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Prevalence, anatomical pattern and modifiable risk factors of peptic ulcer disease among patients undergoing upper gastrointestinal endoscopy in central Nepal: a cross-sectional and nested matched case– control study

Achyoot Sharan Koirala

koiralaachyoot@gmail.com

College of Medical Sciences

Parikshit Prasai

Melamchi Primary Health Center

Ujjwal Kumar Shah

College of Medical Sciences

Garima Wagle

College of Medical Sciences

Shushil Sharma Timilsina

College of Medical Sciences

Bibek Luitel

College of Medical Sciences

Devnarayan Jaisy

College of Medical Sciences

Khus Raj Dewan

College of Medical Sciences

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Abstract

Background

Peptic ulcer disease (PUD) is an important cause of gastrointestinal morbidity in South Asia, yet contemporary endoscopy-based data from Nepal are limited and rarely capture modifiable risk factors. We aimed to estimate the prevalence and anatomical pattern of PUD and to examine its associated risk factors at a tertiary centre in central Nepal.

Methods

We conducted a retrospective cross-sectional study of 1,242 consecutive adults who underwent upper gastrointestinal endoscopy between April and December 2025. PUD was defined as an endoscopically confirmed gastric and/or duodenal ulcer. A nested case–control analysis matched 224 PUD cases to 448 ulcer-free controls (1:2) on age and sex; drug, lifestyle and comorbidity exposures were abstracted from medical records. Associations were assessed using logistic regression.

Results

PUD prevalence was 18.0% (224/1,242). The mean age was 49.6 ± 16.6 years and 50.4% were male. Ulcers were gastric-only in 67.4%, duodenal-only in 18.8% and both in 13.8%; the antrum was involved in 67.9% of cases. Among tested patients, Helicobacter pylori positivity was 94.8% and was near-universal in both cases (97.8%) and controls (95.3%), and therefore did not discriminate cases from controls (odds ratio 1.97; p = 0.19). In an exploratory case–control analysis, NSAID use (adjusted odds ratio [aOR] 7.41, 95% CI 2.09–26.29) and ever-smoking (aOR 2.11, 1.08–4.14) were associated with PUD, whereas alcohol, comorbidities, age and sex were not.

Conclusions

Approximately one in five endoscopy patients had PUD, predominantly antral, with a shift from duodenal towards gastric ulcers across increasing age and in women. Against a background of near-universal H. pylori infection, NSAID use and smoking were associated with ulceration in an exploratory case–control analysis. These contemporary descriptive data provide a baseline for central Nepal and generate hypotheses about modifiable risk factors that warrant confirmation in prospective studies.

Background

Peptic ulcer disease (PUD) is a break in the mucosal lining of the gastrointestinal tract that extends to the muscularis propria, arising from an imbalance between aggressive factors such as gastric acid and pepsin and protective mucosal mechanisms [1]. It most often involves the stomach and proximal

duodenum. Although the global age-standardised incidence, mortality and disability-adjusted life-years attributable to PUD have fallen by roughly 40–50% between 1990 and 2019, the burden remains disproportionately concentrated in low and low-middle sociodemographic-index regions, including South Asia [2].

Helicobacter pylori infection and the use of non-steroidal anti-inflammatory drugs (NSAIDs) are the two dominant causes of PUD, while smoking and alcohol are recognised contributory factors [3]. In high-income countries, widespread H. pylori eradication, declining smoking rates and proton-pump-inhibitor use have driven a marked decline in PUD [4]. In much of South Asia, however, H. pylori prevalence remains very high and unregulated over-the-counter NSAID use is common, so the relative contribution of these factors to ulcer formation may differ from that in Western populations [5,3]. Upper gastrointestinal endoscopy, with a sensitivity and specificity of about 90%, remains the diagnostic gold standard and allows simultaneous assessment of ulcer site, bleeding stigmata using the Forrest classification, and H. pylori status by rapid urease test [1,6].

Despite the clinical frequency of PUD, contemporary endoscopy-based prevalence data from Nepal are limited. Existing Nepali studies report widely varying prevalence figures, from 10.62% to 35.6%, are largely more than a decade old, and rarely characterise the detailed anatomical distribution of ulcers or the contribution of modifiable risk factors such as NSAID use and smoking [7,8]. No previous study has described PUD among endoscopy patients in the Chitwan region of central Nepal. Such local data are needed to inform endoscopic biopsy protocols, H. pylori testing strategies and counselling on modifiable risk factors.

We therefore undertook this study with two aims: (i) to estimate the prevalence and anatomical pattern of PUD among patients undergoing upper gastrointestinal endoscopy at a tertiary care centre in central Nepal; and (ii) using a nested matched case–control design, to identify the modifiable drug, lifestyle and comorbidity risk factors independently associated with PUD in this population.

Materials and methods

Study design and setting

This was a retrospective, record-based study comprising a cross-sectional prevalence component and a nested matched case–control component. It was conducted in the Department of Gastroenterology, College of Medical Sciences and Teaching Hospital (COMS-TH), Bharatpur, Chitwan a tertiary care teaching hospital that serves as the principal gastroenterology referral centre for the Chitwan district and the surrounding Terai belt of central Nepal. The unit performs 5–10 upper gastrointestinal endoscopies per day, six days a week, and maintains an electronic endoscopy database. The study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline [9].

Participants and case definition

All consecutive adult patients (≥ 18 years) who underwent upper gastrointestinal endoscopy for gastrointestinal symptoms between April and December 2025 were eligible. Duplicate records of the same endoscopy episode and records with incomplete endoscopy reports were excluded. PUD was defined as the endoscopic presence of one or more ulcers in the stomach (cardia, fundus, body, antrum or pylorus) and/or duodenum (first or second part). Patients without any endoscopically identified ulcer constituted the ulcer-free group.

Nested case–control sampling

For the risk-factor analysis, all 224 patients with a gastric and/or duodenal ulcer served as cases (patients with ulcers at atypical sites, such as jejunal or anastomotic ulcers, were not included, to maintain a consistent PUD case definition). For each case, two ulcer-free controls were selected from the same endoscopy cohort and matched on age (within approximately five years; achieved mean age difference 2.8 years) and sex, yielding 1:2 matching and 448 controls. Drug history (NSAIDs, aspirin, corticosteroids), personal history (smoking, alcohol, smokeless tobacco) and comorbidities were abstracted from outpatient tickets and admission records. Fields not recorded in the source document were classified as ‘not documented’ and treated as missing rather than as absent; the risk-factor analyses were therefore complete-case analyses for each variable.

Variables and measurements

Age, sex, endoscopic indication, ulcer site, Forrest classification of bleeding ulcers and H. pylori status were recorded for the prevalence cohort. H. pylori status was determined by rapid urease test on antral biopsies obtained as part of routine clinical care; results were categorised as positive, negative or not done. Smoking was classified as ever (current or former) versus never. NSAID, aspirin, corticosteroid and alcohol use were recorded as present, absent or not documented.

Statistical analysis

Continuous variables are summarised as mean ± standard deviation and categorical variables as counts and percentages. PUD prevalence was calculated as the proportion of endoscopy patients with an ulcer, with the denominator being all patients in the cohort. Associations between categorical variables were tested using the chi-square test. For the prevalence cohort, a multivariable logistic regression model estimated the adjusted association of age, sex and H. pylori status with PUD among patients with a documented H. pylori result. For the nested case–control component, univariable odds ratios (ORs) for each exposure were estimated using Firth penalized logistic regression, which yields finite, stable estimates for low-frequency exposures and zero cells; rare individual comorbidities were additionally summarised as a single ‘any comorbidity’ variable. A multivariable logistic regression model adjusted for

age, sex, NSAID use, smoking and alcohol use estimated associations; with 129 cases and five covariates the events-per-variable ratio was approximately 26, above the conventional minimum of ten, and the model was re-estimated using Firth penalized logistic regression to confirm stability. Because complete-case restriction disturbed the 1:2 matching, age and sex were retained as covariates in the multivariable model rather than relying on the matching, and a conditional logistic regression on the intact matched sets was performed as a sensitivity analysis. Odds ratios are presented with 95% confidence intervals (CIs); a two-sided p-value < 0.05 was considered statistically significant. Analyses were performed in Python 3 (NumPy and pandas), with maximum-likelihood logistic regression fitted by iteratively reweighted least squares and Firth penalized models using profile-likelihood confidence intervals.

To assess the robustness of the complete-case findings to missing risk-factor data, we pre-specified three sensitivity analyses. As the primary sensitivity analysis we used multiple imputation by chained equations under a missing-at-random assumption, generating 25 imputed datasets; the imputation models included case status, age, sex and all candidate exposures, and a bootstrap step was used at each imputation to propagate parameter uncertainty (proper imputation). Estimates from the imputed datasets were combined using Rubin's rules. As a worst-case bounding scenario we re-fitted the model after recoding every undocumented exposure as absent ('not documented' = 'No'); this assumes a specific and probably incorrect mechanism and is therefore presented only as an extreme bound. Finally, as a secondary robustness check we fitted a conditional logistic regression restricted to the matched sets that remained intact after excluding members with missing exposures.

Ethical considerations

The study was approved by the Institutional Review Committee of the College of Medical Sciences and Teaching Hospital (COMS-TH-IRC; approval number COMSTH-IRC/2026-023). Because the study used only existing, de-identified medical records and involved no patient contact, the requirement for individual informed consent was waived by the committee. Patient names were replaced with study identifiers before analysis, and the dataset was stored on a single password-protected device. The study was conducted in accordance with the Declaration of Helsinki.

Results

Participant flow and cohort characteristics

Of 1,349 endoscopy records retrieved during the study period, 107 were excluded as duplicates or for incomplete endoscopy reports, leaving 1,242 patients for the prevalence analysis (Fig. 1). The mean age was 49.6 ± 16.6 years, 50.4% (622/1234) were male, and acid peptic disease was the indication for endoscopy in 82.4% of patients (Table 1). A rapid urease test was performed in 883 patients, of whom 94.8% (837/883) were H. pylori positive; the test was not done in 359 patients.

Table 1
Characteristics of the prevalence cohort (N = 1,242).

Characteristic	Value
Total patients, n	1,242
Age, years (mean ± SD)	49.6 ± 16.6
Male, n (%)	622 (50.4%)
Female, n (%)	612 (49.6%)
Acid peptic disease indication, %	82.4%
H. pylori tested, n	883
H. pylori positive (of tested), n (%)	837 (94.8%)
PUD identified, n (%)	224 (18.0%)

SD, standard deviation; PUD, peptic ulcer disease.

Prevalence and anatomical pattern of PUD

PUD was identified in 224 patients, an overall prevalence of 18.0% (224/1,242). Ulcers were confined to the stomach in 151 patients (67.4%), confined to the duodenum in 42 (18.8%) and involved both sites in 31 (13.8%) (Fig. 2). Among the stomach, the antrum was by far the most frequently involved site, affected in 152 patients (67.9% of all PUD cases), followed by the body (16), fundus (13), pylorus (11) and cardia (1) (Fig. 2; Table 2).

Table 2
Anatomical distribution of peptic ulcer disease (n = 224 patients with PUD).

Ulcer location	n	% of PUD cases
Gastric only	151	67.4
Duodenal only	42	18.8
Both gastric and duodenal	31	13.8
Gastric site - antrum	152	67.9
Gastric site - body	16	7.1
Gastric site - fundus	13	5.8
Gastric site - pylorus	11	4.9
Gastric site - cardia	1	0.4

Gastric site categories are not mutually exclusive (some patients had multiple sites involved).

Prevalence by age and sex

PUD prevalence rose with age, from 10.9% in patients younger than 30 years to a peak of 23.7% in those aged 50–59 years (Fig. 3A). Prevalence was substantially higher in men (23.0%) than in women (13.2%) (Fig. 3B). In the multivariable model restricted to patients with a documented H. pylori result (n = 879), older age (aOR per year 1.02, 95% CI 1.01–1.03), male sex (aOR 2.36, 1.68–3.33) and a positive H. pylori test (aOR 3.16, 1.1–9.03) were each independently associated with PUD (Fig. 3A, 3B).

Bleeding stigmata (Forrest classification)

A Forrest grade was documented for 162 of 224 ulcers (72.3%) and was not recorded for 62 (27.7%) (Fig. 3C). Among classified ulcers, the overwhelming majority had a clean base (Forrest III, n = 145; 89.5%), indicating a low risk of rebleeding. A flat pigmented spot (Forrest IIc) was seen in 9 ulcers (5.6%). High-risk stigmata were uncommon: active oozing (Forrest Ib) was recorded in 6 ulcers and an adherent clot (Forrest IIb) in 3; no ulcer showed active spurting (Ia) or a non-bleeding visible vessel (IIa). Overall, 154 of 162 classified ulcers (95.1%) were low-risk lesions and only 9 (5.6%) carried high or intermediate rebleeding risk.

Age- and sex-related variation in ulcer site

The anatomical distribution of ulcers shifted with age. Among the 193 patients whose ulcers were confined to a single region (gastric-only or duodenal-only), the proportion that was duodenal fell progressively from 35.7% in patients younger than 40 years to 19.0% at 40–59 years and 16.4% at 60 years or older (test for trend p = 0.0261). In a logistic model, each additional year of age reduced the odds that an ulcer was duodenal rather than gastric (odds ratio 0.974, 95% CI 0.954–0.995; p = 0.0159), equivalent to an odds ratio of 0.77 per decade (Fig. 4A). A parallel sex difference was evident: the gastric-to-duodenal ulcer ratio was 3.63:1 in women versus 2.09:1 in men, indicating a stronger gastric predominance in women (Fig. 4B). Despite these associations, the multivariable risk models discriminated only modestly between patients with and without PUD (c-statistic 0.65 for the cohort model and 0.62 for the case–control model), indicating that the identified factors are risk markers rather than the basis for an individual-level diagnostic rule.

Nested case–control analysis of risk factors

The case–control component compared 224 PUD cases with 448 age- and sex-matched ulcer-free controls (cases: mean age 53.2 ± 15.8 years, 63.8% male; controls: 53.1 ± 15.8 years, 63.8% male) (Table 3). Of these, 132 cases and 152 controls had at least one risk factor documented and contributed to the univariable analyses (Table 4), while 129 cases and 139 controls (268 patients in total) had complete data for every covariate in the multivariable model (Fig. 1).

Notably, H. pylori positivity was near-universal in both groups 97.8% of cases and 95.3% of controls and therefore did not distinguish cases from controls (OR 1.97, 95% CI 0.72–6.53; p = 0.19). In contrast, the

modifiable factors differed between groups (Table 4). NSAID use was reported in 13.7% of cases versus 2.1% of controls (Firth OR 6.5, 2.26–25.09), and ever-smoking in 26.7% versus 12.1% (OR 2.61, 1.42–4.95). Alcohol use, smokeless-tobacco use and comorbidity whether considered individually or as a composite 'any comorbidity' variable (OR 1.49, 0.94–2.39) were not significantly associated with PUD; the estimate for aspirin use was not reliable because no control was exposed (2/130 cases vs 0/148 controls).

In the multivariable model (n = 268; 129 cases, 139 controls; events-per-variable ≈ 26), NSAID use (aOR 7.41, 95% CI 2.09–26.29; p = 0.002) and ever-smoking (aOR 2.11, 1.08–4.14; p = 0.029) were the only factors independently associated with PUD, whereas alcohol use, age and sex were not (Table 5; Fig. 5). Because complete-case restriction disturbed the matching, age and sex were retained as covariates. Re-estimating the model with Firth penalized logistic regression gave concordant results (NSAID aOR 6.39, 95% CI 2.18–24.99; smoking aOR 2.07, 1.07–4.06), confirming that the estimates were not artefacts of small-sample instability.

These findings were robust across the three sensitivity analyses (Supplementary Table S1). Under multiple imputation (primary sensitivity analysis; 25 datasets, n = 671), the associations were unchanged or strengthened for NSAID use (aOR 8.92, 95% CI 2.48–32.08) and ever-smoking (aOR 2.27, 1.23–4.19), while alcohol use remained non-significant (aOR 1.15). Conditional logistic regression restricted to the 97 intact matched sets gave concordant point estimates (NSAID aOR 8.45, 1.74–40.92; smoking aOR 2.42, 1.08–5.43) with wider confidence intervals reflecting reduced power. Even in the implausible worst-case bound in which every undocumented exposure was treated as absent, the NSAID and smoking associations strengthened (aOR 10.75 and 2.99). Across all approaches, NSAID use and smoking remained the factors most consistently associated with PUD.

Table 3
Characteristics of cases and matched controls in the nested case–control study.

Characteristic	**Cases (n = 224)**	**Controls (n = 448)**
Age, years (mean ± SD)	53.2 ± 15.8	53.1 ± 15.8
Male, %	63.8%	63.8%
H. pylori positive (of tested), %	97.8%	95.3%

Table 4
Risk factors for peptic ulcer disease: univariable odds ratios (Firth penalized logistic regression).

Risk factor	Cases, n/N (%)	Controls, n/N (%)	OR (95% CI)	p
NSAID use	18/131 (13.7%)	3/142 (2.1%)	6.5 (2.26–25.09)	< 0.001
Smoking (ever)	35/131 (26.7%)	18/149 (12.1%)	2.61 (1.42–4.95)	0.002
Alcohol use	61/131 (46.6%)	62/148 (41.9%)	1.21 (0.75–1.94)	0.434
Tobacco chewing	21/131 (16.0%)	14/150 (9.3%)	1.83 (0.91–3.8)	0.092
Aspirin use	2/130 (1.5%)	0/148 (0.0%)	Not estimable*	-
Steroid use	7/128 (5.5%)	2/148 (1.4%)	3.62 (0.95–19.64)	0.061
Diabetes	17/132 (12.9%)	16/151 (10.6%)	1.24 (0.61–2.57)	0.550
Hypertension	44/132 (33.3%)	37/151 (24.5%)	1.54 (0.92–2.58)	0.102
CLD/Cirrhosis	19/130 (14.6%)	21/150 (14.0%)	1.05 (0.54–2.05)	0.878
CKD	4/132 (3.0%)	4/150 (2.7%)	1.14 (0.29–4.5)	0.847
CVD/Stroke	5/132 (3.8%)	3/149 (2.0%)	1.81 (0.47–7.92)	0.388
COPD	8/131 (6.1%)	2/149 (1.3%)	4.06 (1.1–21.76)	0.035
Any comorbidity	67/132 (50.8%)	62/152 (40.8%)	1.49 (0.94–2.39)	0.093
H. pylori positive	174/178 (97.8%)	285/299 (95.3%)	1.97 (0.72–6.53)	0.194

OR, odds ratio; CI, confidence interval; NSAID, non-steroidal anti-inflammatory drug; CLD, chronic liver disease; CKD, chronic kidney disease; CVD, cardiovascular disease; COPD, chronic obstructive pulmonary disease. Odds ratios were estimated by Firth penalized logistic regression with profile-likelihood confidence intervals, which remain stable for low-frequency exposures. ‘Any comorbidity’ denotes one or more of diabetes, hypertension, CLD, CKD, CVD or COPD. *Aspirin use was not reliably estimable because no control was exposed (2/130 cases vs 0/148 controls).

Table 5
Factors associated with peptic ulcer disease: multivariable logistic regression (n = 268; 129 cases, 139 controls).

Variable	Adjusted OR (95% CI)	p
NSAID use	7.41 (2.09–26.29)	0.002
Smoking (ever)	2.11 (1.08–4.14)	0.029
Alcohol use	1.1 (0.64–1.87)	0.736
Male sex	1.09 (0.63–1.88)	0.749
Age (per year)	1.0 (0.99–1.02)	0.758

Maximum-likelihood estimates are shown; events-per-variable ≈ 26. Firth penalized estimates were concordant (NSAID aOR 6.39, 95% CI 2.18–24.99; smoking aOR 2.07, 1.07–4.06; alcohol aOR 1.09, 0.64–1.85).

Discussion

In this study of 1,242 consecutive patients undergoing upper gastrointestinal endoscopy at a tertiary centre in central Nepal, peptic ulcer disease was present in 18.0% of patients, ulcers were overwhelmingly antral, and against a background of near-universal H. pylori infection NSAID use and smoking were the factors that independently distinguished ulcer cases from matched controls. Each of these findings is considered in turn below.

The observed prevalence of 18.0% (approximately one in five patients) lies within the wide range previously reported from Nepal (10.62–35.6%) but is derived from a substantially larger and more recent cohort than earlier single-centre series and represents the first such estimate from the Chitwan region [7,10]. It is also consistent with the elevated PUD burden described for low and low-middle sociodemographic-index regions in the Global Burden of Disease analysis [2]. The rise in prevalence with age and the near two-fold higher prevalence in men mirror established demographic patterns and can help identify patients in whom a lower threshold for endoscopic referral may be warranted [3,4].

The anatomical pattern was striking. Two-thirds of patients with PUD (67.9%) had antral involvement, and the antrum alone accounted for 152/274 (55.5%) of all ulcer instances, a proportion markedly higher than the 30–40% typically reported from Western endoscopy series. Gastric ulcers also outnumbered duodenal ulcers, a distribution that contrasts with the duodenal predominance historically described in some South Asian and African cohorts [11]. This antral and gastric predominance is biologically consistent with antral-predominant H. pylori gastritis, and it has a direct practical implication: it supports protocol-driven antral sampling for H. pylori and histology during the endoscopic evaluation of dyspepsia in this population, with a low threshold for biopsy of antral ulcers to exclude malignancy.

This gastric predominance was not uniform across the population but varied systematically with age and sex. Duodenal ulcers were relatively common in the young (accounting for 35.7% of single-region ulcers in those under 40) but became progressively less frequent with age (down to 16.4% at 60 years or older; odds ratio 0.77 per decade), and the gastric predominance was more marked in women than in men. This age-related transition from a duodenal towards a gastric phenotype echoes patterns reported elsewhere [8] but, to our knowledge, has not previously been quantified in a Nepali endoscopy population. It is biologically coherent: duodenal ulceration in younger patients is consistent with high acid output and antral-predominant H. pylori gastritis, whereas the shift towards gastric (especially non-antral) ulcers with age reflects the progression to atrophic, corpus-predominant gastritis and the accumulating contribution of NSAID exposure. Clinically, it reinforces the need for thorough gastric inspection and biopsy in older patients, in whom a gastric ulcer carries a higher index of suspicion for malignancy.

The Forrest classification provided further clinically relevant insight. Among the 162 ulcers with a documented grade, the overwhelming majority were clean-based (Forrest III) and only a small minority showed high- or intermediate-risk stigmata of recent haemorrhage (Forrest Ib or IIb); no ulcer showed active spurting (Ia) or a non-bleeding visible vessel (IIa). This preponderance of low-risk lesions is expected in a cohort in which the great majority of endoscopies were performed for acid-peptic symptoms rather than for acute upper gastrointestinal bleeding, and it implies that most ulcers in this setting can be managed medically without endoscopic haemostasis, reserving endoscopic therapy and intensive monitoring for the few high-risk (Forrest Ia–IIb) ulcers in accordance with international guidance [6]. Importantly, however, a Forrest grade was not recorded for 62 of 224 ulcers (27.7%); because the Forrest grade directly informs rebleeding risk and the need for intervention, this documentation gap is a tangible target for quality improvement through structured, checklist-based endoscopic reporting.

The nested case–control analysis clarified the role of the principal aetiological agents. H. pylori positivity was extraordinarily high and near-identical in cases and controls (97.8% versus 95.3%), so it did not discriminate patients with ulcers from those without. Importantly, this does not contradict the positive association seen in the prevalence-cohort regression: both point estimates lay above unity (cohort aOR 3.16; case–control OR 1.97) but rested on very few unexposed individuals (only 46 H. pylori-negative patients in the cohort and 18 across the case–control sample), so both estimates were imprecise. The most coherent interpretation is that H. pylori is so prevalent in this population that it cannot, by itself, distinguish who ulcerates, and that its apparent cohort-level association is additionally vulnerable to selection because testing was not universal. Against this near-universal background, NSAID use (adjusted OR 7.41, 95% CI 2.09–26.29) and ever-smoking (adjusted OR 2.11, 1.08–4.14) were the factors most strongly associated with ulceration; given the retrospective design and incomplete documentation, these associations are best regarded as exploratory and hypothesis-generating rather than as established causal effects.

This pattern is biologically coherent and clinically actionable. Where H. pylori infection is almost universal, the additional mucosal insult from NSAIDs and the impaired mucosal defence and healing associated with smoking plausibly tip susceptible individuals towards overt ulceration [3,4]. The implication is that, in settings such as ours, a test-and-treat strategy for H. pylori while still appropriate is unlikely on its own to abolish the ulcer burden. Parallel investment in NSAID stewardship (curbing unregulated over-the-counter analgesic use and co-prescribing gastroprotection for high-risk users) and in smoking cessation is likely to yield substantial additional benefit, and these interventions can be targeted preferentially at older men, in whom prevalence was highest.

Our study has several strengths: a large, consecutive and contemporary cohort; a clear endoscopic case definition; and a matched design that allowed the modifiable risk factors absent from earlier Nepali studies to be examined directly. Several limitations should be acknowledged. First, the retrospective record-based design meant that risk-factor data were frequently undocumented, and missingness was greater among controls than cases that is, it was related to case status. Because case status is observed, this pattern is compatible with a missing-at-random mechanism conditional on the observed data; reassuringly, our primary multiple-imputation analysis, which makes this assumption explicit, yielded estimates essentially identical to the complete-case results, and the associations persisted even under a worst-case bound. Nonetheless, residual missing-not-at-random bias cannot be fully excluded, and power was limited for less prevalent exposures such as aspirin and corticosteroid use, for which associations were suggestive but did not reach significance. Second, H. pylori testing was not performed in all patients, which may bias the cohort-level estimate of its association with PUD. Third, the cross-sectional and case–control design cannot establish causality. Finally, as a single-centre tertiary study, the findings may not generalise to community or primary-care settings. Prospective studies with systematic risk-factor ascertainment and universal H. pylori testing are warranted to confirm these associations.

Conclusions

In this large, contemporary endoscopy series from central Nepal, peptic ulcer disease affected approximately one in five patients and was predominantly antral, with a clear shift from duodenal towards gastric ulcers across age and a stronger gastric predominance in women. These descriptive findings are the study's principal contribution and provide baseline institutional data for the region. Against a background of near-universal H. pylori infection, the exploratory case–control analysis identified NSAID use and smoking as the factors most strongly associated with ulceration; while hypothesis-generating rather than confirmatory, these associations support attention to NSAID stewardship and smoking cessation alongside H. pylori management and warrant prospective evaluation.

Declarations

Ethics approval and consent to participate

The study was approved by the Institutional Review Committee of the College of Medical Sciences and Teaching Hospital (COMS-TH-IRC; approval number COMSTH-IRC/2026-023). As the study used only existing, de-identified medical records with no patient contact, the committee granted a waiver of individual informed consent. The study was conducted in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors have declared that no competing interests exist.

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Author Contribution

ASK (Achyoot Sharan Koirala) conceived and designed the study, collected and curated the data, performed the statistical analysis and drafted the manuscript. KRD (Khus Raj Dewan), as senior author, supervised the study, provided gastroenterological and academic oversight, and critically revised the manuscript for important intellectual content. BL (Bibek Luitel) and DJ (Devnarayan Jaisy) contributed to the study design and methodology, supervised data collection and verification, and critically revised the manuscript. PP (Parikshit Prasai), UKS (Ujjwal Kumar Shah), GW (Garima Wagle) and SST (Shushil Sharma Timilsina) carried out data collection and curation and reviewed the manuscript. ASK and KRD take responsibility for the integrity of the data and the accuracy of the analysis. All authors read and approved the final manuscript.

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Data Availability

All relevant data are within the manuscript. The de-identified dataset underlying the findings is available from the corresponding author on reasonable request, subject to the data-sharing policy of the COMS-TH Institutional Review Committee.

Supplementary material

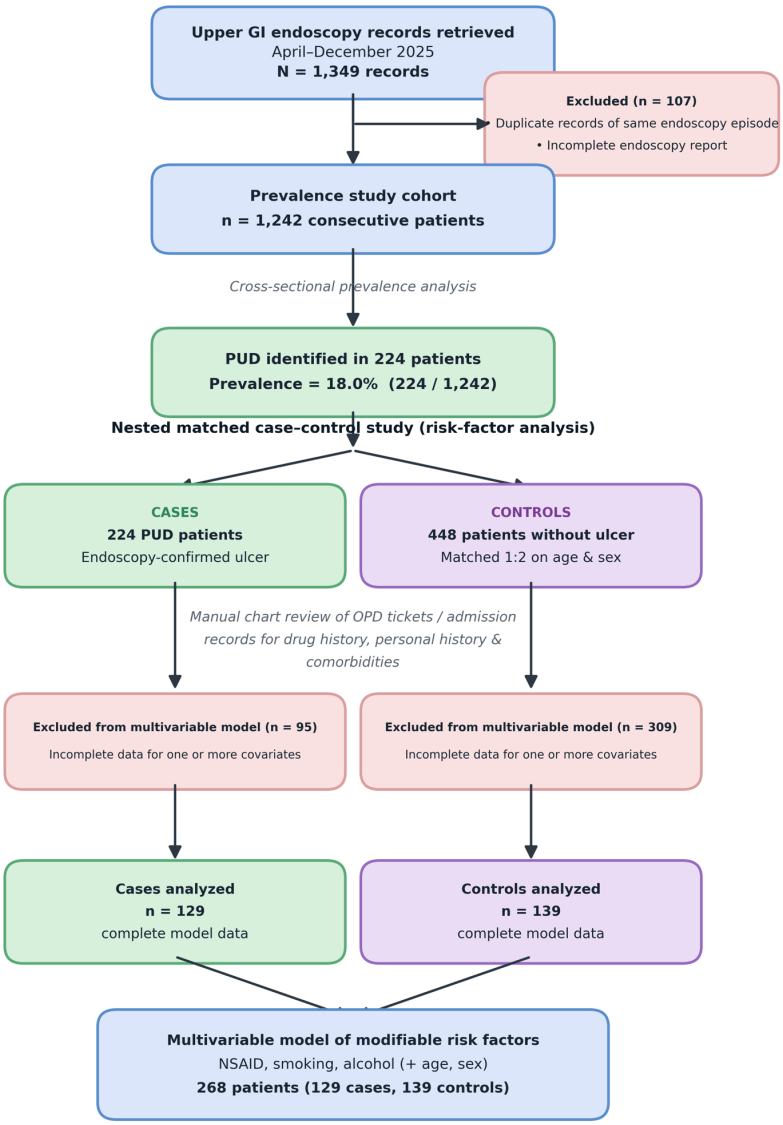
Supplementary Table S1. Sensitivity analyses of the multivariable model for peptic ulcer disease: complete-case, multiple imputation, worst-case (not-documented = ‘No’) bound, and conditional logistic regression on intact matched sets (provided as a separate file).

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Figures

Figure 1. Participant flow through the prevalence and nested case–control study



PUD, peptic ulcer disease; OPD, outpatient department. Undocumented risk-factor fields were treated as missing, not absent (complete-case approach). The multivariable model required complete covariate data; univariable analyses (Table 4) used all participants with the relevant variable documented (132 cases and 152 controls had at least one risk factor documented).

Figure 1

See image above for figure legend.

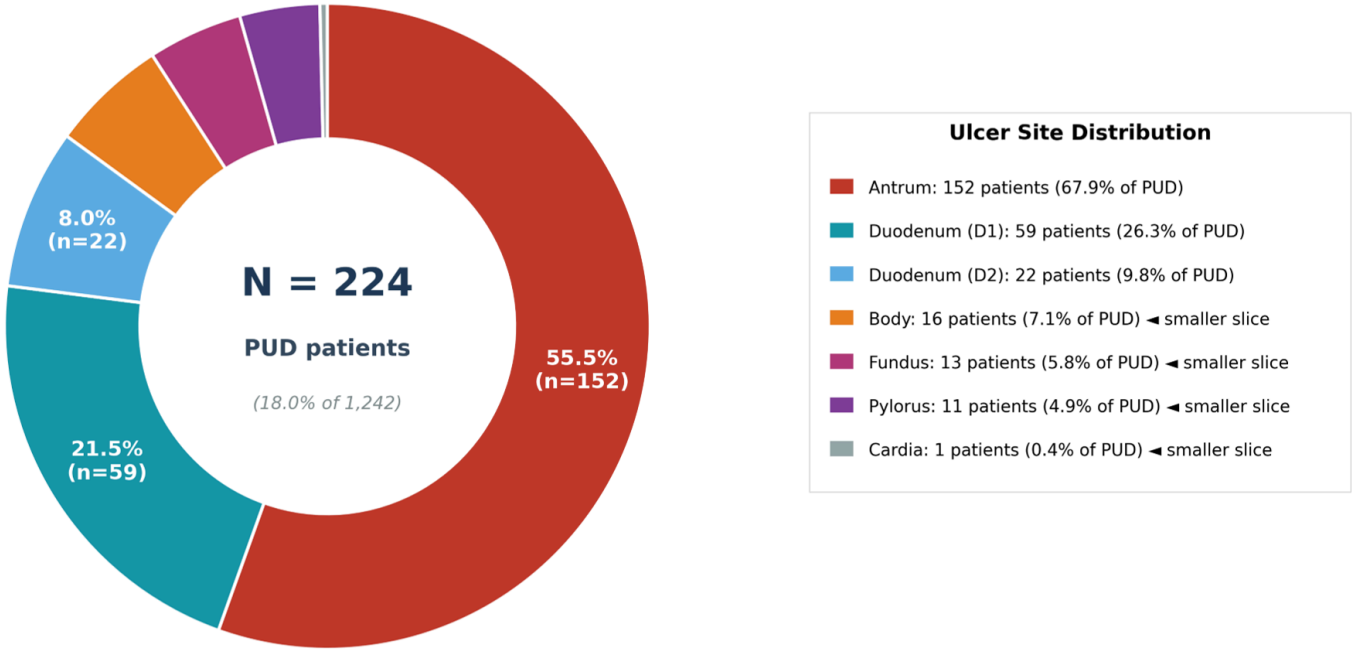


Figure 2

Anatomical distribution of ulcer sites among 224 patients with peptic ulcer disease. The antrum was involved in 67.9% of patients and accounted for 55.5% of all ulcer instances. Site categories are not mutually exclusive; total instances (274) exceed the number of patients (224) because some patients had ulcers at multiple sites.

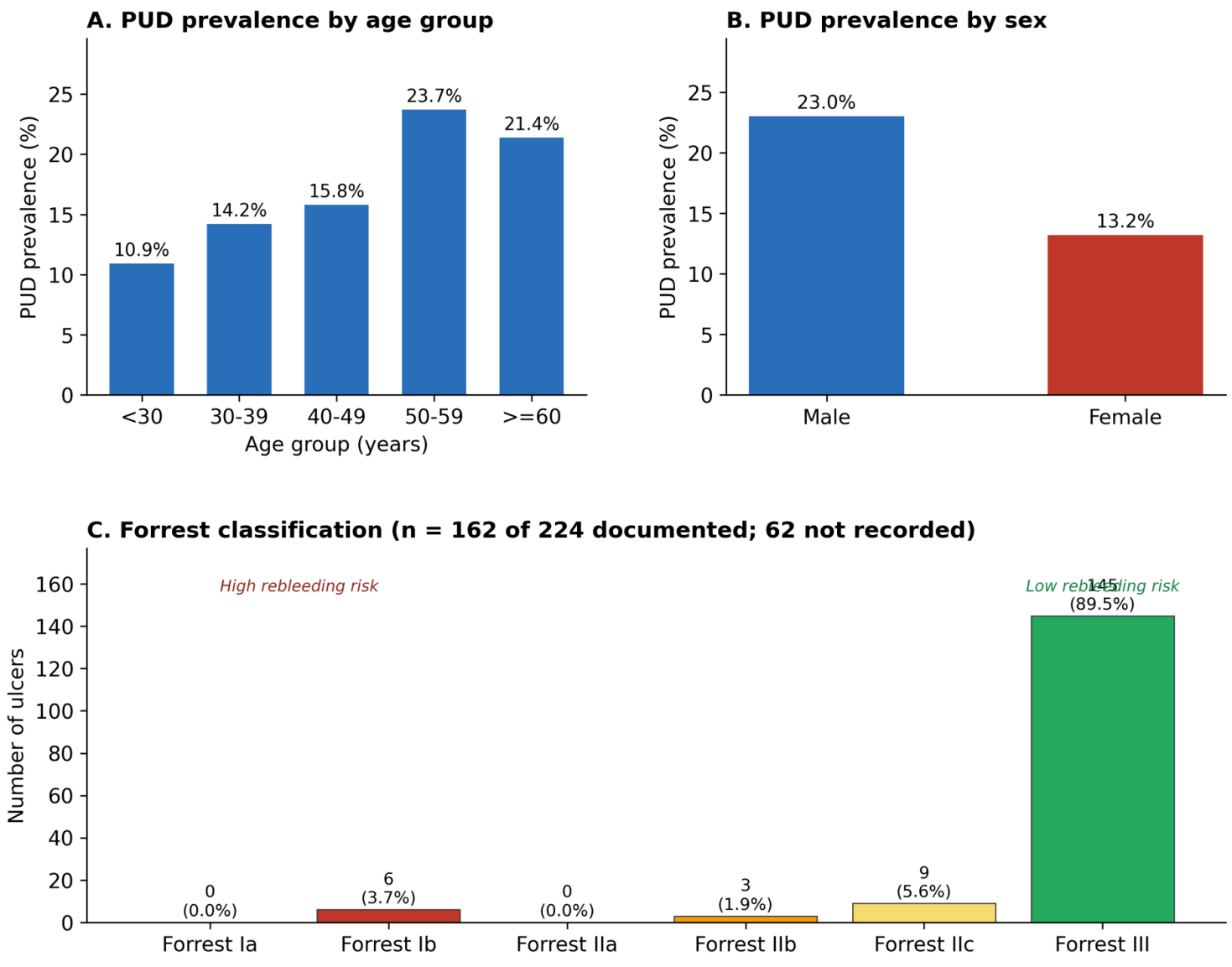


Figure 3

Demographic and endoscopic profile of peptic ulcer disease. (A) PUD prevalence by age group; (B) PUD prevalence by sex; (C) Forrest classification among the 162 ulcers with a documented bleeding grade (grades Ia–IIb, high or intermediate rebleeding risk; IIc and III, low risk).

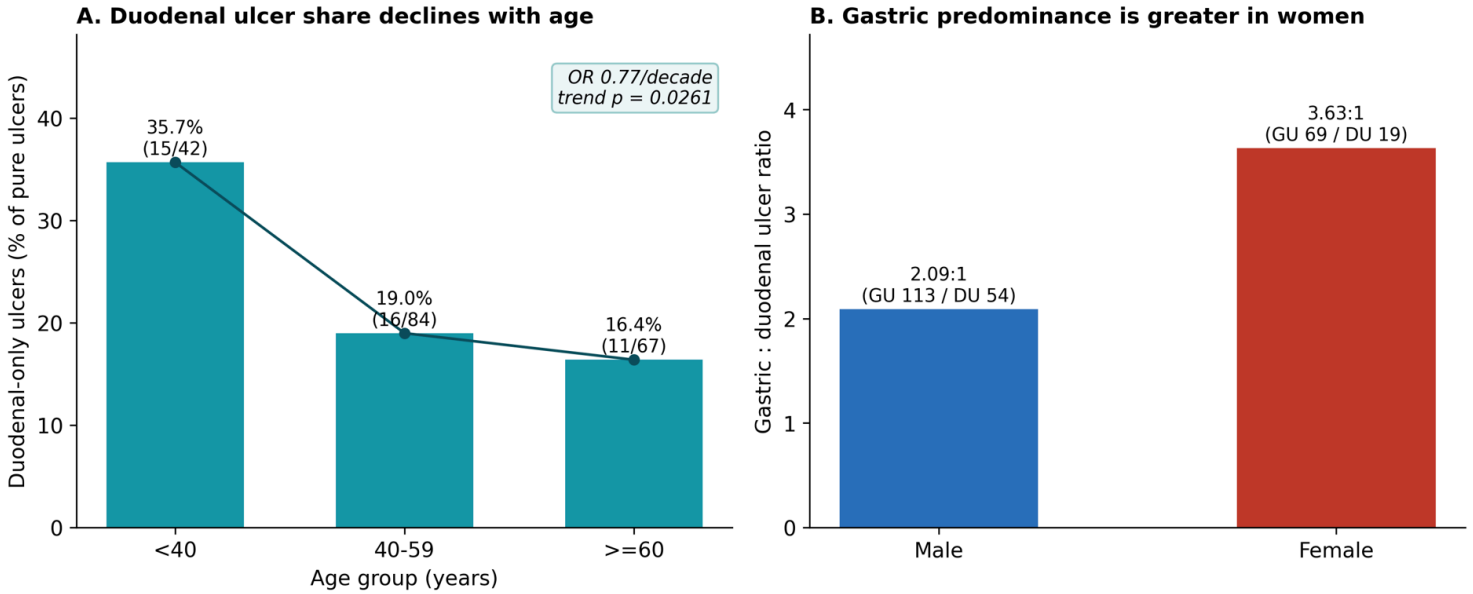


Figure 4

Age- and sex-related variation in ulcer site among patients with peptic ulcer disease. (A) Duodenal ulcers as a proportion of single-region ulcers, by age group (with test-for-trend); (B) gastric-to-duodenal ulcer ratio by sex.

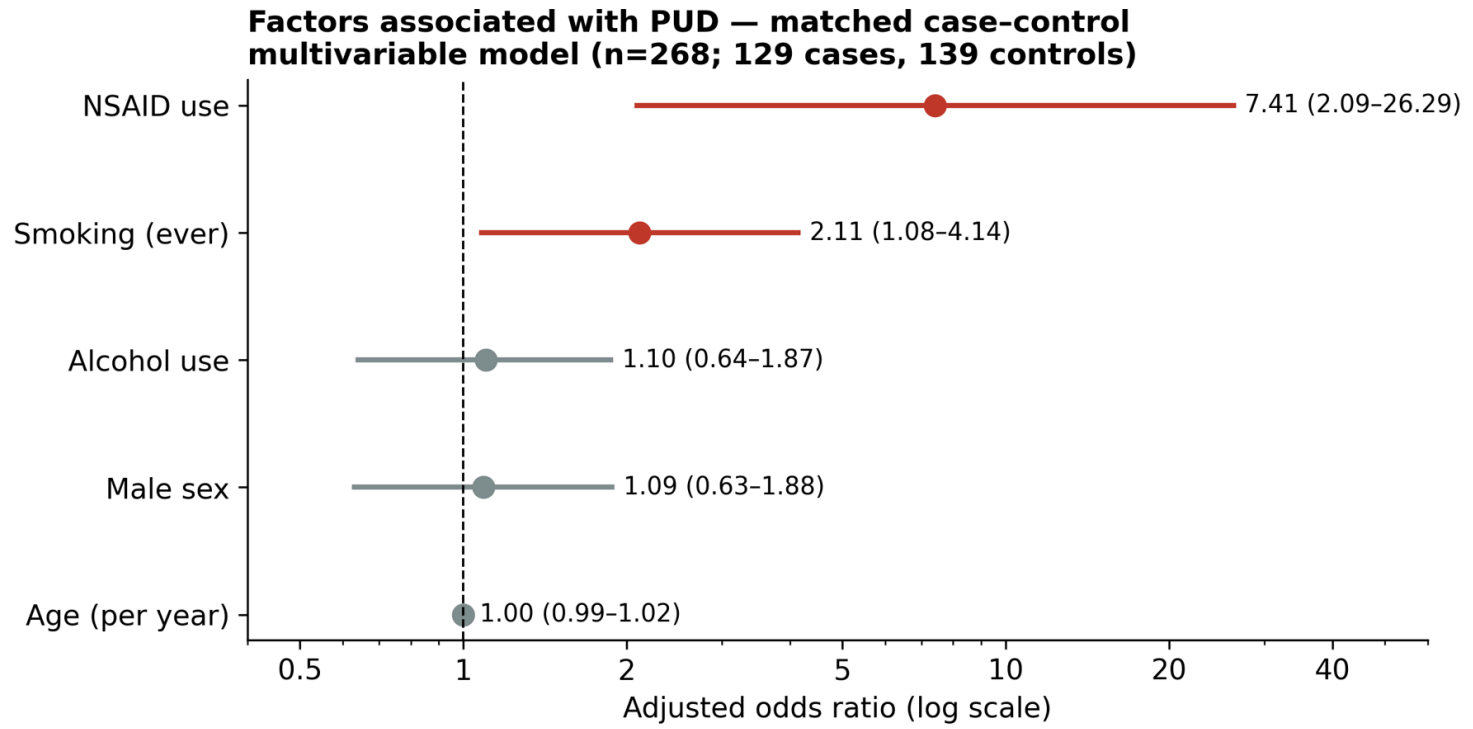


Figure 5

Forest plot of adjusted odds ratios for independent predictors of PUD in the matched case–control analysis.

Supplementary Files

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- Supplementarymaterial.docx