International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

Clinical, Endoscopic and Histopathological Spectrum of Oesophageal Lesions: A Cross-Sectional Study of Endoscopy-Biopsy Concordance at a Tertiary-Care Centre in Jaipur, India

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Abstract: ***Background:*** *Oesophageal lesions encompass inflammatory, premalignant, and malignant conditions with overlapping clinical and endoscopic appearances. Histopathological evaluation remains essential for definitive diagnosis and appropriate management.* ***Objective:*** *To describe the clinical, endoscopic, and histopathological spectrum of oesophageal lesions and assess associations between patient characteristics, endoscopic findings, and broad histopathological categories.* ***Methods:*** *This hospital-based cross-sectional observational study included 80 adults with endoscopically detected oesophageal lesions who underwent biopsy at a tertiary-care centre in Jaipur, India. Demographic data, symptoms, risk factors, radiological findings, endoscopic features, and histopathological diagnoses were recorded. Associations were analysed using the chi-square test, with P<0.05 considered statistically significant.* ***Results:*** *The mean age was 54.21±12.94 years, and 70.0% were men. Dysphagia was the commonest symptom (66.3%), followed by heartburn or regurgitation (61.3%). The lower third was the most frequently involved site (53.8%). Histopathologically, 57.5% of lesions were non-neoplastic, 10.0% premalignant, and 32.5% malignant. Chronic reflux or nonspecific oesophagitis was the commonest diagnosis (35.0%), while squamous cell carcinoma was the predominant malignancy (26.3%). Age, residence, dysphagia, heartburn or regurgitation, weight loss, smoking, tobacco chewing, and gastro-oesophageal reflux disease were significantly associated with histopathological category. Endoscopic impression showed a strong association with histopathology (P<0.001), with 77.5% concordance.* ***Conclusion:*** *Oesophageal lesions showed a broad histopathological spectrum, with inflammatory lesions predominating and squamous cell carcinoma remaining the principal malignancy. Despite good endoscopy–histopathology concordance, biopsy confirmation was indispensable, particularly for suspicious, discordant, or premalignant lesions across routine tertiary-care diagnostic practice settings.*

MeSH Keywords: Esophageal Diseases; Endoscopy, Gastrointestinal; Biopsy; Esophagitis; Esophageal Neoplasms

1. Introduction

Oesophageal lesions comprise a heterogeneous group of disorders ranging from reactive and inflammatory mucosal changes to infectious disease, metaplasia, epithelial dysplasia, benign neoplasms and invasive malignancy. This diversity makes accurate classification essential because lesions with similar clinical or endoscopic appearances may differ substantially in their natural history, treatment and prognosis [1–3].

Gastro-oesophageal reflux disease is among the most frequently encountered oesophageal disorders and develops when reflux of gastric contents produces troublesome symptoms or mucosal injury. Endoscopic manifestations range from erythema and superficial erosions to extensive ulceration, friability and peptic stricture. [3,4].

Other inflammatory oesophageal lesions include eosinophilic and infectious oesophagitis. Eosinophilic oesophagitis is a chronic immune-mediated disorder characterised clinically by symptoms of oesophageal dysfunction and histologically by eosinophil-predominant inflammation. Endoscopic findings such as fixed rings, linear furrows, white exudates, mucosal oedema, narrowing and strictures may suggest the diagnosis, but these features are neither uniformly present nor independently diagnostic. Histological examination of appropriately obtained biopsies is therefore required to establish the diagnosis and assess disease activity [5]. Because symptoms and endoscopic morphology may overlap with reflux, medication-induced injury and neoplasia, biopsy-based histological and microbiological assessment is central to definitive diagnosis [6].

Premalignant and malignant lesions constitute the most clinically consequential part of the oesophageal disease spectrum. Oesophageal cancer remains a major cause of cancer-related mortality worldwide because many patients become symptomatic only after substantial luminal involvement or locally advanced disease. An estimated 604,100 new cases and 544,100 deaths occurred globally in 2020, with squamous cell carcinoma accounting for approximately 85% of cases and adenocarcinoma for most of the remainder [7]. The two major histological subtypes have distinct epidemiological and biological profiles. Squamous cell carcinoma is associated particularly with tobacco exposure, alcohol consumption, nutritional deficiencies and selected dietary or environmental exposures, whereas adenocarcinoma is closely linked to chronic reflux, Barrett's oesophagus, obesity and tobacco use [7,8].

The relative frequency of these malignancies varies considerably across geographical regions. Although adenocarcinoma has become increasingly prominent in several Western populations, squamous cell carcinoma continues to predominate in many Asian and African populations. Indian institutional data have similarly shown continued predominance of squamous cell carcinoma, frequently involving the middle third of the oesophagus, without convincing evidence of a complete transition towards adenocarcinoma [9,10].

Clinical features provide important initial diagnostic information but have limited specificity [3,8,9]. Upper gastrointestinal endoscopy is the principal investigation for direct evaluation of suspected oesophageal lesions. It permits assessment of lesion location, extent, surface characteristics, friability, ulceration, luminal narrowing and contact bleeding and enables targeted tissue acquisition. High-quality mucosal inspection is particularly important because early neoplastic abnormalities may be subtle, flat or difficult to distinguish from inflammatory change [11,12].

Histopathological examination remains necessary for differentiating reactive changes from dysplasia, identifying intestinal metaplasia, confirming infectious organisms, categorising invasive tumours and determining tumour differentiation where adequate tissue is available. Correlation with the endoscopic impression is particularly important because visually suspicious lesions may prove inflammatory or benign, whereas subtle endoscopic abnormalities may contain dysplasia or malignancy [12–14].

In this context, the present study was undertaken to describe the clinical features, endoscopic findings and histopathological spectrum of oesophageal lesions among adult patients undergoing endoscopic biopsy at a tertiary-care institution in Jaipur, India, and to determine the associations between these parameters.

Study Design, Setting, and Population

This hospital-based, cross-sectional observational study was conducted in the Department of Pathology at Sawai Man Singh Medical College and associated hospitals, Jaipur, Rajasthan. The study included adult patients (aged >18 years) with endoscopically detected oesophageal lesions from whom biopsy specimens were obtained. Recruitment and sample collection continued for a maximum of nine months or until the target sample size was achieved. An additional two-month period was allocated for data verification, statistical analysis, and report preparation.

Sample Size and Sampling Technique

The sample size was calculated using an expected carcinoma proportion of 23.8% from a reference study, with a 95% confidence level and 10% absolute allowable error, yielding a minimum of 70 cases. Accounting for potential exclusions due to inadequate tissue or incomplete data, the final sample size was rounded to 80 participants. Consecutive sampling was employed—all eligible patients who provided informed consent were enrolled until the target was reached. No random selection was performed to ensure the study population reflected routine clinical practice.

Eligibility Criteria

Inclusion criteria comprised patients over 18 years with endoscopic oesophageal biopsies, adequate tissue fixation, sufficient material for histopathological evaluation, and complete clinical and endoscopic data. Exclusion criteria included anticoagulant therapy, coagulopathies precluding safe biopsy, inadequate or poorly preserved specimens, missing consent, and unavailable clinical records.

Data Collection

Clinical data were collected using a predesigned, pretested proforma. Information on age, sex, residence, symptoms (including dysphagia grade, odynophagia, heartburn, chest pain, bleeding, and weight loss), risk factors (tobacco use, alcohol, GERD, corrosive ingestion), medical history, BMI, and haemoglobin were recorded. Endoscopy reports provided data on lesion site, size, morphology, and provisional impression. Radiological findings from clinically indicated imaging (barium swallow, CT, PET-CT) were documented when available.

Specimen Processing

Biopsy specimens were immediately placed in 10% neutral buffered formalin and transported to the pathology department with completed requisition forms. Gross examination recorded fragment number, size, colour, and consistency. All tissue was processed via routine paraffin embedding; small fragments were wrapped in filter paper to prevent loss. Sections of 3–5 µm thickness were cut, stained with haematoxylin and eosin, and examined under light microscopy. Additional levels or special stains were used only when clinically required.

Histopathological Examination

Slides were evaluated for epithelial integrity, inflammation, metaplasia, dysplasia, and malignancy. Malignant lesions were subtyped (squamous cell carcinoma, adenocarcinoma, etc.) and graded as well, moderately, or poorly differentiated. Diagnoses were categorised as non-neoplastic, premalignant, or malignant for statistical analysis. Difficult cases were reviewed by a senior pathologist, and accepted departmental diagnostic criteria were applied.

Clinicopathological Correlation

Endoscopic impressions were compared with histopathological diagnoses and classified as concordant or discordant. Histopathology served as the diagnostic reference. Limitations such as sampling error, crush artefact, or tissue heterogeneity were documented.

Statistical Analysis

Data were entered into Microsoft Excel and cross-verified. Categorical variables were summarised as frequencies and percentages; continuous variables as mean (SD) or median (IQR) based on distribution. Associations were assessed using chi-square or Fisher's exact tests. Group comparisons used t-tests, ANOVA, or non-parametric equivalents (Mann–Whitney U, Kruskal–Wallis) as appropriate. A two-tailed p-value <0.05 was considered significant.

Ethical Considerations

The study received Institutional Ethics Committee approval. Written informed consent was obtained from all participants, with assurance of confidentiality, voluntary participation, and no impact on clinical care. No research-specific biopsies were performed. Anonymised data were used for analysis and reporting, and clinically relevant diagnoses were communicated through routine channels.

2. Results

A total of 80 patients with endoscopically detected oesophageal lesions were included in the analysis. The mean age was 54.21 ± 12.94 years (range: 26–78 years), with the largest proportion in the 60–69-year age group (32.5%), followed by 40–49 years (22.5%) and 50–59 years (21.3%). Males constituted 70.0% of the study population; 52.5% were from urban areas and 47.5% from rural areas (Table 1).

The mean duration of symptoms was 10.87 ± 7.82 months, mean dysphagia grade was 1.53 ± 1.39 (range: 0–4), mean BMI was 24.75 ± 3.28 kg/m², and mean haemoglobin was 11.80 ± 1.51 g/dL. A mean of 4.52 ± 1.15 biopsy fragments was obtained per participant (Table 1).

Dysphagia was the most frequent symptom, present in 53 participants (66.3%), followed by heartburn/regurgitation in 49 (61.3%), odynophagia in 29 (36.3%), chest pain in 27 (33.8%), and weight loss in 26 (32.5%). Upper gastrointestinal bleeding and food impaction were reported by 7.5% and 3.8%, respectively (Table 1). Regarding dysphagia severity, 33.8% had no dysphagia, while grades 1, 2, 3, and 4 were observed in 17.5%, 22.5%, 15.0%, and 11.3%, respectively (Table 1).

Tobacco chewing was reported by 38.8%, smoking by 35.0%, and alcohol use by 28.7%. A history of GERD was present in 61.3%, whereas atopy was documented in 3.8% (Table 1).

Radiological investigations were not performed in 31.3%. Among barium swallow findings, irregular shouldered narrowing (13.8%), mild distal mucosal irregularity (12.5%), and irregular distal narrowing with shouldering (5.0%) were most frequent. Small sliding hiatus hernia with reflux was observed in 3.8% (Table 2). Contrast-enhanced CT demonstrated enhancing oesophageal wall thickening without metastasis in 7.5%, asymmetrical mural thickening with luminal narrowing and small paraoesophageal lymph nodes in 5.0%, and distal oesophageal/GOJ wall thickening with regional lymph nodes in 1.3% (Table 2).

The lower third of the oesophagus was the most commonly involved site (53.8%), followed by the middle third (27.5%), upper third (10.0%), and GOJ (8.8%) (Table 2). Oesophagitis was the most frequent endoscopic impression (40.0%), followed by suspected carcinoma (30.0%), benign stricture/ulcer (7.5%), Barrett's oesophagus (6.3%), candidiasis (5.0%), dysplasia/suspicious neoplasia (5.0%), benign polyp/plaque (3.8%), and eosinophilic oesophagitis (2.5%) (Table 2).

Chronic reflux/nonspecific oesophagitis was the most frequent histopathological diagnosis (35.0%). Squamous cell carcinoma was diagnosed in 26.3%, adenocarcinoma in 6.3%, erosive/ulcerative oesophagitis in 7.5%, and candidal oesophagitis in 6.3%. Eosinophilic oesophagitis was diagnosed in 2.5% (Table 3). Barrett's oesophagus without dysplasia was observed in 5.0%, with low-grade and high-grade dysplasia each in 1.3%. Squamous dysplasia was observed in 2.5% (Table 3).

On broad classification, 46 lesions (57.5%) were non-neoplastic, 26 (32.5%) were malignant, and eight (10.0%) were premalignant. No dysplasia was present in 95.0%, while low-grade and high-grade dysplasia were each identified in 2.5% (Table 3). Among malignant lesions, 42.3% were moderately differentiated, 30.8% poorly differentiated, and 26.9% well differentiated. Concordance between endoscopic impression and histopathological diagnosis was observed in 77.5%, with 22.5% discordant (Table 3).

The distribution of histopathological categories differed significantly across age groups (χ^2=33.388, df=10, P<0.001). Of 26 malignant lesions, 38.5% occurred in the 60–69-year group, 34.6% in 50–59 years, and 23.1% in ≥70 years; none occurred in the 18–39-year group. Non-neoplastic lesions were most frequent in the 40–49-year group (34.8%) (Table 4). Although malignant lesions were more common in males (20/56) and all premalignant lesions occurred in males, the association between sex and histopathological category was not statistically significant (χ^2=5.848, df=2, P=0.054) (Table 4).

Residence was significantly associated with histopathological category (χ^2=10.125, df=2, P=0.006); 73.1% of malignant lesions occurred in rural participants, whereas 65.2% of non-neoplastic lesions occurred in urban participants (Table 4).

Dysphagia was significantly associated with histopathological category (χ^2=25.529, df=2, P<0.001), present in all malignant cases compared with 43.5% of non-neoplastic and 87.5% of premalignant cases (Table 5). Heartburn/regurgitation was also significantly associated (χ^2=15.081, df=2, P=0.001), reported by 76.1% with non-neoplastic and 75.0% with premalignant lesions, versus 30.8% with malignant lesions (Table 5). Weight loss showed a significant association (χ^2=34.658, df=2, P<0.001), present in 76.9% of malignant cases compared with 10.9% of non-neoplastic and 12.5% of premalignant cases (Table 5). No significant associations were observed for odynophagia, chest pain, food impaction, or upper gastrointestinal bleeding (Table 5).

Smoking (χ^2=6.567, df=2, P=0.038), tobacco chewing (χ^2=6.068, df=2, P=0.048), and alcohol use (χ^2=5.980, df=2, P=0.050) were significantly associated with histopathological category (Table 5). GERD history was significantly associated (χ^2=8.458, df=2, P=0.015), present in 71.7% of non-neoplastic and 75.0% of premalignant cases, compared with 38.5% of malignant cases. No significant association was observed for atopy (Table 5).

Anatomical site was not significantly associated with histopathological category (χ^2=7.401, df=6, P=0.285). Among malignant lesions, the middle third was most frequently involved (42.3%), followed by the lower third (38.5%), whereas the lower third accounted for 60.9% of non-neoplastic and 62.5% of premalignant lesions (Table 6).

A significant association was observed between endoscopic impression and histopathological category (χ^2=87.513, df=14, P<0.001). Of 24 lesions endoscopically suspected as carcinoma, 22 were histopathologically malignant. Among 32 lesions with an endoscopic diagnosis of oesophagitis, 30 were

non-neoplastic. Of five lesions suspected as Barrett's oesophagus, three were premalignant. Two of four lesions classified as dysplasia/suspicious neoplasia were malignant and two were premalignant (Table 6). Endoscopic morphology was also significantly associated with histopathological category (χ^2=160.000, df=54, P<0.001); circumferential friable growths, infiltrative growths, and ulceroproliferative lesions were predominantly malignant, while erythema and erosions were associated with non-neoplastic lesions (Supplementary Table S1).

3. Discussion

The present study characterised the clinical, endoscopic and histopathological spectrum of oesophageal lesions in 80 adults undergoing endoscopic biopsy. The predominance of patients in the sixth and seventh decades, together with a male proportion of 70%, was consistent with Indian series showing that oesophageal pathology, particularly carcinoma, occurs mainly in older men [9,10,15]. Geetanjali et al. reported a comparable mean age of 56.29 years and male predominance among 200 patients with oesophageal lesions [15]. The concentration of malignant lesions among participants aged 50 years or older in the present study further supports the cumulative influence of prolonged mucosal exposure, lifestyle factors and age-related biological susceptibility.

Non-neoplastic lesions constituted 57.5% of biopsies, with chronic reflux or nonspecific oesophagitis being the most frequent diagnosis. This pattern accords with the clinical burden of reflux-related disease and with previous biopsy-based studies in which inflammatory lesions exceeded neoplastic lesions [3,14]. The lower third was the commonest overall site, reflecting the predominance of reflux-associated pathology and Barrett-related changes in the distal oesophagus. Barrett's oesophagus and dysplasia represented a smaller proportion because intestinal metaplasia constitutes the recognised precursor pathway for oesophageal adenocarcinoma [4]. These findings support careful biopsy of distal mucosal abnormalities even when their endoscopic appearance is not overtly malignant.

Malignancy was identified in 32.5% of cases. Squamous cell carcinoma was considerably more frequent than adenocarcinoma, reproducing the histological pattern reported from most Indian centres [9,10,15]. The middle third was the commonest site among malignant lesions, whereas adenocarcinoma and Barrett-associated lesions were concentrated distally. This anatomical distribution is biologically plausible and resembles earlier Indian clinicopathological observations [15]. Moderately differentiated tumours formed the largest grade category, indicating that many lesions had developed architectural and cytological atypia by the time biopsy was performed. The findings contrast with Western populations in which adenocarcinoma has increased markedly, but remain consistent with the continued predominance of squamous cell carcinoma across much of Asia [2,16].

Dysphagia and weight loss showed the strongest clinical associations with malignant histopathology. Dysphagia was present in every malignant case, while weight loss occurred in more than three quarters. Similar associations were reported by Geetanjali et al., who observed dysphagia in 97.5% of patients with oesophageal carcinoma [15]. These symptoms probably reflect progressive luminal compromise and impaired nutritional intake and should therefore prompt early endoscopy and biopsy. Smoking and tobacco chewing were also significantly associated with histopathological category. Tobacco exposure is an established determinant of squamous carcinogenesis, and Indian case-control evidence has demonstrated increased oesophageal cancer risk with smoking and smokeless tobacco use [8,16,17]. Alcohol showed only a borderline association, which may reflect limited statistical power, exposure misclassification or variation in dose and duration.

Endoscopic impression was strongly associated with histopathological category, and overall concordance was 77.5%. Nevertheless, almost one quarter of cases were discordant, demonstrating that visual assessment alone could not reliably distinguish inflammatory, premalignant and malignant lesions. Comparable studies have reported high concordance for common conditions, while others found high sensitivity, lower specificity and diagnostic accuracy of approximately 82% [18,19]. Endoscopic overestimation may arise from ulceration, friability and inflammatory stricturing, whereas underestimation may result from subtle dysplasia, superficial sampling or heterogeneous tumours. Histopathological confirmation should therefore remain mandatory for all suspicious oesophageal lesions. The study was limited by its single-centre design, modest sample size, consecutive hospital-based sampling and sparse cells in several subgroup analyses; consequently, observed associations should not be interpreted as causal or assumed to represent the wider population. Larger multicentre studies incorporating standardised biopsy protocols, exact tests for sparse data, and follow-up outcomes are required to confirm these clinicopathological relationships and improve generalisability across India.

4. Conclusion

Oesophageal lesions demonstrated a broad histopathological spectrum, with non-neoplastic lesions predominating and chronic reflux oesophagitis being the most frequent diagnosis. Squamous cell carcinoma was the commonest malignancy, particularly among older patients. Dysphagia, weight loss, smoking, tobacco chewing and rural residence were significantly associated with malignant or premalignant pathology. Although endoscopic and histopathological findings showed good overall concordance, the observed diagnostic discordance confirmed that histopathological examination remained essential for definitive diagnosis and appropriate clinical management.

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Tables

Table 1: Demographic, clinical, laboratory, and procedural characteristics of the study participants

Characteristic	Overall (N=80)
Demographic characteristics	
Age, years	54.21 ± 12.94 (26–78)
Age group	
18–29 years	2 (2.5)
30–39 years	10 (12.5)
40–49 years	18 (22.5)
50–59 years	17 (21.3)
60–69 years	26 (32.5)
≥70 years	7 (8.8)
Sex	
Female	24 (30.0)
Male	56 (70.0)
Residence	
Rural	38 (47.5)

Urban	42 (52.5)
Clinical, laboratory, and procedural characteristics	
Symptom duration, months	10.87 ± 7.82 (1–47)
Dysphagia grade	1.53 ± 1.39 (0–4)
Body mass index, kg/m²	24.75 ± 3.28 (18.3–33.5)
Haemoglobin, g/dL	11.80 ± 1.51 (8.4–14.7)
Number of biopsy fragments	4.52 ± 1.15 (3–6)
Presenting symptoms	
Dysphagia	53 (66.3)
Odynophagia	29 (36.3)
Heartburn/regurgitation	49 (61.3)
Chest pain	27 (33.8)
Food impaction	3 (3.8)
Weight loss	26 (32.5)
Upper gastrointestinal bleeding	6 (7.5)
Risk factors and relevant clinical history	
Smoking	28 (35.0)
Tobacco chewing	31 (38.8)
Alcohol use	23 (28.7)
History of gastroesophageal reflux disease	49 (61.3)
History of atopy	3 (3.8)
Dysphagia grade distribution	
Grade 0	27 (33.8)
Grade 1	14 (17.5)
Grade 2	18 (22.5)
Grade 3	12 (15.0)
Grade 4	9 (11.3)

Table 2: Radiological and endoscopic characteristics of the oesophageal lesions

Diagnostic finding	Overall (N=80), n (%)
Barium Swallow findings	
focal mucosal irregularity in the mid oesophagus	2 (2.5)
irregular distal narrowing with shouldering	4 (5.0)
irregular shouldered narrowing	11 (13.8)
mild concentric narrowing without irregular shouldering	1 (1.3)
mild distal mucosal irregularity	10 (12.5)
reflux with distal mucosal irregularity	2 (2.5)
short smooth stricture	2 (2.5)
small sliding hiatus hernia with reflux	3 (3.8)
superficial distal ulceration without shouldering	2 (2.5)
CECT findings	
not indicated/no significant radiological abnormality	7 (8.8)
asymmetrical mural thickening with luminal narrowing and small paraoesophageal nodes	4 (5.0)
distal oesophageal/GEJ wall thickening with few regional nodes	1 (1.3)
enhancing wall thickening without distant metastasis	6 (7.5)
Radiological evaluation not performed	*25 (31.3)*
Anatomical site on endoscopy	
Gastroesophageal junction	7 (8.8)
Lower third	43 (53.8)
Middle third	22 (27.5)
Upper third	8 (10.0)
Endoscopic impression	
Barrett's oesophagus	5 (6.3)
Benign polyp/plaque	3 (3.8)
Benign stricture/ulcer	6 (7.5)
Candidiasis	4 (5.0)
Carcinoma	24 (30.0)
Dysplasia/suspicious neoplasia	4 (5.0)
Eosinophilic oesophagitis	2 (2.5)
Oesophagitis	32 (40.0)

CECT, contrast-enhanced computed tomography; GEJ, gastroesophageal junction.

Table 3: Histopathological spectrum, dysplasia, tumour differentiation, and endoscopy–histopathology concordance

Histopathological or diagnostic variable	n (%)
Detailed histopathological diagnosis, N=80	
Adenocarcinoma	5 (6.3)
Barrett's oesophagus with intestinal metaplasia and no dysplasia	4 (5.0)
Barrett's oesophagus with high-grade dysplasia	1 (1.3)
Barrett's oesophagus with low-grade dysplasia	1 (1.3)
Benign stricture with chronic inflammation	2 (2.5)
Candidal oesophagitis	5 (6.3)
Chronic reflux/chronic nonspecific oesophagitis	28 (35.0)
Eosinophilic oesophagitis	2 (2.5)
Erosive/ulcerative oesophagitis	6 (7.5)
Glycogenic acanthosis	1 (1.3)
Hyperplastic polyp	1 (1.3)
Squamous cell carcinoma	21 (26.3)
Squamous dysplasia	2 (2.5)
Squamous papilloma	1 (1.3)
Histopathological diagnostic group, N=80	
Barrett's oesophagus	4 (5.0)
Benign polyp/plaque	3 (3.8)
Benign stricture/ulcer	2 (2.5)
Candidiasis	5 (6.3)
Carcinoma	26 (32.5)
Dysplasia/suspicious neoplasia	4 (5.0)
Eosinophilic oesophagitis	2 (2.5)
Oesophagitis	34 (42.5)
Broad histopathological category, N=80	
Non-neoplastic	46 (57.5)
Premalignant	8 (10.0)
Malignant	26 (32.5)
Dysplasia grade, N=80	
No dysplasia/not applicable	76 (95.0)
Low-grade dysplasia	2 (2.5)
High-grade dysplasia	2 (2.5)
Tumour differentiation among malignant cases, n=26	
Well differentiated	7 (26.9)
Moderately differentiated	11 (42.3)
Poorly differentiated	8 (30.8)
Endoscopy–histopathology concordance, N=80	
Concordant	62 (77.5)
Discordant	18 (22.5)

Table 4: Association of demographic characteristics with the broad histopathological category

Variable/category	Malignant (n=26)	Non-neoplastic (n=46)	Premalignant (n=8)	Overall (N=80)	χ^2 (df)	P value
Age group						
18–29 years	0 (0.0)	2 (4.3)	0 (0.0)	2 (2.5)	33.388 (10)	<0.001
30–39 years	0 (0.0)	10 (21.7)	0 (0.0)	10 (12.5)	—	—
40–49 years	1 (3.8)	16 (34.8)	1 (12.5)	18 (22.5)	—	—
50–59 years	9 (34.6)	7 (15.2)	1 (12.5)	17 (21.3)	—	—
60–69 years	10 (38.5)	11 (23.9)	5 (62.5)	26 (32.5)	—	—
≥70 years	6 (23.1)	0 (0.0)	1 (12.5)	7 (8.8)	—	—
Sex						
Female	6 (23.1)	18 (39.1)	0 (0.0)	24 (30.0)	5.848 (2)	0.054
Male	20 (76.9)	28 (60.9)	8 (100.0)	56 (70.0)	—	—
Residence						
Rural	19 (73.1)	16 (34.8)	3 (37.5)	38 (47.5)	10.125 (2)	0.006
Urban	7 (26.9)	30 (65.2)	5 (62.5)	42 (52.5)	—	—

Table 5: Association of presenting symptoms and clinical risk factors with the broad histopathological category

Feature present	Malignant (n=26)	Non-neoplastic (n=46)	Premalignant (n=8)	Overall (N=80)	χ^2 (df)	P value
Dysphagia	26 (100.0)	20 (43.5)	7 (87.5)	53 (66.3)	25.529 (2)	<0.001
Odynophagia	12 (46.2)	15 (32.6)	2 (25.0)	29 (36.3)	1.806 (2)	0.405
Heartburn/regurgitation	8 (30.8)	35 (76.1)	6 (75.0)	49 (61.3)	15.081 (2)	0.001
Chest pain	12 (46.2)	11 (23.9)	4 (50.0)	27 (33.8)	4.725 (2)	0.094
Food impaction	0 (0.0)	3 (6.5)	0 (0.0)	3 (3.8)	2.304 (2)	0.316
Weight loss	20 (76.9)	5 (10.9)	1 (12.5)	26 (32.5)	34.658 (2)	<0.001

Upper gastrointestinal bleeding	4 (15.4)	2 (4.3)	0 (0.0)	6 (7.5)	3.637 (2)	0.162
Smoking	12 (46.2)	11 (23.9)	5 (62.5)	28 (35.0)	6.567 (2)	0.038
Tobacco chewing	15 (57.7)	13 (28.3)	3 (37.5)	31 (38.8)	6.068 (2)	0.048
Alcohol use	12 (46.2)	10 (21.7)	1 (12.5)	23 (28.7)	5.980 (2)	0.050
History of gastroesophageal reflux disease	10 (38.5)	33 (71.7)	6 (75.0)	49 (61.3)	8.458 (2)	0.015
History of atopy	0 (0.0)	3 (6.5)	0 (0.0)	3 (3.8)	2.304 (2)	0.316

Table 6: Association of endoscopic site and endoscopic impression with the broad histopathological category

Endoscopic variable/category	Malignant (n=26)	Non-neoplastic (n=46)	Premalignant (n=8)	Overall (N=80)	χ^2 (df)	P value
Anatomical site						
Gastroesophageal junction	1 (3.8)	5 (10.9)	1 (12.5)	7 (8.8)	7.401 (6)	0.285
Lower third	10 (38.5)	28 (60.9)	5 (62.5)	43 (53.8)	—	—
Middle third	11 (42.3)	10 (21.7)	1 (12.5)	22 (27.5)	—	—
Upper third	4 (15.4)	3 (6.5)	1 (12.5)	8 (10.0)	—	—
Endoscopic impression						
Barrett's oesophagus	0 (0.0)	2 (4.3)	3 (37.5)	5 (6.3)	87.513 (14)	<0.001
Benign polyp/plaque	0 (0.0)	3 (6.5)	0 (0.0)	3 (3.8)	—	—
Benign stricture/ulcer	2 (7.7)	4 (8.7)	0 (0.0)	6 (7.5)	—	—
Candidiasis	0 (0.0)	4 (8.7)	0 (0.0)	4 (5.0)	—	—
Carcinoma	22 (84.6)	1 (2.2)	1 (12.5)	24 (30.0)	—	—
Dysplasia/suspicious neoplasia	2 (7.7)	0 (0.0)	2 (25.0)	4 (5.0)	—	—
Eosinophilic oesophagitis	0 (0.0)	2 (4.3)	0 (0.0)	2 (2.5)	—	—
Oesophagitis	0 (0.0)	30 (65.2)	2 (25.0)	32 (40.0)	—	—

Supplementary Table S1. Detailed endoscopic morphology according to the broad histopathological category [ADD THIS AS A SUPPLEMENTARY FILE]

Detailed endoscopic finding	Malignant (n=26)	Non-neoplastic (n=46)	Premalignant (n=8)	Overall (N=80)
Adherent whitish plaques	0 (0.0)	3 (6.5)	0 (0.0)	3 (3.8)
Barrett segment with nodularity	0 (0.0)	0 (0.0)	2 (25.0)	2 (2.5)
Chronic ulcer with fibrotic narrowing	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Circumferential friable growth	5 (19.2)	0 (0.0)	0 (0.0)	5 (6.3)
Congested oedematous mucosa	0 (0.0)	6 (13.0)	0 (0.0)	6 (7.5)
Distal ulceronodular mass on Barrett segment	2 (7.7)	0 (0.0)	0 (0.0)	2 (2.5)
Oedema with concentric rings and furrows	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Erosions with contact bleeding	0 (0.0)	4 (8.7)	0 (0.0)	4 (5.0)
Erythema with friability	0 (0.0)	9 (19.6)	0 (0.0)	9 (11.3)
Flat nodular lesion with friable mucosa	0 (0.0)	0 (0.0)	2 (25.0)	2 (2.5)
Infiltrative growth with contact bleeding	7 (26.9)	0 (0.0)	0 (0.0)	7 (8.8)
Irregular Z-line with short-segment columnar mucosa	0 (0.0)	0 (0.0)	1 (12.5)	1 (1.3)
Linear erosions in the distal oesophagus	0 (0.0)	7 (15.2)	0 (0.0)	7 (8.8)
Los Angeles grade A/B oesophagitis	0 (0.0)	6 (13.0)	0 (0.0)	6 (7.5)
Mucosal ulcer with surrounding erythema	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Multiple slightly raised whitish plaques	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Nodular friable lesion at the distal oesophagus/GEJ	2 (7.7)	0 (0.0)	0 (0.0)	2 (2.5)
Patchy white plaques in the mid oesophagus	0 (0.0)	2 (4.3)	0 (0.0)	2 (2.5)
Salmon-coloured mucosa above the GE junction	0 (0.0)	0 (0.0)	2 (25.0)	2 (2.5)
Shallow ulcerated lesion	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Short benign-appearing stricture with surrounding inflammation	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Short-segment Barrett-appearing mucosa	0 (0.0)	0 (0.0)	1 (12.5)	1 (1.3)
Small exophytic polypoid lesion	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Small sessile polyp at the GE junction	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Trachealisation with rings and linear furrows	0 (0.0)	1 (2.2)	0 (0.0)	1 (1.3)
Ulcerated distal growth with narrowed lumen	1 (3.8)	0 (0.0)	0 (0.0)	1 (1.3)
Ulceronodular lesion	4 (15.4)	0 (0.0)	0 (0.0)	4 (5.0)
Ulceroproliferative growth causing luminal narrowing	5 (19.2)	0 (0.0)	0 (0.0)	5 (6.3)

Pearson χ^2=160.000, df=54, P *<0.001*