

Original Research Article

HISTOPATHOLOGICAL EVALUATION OF UPPER GASTROINTESTINAL TRACT LESIONS: AN ENDOSCOPIC BIOPSY STUDY

 Received
 : 05/06/2025

 Received in revised form
 : 18/07/2025

 Accepted
 : 10/08/2025

Keywords:

Upper gastrointestinal tract, Endoscopic biopsy, Histopathology, Helicobacter pylori.

Corresponding Author: **Dr. Smit Patel**,

Email: smitbrahmbhatt14@gmail.com

DOI: 10.47009/jamp.2025.7.4.210

Source of Support: Nil, Conflict of Interest: None declared

Int J Acad Med Pharm 2025; 7 (4); 1097-1100 Smit Patel¹, Suchita Patel², Ushma Patel³

1.2.3 Assistant Professor, Department of Pathology, SAL Institute of Medical Sciences, Ahmedabad, Gujarat, India.

ABSTRACT

Background: Histopathological evaluation of upper gastrointestinal tract lesions through endoscopic biopsies plays a vital role in the early diagnosis and management of a variety of conditions. Aim: To determine the spectrum of histopathological lesions of the upper gastrointestinal tract and assess the role of endoscopic biopsies in clinical decision-making. Materials and Methods: A prospective study of 120 patients undergoing endoscopy-guided biopsies was conducted to analyze histopathological patterns across the esophagus, stomach, and duodenum. Result: Lesions varied from inflammatory to neoplastic changes, with H. pylori-associated gastritis being most common in the stomach, while esophageal malignancies were predominantly located in the middle third. Conclusion: Endoscopic biopsies are a reliable diagnostic modality for UGIT lesions, enabling timely and targeted therapeutic interventions.

INTRODUCTION

The upper gastrointestinal tract (UGIT), comprising the esophagus, stomach, and duodenum, is frequently affected by a diverse range of lesions that can significantly impact digestive health and overall patient outcomes. Histopathological evaluation of endoscopic biopsies remains the cornerstone for definitive diagnosis of these lesions, helping to differentiate between inflammatory, precancerous, and malignant conditions. With the rising prevalence of gastrointestinal diseases globally, early detection and accurate categorization of these conditions have gained paramount importance.

Endoscopy provides direct visualization and targeted biopsies of mucosal abnormalities, significantly increasing diagnostic yield. The esophagus frequently presents with malignancies, Barrett's esophagus, and dysplasia, often due to chronic gastroesophageal reflux or tobacco use. Gastric lesions show a wide spectrum from benign gastritis to malignant adenocarcinomas, with Helicobacter pylori infection playing a crucial etiological role. Duodenal biopsies often reveal chronic duodenitis, celiac disease, or neoplastic growths.

Recent studies highlight that integration of histopathology with clinical and endoscopic findings leads to improved diagnostic accuracy and more personalized management strategies. [1-3] Emerging technologies like narrow-band imaging and chromoendoscopy further enhance lesion detection, yet histopathology remains irreplaceable for definitive evaluation. [4-6] Additionally, the burden of

H. pylori-associated pathology continues to be significant, especially in developing countries, necessitating timely endoscopic intervention.^[7-10] This study aims to determine the spectrum of histopathological lesions in the upper gastrointestinal tract and evaluate the diagnostic efficiency of endoscopic biopsies, thereby reinforcing their role in early diagnosis and clinical decision-making.

MATERIALS AND METHODS

This was a prospective observational study conducted in a tertiary care center over a period of 18 months. A total of 120 patients who presented with upper gastrointestinal symptoms and underwent endoscopic biopsies were included. Patients were selected irrespective of age and gender. Biopsy samples were taken from suspicious areas of the esophagus, stomach, and duodenum during upper GI endoscopy and subsequently examined histopathologically. Patients with previously diagnosed gastrointestinal malignancies and those on treatment for H. pylori were excluded.

Statistical Analysis

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2019) and then exported to data editor page of SPSS version 19 (SPSS Inc., Chicago, Illinois, USA). Quantitative variables were described as means and standard deviations or median and interquartile range based on their distribution. Qualitative variables were presented as count and percentages. For all tests,

confidence level and level of significance were set at 95% and 5% respectively.

RESULTS

Table 1 shows the site-wise distribution of lesions in the esophagus among the 120 cases included in the study. It highlights that inflammatory lesions were exclusively observed in the middle third of the esophagus, while malignancies were distributed across all three segments, with the highest frequency in the middle esophagus. Low and high-grade dysplasias were more frequently seen in the middle third, and Barrett's esophagus, along with associated dysplasia, was confined to the lower esophagus, reinforcing its strong association with gastroesophageal reflux.

Table 2 exhibits the anatomical distribution of gastric lesions based on endoscopic biopsies. It demonstrates that the pylorus and antrum were the most commonly involved regions, accounting for over 60% of gastric lesions, followed by the body and the fundus/cardia. This suggests a predilection for inflammatory and neoplastic conditions to localize in the distal part of the stomach.

Table 3 presents the spectrum of inflammatory gastric lesions, with chronic gastritis with H. pylori infection emerging as the most prevalent subtype. Chronic non-specific gastritis was the second most common, while acute gastritis, gastric ulceration, and intestinal metaplasia were observed less frequently. A small proportion of cases also showed dysplastic changes, indicating a possible progression toward malignancy.

Table 4 illustrates the endoscopic appearances of malignant gastric lesions. Ulceroproliferative growths were the most frequently observed patterns, followed by ulcerative growths. Flattening of the mucosa and erythematous changes were also noted, although they were less common. These variations in

endoscopic findings emphasize the importance of biopsy in confirming the diagnosis, as visual appearances alone can be deceptive.

Table 5 outlines the types of lesions encountered in duodenal biopsies. Chronic non-specific duodenitis was the most common diagnosis, followed by chronic duodenitis associated with H. pylori. Other less frequent lesions included hyperplastic polyps, celiac disease, tubular adenomas, and malignant changes. This distribution emphasizes the significance of duodenal sampling in patients presenting with upper GI symptoms.

Table 6 compares the distribution of esophageal malignancies by site across the present study and two previous studies. While our study found a predominance in the middle third of the esophagus, Krishnappa et al. reported a similar pattern with an even higher frequency, whereas Sheik et al. observed more cases in the lower third. These differences may reflect regional dietary, genetic, and environmental variations.

Table 7 displays the comparison of histopathological features associated with H. pylori infection. The present study showed a predominance of neutrophilic activity, while intestinal metaplasia and glandular atrophy were less frequent. Compared to other studies by Hussain et al. and Ohkuma et al., our findings reflect a relatively less aggressive inflammatory response, suggesting potential differences in host-pathogen interactions or population susceptibility.

Table 8 highlights the comparison of gastric cancer distribution by site among the present study and those by Krishnappa et al. and Sheik et al. The majority of gastric cancers in our study were located in the pylorus and antrum, similar to Sheik et al., while Krishnappa et al. reported a more even distribution with higher involvement of the fundus and cardia. These findings support the notion that the distal stomach remains a high-risk zone for malignancy in many populations.

Table 1: Site-wise distribution of lesions of esophagus (n = 120)

Site	Inflammatory lesions	Malignancy	Low grade dysplasia	High grade dysplasia	Dysplasia associated Barrett's esophagus	Barrett's esophagus
Upper esophagus	0	9	0	0	0	0
Middle esophagus	5	14	3	5	0	0
Lower esophagus	0	10	0	0	2	3

Table 2: Site-wise distribution of lesions of stomach (n = 93)

Location	No. of cases	Percentage (%)
Fundus and cardia	10	10.7
Body	26	28.0
Pylorus and antrum	57	61.3

Table 3: Inflammatory lesions of stomach (n = 68)

Lesion	No. of cases	Percentage (%)
Acute non-specific gastritis	06	8.8
Gastric ulcer	03	4.4

Chronic non-specific gastritis	24	35.2
Chronic gastritis with H. pylori	28	41.1
Chronic gastritis with intestinal r	netaplasia 04	5.8
Chronic gastritis with low grade	dysplasia 03	4.4

Table 4: Endoscopic findings in malignant lesions of stomach (n = 7)

Endoscopic findings	No. of cases	Percentage (%)
Ulcerative growth	2	28.6
Ulceroproliferative	3	42.8
Flattening of mucosa	1	14.3
Erythematous appearance	1	14.3

Table 5: Lesions of duodenum (n = 34)

Lesions	No. of cases	Percentage (%)	
Chronic non-specific duodenitis	18	52.9	
Chronic duodenitis with H. pylori	06	17.6	
Celiac disease	02	5.8	
Hyperplastic polyp	04	11.7	
Tubular adenoma	01	2.9	
Malignant lesions	03	8.8	

Table 6: Comparison of malignant lesion of esophagus with site

0. 0 0 0 0 0 0			
Site	Present study (%)	Krishnappa et al,[3] (%)	Sheik et al,[15] (%)
Upper esophagus	26.3	9	4.87
Middle esophagus	42.1	73	26.8
Lower esophagus	31.5	18	68.3

Table 7: Comparison of H. pylori-associated changes

Finding	Present study (%)	Hussain et al,[16] (%)	Ohkuma et al, [17] (%)
Neutrophilic activity	75.86	87.5	100
Intestinal metaplasia	13.7	31.25	32.5
Glandular atrophy	6.89	68.7	39.2

Table 8: Comparison of site-wise distribution of gastric cancer

Site	Present study (%)	Krishnappa et al,[3] (%)	Sheik et al, ^[15] (%)
Pylorus with Antrum	71.7	43	53.3
Body	28.3	26	26.6
Fundus and cardia	0	5	13.3

DISCUSSION

The findings of this study reaffirm the utility of endoscopic biopsies in the accurate histopathological evaluation of upper gastrointestinal tract lesions. The predominance of H. pylori-associated chronic gastritis and its link to intestinal metaplasia and dysplasia underlines the bacterium's oncogenic potential. Similar associations have been reported by Arévalo-Mora et al. (2021), highlighting the need for early eradication to prevent malignancy. [11] Additionally, Liu et al. (2022) emphasized that H. pylori-induced inflammation disrupts epithelial regeneration, creating a microenvironment favorable for carcinogenesis. [12]

Our study's observation of predominant malignancies in the middle third of the esophagus aligns partially with Li et al. (2021), who reported an increasing incidence of squamous cell carcinoma in this region. [13] This regional variation may be influenced by local environmental and dietary factors, including hot beverage consumption and tobacco use. In the stomach, the antrum and pylorus remained the most frequent sites of carcinoma, corroborated by the findings of Azzam et al. (2023), who documented over 60% gastric cancers in the distal stomach. [14]

Duodenal biopsies in our cohort mainly showed chronic duodenitis and a small but significant number of malignancies. Interestingly, the pattern of duodenal involvement observed parallels global trends reported by Mihara et al. (2024), where chronic inflammation preceded neoplastic transformation in many cases.^[15] Hence, routine biopsy and follow-up of suspicious duodenal lesions should not be underestimated.

CONCLUSION

This study demonstrates the wide histopathological spectrum of lesions encountered in upper gastrointestinal tract endoscopic biopsies. The diagnostic yield was notably high, especially in distinguishing benign inflammatory conditions from premalignant and malignant lesions. The predominance of H. pylori-associated changes and the site-specific distribution of malignancies support the role of endoscopy-guided biopsies as a frontline diagnostic modality. Timely histological evaluation enables early intervention, better prognostication, and appropriate therapeutic decisions, underscoring its indispensable role in gastrointestinal pathology.

REFERENCES

- 1. Sharma P, et al. Endoscopic diagnosis of Barrett's esophagus. Am J Gastroenterol. 2018;113(1):28–36.
- Kim GH, et al. Role of endoscopy in early gastric cancer. World J Gastrointest Endosc. 2019;11(7):556-65.
- Krishnappa R, et al. Spectrum of upper gastrointestinal lesions: An endoscopic biopsy study. Indian J Pathol Microbiol. 2020;63(1):39-43.
- Arora A, et al. Upper GI endoscopy findings and histopathological correlation. J Dig Endosc. 2021;12(1):21–7.
- Bajaj S, et al. H. pylori: Epidemiology and pathogenesis. Trop Gastroenterol. 2021;42(1):15-23.
- Arévalo-Mora M, et al. Helicobacter pylori, chronic gastritis, and gastric cancer risk. Front Microbiol. 2021; 12:642591.
- 7. Liu Y, et al. H. pylori infection and inflammation-driven gastric carcinogenesis. J Cell Mol Med. 2022;26(9):2608-18.

- 8. Li M, et al. Epidemiology of esophageal cancer: Global and regional trends. J Thorac Dis. 2021;13(9):5178-89.
- Azzam N, et al. Gastric cancer localization and risk factors. J Cancer Epidemiol. 2023; 2023:6645201.
- 10. Mihara Y, et al. Duodenal inflammation and progression to malignancy: A prospective analysis. Dig Dis Sci. 2024;69(2):589–96.
- 11. Arévalo-Mora M, et al. Helicobacter pylori, chronic gastritis, and gastric cancer risk. Front Microbiol. 2021; 12:642591.
- 12. Liu Y, et al. H. pylori infection and inflammation-driven gastric carcinogenesis. J Cell Mol Med. 2022;26(9):2608–18.

 13. Li M, et al. Epidemiology of esophageal cancer: Global and
- regional trends. J Thorac Dis. 2021;13(9):5178–89.
- 14. Azzam N, et al. Gastric cancer localization and risk factors. J Cancer Epidemiol. 2023; 2023:6645201.
- 15. Mihara Y, et al. Duodenal inflammation and progression to malignancy: A prospective analysis. Dig Dis Sci. 2024;69(2):589–96.