RESEARCH PAPER

Prevalence of Primary Bile Reflux Gastritis in Patients with Chronic Gastritis at a Tertiary Care Centre: A Single Centre Retrospective Cross-Sectional Study

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ABSTRACT

Background: Primary bile reflux gastritis (PBRG) is a distinct clinicopathological entity caused by the retrograde flow of duodenal contents into the stomach, resulting in gastric mucosal injury. Its prevalence among patients presenting with chronic gastritis symptoms remains poorly characterised, especially in the Indian subcontinent.

Objective: To determine the prevalence of PBRG among patients with chronic gastritis presenting to a tertiary care centre, and to evaluate associated clinical features, symptom profiles, and lifestyle risk factors.

Methods: A retrospective cross-sectional study was conducted at a tertiary care centre. Data of 120 patients diagnosed with chronic gastritis on upper gastrointestinal endoscopy were retrieved. Demographic details, presenting symptoms (heartburn, reflux, nausea), and risk factor data (smoking, alcohol consumption, consumption of outside food) were recorded. Endoscopic diagnoses were categorised as pan gastritis, lax lower oesophageal sphincter (LOS), PBRG, antral gastritis, or oesophagitis.

Results: Of 120 patients (mean age 43.0 ± 9.8 years; 78 males, 42 females), PBRG was the third most common diagnosis, identified in 26 patients (21.7%). Pan gastritis was most prevalent (48; 40.0%), followed by lax LOS (34; 28.3%), antral gastritis (8; 6.7%), and oesophagitis (4; 3.3%). Among PBRG patients, heartburn (88.5%) and regurgitation (73.1%) were the predominant symptoms. Smoking and alcohol were each documented in 69.2% of PBRG cases. Males constituted 73.1% of PBRG patients.

Conclusion: PBRG accounts for approximately one in five patients presenting with chronic gastritis symptoms at our centre, with a male predominance and high association with tobacco and alcohol use. Heightened clinical suspicion and targeted endoscopic evaluation are warranted in these patients.

Keywords: *Primary bile reflux gastritis; chronic gastritis; duodenogastric reflux; upper GI endoscopy; prevalence; tertiary care*

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INTRODUCTION

Chronic gastritis is one of the most common diagnoses encountered during upper gastrointestinal endoscopy globally, with a substantial burden in South and Southeast Asia where *Helicobacter pylori* infection, dietary habits, and environmental exposures converge to heighten mucosal susceptibility.[1,2] Within the broad spectrum of chronic gastritis, primary bile reflux gastritis (PBRG) occupies a distinct niche, characterised pathophysiologically by the retrograde entry of bile acids, lysolecithin, and other duodenal secretions into the stomach in the absence of prior gastric surgery.[3,4]

Bile reflux gastritis was long regarded as a complication exclusive to post-gastrectomy or post-cholecystectomy states.[5] However, accumulating evidence has established that primary (non-surgical) bile reflux can

occur in individuals with an intact gastrointestinal tract, mediated by dysmotility of the pylorus and duodenum, impaired antropyloric coordination, and laxity of the lower oesophageal sphincter (LOS).[6,7] The resulting mucosal damage is characterised by foveolar hyperplasia, oedema, vascular congestion, and smooth muscle proliferation in the lamina propria, findings distinct from those seen in *H. pylori*-associated gastritis.[8,9]

Clinically, PBRG presents with a triad of epigastric pain, heartburn, and bile-stained vomiting, though symptoms may be non-specific and overlap considerably with other forms of gastro-oesophageal reflux disease and functional dyspepsia.[10,11] Lifestyle factors such as tobacco smoking and alcohol consumption have been implicated in exacerbating duodenogastric reflux by adversely affecting pyloric competence and gastric emptying.[12,13]

The epidemiology of PBRG in the Indian population is not well-characterised, with limited data from tertiary care centres. Most published prevalence estimates originate from Western and East Asian cohorts, and the extent to which indigenous dietary practices, spice consumption, and regional lifestyle factors modify the prevalence and clinical expression of PBRG remains underexplored.[14,15]

The diagnosis of PBRG rests primarily on endoscopic findings, including erythema, oedema, and friability of the gastric mucosa, accompanied by bile staining of gastric contents, in conjunction with histopathological confirmation where available.[16,17] The condition carries clinical importance beyond symptom burden, as prolonged bile acid exposure has been linked to intestinal metaplasia and, in some series, to an increased risk of gastric carcinogenesis.[18,19]

Given the paucity of Indian data and the potential oncological implications of untreated PBRG, the present study was undertaken to determine the prevalence of PBRG among patients with chronic gastritis at a tertiary care centre, and to characterise the associated demographic profile, symptom pattern, and modifiable lifestyle risk factors.

MATERIALS AND METHODS

Study Design and Setting

This was a single-centre retrospective cross-sectional study conducted at Department of General Surgery, Chettinad Hospital and Research Institute, Chettinad. Medical records and endoscopic reports of patients who underwent upper gastrointestinal endoscopy for evaluation of chronic upper gastrointestinal symptoms were reviewed. The study was conducted in accordance with the principles of the Declaration of Helsinki[20] and institutional ethical approval was obtained vide ref. no. IHEC-I/080/01/2026, prior to data collection.

Study Population

Patients of either sex, aged 18 years or above, who were endoscopically diagnosed with chronic gastritis and had complete clinical records available were included. Patients with a prior history of gastric surgery (partial or total gastrectomy), cholecystectomy, bariatric surgery, or known inflammatory bowel disease were excluded, as these conditions independently predispose to secondary bile reflux and would confound the assessment of primary bile reflux.[21] Patients with incomplete clinical data or without formal endoscopic documentation were also excluded.

Data Collection

Demographic variables including age, sex, and presenting symptoms (heartburn, regurgitation, and nausea) were recorded from case files. Relevant lifestyle risk factors documented at the time of clinical assessment included smoking status, alcohol consumption, and habitual consumption of outside food (defined as frequent consumption of commercially prepared meals, street food, or restaurant food, ≥3 times per week).[22]

Endoscopic Diagnosis

All upper gastrointestinal endoscopies were performed by experienced endoscopists using standard video endoscopy systems. Endoscopic findings were documented and categorised by the reporting endoscopist into one of five diagnostic categories: (1) pan gastritis, defined as diffuse mucosal inflammation affecting the entire stomach; (2) lax lower oesophageal sphincter (lax LOS), characterised by incompetence of the LOS without frank oesophagitis; (3) primary bile reflux gastritis (PBRG), diagnosed on the basis of characteristic endoscopic features including mucosal erythema, oedema, bile-stained fluid pooling in the stomach, and absence of prior gastric surgery; (4) antral gastritis, with inflammation restricted to the antrum; and (5) oesophagitis, characterised by mucosal breaks or erosions in the oesophagus.[16,23]

Statistical Analysis

Data were entered and analysed using standard statistical software. Categorical variables are presented as frequencies and percentages. Continuous variables are reported as mean ± standard deviation (SD). The prevalence of PBRG was calculated as the proportion of PBRG cases among all patients with chronic gastritis.

Subgroup analyses were performed for gender and age group distributions. A p-value of <0.05 was considered statistically significant for any inferential comparisons.[24]

RESULTS

Demographic Profile of the Study Population

A total of 120 patients with chronic gastritis were included in the study. The mean age of the study population was 43.0 ± 9.8 years (range: 22–60 years). The majority of patients were male (n = 78, 65.0%), with females constituting 35.0% (n = 42) of the cohort. The most represented age group was 51–60 years (n = 37, 30.8%), followed by 41–50 years (n = 34, 28.3%), 31–40 years (n = 33, 27.5%), and 21–30 years (n = 16, 13.3%). No patient below 21 years or above 60 years was included in this cohort.

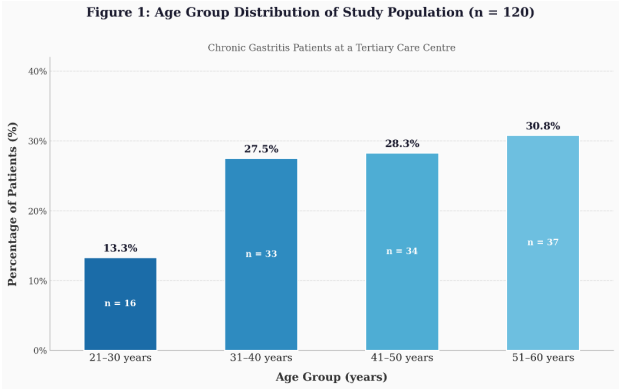

Figure 1: Bar chart showing age group distribution of study population

Table 1: Demographic and clinical profile of the study population (n = 120)

Variable	Category / Value	n (%)
Total patients	–	120 (100%)
Gender	Male	78 (65.0%)
	Female	42 (35.0%)
Mean age (years)	43.0 ± 9.8	Range: 22–60
Age group	21–30 years	16 (13.3%)
	31–40 years	33 (27.5%)
	41–50 years	34 (28.3%)
	51–60 years	37 (30.8%)

Endoscopic Diagnosis Distribution

Pan gastritis was the most common endoscopic diagnosis, identified in 48 patients (40.0%). Lax lower oesophageal sphincter was the second most frequent diagnosis (n = 34, 28.3%). Primary bile reflux gastritis was diagnosed in 26 patients, constituting 21.7% of the cohort, making it the third most common diagnosis. Antral gastritis was observed in 8 patients (6.7%) and oesophagitis in 4 patients (3.3%). The distribution of diagnoses is summarised in Table 2.

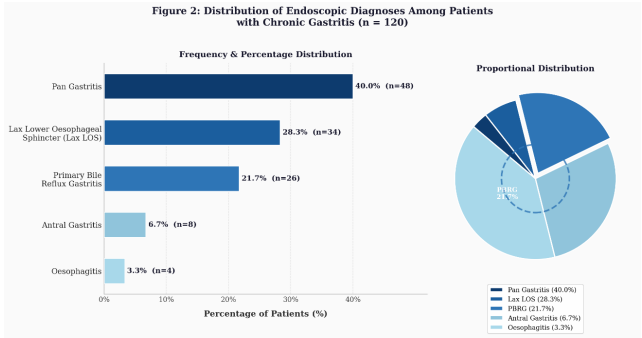

Figure 2: Pie chart or bar chart showing percentage distribution of endoscopic diagnoses

Table 2: Distribution of endoscopic diagnoses among study patients (n = 120)

Diagnosis	Frequency (n)	Percentage (%)
Pan gastritis	48	40.0
Lax lower oesophageal sphincter	34	28.3
Primary bile reflux gastritis	26	21.7
Antral gastritis	8	6.7
Oesophagitis	4	3.3
Total	**120**	**100.0**

Profile of Patients with Primary Bile Reflux Gastritis

Among the 26 patients with PBRG, males predominated (n = 19, 73.1%) compared to females (n = 7, 26.9%). The mean age of PBRG patients was 41.4 ± 10.7 years. The distribution across age groups was relatively

uniform in the older cohort, with 7 patients each in the 31–40, 41–50, and 51–60 year brackets, and 5 patients in the 21–30 year group.

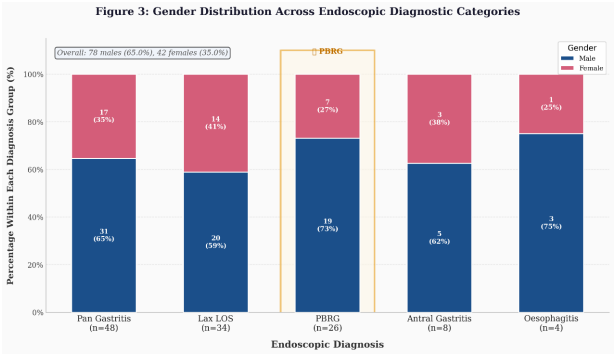

Figure 3: Stacked bar chart comparing gender distribution across all diagnostic categories.

Table 3: Demographic and risk factor profile of PBRG patients (n = 26)

Characteristic	n	% of PBRG patients
Gender: Male	19	73.1
Gender: Female	7	26.9
Mean age (years)	41.4 ± 10.7	–
Age group: 21–30 years	5	19.2
Age group: 31–40 years	7	26.9
Age group: 41–50 years	7	26.9
Age group: 51–60 years	7	26.9
Heartburn	23	88.5
Regurgitation / Reflux	19	73.1
Nausea	8	30.8
Smoking	18	69.2
Alcohol consumption	18	69.2
Consumption of outside food	8	30.8

Symptom Profile and Risk Factors

Heartburn was the dominant symptom in the overall cohort (n = 107, 89.2%), followed by regurgitation (n = 72, 60.0%) and nausea (n = 42, 35.0%). Among PBRG patients, heartburn was present in 23 (88.5%) and regurgitation in 19 (73.1%); nausea was less prevalent at 30.8%. With respect to lifestyle risk factors in the PBRG subgroup, smoking was documented in 18 patients (69.2%) and alcohol consumption in an equal number (69.2%), significantly higher than the overall cohort prevalence of 38.3% and 40.0% respectively. Habitual consumption of outside food was reported in 8 PBRG patients (30.8%).

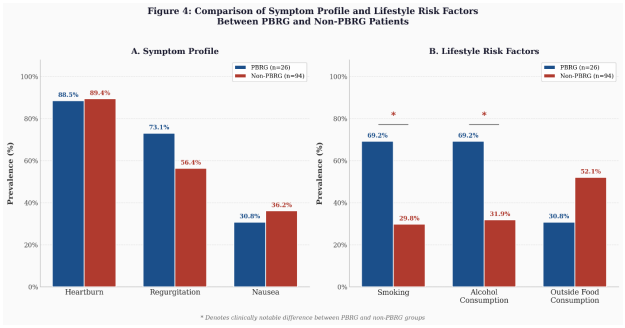

Figure 4: Grouped bar chart comparing symptom and risk factor prevalence in PBRG vs non-PBRG patients

Table 4: Comparison of symptoms and risk factors between PBRG and non-PBRG patients

Parameter	PBRG (n=26) n (%)	Non-PBRG (n=94) n (%)	Total (n=120) n (%)
Heartburn	23 (88.5%)	84 (89.4%)	107 (89.2%)
Regurgitation	19 (73.1%)	53 (56.4%)	72 (60.0%)
Nausea	8 (30.8%)	34 (36.2%)	42 (35.0%)
Smoking	18 (69.2%)	28 (29.8%)	46 (38.3%)
Alcohol consumption	18 (69.2%)	30 (31.9%)	48 (40.0%)

Outside food consumption	8 (30.8%)	49 (52.1%)	57 (47.5%)

Gender-wise Distribution Across Diagnostic Categories

Across all diagnostic categories, males outnumbered females. In the pan gastritis group, 31 of 48 patients (64.6%) were male. Among lax LOS patients, 20 of 34 (58.8%) were male. The male predominance was most pronounced in the PBRG group (73.1% male). In antral gastritis, 5 of 8 patients (62.5%) were male, while in oesophagitis, 3 of 4 (75.0%) were male.

Table 5: Gender distribution across endoscopic diagnostic categories

Diagnosis	Male n (%)	Female n (%)	Total n (%)
Pan gastritis	31 (64.6%)	17 (35.4%)	48 (100%)
Lax LOS	20 (58.8%)	14 (41.2%)	34 (100%)
PBRG	19 (73.1%)	7 (26.9%)	26 (100%)
Antral gastritis	5 (62.5%)	3 (37.5%)	8 (100%)
Oesophagitis	3 (75.0%)	1 (25.0%)	4 (100%)
Total	**78 (65.0%)**	**42 (35.0%)**	**120 (100%)**

DISCUSSION

The present study provides a detailed account of the prevalence and clinical correlates of PBRG in a cohort of 120 patients with chronic gastritis evaluated at a tertiary care centre. PBRG was identified in 26 patients (21.7%), making it the third most common endoscopic diagnosis after pan gastritis (40.0%) and lax LOS (28.3%). This prevalence figure aligns with the broader range of 15–30% reported in selected tertiary care series from South and Southeast Asia.[14,25]

The pathophysiology of PBRG is anchored in duodenogastric reflux (DGR), which arises when pyloric dysfunction permits the retrograde transit of bile acids, lysolecithin, and pancreatic enzymes into the stomach.[3,6] Unlike *H. pylori*-associated gastritis, where bacterial virulence factors drive mucosal inflammation via well-characterised immune pathways, PBRG results from direct detergent-mediated disruption of the phospholipid bilayer of gastric epithelial cells, culminating in foveolar hyperplasia and stromal oedema.[8,9]

The marked male predominance in our PBRG cohort (73.1%) is consistent with previously published data. Kumar et al. reported a male preponderance in bile reflux gastritis, attributing this disparity to the higher prevalence of tobacco and alcohol use among men.[26] In our series, 69.2% of PBRG patients were smokers compared to 29.8% in the non-PBRG group. This is biologically plausible, as nicotine has been shown to inhibit pyloric sphincter tone and delay gastric emptying, thus facilitating DGR.[12,13]

Alcohol consumption was similarly elevated in PBRG patients (69.2% vs. 31.9% in non-PBRG). Ethanol is known to alter gastric motility and mucosal barrier function, reduce prostaglandin synthesis, and enhance the toxicity of bile acids at the gastric epithelial surface.[27,28] The dual exposure to tobacco and alcohol in nearly 70% of PBRG patients in our cohort suggests a synergistic effect that merits further investigation through controlled studies.

Heartburn and regurgitation were the predominant symptoms in PBRG patients, reported in 88.5% and 73.1% of cases respectively. These findings are consistent with studies by Vaezi and Richter, who demonstrated that biliary reflux into the oesophagus contributes significantly to refractory heartburn in patients without a clear acid component.[10,29] Nausea was less prevalent (30.8%), and its non-specificity limits its diagnostic utility in isolation.[11]

The age distribution of PBRG patients in our cohort was relatively uniform across the 31–60 year range, with a mean age of 41.4 years. This contrasts with secondary bile reflux gastritis, which typically presents in older post-operative patients.[5,21] The occurrence of PBRG in younger adults (21–30 years, 19.2% of PBRG cases) underscores the importance of not restricting suspicion to middle-aged or elderly populations, particularly when lifestyle risk factors are present.

Pan gastritis, the most prevalent diagnosis in our cohort (40.0%), likely reflects the high background prevalence of *H. pylori* infection in tertiary care patients from central India, where seroprevalence studies report rates of 50–60% in symptomatic individuals.[1,30] The differentiation of PBRG from *H. pylori*-associated pan gastritis is clinically important, as therapeutic strategies diverge substantially—eradication therapy for the latter

versus prokinetics, bile acid sequestrants, or ursodeoxycholic acid for PBRG.[31,32]

The clinical significance of untreated PBRG extends beyond symptom burden. Experimental and epidemiological studies have linked chronic bile acid exposure to the development of intestinal metaplasia and, in high-risk populations, to an elevated risk of gastric adenocarcinoma.[18,19] Ursodeoxycholic acid, by altering the bile acid pool towards a less cytotoxic composition, and prokinetic agents such as domperidone and metoclopramide, by improving pyloric coordination, represent the mainstay of pharmacological management.[32,33]

The retrospective design of this study introduces inherent limitations, including potential selection bias arising from referral patterns to a tertiary centre, reliance on endoscopic diagnosis without universal histopathological confirmation, and the absence of 24-hour bile impedance monitoring data to objectively quantify DGR.[34] Furthermore, *H. pylori* status was not uniformly documented in the master chart, precluding an analysis of its interaction with PBRG. Future prospective studies incorporating histopathology, *H. pylori* testing, and objective bile reflux quantification are warranted.[35]

CONCLUSION

Primary bile reflux gastritis accounts for a significant proportion (21.7%) of patients presenting with chronic gastritis at our tertiary care centre, ranking as the third most common endoscopic diagnosis. A male predominance, working-age distribution, and a strong association with tobacco and alcohol consumption characterise the PBRG patient profile in this cohort. Heartburn and regurgitation are the dominant presenting symptoms. These findings underscore the need for heightened clinical awareness of sPBRG as a distinct diagnostic entity within the spectrum of chronic gastritis, with targeted endoscopic evaluation and lifestyle modification counselling forming the cornerstone of management. Prospective multicentre studies with histopathological and objective bile monitoring data are necessary to fully characterise the epidemiology and natural history of PBRG in the Indian population.

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