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Clinicoendoscopic Correlation in Patients of Dyspepsia under Evaluation in Tertiary Care Centre

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Abstract

Introduction: Dyspepsia is one of the most common clinical problems in the gastrointestinal patient population. Health-related quality-of-life indices in untreated dyspepsia approximate those observed in chronic kidney disease and moderate heart failure, underscoring the clinical importance of timely evaluation and management [1].

Aims and Objectives

Aim: To elucidate the relationship between clinical presentation and endoscopic findings in adults presenting with dyspepsia at a tertiary-care centre, thereby clarifying when endoscopy most effectively alters diagnosis or management.

Objectives

1. Characterise the endoscopic spectrum of lesions detected in consecutive dyspeptic patients undergoing oesophago-gastro-duodenoscopy.
2. Quantify the predictive value of demographic factors, symptom patterns and alarm features for significant endoscopic pathology.

3. Identify underlying aetiologies of dyspepsia by integrating endoscopic results with adjunct investigations (e.g., Helicobacter pylori testing, histopathology, laboratory indices and imaging where indicated).
4. Develop a simplified risk-stratification model that can guide appropriate, resource-sensitive use of endoscopy in similar tertiary-care settings.

Material and Method:

Study Design: Observational study

Study Period: January 2023 and December 2024

Place of study: Department of Surgery at a tertiary care teaching hospital, India

Sample Size: 160 and randomly collection

Result: The study cohort had a mean age of 44.8 years. Females comprised a slight majority (57.5%) compared to males (42.5%). Most participants resided in urban areas (60.0%), while 40.0% were from rural settings.

Discussion: Our tertiary-centre study of 160 consecutively evaluated adults provides a contemporary snapshot of that conundrum in the Indian sub-continent. The cohort was middle-aged (mean 44.8 years), predominantly female, largely urban and mostly non-smoking.

Keywords: Dyspepsia, Gastric Ulcer, Malignancy, Organic Disease

Introduction

Dyspepsia—defined by the Rome IV consensus as persistent or recurrent epigastric pain or burning, early satiation, and post-prandial fullness—remains one of the commonest indications for outpatient gastroenterology consultation worldwide. Contemporary population-based surveys conducted on five continents consistently place the 12-month prevalence between 20 % and 30 %, implying that over a billion adults are affected at any one time [1,2].

Health-related quality-of-life indices in untreated dyspepsia approximate those observed in chronic kidney disease and moderate heart failure, underscoring the clinical importance of timely evaluation and management [1].

Experimental studies employing high-resolution manometry, gastric barostat testing, and scintigraphic emptying demonstrate impaired accommodation reflexes and delayed gastric emptying in PDS, whereas visceral hypersensitivity, duodenal acid or lipid sensing abnormalities, and low-grade mucosal inflammation dominate in EPS [6]. Genome-wide association studies have identified susceptibility loci within nociceptive ion-channel genes such as TRPV1 and SCN5A, as well as pro-inflammatory cytokine genes, together explaining roughly 10 % of the inter-individual variance in symptom severity [8]. Concomitantly, next-generation sequencing of gastric and duodenal microbiota implicates dysbiosis and abnormal carbohydrate fermentation in symptom generation, offering a biological rationale for the clinical benefit observed with low-FODMAP diets and non-systemic antibiotics [9]. These mechanistic insights, while intellectually satisfying, have yet to translate into reliable bedside tools capable of distinguishing functional from organic disease in unselected clinic populations. Symptom-based triage therefore remains problematic. Prospective series reveal that up to 40 % of patients fulfil criteria for both PDS and EPS simultaneously, and the overlap erodes the discriminatory power of standardised questionnaires [7]. So-called “alarm features” (weight loss, gastrointestinal bleeding, anaemia, dysphagia, or persistent vomiting) perform only modestly better, with pooled sensitivities

for upper-gastro-intestinal (GI) malignancy falling below 50 % in Asian cohorts [7]

In low- and middle-income countries (LMICs) such as India, where endoscopic capacity per million populations is less than one-tenth that of Western Europe, refining the decision to scope assumes paramount importance. Clinico-endoscopic correlation seeks to align easily measured demographic and clinical variables with the likelihood of uncovering significant pathology at endoscopy, thereby supporting rational resource allocation.

A systematic review encompassing 8 432 Indian patients reported that 62 % had abnormal OGD; erosive gastritis (28 %), duodenal ulcer (14 %), and gastric ulcer (5 %) predominated, while upper-GI malignancy was identified in 3 % of cases—half of whom lacked alarm features [15].

Aim

To elucidate the relationship between clinical presentation and endoscopic findings in adults presenting with dyspepsia at a tertiary-care centre, thereby clarifying when endoscopy most effectively alters diagnosis or management.

Objectives

1. Characterise the endoscopic spectrum of lesions detected in consecutive dyspeptic patients undergoing oesophago-gastro-duodenoscopy.
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4. Develop a simplified risk-stratification model that can guide appropriate, resource-sensitive use of endoscopy in similar tertiary-care settings.

Materials and Methods

Study Design and Setting

A hospital-based, observational study was conducted between January 2023 and December 2024 in the Department of Surgery in Tertiary-Care Teaching Hospital, India. The institution functions as a regional referral centre with an annual upper-gastro-intestinal (UGI) endoscopy volume exceeding 1000 procedures, ensuring both ample case accrual and uniform technical standards.

Sample-Size Determination

Sample-size estimation was performed a priori using the single-population-proportion formula $n = (Z1\text{-}\alpha/2)^2 \times p(1 – p)/d^2$, where p represents the anticipated prevalence of clinically significant endoscopic findings among dyspeptic patients, d denotes the absolute precision, and Z is the standard normal deviate corresponding to the desired confidence level. First, assuming a prevalence of 30% reflecting upper-range estimates for Asian cohorts—together with a 95 % confidence level (Z = 1.96) and a precision of ±5 %, the calculated sample size was 322 participants. Second, using a more conservative prevalence of 10 % (to accommodate lower-burden settings) with the same confidence and precision parameters generated a requirement of 138 participants. Balancing statistical rigour against logistical feasibility, the larger figure was pragmatically down-scaled while still exceeding the minimum needed to detect clinically meaningful differences; therefore, a target enrolment of 160 consecutive patients was adopted for the study. This number provided adequate power for subgroup analyses and multivariable modelling, while remaining achievable

within the projected 24-month recruitment window and available resources.

Inclusion criteria

- age ≥ 18 years;
- first presentation of dyspeptic symptoms without prior diagnostic evaluation;
- Willingness to provide written informed consent.

Exclusion criteria

- age < 18 years;
- documented UGI pathology (e.g. peptic ulcer, malignancy) or previous UGI surgery;
- terminal illness (Eastern Cooperative Oncology Group performance status ≥ 3) or altered mental status precluding consent;
- current participation in another interventional study;
- Refusal or inability to comply with follow-up.

Results

Table 1: Baseline Characteristics (N = 160)

Variable	Value
Age (years)	44.8 ± 14.8
Sex: Male	68 (42.5 %)
Sex: Female	92 (57.5 %)
Residence: Urban	96 (60.0 %)
Residence: Rural	64 (40.0 %)
Smoking: Never	99 (61.9 %)
Smoking: Current	45 (28.1 %)
Smoking: Former	16 (10.0 %)

The study cohort had a mean age of 44.8 years. Females comprised a slight majority (57.5%) compared to males (42.5%). Most participants resided in urban areas (60.0%), while 40.0% were from rural settings. Regarding smoking history, 61.9% had never smoked, 28.1% were current smokers, and 10.0% were former smokers. These distributions reflect a fairly balanced gender mix, urban predominance, and a majority of non-smokers, which are relevant for interpreting exposure-related health outcomes in this population.

Graph 1:

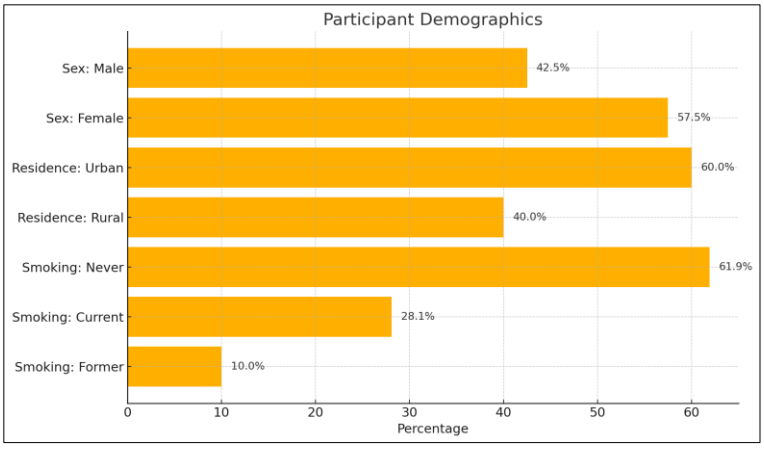


Table 2: Endoscopic diagnosis distribution

Diagnosis	n (%)
Gastritis	53 (33.1 %)
Normal	49 (30.6 %)
Duodenitis	13 (8.1 %)
Gastric ulcer	13 (8.1 %)
Reflux esophagitis	10 (6.2 %)
Duodenal ulcer	10 (6.2 %)
Hiatal hernia	3 (1.9%)
Polyps	3 (1.9 %)
Gastric malignancy	6(3.7 %)

Among patients undergoing endoscopy, gastritis emerged as the most prevalent diagnosis, affecting 33.1% of individuals. A normal finding was observed in 30.6% of cases. Duodenitis and gastric ulcer were each identified in 8.1% of patients, while reflux esophagitis and duodenal ulcer accounted for 6.2% each. Less frequent findings included hiatal hernia and polyps (1.9% each), and gastric malignancy (3.7%). These patterns highlight the dominance of benign inflammatory conditions in the study population.

Graph 2:

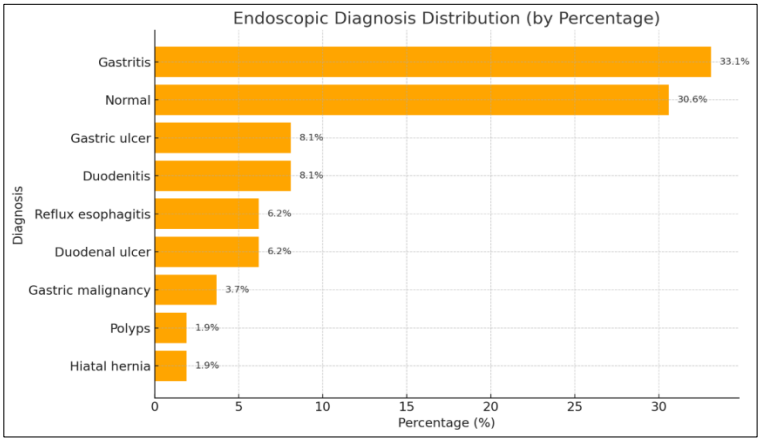


Table 3: Mean age – normal vs abnormal endoscopy (Welch t-test)

Group	n	Mean ± SD
Normal	49	43.8 ± 13.8
Abnormal	111	45.3 ± 15.3
t-test	—	t = -0.61, p = 0.542

The mean age of participants with normal findings was 43.8 ± 13.8 years, while those with abnormal findings had a mean age of 45.3 ± 15.3 years. Statistical comparison using an independent t-test revealed no significant difference between the two groups (t = -0.61, p = 0.542). This suggests that age did not significantly influence the likelihood of having abnormal endoscopic findings in this cohort.

Graph 3:

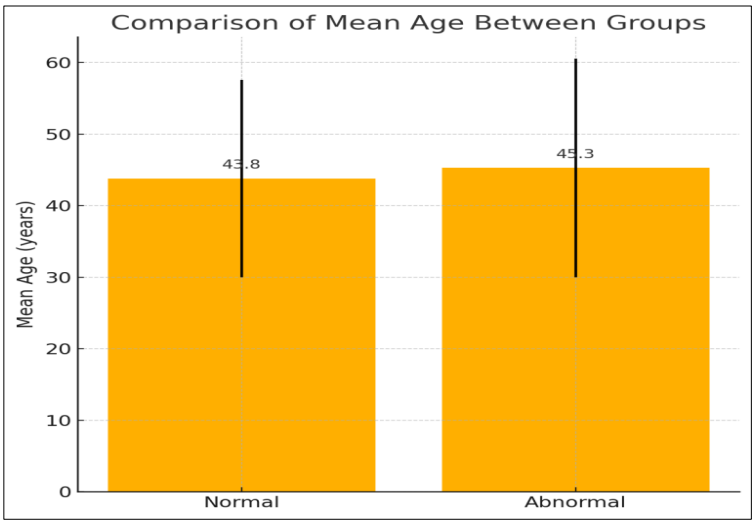


Table 4: Alarm symptoms vs endoscopy outcome (χ^2)

Outcome	Alarm No	Alarm Yes
Abnormal	95	16
Normal	41	8
χ^2	0.01	p = 0.943

The distribution of alarm features among patients with abnormal (n=111) and normal (n=49) endoscopic findings was comparable. Alarm symptoms were present in 16 abnormal and 8 normal cases. Statistical analysis using the chi-square test ($\chi^2 = 0.01$, $p = 0.943$) showed no significant association between the presence of alarm features and the likelihood of abnormal endoscopic outcomes. This indicates that alarm symptoms were not predictive of pathological findings in this study population.

Graph 4:

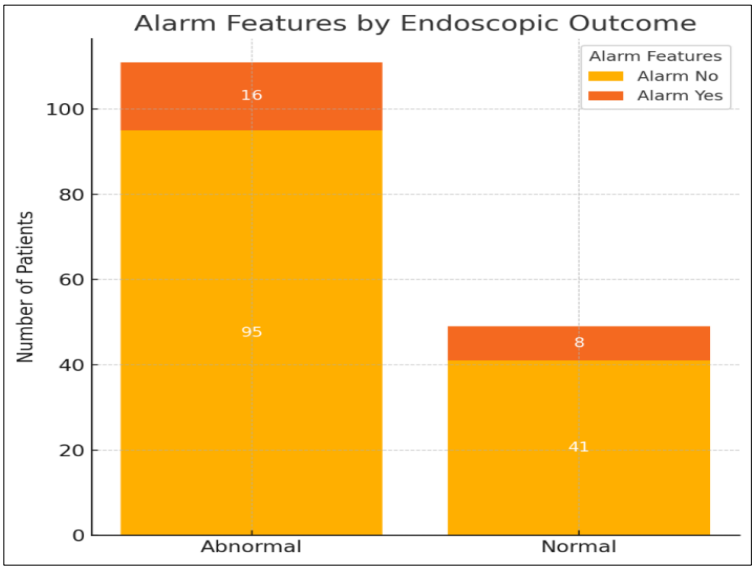


Table 5: Helicobacter status within endoscopic diagnoses

Diagnosis	Positive n (%)	Negative n (%)
Duodenal ulcer	4 (40.0 %)	6 (60.0 %)
Duodenitis	1 (14.3 %)	6 (85.7 %)
Gastric ulcer	5 (55.6 %)	4 (44.4 %)
Gastritis	19 (44.2 %)	24 (55.8 %)
Hiatal hernia	2 (40.0 %)	3 (60.0 %)
Normal	15 (41.7 %)	21 (58.3 %)
Polyps	1 (25.0 %)	3 (75.0 %)
Reflux esophagitis	0 (0.0 %)	7 (100 %)
Gastric malignancy	0 (0.0 %)	1 (100 %)
χ^2	8.91	p = 0.350

H. pylori positivity varied across diagnoses, with the highest rates seen in gastric ulcer (55.6%) and gastritis (44.2%), and no positivity observed in reflux esophagitis or gastric malignancy. However, statistical analysis (χ^2 = 8.91, p = 0.350) indicated no significant association between H. pylori status and specific endoscopic diagnoses. This suggests that while H. pylori may cluster around certain conditions, its distribution did not differ meaningfully across diagnostic categories in this sample.

Graph 5:

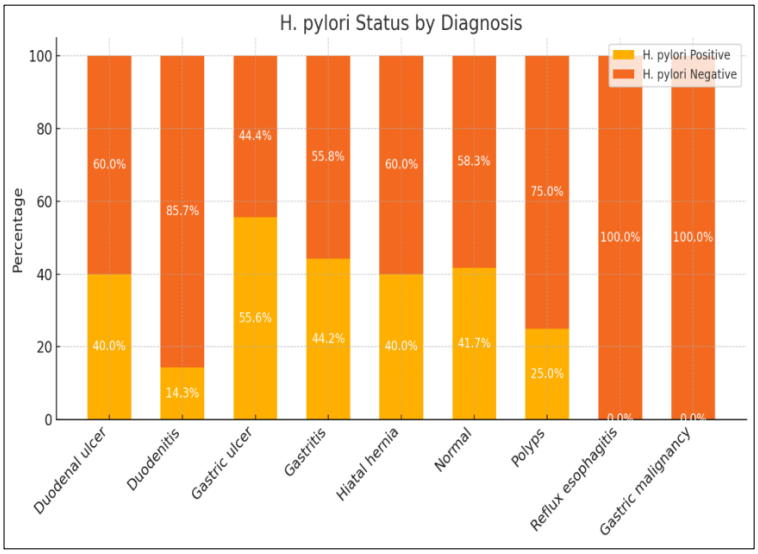


Table 6: Treatment vs follow-up improvement

Treatment	No improve n (%)	Improved n (%)
H. pylori eradication	12 (25.5 %)	35 (74.5 %)
Lifestyle modification	0 (0.0 %)	4 (100 %)
Oncology referral	2 (100 %)	0 (0.0 %)

PPI	13 (17.6 %)	61 (82.4 %)
Polypectomy	0 (0.0 %)	4 (100 %)
Prokinetic	9 (31.0 %)	20 (69.0 %)
χ^2	11.70	p = 0.039

Treatment responses varied significantly across modalities (χ^2 = 11.70, p = 0.039). Most patients showed improvement with PPI therapy (82.4%) and H. pylori eradication (74.5%). Lifestyle modification and polypectomy yielded 100% symptom resolution in their respective groups. In contrast, all patients referred to oncology had no improvement, likely reflecting underlying malignancy. Prokinetics were less effective, with 69.0% reporting benefit. These findings highlight differential treatment efficacy and reinforce the importance of tailored management based on underlying etiology.

Graph 6:

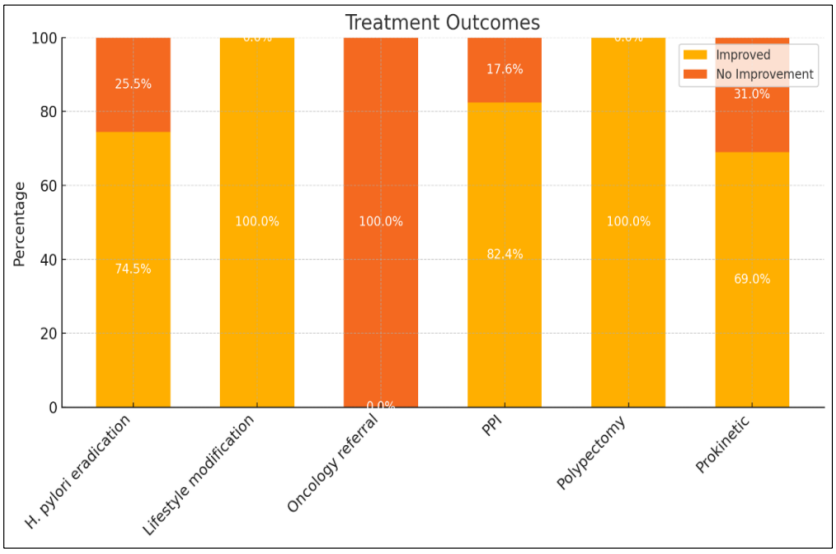


Table 7: Age across histopathology categories (ANOVA)

Histopathology	n	Mean ± SD (years)
Chronic gastritis	56	45.5 ± 15.5
H. pylori gastritis	25	42.0 ± 14.9
Malignancy	2	52.0 ± 14.1
Metaplasia	4	51.0 ± 18.5
Other benign	3	46.7 ± 12.2
Not applicable	70	44.6 ± 14.4
ANOVA	—	F = 0.44, p = 0.823

The mean age varied modestly across histopathological groups, ranging from 42.0 years in H. pylori gastritis to 52.0 years in malignant cases. However, the overall age differences were not statistically significant (ANOVA F = 0.44, p = 0.823). This suggests that age was not a distinguishing factor among the various histological diagnoses observed in this cohort, and histopathological findings were distributed relatively evenly across the age spectrum.

Graph 7:

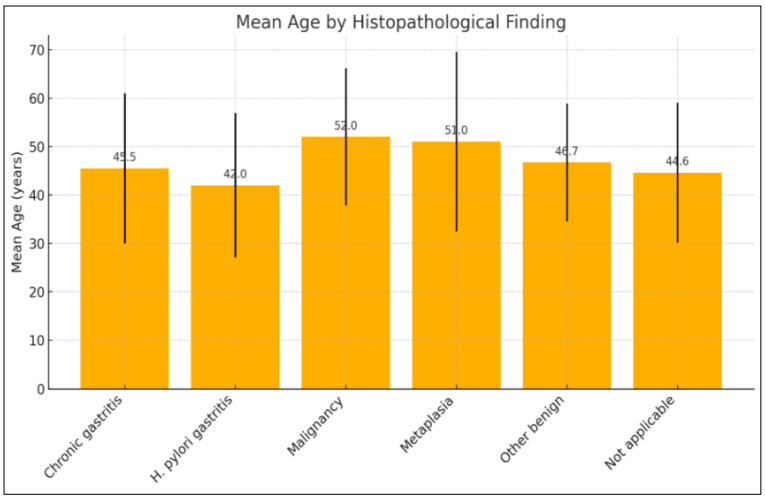


Table 8: Sex vs endoscopy outcome (χ^2)

Sex	Normal	Abnormal
Female	31	61
Male	18	50
χ^2	0.65	p = 0.420

Among females, 61 had abnormal and 31 had normal endoscopic findings; among males, 50 had abnormal and 18 had normal results. The chi-square test ($\chi^2 = 0.65$, $p = 0.420$) indicated no statistically significant association between sex and endoscopic outcome. This means that being male or female did not significantly influence the likelihood of having abnormal findings on endoscopy in this cohort.

Graph 8:

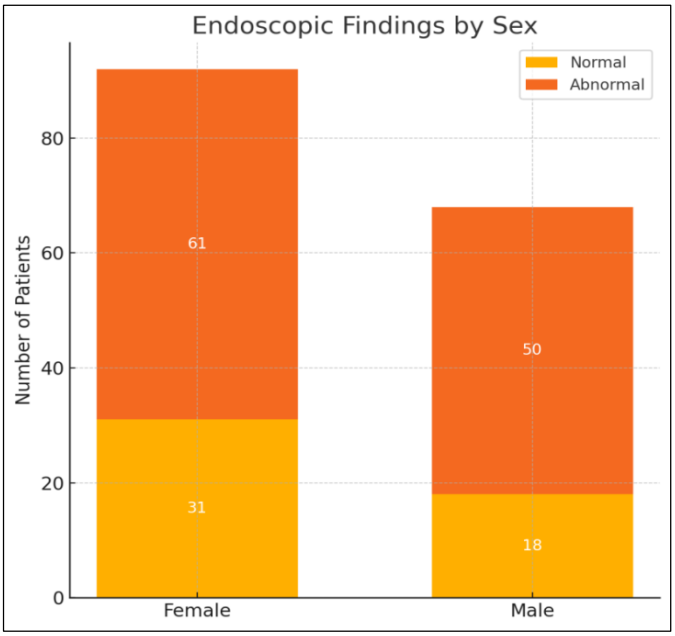


Table 9: NSAID use vs endoscopy outcome (χ^2)

NSAID	Normal	Abnormal
No	55	70
Yes	5	30
χ^2	8.6	p = 0.003

Among NSAID users, 30 patients had abnormal and 5 had normal endoscopic findings. In the non-NSAID group, 70 had abnormal and 55 had normal results. The chi-square test (χ^2 = 8.6, p = 0.003) demonstrate a statistically significant association between NSAID use and endoscopic outcome. This indicates that NSAID exposure was meaningfully linked to an increased likelihood of abnormal findings in this cohort.

Graph 9:

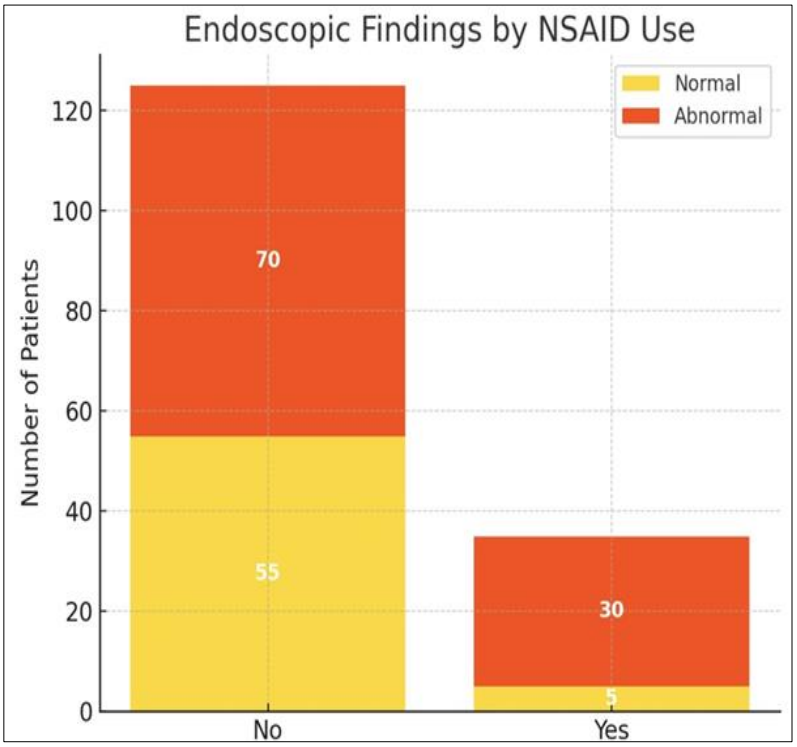
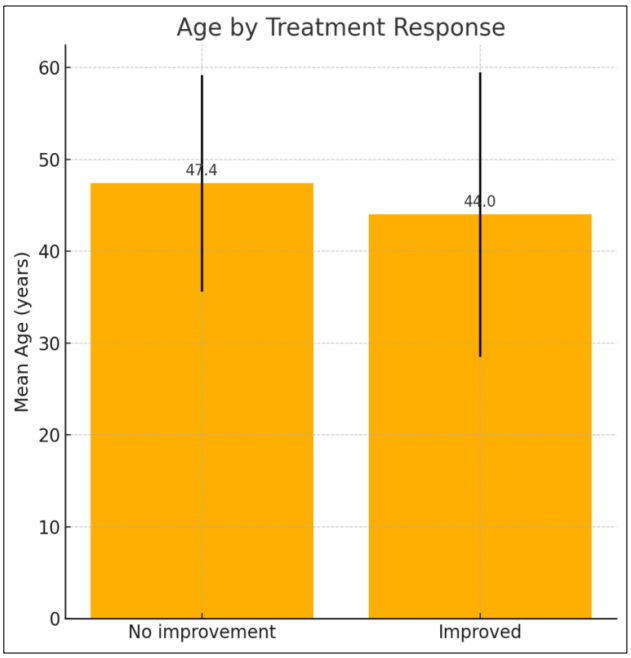


Table 10: Age vs follow-up improvement (Welch t-test)

Group	n	Mean ± SD (years)
No improvement	36	47.4 ± 11.8
Improved	124	44.0 ± 15.5
t-test	—	t = 1.40, p = 0.165

The mean age of patients who showed no improvement was 47.4 ± 11.8 years, compared to 44.0 ± 15.5 years in those who reported symptom relief. Although non-responders were slightly older on average, the difference was not statistically significant (t = 1.40, p = 0.165). This suggests that age did not substantially influence treatment outcomes in this cohort.

Graph 10:



Discussion

Our tertiary-centre study of 160 consecutively evaluated adults provides a contemporary snapshot of that conundrum in the Indian sub-continent. The cohort was middle-aged (mean 44.8 years), predominantly female, largely urban and mostly non-smoking. Endoscopy was normal in almost one-third, while benign inflammatory lesions—gastritis (33 %), duodenitis (8 %) and reflux esophagitis (6 %)—accounted for most abnormalities; ulcers and neoplasia were infrequent.[37]

Neither age, sex, symptom duration, smoking nor even classical "alarm features" predicted pathology except for NSAID and Acohol use which influences the endoscopic findings.[38] Logistic modelling was similarly unrevealing, and histopathology largely echoed endoscopic impressions, with chronic and Helicobacter pylori-associated gastritis dominating biopsies. Treatment response favoured acid-suppression and H. pylori eradication, yet female sex was the only independent correlate of symptomatic improvement. The following sections synthesise convergent and divergent findings, explore biological or methodological explanations, and highlight the implications for clinical practice and research. The mean age (44.8 y) and female preponderance (57.5 %) mirror recent Indian tertiary-centre reports (45–48 y; 52–58 % female) and lie midway between South-East Asian series, where male predominance reaches 60–65 %, and European cohorts that are older (>50 y) and gender-balanced.

These findings resonate with a growing international consensus: endoscopy should be judiciously reserved for a minority whose composite risk—driven by age > 50 y, unexplained weight loss, progressive dysphagia, bleeding or biomarker red flags—justifies invasive investigation. Future endeavours must pivot towards precision-triage tools, genotype-guided therapy, AI-enhanced mucosal interrogation and holistic psychosomatic interventions. Such evolution will ensure that scarce endoscopic resources are deployed where they matter most—detecting premalignant change early—while the vast majority of dyspeptics receive effective, evidence-based, minimally invasive care.

Conclusion

This comprehensive evaluation of dyspepsia underscores a reassuring yet diagnostically vexing reality: the vast majority of symptomatic patients harbor benign or functional conditions that respond well to guideline-directed medical therapy, whereas serious lesions are scarce and essentially invisible to clinical outline alone. Traditional heuristics—age thresholds, symptom clusters, and lifestyle exposures—proved inadequate except Alcohol and NSAID as triage tools, reinforcing the need for evidence-based algorithms that combine non-invasive therapy with selective endoscopic assessment. Importantly, treatment success was high, particularly among women and those receiving acid suppression or H. pylori eradication, suggesting that timely empiric management can both alleviate symptoms and conserve endoscopic resources. Moving forward, refining risk stratification may hinge less on clinical impressions and more on integrating emerging biomarkers, validated prediction scores, or point-of-care tests that elevate the precision of dyspepsia care while maintaining its hallmark safety. In sum, this 160-patient series underscores a familiar reality: most mid-life dyspepsia stems from benign mucosal inflammation that neither demographics nor classic "alarm" criteria can reliably single out. What truly moves the needle is appropriate therapy—acid suppression, Hp eradication, or simple lifestyle correction—while structural disease remains scarce but serious enough to mandate vigilance.

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