \pm 0.023 (n=160)	0.2246

Comparison of laboratory values between patients with and without Foveolar Hyperplasia
 Abbreviations: WBC, white blood cell; CRP, C-reactive protein; NLR, neutrophil-to-platelet ratio; SD, standard deviation.

Table IV. ROC AUC Results

Variable	AUC	95% CI	p-value	n
Platelets	0.571	0.510–0.632	0.0222	336
CRP	0.563	0.473–0.653	0.1702	157
Albumin	0.458	0.387–0.530	0.2525	251
Neutrophil/Platelet Ratio	0.455	0.393–0.516	0.1490	335

Abbreviations: ROC, receiver operating characteristic; AUC, area under the curve; CI, confidence interval.

In multivariate logistic regression, platelet count (p=0.019) and negative *H. pylori* status (p=0.034) were found to be independent predictors of foveolar hyperplasia. Female sex and glandular atrophy were not statistically significant predictors (Table V).

Table V. Logistic Regression Results

Variable	β (Coef)	p-value	OR (Exp(β))	95% CI (Exp(β))
Intercept	-0.435	0.660	0.647	(0.093 – 4.498)
Platelets (PLT)	0.000005	0.019	1.000	(1.000 – 1.000)
Female sex	-0.171	0.646	0.842	(0.406 – 1.749)
<i>H. pylori</i> (+)	-0.932	0.034	0.394	(0.167 – 0.932)
Glandular atrophy	+0.868	0.119	2.383	(0.800 – 7.104)

Independent predictors of Foveolar Hyperplasia
 Abbreviations: OR, odds ratio; CI, confidence interval.

Discussion and Conclusion

In this retrospective study of patients with biopsy-proven foveolar hyperplasia, we found that platelet counts were significantly higher and *Helicobacter pylori* positivity was significantly lower in the foveolar hyperplasia group, whereas most clinical symptoms and systemic inflammatory markers did not differ between groups. Histopathological features such as glandular atrophy were also less frequent in these patients. These findings suggest that foveolar hyperplasia, when identified in endoscopically normal or mildly edematous corpus mucosa, may represent a localized histological alteration rather than a manifestation of overt inflammatory or hypertrophic gastropathy.

Foveolar hyperplasia is a common histopathological finding in gastric biopsies, but its clinical significance often remains uncertain^{1,13}. Although it is frequently observed in protein-losing gastropathies such as Ménétrier’s disease, it may also appear in various clinical contexts as an isolated finding. In this study, we investigated the presence of foveolar hyperplasia in patients with hypertrophic gastric folds identified during endoscopy but without a diagnosis of Ménétrier’s disease, and we comparatively evaluated the clinical, laboratory, and radiological characteristics of these patients^{14,15}. There are limited studies in the literature specifically addressing this condition³.

Foveolar hyperplasia is a commonly reported histopathological finding in gastric biopsy samples, yet its clinical significance in patients without overt hypertrophic gastropathy remains unclear¹⁶. In our cohort, all patients had biopsies taken from the gastric corpus mucosa, which appeared either normal or mildly edematous during endoscopy. The reported prevalence of foveolar hyperplasia in routine gastric biopsies varies widely in the literature, ranging from 10% to 30%, depending on the population and indication for endoscopy^{13,17}. In our analysis, the presence of foveolar hyperplasia did not correlate significantly with age, sex, or smoking status. Similarly, common presenting complaints such as dyspepsia or reflux symptoms were not found to differ between groups. These findings suggest that foveolar hyperplasia, when identified in otherwise endoscopically unremarkable mucosa, does not exhibit a distinct clinical pattern.

Histopathological findings such as glandular atrophy, inflammation, and *H. pylori* infection are often evaluated alongside foveolar hyperplasia, particularly in the context of hypertrophic gastropathies¹⁸. In our study, glandular atrophy was observed less frequently in patients with foveolar hyperplasia compared to those without, reaching statistical significance. Similarly, *H. pylori* positivity was significantly lower

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in the foveolar hyperplasia group. While inflammation was present in the vast majority of patients in both groups, the absence of a significant difference suggests that foveolar hyperplasia may not directly reflect the degree of mucosal inflammation. Gastropathy was reported less frequently in foveolar cases, and radiological assessment showed a lower rate of gastric wall thickening on CT in this group, supporting the view that foveolar hyperplasia may occur independently of gross inflammatory or hypertrophic changes.

One of the most noteworthy findings in our study was the inverse association between *Helicobacter pylori* infection and foveolar hyperplasia. While several studies in the literature have reported a positive correlation between *H. pylori* colonization and mucosal changes such as surface hyperplasia or increased mucin expression^{13,19,20}, other reports have noted conflicting or even negative associations, particularly when analyzing specific histological subtypes^{20,21}. *H. pylori* produces urease, an enzyme that locally increases pH in the gastric submucosa, thereby mitigating the damaging effects of gastric acid exposure. This buffering effect has been proposed to protect against acid-induced mucosal injury²²⁻²⁴. Conversely, it has been hypothesized that sustained exposure to gastric acidity may stimulate mucinous differentiation and foveolar cell proliferation, potentially contributing to the development of foveolar hyperplasia in *H. pylori*-negative settings^{25,26}. In this context, the lower prevalence of *H. pylori* in our foveolar hyperplasia group may suggest a non-inflammatory, acid-mediated proliferative mechanism, although causality cannot be established in this retrospective setting. Additional studies, including mechanistic or molecular investigations, are needed to better elucidate the role of acid-mucin interactions in foveolar remodeling.

In the comparison of laboratory parameters, platelet count was significantly higher in patients with foveolar hyperplasia, while other markers such as hemoglobin, CRP, albumin, ferritin, iron, and neutrophil counts did not differ significantly between groups. The neutrophil-to-platelet ratio, an emerging indicator in various inflammatory conditions, also showed no significant association with foveolar hyperplasia in this cohort. These findings suggest that, aside from a mild thrombocytosis, foveolar hyperplasia does not appear to correlate with systemic inflammatory or nutritional markers, further supporting its potentially localized and non-reactive nature. Although hypoalbuminemia was not observed in our population, previous case reports in Ménétrier's disease have linked protein-losing gastropathy to hypercoagulability and reactive thrombocytosis²⁷. Whether the mild elevation in platelet count observed in our study reflects a subtle compensatory response

or a coincidental laboratory variation remains unclear and warrants further investigation.

Diagnostic performance analysis showed that platelet count had the highest area under the curve (AUC=0.571, 95% CI: 0.510–0.632, p=0.022) among the laboratory parameters evaluated; however, its discriminatory power remained modest. CRP, albumin, and the neutrophil-to-platelet ratio did not demonstrate statistically significant discriminatory ability. Although platelet count reached statistical significance in multivariate analysis, its diagnostic performance was limited, indicating that the clinical utility of this finding is weak and should be interpreted cautiously. In multivariate logistic regression, higher platelet count and negative *H. pylori* status emerged as independent factors associated with the presence of foveolar hyperplasia. Other variables such as female sex and glandular atrophy were not independently significant. These findings should be interpreted in the context of the retrospective design of the study.

In this study, we evaluated the clinical, laboratory, histopathological, and radiological characteristics of patients with biopsy-proven foveolar hyperplasia in the absence of overt hypertrophic gastropathy or a diagnosis of Ménétrier's disease. Our findings suggest that foveolar hyperplasia is a relatively common histological finding that does not appear to correlate with specific clinical symptoms or systemic inflammatory markers. Platelet count and *H. pylori* negativity were independently associated with foveolar hyperplasia, although their diagnostic performance was limited. These results support the view that foveolar hyperplasia, when identified incidentally in gastric biopsies, may represent a benign and localized mucosal change rather than a marker of significant pathology. Further prospective studies are needed to better define its clinical implications.

Limitations

This study has several limitations. First, the retrospective design limits the ability to establish causal relationships between foveolar hyperplasia and the associated clinical or laboratory findings. As all data were obtained from existing records, variability in biopsy sampling, histopathological reporting, and documentation of clinical features may have introduced information bias. The absence of central pathology review and interobserver variability among reporting pathologists may also have influenced diagnostic consistency. Additionally, since most patients were evaluated in an outpatient setting, detailed information regarding medication use was not consistently available. In particular, exposure to

proton pump inhibitors (PPIs), which may alter gastric acidity and potentially influence mucosal remodeling, could not be reliably assessed. This limitation may have introduced residual confounding in interpreting the proposed acid-mediated mechanism. Furthermore, laboratory parameters such as ferritin and iron levels were not available for all patients, which may have limited the power to detect associations. Lastly, as the study was conducted in a single tertiary center, the generalizability of the findings may be limited.

Researcher Contribution Statement

Idea and design: Ö.K., M.S.; Data collection and processing: Ö.K., U.A.; Analysis and interpretation of data: M.S., U.A.; Writing of significant parts of the article: Ö.K., M.S.; Critical revision of the manuscript: U.A., A.B.; Final review and approval of the manuscript: A.B.

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There are no acknowledgements to declare.

Conflict of Interest Statement

The authors of the article have no conflict of interest declarations.

Ethics Committee Approval Information

Approving Committee: Ondokuz Mayıs University Clinical Research Ethics Committee

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