Histopathologic Study of Gastritis and Helicobacter Pylori Infection at a Tertiary Centre in Kathmandu

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INTRODUCTION

Gastritis is gastric inflammation associated with mucosal injury. It is a major cause of morbidity and mortality. It can be categorized into acute and chronic gastritis. Acute gastritis refers to acute mucosal damage after acute alcohol intoxication, drugs, corrosive substance, gastric irradiation etc. Chronic gastritis is a chronic inflammation of the gastric mucosa characterized by infiltration of the superficial mucosa by chronic inflammatory cells and with progressive spread to involve the whole mucosa leading to atrophy and intestinal metaplasia and may even progress to the development of carcinoma. Chronic infection with Helicobacter pylori (H. Pylori) is believed to be the major causative agent in the pathogenesis of chronic active gastritis, duodenal and gastric ulcer along with gastric carcinoma. Sydney system classifies gastritis by the intensity of mononuclear inflammatory infiltrates, activity of polymorphs, atrophy, metaplasia and dysplasia and presence of H. pylori.

METHODS:
Observational, descriptive, cross-sectional retrospective study conducted at Department of Pathology, Kathmandu Model Hospital, Kathmandu, Nepal from January 2015 to December 2015. Record of computerized histopathology reports of all cases of endoscopic gastric biopsies were retrieved from the laboratory. The biopsy samples obtained from both body and antrum were included in the study. The grades of mononuclear cells infiltration, neutrophilic activity, metaplasia, dysplasia and glandular atrophy were determined using the Sydney system classification. The presence or absence of H. pylori was noted in the slides stained with Giemsa stain. Data analysis was done using a Microsoft Excel file.

RESULTS:
Of 128 samples studied, the most common endoscopic findings were erythematous lesions and mononuclear infiltrate was seen in all cases. 61.7% showed moderate inflammation. The activity was seen in 35.9% of cases. H. pylori organism was noted in 48.4% of cases.

CONCLUSIONS:
The severity of chronic inflammation and neutrophilic activity are significantly associated with infection by this microorganism.

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mucosa by chronic inflammatory cells and with progressive spread to involve the whole mucosa leading to atrophy and intestinal metaplasia. These changes may progress to dysplasia with the eventual development of carcinoma. Chronic infection with Helicobacter pylori (H. Pylori) is believed to be the major causative agent in the pathogenesis of chronic active gastritis, duodenal and gastric ulcers. It is also associated with gastric carcinoma and low-grade mucosa-associated lymphoid tumor (MALT) lymphoma.

H. pylori are a gram-negative microaerophilic spiral bacterium first isolated in 1982. It is a curved to wavy bacterium that can be identified by hematoxylin and eosin stains when numerous and readily recognized by other histochemical stains like Giemsa, Warthin-starry or silver stains.

Assessing gastritis involves clinical examination along with endoscopic and histopathological examination. Grading of chronic gastritis is done by the Sydney grading system and its updated Houston version (1996). Sydney system classifies gastritis by the intensity of mononuclear inflammatory infiltrates, activity of polymorphs, atrophy, metaplasia and dysplasia and presence of H. pylori.

METHODS

The present study is an observational, descriptive, cross-sectional retrospective study conducted at the Department of Pathology, Kathmandu Model Hospital, Kathmandu, Nepal from January 2015 to December 2015. A record of computerized histopathology reports of all cases of endoscopic gastric biopsies was retrieved from the laboratory. The biopsy samples obtained from both body and antrum were included in the study. The samples obtained from only one anatomical site of the stomach were excluded from the study.

The patient’s age, sex, and upper GI findings were recorded. The diagnoses were made on histologic examination of processed tissue. The tissue was processed in an automated tissue processor as per standard protocol. The slides were stained with Hematoxylin and Eosin as per standard protocol. All slides were stained with Giemsa stain for the detection of Helicobacter pylori as per standard protocol.

Finally, the slides were examined under a microscope and histopathological findings were noted. The grades of mononuclear cells infiltration, neutrophilic activity, metaplasia, dysplasia and glandular atrophy were determined using the Sydney system. Presence or absence of H. pylori was noted in the slides stained with Giemsa stain. Data analysis was done using a Microsoft Excel file.

RESULTS

Altogether 128 samples were studied. The age of the patient ranged from 18 to 82 years (Figure 1). 64.8% were female while 35.2% were male. The most common endoscopic findings were erythematous lesions (57.8%) followed by the erosive lesion (38.2%). 3.1% had ulcerative lesions while only one case showed the presence of a mass (Figure 3).

On microscopy, the mononuclear infiltrate was seen in all cases where 29% of the biopsies showed mild inflammation, 61.7% showed moderate inflammation and 9.3% showed severe inflammation (Figure 2).
The activity was seen in 35.9% of cases. H. pylori organism was noted in 48.4% of cases. 67.7% of all cases positive for H. pylori showed activity while 32.3% of cases did not show activity despite having H. pylori. 4 cases did not show H. pylori in tissues showing activity. The odds ratio of having active infection in presence of H. pylori is 32.55 (Table 1).

Metaplasia was seen in 9.3% of cases of which 7.8% had H. pylori and 1.5% did not show H. pylori. 50% of all metaplasia were pseudo pyloric metaplasia and 50% were intestinal metaplasia. Metaplasia with activity was seen in 8 cases, all of which showed the presence of H. pylori. Metaplasia without activity was seen in 4 cases, one of which showed the presence of H. pylori. The odds ratio of having metaplastic glands in presence of H. pylori is 4.03 (Table 2). Only 1 case showed the presence of carcinoma.

**DISCUSSION**

Age group shown by many studies show that patients with gastritis fall between the ages 16-81 years. This is similar to our study where the patients were between 18-82 years. There are 6 patients below the age of 20 years of which 50% of cases showed colonization for H. pylori. Maharjan S et al in their study found only 2 children with gastritis without H. pylori colonization.5 A study done in Columbia showed 59% of the affected children with H. pylori infection.6 Many studies showed more male patients than female patients in contrast to our study where there is a female preponderance. 1, 7, 8 A study by Pasechnikov VD et al had more female patients, similar to our study.3 Antral biopsy was the most common site of lesion in studies done by Archila P et al and Maharjan S et al. 5, 6 The site of biopsy is important for an accurate diagnosis.9 The Sydney system of classification of gastritis emphasizes the importance of topographical, morphological and etiological information.10 According to the updated Sydney system, 5 sites are recommended; 2 from the antrum, 2 from the body and one from incisura angularis.4 In our study, we have studied samples only where at least two biopsy sites were included in the biopsy.

Increased documentation and detection of gastritis have been possible after the introduction of the use of endoscopy and biopsy.8 Introduction of the Sydney system for the classification of gastritis and H. pylori has improved the knowledge and reproducibility of the classification of gastritis.7 Endoscopy showed an erythematous lesion in 57.8% of cases in our study. Similar findings were noted by Garg et al, Khakoo et al and Calabrese et al.1, 11, 12
Chronic inflammatory activity was moderate in 61.7% of cases followed by mild inflammation in 29%. A study by Suzana et al and Garg et al showed more of mild inflammation on microscopy.\(^1\)\(^,\)\(^13\) el-Zimaity had 91.5% with moderate to severe chronic inflammation.\(^14\)

Patients with chronic gastritis will progress to atrophic gastritis in its natural course which is characterized by atrophy and metaplasia.\(^4\) Gastric atrophy is defined as the loss of appropriate glands in a given compartment.\(^15\) Atrophic changes in the gastric epithelium are associated with the development of gastric carcinoma.\(^13\) Our study showed atrophy of mucosa in 28.1% of total patients. Suzana et al showed atrophy in 44.93% of their study. Garg et al showed atrophy in 12.3% of cases while Maharjan et al showed atrophy in only 5.5% cases.\(^5\)\(^,\)\(^17\)\(^,\)\(^18\)

Active inflammation is the presence of neutrophils in lamina propria or glands and the density of intraepithelial neutrophils correlates well with the extent of mucosal damage and H. pylori infection. The presence of H. pylori is associated with tissue damage and histologic finding of chronic gastritis.\(^16\) Our study showed active inflammation in 35.9% of cases. It is similar to the study done by Maharjan et al while Dhakhwa et al and Park et al had higher neutrophilic activity.\(^5\)\(^,\)\(^17\)\(^,\)\(^18\) H. pylori organism was noted in 48.4% of cases. Histological examination and culture are the gold standards for the detection of H. pylori infection.\(^1\)\(^,\)\(^13\) el-Zimaity had 91.5% with moderate to severe chronic inflammation.\(^14\)

Our study shows that neutrophilic activity is more prevalent in those cases with the presence of H. pylori.\(^5\) Metaplastic changes occur in two forms in the stomach as intestinal metaplasia and pseudo pyloric metaplasia. It reflects some degree of mucosal damage by chronic gastritis.\(^4\) Metaplasia was seen in 9.3% of cases. 50% of all metaplasia were pseudo pyloric metaplasia and 50% were intestinal metaplasia. Udoh et al found metaplasia in 16.6% of cases.\(^22\) Intestinal metaplasia is considered a negative indicator for H. pylori colonization.\(^1\) Of 9.3% metaplasia in our study, 7.8% had H. pylori and 1.5% did not show H. pylori. Metaplasia with activity was seen in 8 cases, all of which showed the presence of H. pylori. Metaplasia without activity was seen in 4 cases, one of which showed the presence of H. pylori. Chronic infiltrates, activity, H. pylori density and lymphoid follicles had a strong correlation with surface epithelial damage, while glandular atrophy and intestinal metaplasia had no association in the study done by Garg B et al.\(^1\)

CONCLUSIONS

Helicobacter pylori infection is common in chronic gastritis. The severity of chronic inflammation and neutrophilic activity are significantly associated with infection by this microorganism. There were few cases of intestinal metaplasia and only one case of malignancy.

REFERENCES


