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The burden of intestinal metaplasia in chronic gastritis - A case study

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Abstract

Gastric cancer is one of the leading causes of cancer-related death worldwide, particularly in Asian countries (GC). Gastric adenocarcinoma develops after a series of mucosal changes, including intestinal metaplasia (IM), dysplasia, and adenocarcinoma, according to Correa's cancer cascade. These modifications can range from non-atrophic to atrophic gastritis (AG). As a result, pre-neoplastic gastric lesions include IM and atrophic gastritis. Helicobacter pylori (H. pylori) infection initiates the series of events leading to stomach adenocarcinoma in Correa's carcinogenesis cascade. Long-term data showed that removing H. pylori reduced the likelihood of cancer developing later. However, it is unclear whether eradication of the bacterium in pre-neoplastic gastric lesions can reverse these changes and prevent further progression to cancer. Even if H. pylori eradication can reverse AG, the presence of IM may be a point of no return in this cascade. As a result, periodic endoscopic surveillance may be indicated in cases of extensive or incomplete IM, particularly in populations at high GC risk. Future research will be required to determine the best surveillance endoscopy interval and tool.

Keywords: Helicobacter Pylori, Gastric Cancer, Intestinal Metaplasia, Treatment, Surveillance

Introduction

Gastric cancer is still the third leading cause of cancer-related mortality and the fifth most common cancer in the world, with an estimated 1 million new cases and over 780,000 deaths in 20181. The most prevalent type of gastric cancer is gastric adenocarcinoma, which has two histologic subtypes: intestinal-type and diffuse-type. The Correa cascade, which starts with gastritis caused by Helicobacter pylori and progresses through chronic inflammation, dysplasia, and cancer, ends with intestinal-type gastric adenocarcinoma2. For intestinal-type gastric adenocarcinoma, atrophic gastritis and gastric intestinal metaplasia (GIM) are recognized pre-neoplastic lesions.

The accumulated incidence rate for the progression of GIM in the absence of dysplasia to gastric cancer was 12.4 (95% confidence interval, 10.7-14.3) per 10,000 person-years, according to the American Gastroenterological Association Technical Review that served as the basis for the guidelines for GIM surveillance2. This was based on 10 cohort studies that included nearly 26,000 patients with non-dysplastic GIM. At this time, neither the occurrence of pre-neoplasia nor the progression of neoplastic disease can be predicted. Therefore, concentrated efforts are required right away to identify and further define predictive determinants3.

Nevertheless, the gradual, stepwise development of gastric pre-neoplasia into neoplasia at least permits targeted surveillance and the chance for early detection and resection of neoplastic lesions4. The histologic subtyping of GIM into incomplete and complete types is one biomarker for further stratifying this already at-risk group, even though there are no established predictive biomarkers that are routinely used in clinical settings5. Based on a meta-analysis of 7 studies that included 2014 people with GIM and no concurrent neoplasia (929 with incomplete GIM, 1112 with complete GIM) who developed 74 gastric cancers over the course of 3–12.8 years of follow-up, incomplete GIM was linked to a 3.33-fold (95% confidence interval, 1.96–5.64) higher risk of incident gastric cancer than people with complete GIM6-9.

Histologic subtyping is still incredibly underutilized, especially in India despite data showing a differential risk of gastric cancer. The underutilization is undoubtedly multifactorial, with the heterogeneous and sparse body of research demonstrating the prognostic value of GIM subtyping and the lack of knowledge of its clinical utility each making a significant contribution10-11.

Materials and Methods

This is a retrospective cross-sectional study conducted at the Department of Pathology, Tagore Medical College and Hospital. All gastric biopsies with a histological diagnosis of chronic gastritis received from the clinical department during the study period were included in the study. Excluded were autolyzed specimens and sections with insufficient material.

The paraffin blocks were sectioned and stained with H&E and Giemsa at a thickness of 3 microns. The sections were meticulously analyzed for the presence of Intestinal metaplasia and classified according to the "Updated Sydney System" for chronic gastritis. Comparing the findings of H&E and Alcian blue stains for the detection of GIM. Clinical factors were associated with histopathological findings.

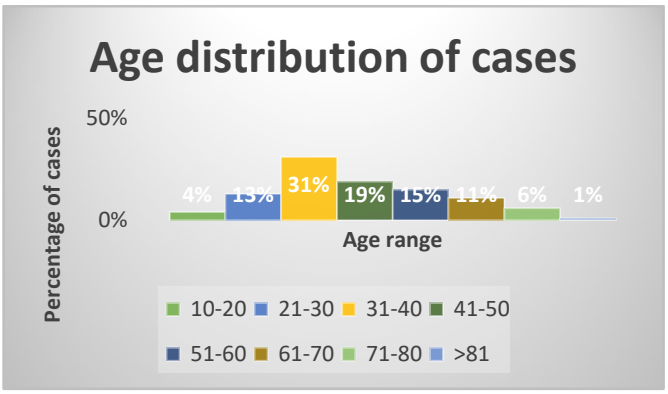
Results

The current study included 80 cases of chronic gastritis that had previously been diagnosed. Patients' ages ranged from 14 to 82, with a mean of 46 years. Males (64%) were disproportionately affected compared to females (36%). Most of the endoscopic biopsies were obtained from antrum (56.3%) followed by pylorus (30%). All the cases were classified as per the ‘Updated Sydney system of classification’ for chronic gastritis into mild, moderate, and severe.

Age Group	Frequency	Percentage of cases
10-20	3	4%
21-30	10	13%
31-40	25	31%
41-50	15	19%
51-60	12	15%
61-70	9	11%

71-80	5	6%
>81	1	1%
Total	80	100%

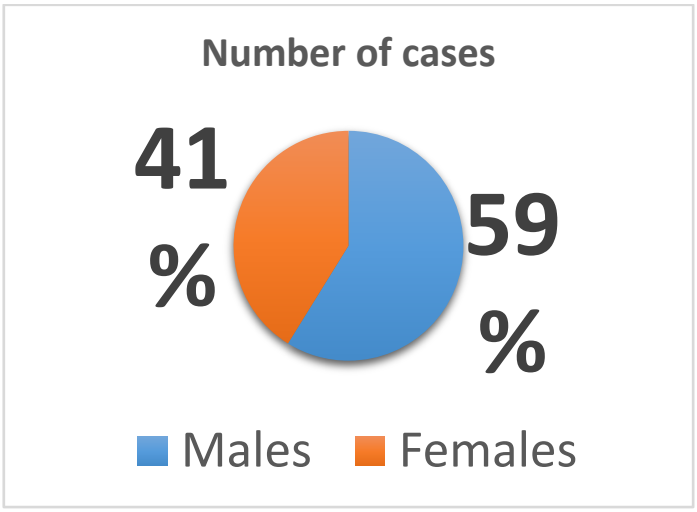
Table 1: Age distribution of cases



Graph 1: Bar graph portraying age distribution among the cases

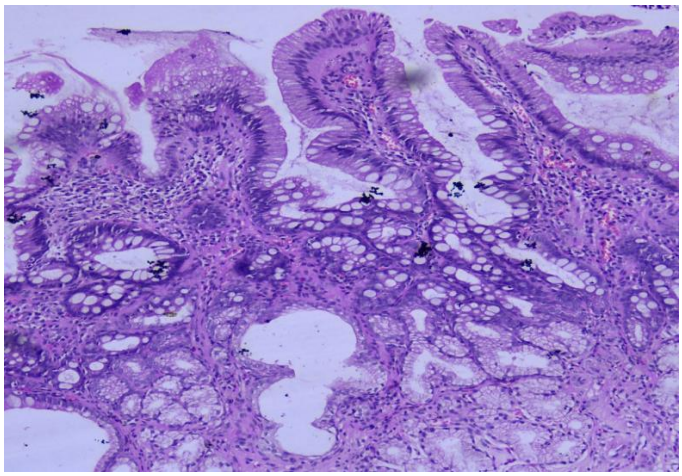
Site	Frequency	Percent
Antrum	45	56.30%
Body	4	5%
Body & Antrum	5	6.30%
Pylorus	24	30%
Pylorus & Body	2	2.50%
Total	80	100

Table 2: Biopsy sites

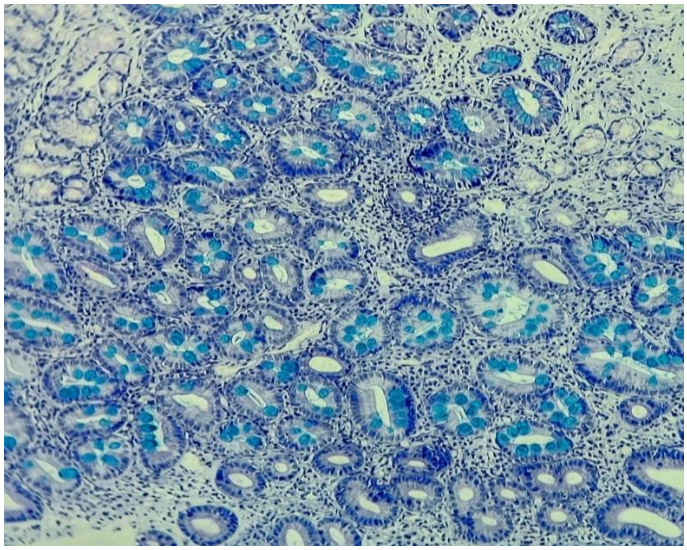


Graph 2: Pie chart illustrating gender distribution.

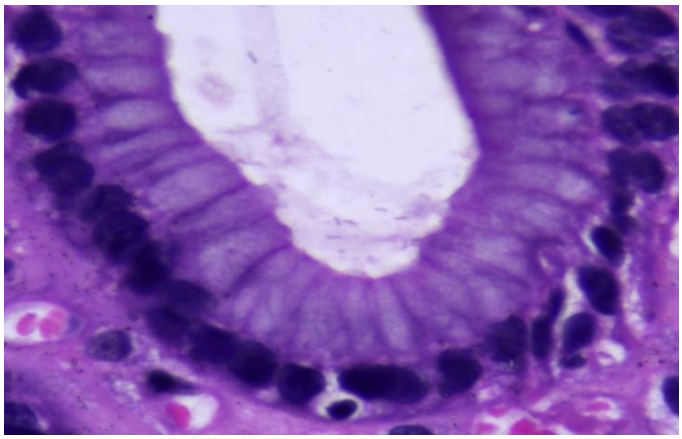
Out of 80 biopsies with chronic gastritis, 38 (48%) biopsies were reported as mild chronic gastritis. Moderate and severe chronic inflammatory infiltrate was observed in 37 (46%) and 5 (6%) biopsies respectively. Active inflammatory infiltrate (neutrophils) was encountered in 57 (71%) biopsies, whereas intestinal metaplastic and atrophic changes were seen in 17 (21%) and 2 (3%) biopsies respectively.



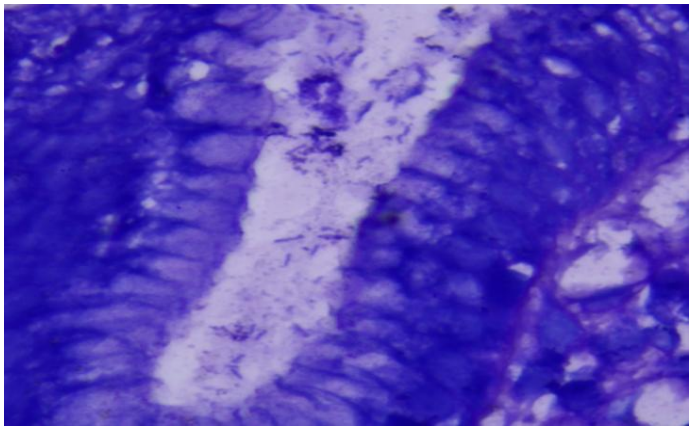
Severe Intestinal metaplasia (H&E) (10x)



Severe Intestinal metaplasia (Alcian Blue) (10x)



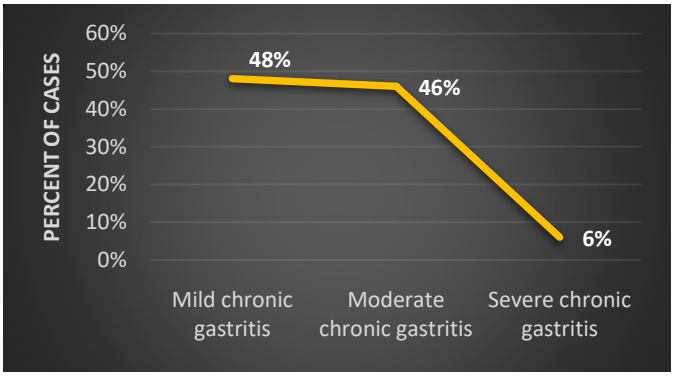
H. pylori – Antral biopsy (H&E) (100x)



H. pylori – Antral biopsy (Giemsa) (100x)

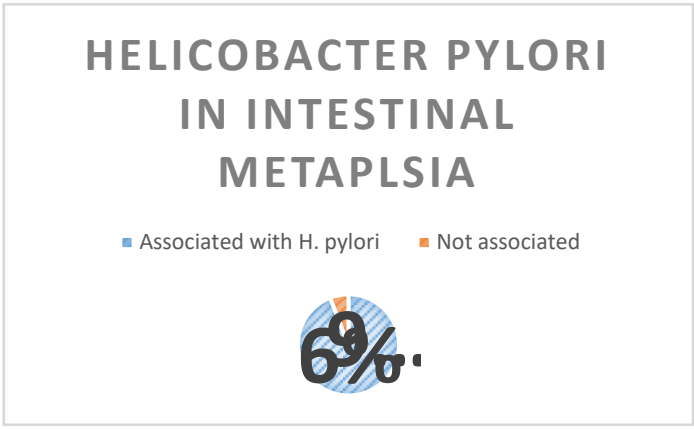
Diagnosis	Frequency	Percent
Mild chronic gastritis	38	48%
Moderate chronic gastritis	37	46%
Severe chronic gastritis	5	6%
Total	80	100%

Table 3: Distribution of cases involved in the study

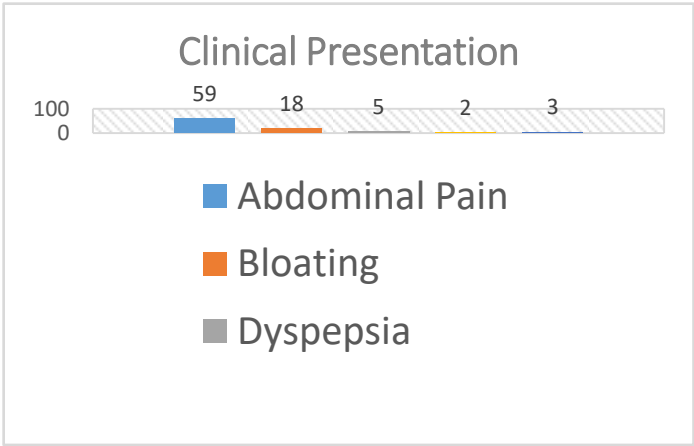


Graph 3: Line diagram of cases involved in study

Out of 80 cases, 17 cases were associated with intestinal metaplasia with the prevalence of 21%. Most of the cases (76%) showed mild GIM with 18 % cases showed moderate GIM and 6 % cases showed severe GIM. Out of 17 cases, 16 (94%) showed evidence of H. pylori. Among the 17 cases with intestinal metaplasia, 10 were seen among males and the remaining 7 were among females symbolizing the increased male predisposition.



Graph 4: Pie chart representing the association between Intestinal metaplasia and H. pylori



In terms of clinical presentation 59 (67.8%) patients presented with abdominal pain, 18 (20.7%) presented with bloating, 5(5.7%) presented with dyspepsia, 2 (2.3%) presented with severe vomiting, and 3 (3.4%) were part of routine health checkup.

As per “Updated Sydney System” of chronic gastritis9; active gastritis, atrophic and intestinal metaplastic

changes were further subdivided as per degree of severity of these changes and H. Pylori positive biopsies were classified according to degree of bacillary colonisation.

	Activity	Intestinal metaplasia	Atrophy	H. pylori infestation
None	23 (29%)	64 (80%)	78 (98%)	21 (26%)
Mild	30 (38%)	13 (16%)	1 (1%)	51 (64%)
Moderate	26 (33%)	2 (3%)	0	5 (6%)
Severe	1 (1%)	1 (1%)	1 (1%)	3 (4%)

Table 4: Severity stratification of cases according to Sydney system

Discussion

In this retrospective study done in chronic gastritis cases (80 cases) received over a period of 6 months in a tertiary care center, out of 87 cases of chronic gastritis 17 shows features of intestinal metaplasia. Almost all are associated with evidence of H. pylori infection with a male preponderance and presented with various clinical manifestations predominantly abdominal pain. It remains controversial whether the presence of GIM signifies the point of no return in the carcinogenesis cascade. In contrast, encouraging results are seen in some studies with early treatment of H. pylori in patients with advanced lesions. The updated Sydney system provides a comprehensive endoscopic sampling protocol with standardized biopsy sites consist of two biopsies from antrum (along the lesser and greater curvature), two from body (along the lesser and greater curvature) and one biopsy from incisura angularis. The scoring of GIM occurs on a visual-analogue scale, with values of none, mild, moderate and marked. Operative Link for Gastric Intestinal Metaplasia (OLGIM) is Sydney system based scoring system to avoid inter-observer variability, scored as Stage 0 to Stage IV

Conclusion

In view of the increased malignant transformation rates associated with intestinal metaplasia, an extensive biopsy sampling method should be incorporated in high-risk cases. There is an increasing need for further stratification of intestinal metaplasia in terms of morphology, histochemistry, and molecular defects along with standardization. Gastric IM can be readily diagnosed by endoscopic biopsy samples. Thus, in an ethnically diverse country like India identification of gastric IM provides an excellent opportunity for early detection and intervention of gastric carcinomas.

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