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Clinical and histopathological evaluation of oesophageal inlet patch: A case-control study

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Abstract

The oesophageal inlet patch (OIP) is a congenital heterotopic gastric mucosal anomaly located in the upper oesophagus and is often incidentally detected during endoscopy. Despite its conventionally benign classification, the potential associations of this condition with gastrointestinal pathology and systemic biochemical changes remain unclear. The objective of this study was to evaluate the clinical, biochemical and histopathological characteristics of patients with OIP in comparison to a control group. A retrospective case-control study was conducted at a tertiary care centre. Patients presenting with dyspeptic complaints who underwent upper gastrointestinal endoscopy were reviewed. The study group comprised fifty patients with endoscopically identified oesophageal inlet patches, while the control group consisted of fifty age- and symptom-matched patients without inlet patches. A comparative analysis was conducted of demographic characteristics, haematological and biochemical parameters, and histopathological findings from gastric antrum and duodenal biopsies between the groups. There were no statistically significant differences between the groups in terms of gender distribution or mean age ($p>0.05$). The majority of haematological and biochemical parameters showed no significant differences; however, mean corpuscular volume, direct bilirubin, albumin, ferritin ($p=0.000$), and CRP levels ($p<0.05$) differed significantly between the groups. The positivity rate for Helicobacter pylori was found to be considerably higher in the OIP group (compared to the control group ($p<0.05$). Additionally, the incidence of intestinal metaplasia was found to be significantly higher in the OIP group ($p<0.05$). The rates of atrophic gastritis and coeliac disease-related findings did not differ significantly. Patients with oesophageal inlet patch exhibit distinct histopathological features, including a higher prevalence of Helicobacter pylori infection and intestinal metaplasia. These findings suggest that OIP may be associated with widespread changes in the gastric mucosa. Further prospective studies are required to clarify the clinical significance and long-term implications of this condition.

Keywords: Oesophageal inlet patch, upper gastrointestinal endoscopy, dyspepsia, helicobacter pylori, intestinal metaplasia

Introduction

The oesophageal inlet patch (OIP), also referred to as heterotopic gastric mucosa of the upper oesophagus, is a congenital anomaly characterised by the presence of ectopic gastric mucosal tissue, most commonly located just below the upper oesophageal sphincter. The inlet patch was initially described by Schmidt in 1805 and subsequently defined in more detail by Stoerk in 1896. However, it remains a frequently overlooked entity during upper gastrointestinal endoscopy due to its typical location and often subtle endoscopic appearance [1].

The prevalence of OIP reported in the literature varies significantly, ranging from 0.1% to 10%, depending to a great extent on the endoscopist's vigilance and the use of high-definition endoscopic techniques with careful inspection of the proximal oesophagus. The true prevalence of the condition may be underestimated in routine clinical practice, according to studies employing narrow-band imaging (NBI) or chromoendoscopy [2].

Etiologically, OIP is believed to result from incomplete squamous epithelialization of the proximal oesophagus during embryogenesis, leaving behind islands of columnar epithelium with gastric differentiation. Histologically, these patches most commonly contain oxyntic (acid-secreting) mucosa,

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though cardiac and antral types have also been documented. Physiologically, the inlet patch consists of heterotopic gastric mucosa that may include functional parietal cells capable of secreting hydrochloric acid. This ectopic acid secretion has the potential to disrupt the local squamous epithelium of the proximal oesophagus, which may result in chronic irritation, inflammation, and even metaplastic changes in adjacent tissues. Furthermore, the absence of protective mechanisms such as mucus and bicarbonate secretion, which generally accompany gastric acid in the stomach, may render the oesophageal mucosa particularly sensitive to acid-related injury when exposed to ectopic secretion from inlet patches. Although acid secretion from these ectopic glands is typically asymptomatic, it can lead to local mucosal irritation and downstream pathologies [3].

Clinically, the condition is usually asymptomatic, and the patch is often an incidental finding. However, in some instances, OIP has been associated with globus sensation, chronic cough, sore throat, hoarseness, dysphagia, or even odynophagia (Smith et al., 2022). Complications like ulceration, strictures and malignant transformation to adenocarcinoma have been reported, although that is rare [4].

From a diagnostic perspective, OIP manifests as a well-circumscribed, flat or slightly elevated, salmon-coloured patch in the proximal oesophagus, typically located 15–20 cm from the incisors. The use of advanced imaging techniques, including narrow-band imaging (NBI), magnification endoscopy, and targeted biopsies, has been shown to enhance diagnostic efficacy. Histopathological examination is a definitive diagnostic tool, often revealing gastric-type mucosa with or without parietal cells [5].

There is currently no standard treatment for asymptomatic OIP. For patients presenting with symptoms, proton pump inhibitors (PPIs) are frequently used to reduce acid-related symptoms. In cases that are more persistent or complicated, endoscopic ablation techniques, including argon plasma coagulation (APC), radiofrequency ablation, or even endoscopic mucosal resection, may be considered [6].

The systemic effects of OIP remain to be fully clarified. However, the potential for acid secretion and the occurrence of mucosal changes may render patients susceptible to proximal oesophageal inflammation. Furthermore, the presence of the condition could be indicative of a more extensive mucosal weakness within the gastrointestinal tract. Several studies have investigated the potential relationships between OIP and Helicobacter pylori (H. pylori) infection, gastric metaplasia, and other histopathological abnormalities. However, the data remain inconclusive [7].

Given these uncertainties, the clinical significance of OIP and its potential correlations with gastric and duodenal histopathology and biochemical parameters require further investigation. Despite the growing awareness of OIP as an endoscopic finding, the literature still lacks comprehensive data comparing the systemic and mucosal profiles of patients with inlet patches to those without. The majority of extant studies are limited in scope, focusing on endoscopic appearance or symptomatology, and frequently overlooking potential associations with broader gastric or duodenal pathology, biochemical markers, or infection status. Furthermore, there is a necessity for further exploration of the potential of OIP to serve as a clinical indicator of mucosal vulnerability or systemic inflammatory changes. In light of this evidence gap, the present study aims to contribute to the existing body of knowledge by offering a multifaceted comparison between affected and unaffected patients.

Material and Methods

The study protocol was approved by Giresun Training and Research Hospital Scientific Research Ethics Committee on June 11, 2025, with decision number 11.06.2025/07. The study was retrospective, so informed consent was not required. This study was conducted in accordance with the relevant ethical principles of the Declaration of Helsinki, revised in 2013.

This retrospective, case-control study was conducted at a tertiary care hospital and involved patients who presented to the general surgery, internal medicine, and gastroenterology outpatient clinics with complaints of dyspepsia. All patients underwent upper gastrointestinal endoscopy between October 2020 and January 2025. Patients with endoscopically visible oesophageal inlet patches (OIP) constituted the study group. A control group was formed by randomly selecting patients who had similar symptoms and were in the same age range but had no endoscopic evidence of inlet patches. Each group consisted of 50 patients, resulting in a total of 100 participants included in the analysis.

Inclusion criteria for both groups were as follows: age 18 years or older, presentation with dyspeptic symptoms, having undergone diagnostic upper gastrointestinal endoscopy with complete visualisation of the proximal oesophagus, and the availability of complete laboratory and histopathological data. Patients in the study group were required to have a documented endoscopic finding of an oesophageal inlet patch. Exclusion criteria included a history of upper gastrointestinal surgery, use of antibiotics, proton pump inhibitors, or bismuth compounds within four weeks before endoscopy, inadequate visualisation of the upper oesophagus, incomplete clinical or laboratory records, or a diagnosis of malignancy, inflammatory bowel disease, or known coeliac disease.

Upper gastrointestinal endoscopy was performed using high-resolution video endoscopes (Fujifilm® EPX3500 HD series) by experienced gastroenterologists. Particular attention was given to the evaluation of the proximal oesophagus, specifically the region 15–20 cm from the incisors, where inlet patches typically appear. These were identified as well-circumscribed, salmon-colored mucosal patches. Biopsies were routinely obtained from the gastric antrum and the second portion of the duodenum in all patients as part of the diagnostic workup for dyspepsia. By

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institutional protocol, inlet patches were not routinely biopsied.

Histopathological analysis was performed on all gastric and duodenal biopsy specimens. The tissues were fixed in 10% formalin, embedded in paraffin, sectioned at a thickness of 4 microns, and stained with hematoxylin and eosin (H&E). Giemsa staining was also employed for the detection of H. pylori. All slides were reviewed by a pathologist blinded to the clinical grouping. The presence of H. pylori, intestinal metaplasia, atrophic gastritis, and any histological features suggestive of coeliac disease were recorded.

Laboratory data collected at the time of endoscopy included a comprehensive panel of haematological and biochemical parameters. Haematological variables included leukocytes, neutrophils, lymphocytes, monocytes, eosinophils, haemoglobin, haematocrit, mean corpuscular volume (MCV), platelet count, and mean platelet volume (MPV). Biochemical parameters included urea, creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total and direct bilirubin, alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), lactate dehydrogenase (LDH), amylase, total protein, albumin, sodium, potassium, chlorine, ferritin, vitamin B12, folic acid, and C-reactive protein (CRP).

Statistical Analysis

Statistical analysis of the data obtained in this study was performed using SPSS version 22.0 (SPSS for Windows, Statistical Package for the Social Sciences, IBM Corp., Armonk, NY). The normality of the variables was evaluated using the Shapiro-Wilk test. Continuous variables are expressed as mean ±standard, while categorical variables are given as frequency and percentage (n, %). The comparisons between the two groups were made using the Mann-Whitney U test, the Chi-square test and Fisher's exact test. $p<0.05$ values were considered statistically significant.

Result

A total of 100 patients were included in the study, with 50 patients comprising the oesophageal inlet patch (OIP) group and 50 patients comprising the control group. In terms of gender distribution, females were predominant in both groups, although the difference was not statistically significant ($p>0.05$). Age distribution was also comparable between the groups. The mean age of the OIP group was 50.72±16.46 years (median:52.5; range:23–78), while the mean age in the control group was 47.28±16.77 years (median:48; range:18–84), with no statistically significant difference ($p>0.05$). The comparison of gender and age variables between groups is shown in Table 1.

Table 1. The comparison of gender and age variables between groups

Variables		OIP Group (n=50)		Control Group (n=50)		*p*
		Number	%	Number	%	
Gender	**Female**	30	60.0	25	50.0	0.421
	Male	20	40.0	25	50.0	
Age	**≤65**	40	80.0	42	84.0	0.795
	>65	10	20.0	8	16.0	
		Mean±S.D. Med. (Min.-Max.)		**Mean±S.D. Med. (Min.-Max.)**		
Age[t]		50.72±16.46 52.5 (23-78)		47.28±16.77 48 (18-84)		0.303

*p<0.05, **p<0.01, X^2: Chi-square test (categorical data), t: Independent sample T test, Med.:Median, S.D.: Standard deviation, Min.: Minimum, Max.: Maximum

When haematological and biochemical parameters were analysed, most variables—including leukocyte count, neutrophil count, lymphocyte count, monocyte count, eosinophils, haemoglobin, haematocrit, platelet count, MPV, urea, creatinine, ALT, AST, ALP, GGT, LDH, amylase, total protein, sodium, potassium, chlorine, vitamin B12, and folic acid—showed no statistically significant differences between the two groups. However, statistically significant differences were observed in several parameters. MCV, direct bilirubin, albumin, ferritin, and CRP levels were significantly different between the groups. Specifically, ferritin levels were significantly higher in the control group compared to the OIP group ($p=0.000$). MCV, direct bilirubin, albumin, and CRP levels also showed statistically significant differences, with CRP being higher in the OIP group ($p<0.05$). Table 2 compares laboratory parameters between groups.

Histopathological analysis of gastric and duodenal biopsies revealed important differences between the groups. The presence of H. pylori was significantly more frequent in the OIP group, detected in 50% of patients, compared to 24% in the control group ($p<0.05$). Similarly, the incidence of intestinal metaplasia was higher in the OIP group (30%) compared to the control group (6%), and this difference was statistically significant ($p<0.05$). In contrast, the prevalence of atrophic gastritis did not differ significantly between the two groups ($p>0.05$). Additionally, no histopathological findings suggestive of coeliac disease were observed in any of the duodenal biopsies in either group. Table 3 compares histopathological findings from gastric and duodenal biopsies between groups.

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Table 2. The comparison of laboratory parameters between groups

Laboratory parameters	OIP Group (n=50)	Control Group (n=50)	*p*
	Mean±S.D. Med. (Min.-Max.)	Mean±S.D. Med. (Min.-Max.)	
Leukocytes[t]	7334.8±2085.89 7220 (3050-12940)	7182.1±2591.44 6765 (3990-16330)	0.746
Lymphocytes[t]	2.12±0.89 2.03 (0.15-3.95)	1.88±0.67 1.8 (0.78-4.03)	0.132
Monocytes[t]	0.47±0.21 0.42 (0.07-1.26)	0.49±0.21 0.43 (0.16-0.99)	0.658
Neutrophils[t]	4.46±1.73 4.02 (1.6-9.08)	4.04±1.47 4.14 (0.58-7.53)	0.198
Eosinophils[z]	0.18±0.15 0.14 (0.01-0.85)	0.2±0.22 0.13 (0-0.92)	0.425
Haemoglobin[t]	12.9±1.54 12.95 (7.7-16.2)	13.03±1.84 13 (8-16.8)	0.711
Haematocrit[t]	39.4±4.19 38.95 (26.7-49.1)	39.39±4.83 39.45 (27.1-47.6)	0.989
MCV[z]	86.83±5.83 87.8 (62.5-98)	84.32±7.13 85.4 (62.6-94.1)	**0.043***
Platelets[t]	268.08±77.44 272.5 (139-496)	273.68±81.8 259 (53-504)	0.726
MPV[t]	9.42±1.01 9.4 (7.5-12.1)	9.11±1.04 8.9 (7.6-12.8)	0.133
Urea[t]	29.09±8.34 29 (13-53)	28.18±10.82 25 (15-56)	0.638
Creatinine[z]	0.77±0.19 0.74 (0.49-1.51)	0.77±0.21 0.73 (0.41-1.4)	0.836
ALT[z]	16.64±9.16 15 (5-62)	20.63±11.46 19 (7-52)	0.061
AST[z]	18.24±4.97 17 (11-33)	22.28±11.26 20 (9-58)	0.094
Total bilirubin[z]	0.49±0.24 0.45 (0.14-1.06)	0.61±0.35 0.57 (0.2-2.14)	0.052
Direct bilirubin[t]	0.20±0.09 0.2 (0.02-0.4)	0.28±0.14 0.26 (0.1-0.71)	**0.002****
ALP[t]	68.88±20.48 64 (37-140)	73.34±21.81 71.5 (29-124)	0.294
GGT[z]	21.24±18.43 16 (6-96)	23.06±16.51 19 (7-92)	0.197
LDH[z]	165.88±32.7 165.5 (103-232)	175.78±52.5 172.5 (95-408)	0.501
Amylase[t]	58.04±18.39 57.5 (27-99)	65.66±20.76 65 (20-117)	0.055
Total protein[t]	72.9±5.07 72.09 (61.06-84.17)	73.92±4.63 72.96 (65.91-88.78)	0.296
Albumin[t]	44.31±5.71 45.97 (27.66-52.2)	46.38±4.64 47.1 (29.36-55.1)	**0.050***
Sodium[t]	140.56±2.52 140 (135-145)	140±2.69 140 (135-147)	0.286
Chlorine[t]	102.65±2.94 103 (96-109)	102.26±2.69 101.5 (98-112)	0.490
Potassium[t]	4.33±0.45 4.4 (3.5-5.9)	4.3±0.43 4.3 (3.5-5.5)	0.752
Ferritin[t]	72.23±48.68 63.97 (10.8-227.4)	118.87±77.46 101.74 (6.56-352.6)	**0.000****
Vitamin B12[t]	327.57±110.9 323 (127-652)	368.86±148.58 344.5 (149-767.6)	0.119
Folic acid[t]	9.02±3.44 8.4 (4.39-17.1)	9.17±3.61 9.3 (3.04-17.3)	0.833
CRP[z]	5.74±10.8 2.81 (0.2-72)	2.28±3.7 1.05 (0.1-18.01)	**0.002****

*p<0.05, **p<0.01, t: Independent sample T test, z: Mann Whitney U Test, Med.:Median, S.D.: Standard deviation, Min.: Minimum, Max.: Maximum

Table 3. The comparison of gastric biopsy results between the two groups

Variables		OIP Group (n=50)		Control Group (n=50)		*p*
		Number	%	Number	%	
H.pylori	**Negative**	25	50.0	38	76.0	0.013*
	Positive	25	50.0	12	24.0	
Atrophic gastritis	**Negative**	38	76.0	40	80.0	0.809
	Positive	12	24.0	10	20.0	
Intestinal metaplasia	**Negative**	35	70.0	47	94.0	0.004**
	Positive	15	30.0	3	6.0	
Duodenal biopsy	**Negative**	50	100.0	50	100.0	-
	Positive	-	-	-	-	

*p<0.05, **p<0.01, X^2: Chi-square test (categorical data)

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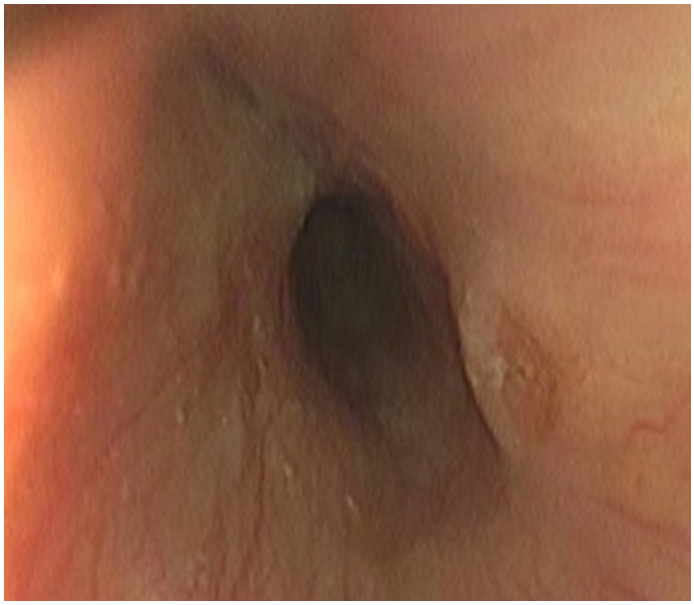

Figure 1. Endoscopic image of the inlet patch in the upper oesophagus

Discussion

The present study provides a comprehensive evaluation of patients with oesophageal inlet patches, examining their clinical, biochemical, and histopathological characteristics in comparison with a matched control group. While OIP has long been regarded as a benign and incidental endoscopic finding, the results of this study underscore its potential clinical significance, particularly its association with underlying gastric pathology and subtle biochemical alterations.

A notable finding of this study is the significantly higher prevalence of H. pylori infection in the OIP group compared to the control group (50% vs. 24%, $p<0.05$). This association has been previously observed in studies conducted by Guiterrez et al., which suggest that the gastric-type mucosa within the inlet patch may act as a habitat for H. pylori colonisation [8]. The present findings lend support to this hypothesis and contribute to the argument that inlet patches are not only anatomical curiosities but may also play a role in broader gastric mucosal pathophysiology.

The markedly elevated rate of intestinal metaplasia observed in the OIP group (30% vs. 6%) is of particular concern. Although the inlet patch itself is rarely reported to undergo malignant transformation, the observed histological changes in the gastric mucosa may reflect a field effect or shared pathophysiological environment influenced by chronic inflammation and H. pylori-associated damage. Mion et al. have reported similar histopathological changes in the vicinity of inlet patches, suggesting that these patients may warrant closer surveillance, especially when other risk factors for gastric carcinogenesis are present [9].

Conversely, the incidence of atrophic gastritis did not exhibit a significant difference between the groups. This finding is consistent with the results of previous studies, although other studies have reported variable results [10]. It is conceivable that the emergence of intestinal metaplasia in this demographic signifies an early or isolated manifestation of gastric mucosal injury, not yet accompanied by diffuse glandular atrophy.

From a biochemical perspective, our study identified significant differences in mean corpuscular volume (MCV), direct bilirubin, albumin, ferritin, and CRP levels between the groups. These findings are original and justify further investigation. Notably, ferritin levels were significantly lower in the OIP group, which suggests the possibility that occult blood loss, chronic inflammation, or iron absorption abnormalities may be present in this population. Although overt anaemia was not observed, these subtle differences may reflect subclinical processes linked to chronic gastric irritation or altered systemic iron metabolism. The elevated levels of C-reactive protein (CRP) observed in the OIP group further substantiate the hypothesis of a low-grade inflammatory response in these patients. Although CRP is a nonspecific marker, its increase aligns with the presence of H. pylori infection and intestinal metaplasia, both of which are known to induce mucosal inflammation [11]. Decreased albumin levels may similarly reflect either nutritional impact or systemic inflammatory status, although this interpretation should be approached with caution given the retrospective nature of the study.

It is interesting to note that, in contrast to the findings of previous reports, which emphasised respiratory and otolaryngological symptoms associated with inlet patches, such as chronic cough, globus sensation, or hoarseness, our study focused primarily on gastrointestinal and histopathological correlates [12]. Nevertheless, the potential systemic effects of acid-secreting ectopic mucosa in the proximal oesophagus, including the possible contributions to supra-oesophageal symptoms, remain an area for future research.

This study makes several meaningful additions to the extant literature. Firstly, the present study serves to reinforce the potential association between OIP and H. pylori infection. Secondly, it is one of the few studies to demonstrate a significantly increased prevalence of intestinal metaplasia in patients with OIP, suggesting a possible role for inlet patches in gastric mucosal pathology. Thirdly, it introduces a novel perspective by exploring differences in systemic biochemical markers between patients with and without inlet patches, raising important questions about the broader physiological impact of this finding.

Study Limitations

This study has several limitations that should be acknowledged. First, its retrospective design inherently limits causal inference and introduces potential selection bias. As inlet patches were identified based on endoscopic records, there is a risk of underdiagnosis or misclassification, especially if the proximal oesophagus was not thoroughly examined in all patients.

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Additionally, biopsies were not taken directly from the inlet patches, in accordance with institutional protocol, which restricts histological confirmation of the inlet mucosa and precludes evaluation of local inflammatory or metaplastic changes. In addition, the present study did not provide data concerning patients' comorbidities or medication use. The lack of symptom-specific data, such as the presence of globus sensation, cough, or laryngopharyngeal symptoms, also limits the ability to correlate endoscopic findings with clinical presentation. Furthermore, the sample size, while adequate for preliminary comparison, may not be sufficient to detect subtle differences in some parameters. Finally, as this was a single-centre study, the generalizability of the findings to other populations or settings may be limited. Despite these constraints, the study provides important insights and generates hypotheses for future prospective research.

Conclusion

The oesophageal inlet patch should not be disregarded as a benign incidental finding. The present study highlights its association with significant histopathological changes, particularly H. pylori infection and intestinal metaplasia, as well as subtle yet significant differences in systemic biochemical markers. These findings suggest that OIP may serve as an indicator of broader gastrointestinal mucosal vulnerability. Prospective studies with long-term follow-up and targeted biopsies are needed to clarify the clinical implications and determine whether patients with OIP would benefit from closer surveillance or tailored management strategies.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical Approval
The study received ethical approval from the Giresun Training and Research Hospital Scientific Research Ethics Committee on 11.06.2025 (Approval No: 11.06.2025/07)

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