Evaluation of chronic gastritis with Helicobacter pylori using updated Sydney system

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ABSTRACT

Background: Helicobacter pylori has been established as a major etiological factor in the pathogenesis of chronic gastritis. The aim of the study was to interpret the histopathological changes in chronic gastritis using updated Sydney system and the association with H. pylori infection.

Methods: This was a 3 years study in which 62 gastric endoscopic mucosal biopsies taken from patients presenting with dyspepsia were included. Slides were stained with routine H and E and Giemsa for H. pylori detection in chronic gastritis cases. Grading of the variables were done with reference to Sydney system of classification.

Results: Out of 62 gastric biopsy specimens, 55 cases (88.7%) were histopathological diagnosed as chronic gastritis. Among chronic gastritis, 21 (38%) cases showed H. pylori and majority of these being moderately (2+) positive. 27 (49%) cases showed neutrophilic activity with most of them showed mild (1+) activity. Chronic inflammation was seen 52 (94.5%) with majority of these graded as moderate (2+) activity. Intestinal metaplasia was seen in 8 (14.5%) of cases with majority being mild (1+). Atrophy was seen only in 3 (5.4%) of cases with majority being mild (1+). Significant statistical association was found between H. pylori and neutrophilic activity (p<0.001).

Conclusions: Histological evaluation of chronic gastritis using updated Sydney system of classification helps in detection of H. pylori infection and prevents further progression of the disease.

Keywords: Updated Sydney system of classification, Chronic gastritis, H. pylori

INTRODUCTION

In 1983, Warren and Marshall documented unknown curved bacilli in gastric antral biopsies from patients with active gastritis and peptic ulcer. This bacterium was Campylobacter pylori. The name was later changed to H. pylori.1 It is widely accepted that colonization of the gastric surface epithelium by H. pylori is commonly associated with chronic gastritis which was documented in 77% cases of gastritis.2 In order to avoid confusions in diagnosis of various forms of gastritis, the Sydney system was introduced to produce a standardized approach to the histological interpretation of gastric biopsies.3 The updated Sydney system provides guidelines for classifying, grading and reporting on endoscopic and histological assessment of gastric mucosa.4 Because it is clear that the extent and distribution of the H. pylori gastritis determine the clinical outcome, the updated Sydney system emphasizes the importance of combining topographical, morphological, and etiological information in order to generate diagnosis.3 The aim of the study was to interpret the histopathological changes in chronic gastritis using updated Sydney system and to correlate these pathological features with H. pylori infection.

METHODS

This study was both retrospective and prospective conducted in the department of pathology, KIMS Hubli for
a period of 3 years from January 2016 to December 2018. All biopsies obtained for various symptoms of dyspepsia like abdominal pain, nausea, vomiting, bloating, heartburn, were included in the study. Exclusion criteria included absolute/relative contraindication to upper GI endoscopy and patients put on H. pylori eradication or acid suppressive therapy in the last 2 weeks.

Written informed consent was taken from all the patients for the study. Approval was obtained from institutional ethical committee prior to conducting the study. Upper gastrointestinal endoscopic biopsy samples received in 10% formalin were taken for the study. While embedding care was taken to see that the mucosal surface was placed 90 degree to the cutting surface. 4-5 µm thick sections were cut on a Leica microtome. Routine haematoxylin and eosin staining was done on all biopsies. Special stain like Giemsa was done for the detection of H. pylori in chronic gastritis cases.

RESULTS

A total of 62 gastric biopsy samples of dyspepsia were included in the study. The endoscopic mucosal biopsies were taken for histopathological evaluation. Out of 62 patients, chronic gastritis was observed in 55 (88.7%) cases, dysplasia in 2 (3.2%), gastric ulcer in 2 (3.2%), polyp in 1 (1.6%), adenocarcinoma in 1 (1.6%) and no significant pathology in 1 (1.6%) case. 55 chronic gastritis cases were further evaluated. Among chronic gastritis cases, majority of cases were seen in the 41-50 years age group followed by 51-60 years age group (Table 1). There were 36 males and 19 female patients with an M: F ratio of 1.9: 1. Most frequent site sampled for biopsy was from gastric antrum constituting 46 cases (83.6%).

Histological grading of chronic gastritis was done by updated Sydney system (Table 2). Chronic inflammation was present in 52 (94.5%) cases of chronic gastritis out of which, 8 (15.3%) had mild, 34 (65.3%) had moderate and 12 (23%) had severe chronic inflammation. Neutrophilic activity was seen in 27 (49%) cases of which 20 (74%) had mild, 5 (18.5%) had moderate and 2 (7.4%) had severe neutrophilic activity. Intestinal metaplasia was seen in 8 (14.5%) cases with 7 cases (87.5%) having mild and 1 case (12.5%) having moderate intestinal metaplasia. Glandular atrophy was seen in 3 (5.4%) cases which were of mild grade.

H. pylori was identified in 21 (38%) cases of chronic gastritis on gastric mucosal biopsies. Out of these 8 (38%) had mild, 11 (52.3%) had moderate and 2 (9.5%) had severe H. pylori colonization. Further, association of H. pylori with neutrophils, lymphoid aggregates, intestinal metaplasia and atrophy in chronic gastritis was also evaluated (Table 3). 27 cases of chronic gastritis showed neutrophilic activity. 24 of these 27 cases (88.8%) were positive for H. pylori. The association was clinically significant (p<0.001). 52 cases of chronic gastritis showed lymphoid aggregates. 18 of these 52 cases (34.6%) were positive for H. pylori. 8 cases of chronic gastritis showed intestinal metaplasia and 2 out of these 8 cases (25%) were positive for H. pylori. 3 cases of chronic gastritis showed atrophy. Out of these 3 cases none were positive for H. pylori.

Table 1: Age distribution in chronic gastritis.

| Age groups (years) | No. of cases | Percentage (%) |
|-------------------|-------------|----------------|
| 11-20             | 3           | 5.45           |
| 21-30             | 6           | 10.9           |
| 31-40             | 9           | 16.36          |
| 41-50             | 18          | 32.72          |
| 51-60             | 12          | 21.8           |
| 61-70             | 4           | 7.27           |
| >70               | 3           | 5.45           |

Table 2: Histological grading of chronic gastritis by updated Sydney system.

| Variables in chronic gastritis | Mild/1+ | Moderate/2+ | Severe/3+ | Total | Percentage (%) |
|-------------------------------|--------|-------------|-----------|-------|----------------|
| H. pylori                     | 8      | 11          | 2         | 21    | 38             |
| Neutrophils                   | 20     | 5           | 2         | 27    | 49             |
| Chronic inflammation          | 8      | 34          | 12        | 52    | 94.5           |
| Intestinal metaplasia         | 7      | 1           | -         | 8     | 14.5           |
| Atrophy                       | 3      | -           | -         | 3     | 5.4            |
DISCUSSION

Gastritis classification has evolved over the years, taking into account topography, epidemiology, morphology and endoscopy. However, this was not forthcoming since most of the gastritis was considered idiopathic until the discovery of H. pylori. Due to the various grading systems of chronic gastritis, it was difficult for pathologists to reliably identify chronic gastritis and compare their findings. In 1994, team of twenty gastrointestinal pathologists from different parts of the world gathered to re-evaluate the Sydney model four years after its implementation. A modified Sydney system and a visual analogue scale was devised as shown in Figure 1.

The histological division of this classification utilizes graded variables which include chronic inflammation, neutrophilic activity, glandular atrophy, intestinal metaplasia and H. pylori density.

In the present study, chronic gastritis was noticed in a wide age group ranging from 12-80 years with a mean age of 49.6 years. This is similar to other studies in which mean age was 47 years and 48 years in another study. The M: F ratio in our study is 1.9: 1 which is similar to studies done by Chen et al, Pruthi et al and where they have reported a M: F ratio of 1.8: 1, 2.3: 1 respectively. The most common site of gastric biopsies was antrum in the present study, which is in line with studies conducted by Garg et al and Park et al. H. pylori was positive in 21 (38%) cases of total chronic gastritis cases. This is almost consistent with studies done by Pruthi et al, and Dhakhwa et al, in which H. pylori was positive in 47% and 44% of cases respectively. However H. pylori positivity was higher in study done by Qureshi et al and lesser in study done by Hassan et al which were 15.5% and 93.7% respectively. These variations in the result can be attributed to biopsy sampling, where multiple biopsies are required to improve the results. The use of immunostains are also helpful for better detection of H. pylori.

Neutrophilic infiltration was seen in 27 (49%) cases with majority of them showing mild activity. 24 of these 27 cases (88.8%) were positive for H. pylori. The association

Table 3: Association of H. pylori with neutrophilic activity, lymphoid aggregates, intestinal metaplasia and atrophy in chronic gastritis.

| Histological variables     | No. of cases | H. pylori positive | Percentage (%) |
|----------------------------|--------------|--------------------|----------------|
| Neutrophilic activity      | 27           | 24                 | 88.88          |
| Lymphoid aggregates        | 52           | 18                 | 34.61          |
| Intestinal metaplasia      | 8            | 2                  | 25             |
| Atrophy                    | 3            | -                  | -              |

Figure 1: Visual analogue scale for grading of chronic gastritis: the up-dated Sydney system.
was clinically significant (p<0.001) which was proved in other studies as well.\textsuperscript{7,9,12} Stolte et al stated that neutrophilic activity is an almost universal phenomenon in H. pylori gastritis. Neutrophils are a very sensitive indicator for the presence of H. pylori and disappear within few days of cure of infection.\textsuperscript{16,52} (94.5\%) cases of chronic gastritis showed moderate to marked chronic inflammation. However, a statistically significant relationship was not demonstrated between H. pylori infection and grade of chronic inflammation. This contrasts with the study done by Genta et al in which 91.8\% patients with H. pylori gastritis showed lymphoid aggregates and was concluded as statistically significant.\textsuperscript{17}

8 (14.5\%) cases of chronic gastritis showed intestinal metaplasia. This is similar to the study done by Hassawi et al in which 15\% of cases showed this change.\textsuperscript{16} However 5\% of cases showed intestinal metaplasia in the study done by Dhakhwa et al and in contrary 23\% of cases showed intestinal metaplasia in study done by Nuaimy et al.\textsuperscript{15,19}

The latter may be attributed to the fact that the diagnostic rate of intestinal metaplasia could be improved by the use of special stain for mucin. In addition, biopsy taken from the area of incisura angularis also increases the rate of detection of intestinal metaplasia as it initially develops in this region.\textsuperscript{20} There was no significant statistical association between H. pylori and intestinal metaplasia which is in line with other studies.\textsuperscript{12,14}

Only 3 (5.4\%) cases of chronic gastritis showed atrophy. None of them were positive for H. pylori. Likewise, it was concluded the association was insignificant in studies conducted by Garg et al and Maharjan et al.\textsuperscript{7,14}

The limitation in the study was the inability to obtain biopsies from intended topographic sites according to the updated Sydney system for detection of H. pylori, neutrophilic infiltration, mononuclear infiltration, gastric atrophy or intestinal metaplasia, yet significant association of neutrophilic activity and H. pylori infection was noted in chronic gastritis cases. An improved sample size could provide a better correlation to the statistical analysis of this study.
CONCLUSION

Histopathological evaluation of endoscopic gastric biopsy using updated Sydney system of classification is of value in detection of H. pylori and various histological changes of chronic gastritis. In our study neutrophilic activity was significantly associated with H. pylori infection. Thus, search for H. pylori should be initiated if neutrophils are seen in the antral biopsies. The detection of H. pylori can help in early treatment and further advancement of disease.

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