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Clinicopathological study of gastric biopsy in gastritis patients

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Abstract: *Background:* Gastritis is the inflammation of gastric mucosa characterized by morphological and histopathological changes. This study was undertaken mainly to study the histo- morphological features of gastritis patients. *Material and Methods:* A cross sectional study was conducted in 100 cases of gastric biopsies in a tertiary care hospital in North Karnataka. The biopsies were fixed and sections were made from paraffin blocks. Two slides with three levels in each slide were examined under light microscope stained with Haematoxylin& Eosin and Giemsa. The slides were examined by two pathologists and findings were documented, compiled and analysed using statistical software. *Results:* This study had shown that 43.0% of the cases had gastric ulcer, 42.0% had chronic gastritis and acute gastritis in 15.0% of the cases. H. Pylori was found in 36.0% of the cases. The histological features included mild, moderate and severe inflammation, neutrophilic activity, mild mucosal atrophy, eosinophilic infiltration, lymphoid follicles and dysplasia. *Conclusion:* This study had shown morphological changes and histopathological changes along with H Pylori infection in cases with gastritis.

Keywords: Gastritis, Histopathological examination, Morphology, H. Pylori, Dysplasia.

Introduction

The human gastrointestinal system is a lengthy, winding organ that frequently develops lesions from inflammatory, neoplastic, and congenital disorders. Inflammation of the Gastric mucosa is called gastritis. Acute inflammation is characterised by neutrophil infiltration, while chronic inflammation is characterised by lymphocytes and plasma cells and is linked to intestinal shrinkage and metaplasia [1].

Although there are a number of potential causes, Helicobacter pylori (H. pylori) is the most frequent one. H. pylori is a gram-negative, curved, flagellated shape bacilli that is positive for urease, catalase, and oxidase. It mostly colonises the stomach's antrum, although it can also be found elsewhere in the body; it causes peptic ulcers and gastritis. It is found in almost half of the human population and possesses characteristics that allow it to adapt to the colonisation of the stomach [2]. Reflux of bile salts or the consumption of alcohol, salicylates, and other anti-inflammatory medications can cause acute gastroenteritis [3].

It is responsible for a broad range of morphological gastric diseases, including intestinal metaplasia, atrophy and mild, moderate and severe active gastritis. Infection has been found to be common in over 75% of cases of duodenal ulcers and 17% of instances of gastric ulcers. It is the primary cause of B-cell mucosa-associated lymphoid tissue (MALT) lymphoma and distal stomach adenocarcinoma. Additionally, it has been connected to extra-gastric symptoms such as iron-deficiency anaemia, vitamin B_{12} deficiency, and idiopathic thrombocytopenic purpura, which are frequently but not always observed in conjunction with autoimmune atrophic gastritis [4-5].

The complimentary uses of histology and endoscopy aid in the diagnosis of various lesions. Despite being an invasive method, diagnostic endoscopy has shown to be a straightforward, secure, and well-tolerated process. Histology is frequently regarded as the "gold standard" in ordinary clinical practice, to which other tests are compared. A biopsy offers a great chance for the

histopathologist and the clinician to link the pathological lesions, endoscopic findings, and clinical data. The hazards of sampling error and taking numerous biopsy samples from the corpus and antrum sections of the stomach mucosa are decreased when using a 2.8 mm channel endoscope. False positive histology results may arise from improperly cleaned equipment, while false negative histology results may be the result of sample mistake and recent or on-going medication therapy [6-7]. The gold-standard test for diagnosing cancer, intestinal metaplasia, acute or chronic inflammation, gastritis, neutrophilic infiltration, and the density of H. pylori colonisation is biopsy. There are fewer studies available in this part of the country about the histo-morphological features of Gastritis. In light of these factors, this study aims to characterize the histo-morphological changes and the degree of inflammation.

Material and Methods

A cross sectional study was conducted in Department of Pathology of S Nijalingappa Medical College, Bagalkot. Clearance from institutional ethical committee was obtained before the study was started. An informed consent was obtained from the all the patients before obtaining the biopsy. This study included 100 gastric biopsies of patients undergoing upper gastrointestinal evaluation for gastritis. The samples obtained from ambiguous biopsy sites and biopsies with poor preservation were excluded from the study. The biopsies obtained from cases of all age group and both sexes were included in the study.

The biopsies were first fixed with 10% formalin and processed routinely to prepare paraffin blocks. Sections of thickness 3 – 4 µm were prepared by cutting the paraffin blocks. Two slides with three levels in each slide were stained with haematoxylin and eosin (H & E). Two pathologists evaluated the slides under a light microscope. The microscopic examination was done using routine H & E staining. Giemsa stain was used to study H. Pylori in each cases. The assessment of H. pylori on Giemsa stain was conducted using a quantitative assessment method. Dispersed organisms identified in less than one-third of the surface epithelium were classified as mild colonisation. Severe colonisation was defined as the presence of huge clusters or continuous layers of organisms covering two thirds of the surface.

The middle ground between the two states mentioned above was moderate colonisation. The biopsies were also evaluated for presence and intensity of mononuclear inflammatory cell infiltrate, inflammatory activity, glandular atrophy, intestinal metaplasia and lymphoid aggregates. The age, gender, clinical history and histological diagnosis of the patients from which the sample is taken were recorded as demographic factors. The data thus obtained was entered in a predesigned proforma and analysed using Statistical Package for social services (SPSSver 20). The data was presented as frequencies and percentages. Chi square test was used as test of significance. A p value of less than 0.05 was considered as statistically significant.

Results

About 39.0% of the study subjects were aged more than 60 years in this study of which 54.0% of the cases were males. This study had shown that gastric ulcer was present in 43.0% of the cases, chronic gastritis was present in 42.0% of the cases (Fig 1), acute gastritis in 15.0% of the cases, fungi (Fig 2), Barrett's oesophagus (Fig 3), squamous cell carcinoma and adenocarcinoma (Fig 4) in one case each (Table 1).

Table-1: Demographic and Histo-morphological features of the study subjects

	Number	Percentage
Age		
21 – 30 years	01	1.0
31 – 40 years	04	4.0
41 – 50 years	25	25.0
51 – 60 years	31	31.0
More than 60 years	39	39.0
Sex		
Male	54	54.0
Female	46	46.0
Morphological findings		
Gastric ulcer	43	43.0
Chronic gastritis	42	42.0
Acute gastritis	15	15.0

	Number	Percentage
Histopathological findings		
Fungi	01	1.0
Barrett's oesophagus	01	1.0
Squamous cell carcinoma	01	1.0
Adenocarcinoma	01	1.0
H. Pylori	36	36.0
Mild inflammation	24	24.0
Moderate inflammation	16	16.0
Severe inflammation	09	9.0
Neutrophilic activity	36	36.0
Mild mucosal atrophy	24	24.0
Eosinophils	9	9.0
Lymphoid follicles	27	27.0
Dysplasia	8	8.0

Fig-1: 20X -H &E stain: Chronic Gastritis

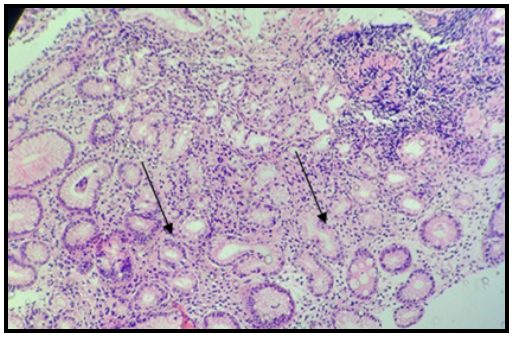


Fig-2: 40x - H & E stain: Aspergillosis Fungal hyphae seen

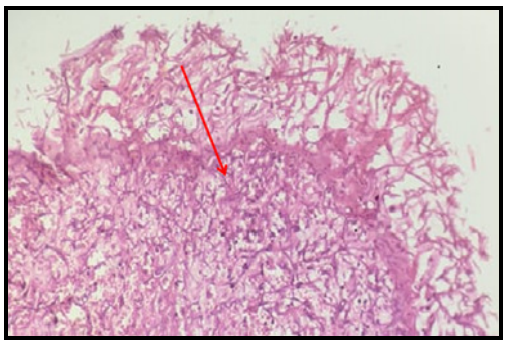


Fig-3: 10X – H & E stain: Barrett's Esophagus; Stratified Squamous Epithelium (⬆) with Columnar Metaplasia (⇩)

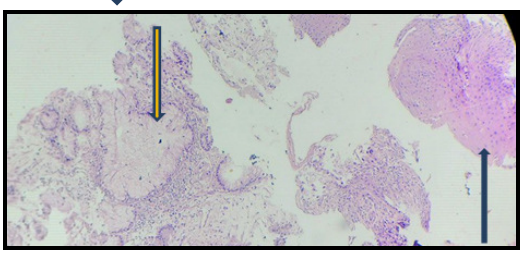
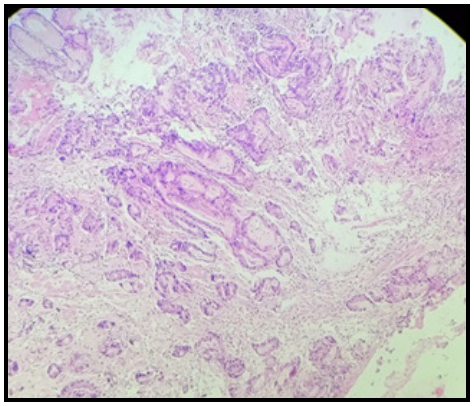


Fig-4: 10x - H & E stain: Adenocarcinoma



Helicobacter Pylori were detected in 33.3% of the gastric ulcer (Fig 5), 52.8% of the chronic gastritis patients and 13.9% of the acute gastritis patients which was statistically not significant (Table 2).

Fig-5: 100X – Giemsa stain: H. Pylori – Long slender bacilli inside the glands

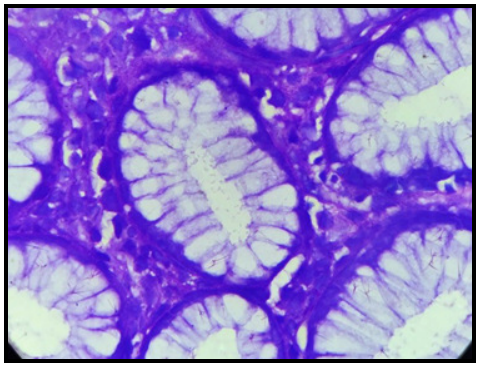


Table-2: Gastric morphology in relation to H Pylori infection

Morphology	H Pylori		P value, sig
	Negative (%)	Positive (%)	
Gastric ulcer	31 (48.4)	12 (33.3)	0.143, NS
Chronic gastritis	23 (35.9)	19 (52.8)	0.101, NS
Acute gastritis	10 (15.6)	5 (13.9)	0.815, NS

This study had shown that, the H. Pylori infection was demonstrated in 27.8% of the cases with mild inflammation, 16.7% with moderate and 2.8% with severe inflammation. This difference was not statistically significant (Table 3).

Table-3: Chronic inflammation in relation to H. Pylori infection			
Chronic inflammation	H Pylori		P value, sig
	Negative (%)	Positive (%)	
Absent	32 (50.0)	19 (52.8)	0.423, NS
Mild	14 (21.9)	10 (27.8)	
Moderate	10 (15.6)	6 (16.7)	
Severe	8 (12.5)	1 (2.8)	

The H. Pylori infection was found in 63.9% of cases without neutrophilic activity and 36.1% with neutrophilic activity. This difference was not statistically significant (Table 4).

Table-4: Neutrophilic activity in relation to H. Pylori infection			
Neutrophilic activity	H Pylori		P value, sig
	Negative (%)	Positive (%)	
Absent	41 (64.1)	23 (63.9)	0.986, NS
Present	23 (35.9)	13 (36.1)	

H.Pylori was demonstrated in 27.8% of the cases with mild mucosal atrophy, 16.7% of the cases with moderate and 11.1% with severe mucosal atrophy. This difference was not statistically significant (Table 5).

Table-5: Mucosal atrophy in relation to H. Pylori infection			
Mucosal atrophy	H Pylori		P value, sig
	Negative (%)	Positive (%)	
Absent	35 (54.7)	16 (44.4)	0.684, NS
Mild	14 (21.9)	10 (27.8)	
Moderate	12 (17.2)	6 (16.7)	
Severe	4 (6.2)	4 (11.1)	

Discussion

This study was undertaken to study the histo-morphology of gastric mucosa in cases with gastritis. This study had shown that majority of the cases with deranged histo-morphology were aged more than 60 year and male patients. These socio demographic features were in concordance with the available studies. In a study by Rani et al, the male to female ratio was 1.5:1 [8].

Upper gastrointestinal tract lesions were more common in males when compared to females and maximum number of cases were during 5^{th} to 7^{th} decade of life which was similar to the results of this study. This study had also shown that, the stomach was the most common site for the lesions in comparison with oesophagus and duodenum.

Gastric ulcer was found in 43.0% of the cases, chronic gastritis in 42.0% of the cases and acute gastritis in 15.0% of the cases.Gastric ulcer was more common manifestation this study. In a study by Atiq et al, mild active gastritis was seen in 19.6% of the cases, moderate active gastritis in 10.5% of the cases and severe active gastritis in 3.9% of the cases.[8] Chronic gastritis is often a most common histopathological finding in gastric biopsies. A study by Jayanthi et al also reported similar findings, chronic inflammation was reported in majority of the cases in their study [9].

H.Pylori infection was demonstrated in 36.0% of the cases in this study. In a study by Atiq et al, 40.5% of the cases had H Pylori activity [9]. In a study by Jayanthi et al, H. Pylori was noted in 35.6% of the cases [10]. H.Pylori infection in most common etiology in chronic gastritis. Histopathological examination remains the common mainstay for detection of H. Pylori infection. The studies have shown the severity of gastritis was directly proportional to the degree of colonization of Helicobacter Pylori. Mild colonization usually demonstrates the milder form of the disease and severe colonization manifests with severe forms of gastritis [11].

The mucous layer of the stomach mucosa coats the epithelial surface and has a crucial protective function. It is an insoluble mucus with a high bicarbonate concentration. Since the bacteria only become pathogenic when they infiltrate the surface epithelial cells and harm the gastric mucosa, it is impossible to establish whether HP that is present in the mucous layer is harmful. Additionally, it was discovered that HP can influence the cervical mucous glands in addition to adhering precisely and selectively to the cytoplasm of mucous surfaces. From a cytological

perspective, the nucleus of the surface mucus cells loses the oval and spherical particles containing mucus that are encased in the bounded membrane.

The cytoplasm then displays spiderweb-like vacuolar degeneration prior to the surface mucous cells proliferating and transforming into intraepithelial neoplasia. Mucosal ulcers, mucosal lymphoid tissue proliferation, gland atrophy, intestinal epithelial metaplasia, MALT lymphoma, and adenocarcinoma can all happen during the histomorphological phase.

Common histological findings included chronic inflammation neutrophilic activity, Mucosal atrophy of mild form, Eosinophils, lymphoid follicles and Dysplasia of the gastric mucosa. In a study by Rani et al, majority of the esophageal biopsies were neoplastic [8]. A study by Atiq et al, showed 33% of the cases had shown atrophic changes and chronicity was demonstrated in 55% of the cases [9]. In a study by Jayanthi et al, the ploymorphonuclear activity was seen in 50.4% of the cases. The chronic gastritis with mild variety was found in 16.4% of the cases which outnumbered moderate and severe activity in their study [10].

In another study by Writteman et al, found a persistent Neutrophilic infiltration in every biopsy, with the majority exhibiting a moderate level of inflammation [12]. A study by Mysorekar had shown that the H Pylori colonisation was associated with the severe antral chronic active gastritis, lymphoid follicles, intestinal metaplasia and dysphagia [13]. Similar to research by Garg et al, a statistically significant correlation was found between acute inflammatory infiltration, lymphoid follicle, and density of HP and chronic inflammation [14]. A study by Wang et al, Mucosal ulcers, mucosal lymphoid tissue proliferation, intestinal epithelial metaplasia, gland atrophy, mucosa-associated lymphoid tissue lymphoma, and adenocarcinoma can all arise during the histomorphology process. The presence of lymphoid follicle often denotes the active inflammation and infection. Based on the histopathological features of HP infection-induced gastric mucosal damage, the degree of gastric mucosal damage, and the depth of involvement, the condition was categorised into five stages in this study: mucus infection, surface epithelial cell infection, lamina propria lesion, mucosal atrophy, and intraepithelial neoplasia [15].

Conclusion

In conclusion, the histomorphological traits, immunophenotype, and pathological phases of gastric mucosal HP infection were identified in this investigation. Based on the extent and severity of the gastric mucosal damage brought on by the HP infection, as well as the regularity of the occurrence and the development of lesions, these aspects were examined in terms of the process that started with the occurrence of a bacterial infection and developed into intraepithelial neoplasia.

This study had a limitation since all the biopsies with ambiguity were excluded and biopsies with poor preservation were also excluded from the study.

This study has shown the histopathological features of the cases with gastritis which included gastric ulcer, acute gastritis and chronic gastritis. The histopathological features include chronic inflammation, neutrophil, eosinophil & lymphocyte activity. H. Pylori was the common organism which was isolated in many samples in this study.

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