Histopathological analysis of chronic gastritis and correlation of pathological features with each other and with endoscopic findings

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Chronic gastritis is a very common disorder which is commonly caused by *Helicobacter pylori*. Histopathological examination is the mainstay of the diagnosis. We evaluated 300 gastric antral biopsies using revised Sydney system and concluded that it helps to analyse gastric biopsies in a very comprehensive manner. It was seen that *H. pylori*, chronic inflammatory infiltrate, neutrophilic infiltration, presence of lymphoid follicles and aggregates and surface epithelial damage are strongly associated with each other. The presence of one of these histological feature is a strong indicator for presence of other features. On the other hand intestinal metaplasia and atrophy of gastric glands was not associated with the above mentioned histological features. In the presence of dense chronic inflammation and infiltration by neutrophils one should carefully search for *H. pylori* organism. We also correlated histological features with endoscopy and found that cases of duodenal ulcer had more severe antral gastritis on histology as compared to those with hyperemia or antral erosions.

Key words: *Helicobacter pylori*, gastritis, inflammation.

Introduction

Gastritis, perhaps best defined as the inflammatory response of the gastric mucosa to injury, is an extremely common condition worldwide. The fundamental histologic change is an increase in the inflammatory cells in the gastric mucosa. Acute gastritis refers to acute mucosal damage after acute alcohol intoxication, drugs, corrosive substance, gastric irradiation etc. Chronic gastritis is mainly characterized by two main features, i.e. infiltration of lamina propria by chronic inflammatory cells and glandular atrophy. For the past 20 years the increased use of endoscopy with biopsy has revolutionized our understanding of the nature and significance of chronic gastritis. Chronic infection with *Helicobacter pylori* is now believed to account for the majority of cases of chronic gastritis. It is also linked to acute gastritis, dyspepsia, peptic ulcer, gastric adenocarcinoma and gastric non-Hodgkin's lymphoma of mucosa-associated lymphoid tissue (MALT).

In spite of numerous flaws, histopathological examination remains the gold standard for diagnosis. *Helicobacter pylori* is a curved to wavy bacterium, which can be identified by hematoxylin and eosin stain when numerous, but is more reliably recognized by other histochemical stains like Giemsa, Warthin-Starry or silver stains. The pattern of inflammation is usually characterized by a mixed acute and chronic inflammatory cell infiltrate in the lamina propria frequently accompanied by intraepithelial infiltration of neutrophils. As there is an effective specific treatment for *H. pylori*-associated gastroduodenal disorders, pathologists are usually requested to identify the organism in endoscopic biopsies.

In 1990, the Sydney system [1] for grading and classifying chronic gastritis was devised to provide a stan-

dardized approach to interpretation of antral biopsies. It emphasizes the importance of combining topographical, morphological and etiological information which would help to generate reproducible and clinically useful diagnosis. This system was reappraised and upgraded in 1994 in a workshop in Houston, Texas [2]. The aims of this workshop were to establish an agreeable terminology of gastritis and to resolve problems of the Sydney system. This system was based on both endoscopic and histologic findings. This system classified gastritis as acute gastritis, chronic gastritis and a special form of gastritis. Since the Sydney system provides a relatively subjective score, in 2002, a modification with a more objective score was proposed by Aydin *et al.* [3]. This technique was more simple and a rapid evaluation of the biopsy was done. Since density of *H. pylori* and the different inflammatory factors appear related to each other, it should be possible to predict presence or absence of one inflammatory parameter based on the presence or absence of a different factor which would also enable the pathologist to give the clinician an overall grade of severity of gastritis.

This study was designed with the following aim: (i) to evaluate histopathological parameters of chronic gastritis according to the revised Sydney system by Aydin *et al.* [3], (ii) to correlate histopathological parameters with endoscopic findings, and (iii) to study the relationship between various histological parameters.

Material and methods

The present study is a one and a half years' prospective study (from January 2008 to June 2009), in which all endoscopic antral biopsies received in the department of Pathology, Dayanand Medical College & Hospital, Ludhiana (India) were analysed with reference to light microscopic findings.

Inclusion criteria for the study were as follows: all patients presenting with chronic ulcer as well as nonulcer dyspepsia. Exclusion criteria included patients with a present or past history of either gastric neoplasms, gastric surgery or long-term therapy with non-steroidal anti-inflammatory drugs, etc. All subjects had given informed consent to study inclusion and a local ethics committee had approved the protocol. Endoscopy was performed and findings, i.e. hyperemia, erosions, ulcerations etc. were recorded and endoscopic biopsies were taken. These were immediately fixed in formalin for histopathological examination.

All biopsies were oriented and processed by routine techniques. 3-4 micrometer thick sections were cut from each block and were stained using hematoxylin and eosin stain. Giemsa stain was done on all biopsies to identify the *H. pylori* organism. The slides were evaluated by two pathologists and a score of 0 to 3 (absent, mild, moderate, marked) was assigned to each morphological variable. *Helicobacter pylori* density, density

Table I. Grading criteria of gastric biopsies according to revised Sydney System by Aydin *et al.* [3]

TYPE OF FEATURE	DENSITY OF THE HISTOLOGICAL FEATURE	GRADE
chronic inflammation (lymphocytes and plasma cells)	2-3 chronic inflammatory cells scattered randomly in the biopsy	nil (0)
	10-15 chronic inflammatory cells/hpf	mild (1)
	some areas with dense chronic inflammatory cells	moderate (2)
	diffuse infiltration with dense chronic inflammatory cells	marked (3)
neutrophilic infiltration	no neutrophils anywhere in the biopsy	nil (0)
	scattered neutrophils in the biopsy	mild (1)
	foci of dense neutrophilic infiltrate with scattered neutrophils in the rest of the biopsy	moderate (2)
	several foci of dense inflammatory infiltrate in the biopsy with involvement of crypts	marked (3)
atrophy	no evidence of gastric gland loss	nil (0)
	small areas where gastric glands have disappeared (< 25%)	mild (1)
	25-50% of the biopsy shows loss of gastric glands	moderate (2)
	> 50% of the biopsy shows loss of gastric glands	marked (3)
intestinal metaplasia	no intestinal metaplasia	nil (0)
	focal areas of intestinal metaplasia (1-4 crytps)	mild (1)
	multiple foci involving > 4 crypts but < 50% of the biopsy	moderate (2)
	intestinal metaplasia involving > 50% of the biopsy specimen	marked (3)
H. pylori density	no *H. pylori* anywhere in the biopsy	nil (0)
	only a few *H. pylori* seen in single or multiple foci	mild (1)
	numerous *H. pylori* seen in separate areas of foci	moderate (2)
	> 50% of the surface area covered with *H. pylori*	marked (3)

Table II. Histological scoring index as proposed by Wootherspoon *et al.* [4]

Grade	Description	Histological features
0	normal	scattered plasma cells in lamina propria, no lymphoid follicles
1	chronic active gastritis	small clusters of lymphocytes in lamina propria, no lymphoid follicles, no LELs
2	chronic active gastritis with florid lymphoid follicles formation	prominent lymphoid follicles with surrounding mantle zone and plasma cells no LELs
3	suspicious lymphoid infiltrate in lamina propria, probably reactive	lymphoid follicles surrounded by small lymphocytes that infiltrates diffusely in lamina propria and occasionally into epithelium
4	suspicious lymphoid infiltrate in lamina propria, probably lymphoma	lymphoid follicles surrounded by CCL cells that infiltrate diffusely in lamina
5	low grade B-cell lymphoma of MALT	presence of dense diffuse in filtrate of CCL cells in lamina propria with prominent LELs

LEL – lymphoepithelial lesion, CCL – centrocyte like lesion

Table III. Age wise distribution of chronic gastritis

Age group	Total No. of cases	Percentage
< 15	3	1%
16-30	64	21.33%
31-45	73	24.33%
46-60	94	31.33%
61-75	55	18.33%
> 75	11	3.67%

Table IV. Endoscopic findings in all the patients

Endoscopic findings	No. of cases	% age
antral hyperemia	204	68
antral erosion	21	7
duodenal ulcer	15	5
normal gastric mucosa	60	20

Hypermia was most commonly encountered endoscopically.
Histological features in all 300 biopsies in accordance with revised Sydney classification.

of neutrophils, lymphocytes and plasma cells were recorded. Glandular atrophy and intestinal metaplasia were also noted and graded according to the above three categories. The grading of presence of lymphoid follicles was done per histologic scoring index as proposed by Wotherspoon *et al.* [4].

For each variable, the highest score was given among antral biopsies according to the scoring system used by Aydin *et al.* [3] (Table I). Grading of lymphoid follicles was done by the index proposed by Wotherspoon *et al.* [4] (Table II).

Statistical analysis

The association between histological findings was assessed by means of χ^2 test for trend [5]. A probability value of $p = 0.05$ was considered statistically significant.

Results

In the present study, 400 endoscopic antral biopsies were received in our department between January 2008 and June 2009; out of which 100 patients had either a history of gastric neoplasm, surgery or long treatment by NSAIDS. So, these 100 patients were ex-cluded from the study. Hence, 300 antral biopsies were analysed taking into consideration the endoscopic findings and various histological parameters such as chronic inflammatory infiltrate, inflammatory activity, glandular atrophy, intestinal metaplasia, surface epithelial damage, *H. pylori* and intestinal metaplasia.

Number of endoscopic antral biopsies analysed = 300

Most common age group affected by chronic gastritis was 46-60 years (Table III). There were 204 males and 96 females with an M : F ratio of 2.1 : 1.

The endoscopic findings

Chronic inflammatory activity was seen maximally in duodenal ulcer (60%), followed by antral erosions and then hyperemia. In 22% of cases having a normal looking mucosa on endoscopy, inflammatory activity was seen. Histologically *H. pylori* density and lymphoid follicles was maximum in duodenal ulcer. In most cases, glandular atrophy was noted in endoscopically normal looking mucosa, while intestinal metaplasia was maximally seen in cases having antral hyperemia.

Table V. Histological grading of all biopsies according to revised Sydney system [3]

HISTOLOGICAL FEATURES	GRADE	NUMBER OF CASES
chronic inflammation	nil	0 (0%)
	mild	210 (70%)
	moderate	82 (27.33%)
	marked	8 (2.6%)
neutrophilic infiltration (activity)	nil	200 (66.67%)
	mild	63 (2%)
	moderate	35 (11.67%)
	marked	2 (0.67%)
glandular atrophy	nil	263 (87.67%)
	mild	30 (10.00%)
	moderate	7 (2.33%)
	marked	0 (0.00%)
intestinal metaplasia	nil	279 (93%)
	mild	16 (5.33%)
	moderate	5 (1.67%)
	marked	0 (0.00%)
density of *H. pylori*	nil	169 (56.33%)
	mild	121 (40.33%)
	moderate	10 (3.33%)
	marked	0 (0.00%)
surface epithelial damage	nil	4 (1.33%)
	mild	258 (86.00%)
	moderate	3 (1.00%)
	marked	35 (11.67%)

Table VI. Distribution of cases on basis of grade of lymphoid follicles as per the histological scoring index proposed by Wootherspoon *et al*. [4]

GRADE	NUMBER OF CASES WITH % AGE
0	163 (54.33%)
1	76 (25.33%)
2	59 (19.66%)
3	0 (0.00%)
4	0 (0.00%)
5	2 (0.67%)

($p = 0.00$); density of *H. pylori* ($p = 0.00$), surface epithelial damage ($p = 0.01$) and lymphoid follicles ($p = 0.00$). No significant association of chronic inflammatory infiltrates with glandular atrophy ($p = 0.10$) and intestinal metaplasia ($p = 0.15$) was observed.

B. Inflammatory activity and its correlation with other histological parameters

A statistically significant relation between inflammatory activity and density of *H. pylori* ($p = 0.00$); surface epithelial damage ($p = 0.02$) and lymphoid follicles ($p = 0.00$) was observed. There was no significant association between inflammatory infiltrate and glandular atrophy ($p = 0.58$) along with intestinal metaplasia ($p = 0.99$).

C. Correlation of *H. pylori* with various other histological parameters

There was a very strong association between chronic inflammatory infiltrates, activity, surface epithelial damage and lymphoid follicles; while glandular atrophy and intestinal metaplasia showed no correlation.

D. Correlation of glandular atrophy with other histological parameters

There was a statistically significant correlation between glandular atrophy and intestinal metaplasia; while there was no correlation with chronic infiltrate,

Intercorrelation among various histological parameters

A. Correlation of chronic inflammatory infiltrate with other histological parameters

The density of chronic infiltrate was strongly associated with the presence of inflammatory infiltrate

Table VII. Endoscopic and histological correlation

HISTOLOGICAL FEATURES	ANTRAL HYPEREMIA (TOTAL CASES = 204)	ANTRAL EROSION (TOTAL CASES = 21)	DUODENAL ULCERS (TOTAL CASES = 15)	NORMAL (TOTAL CASES = 60)
chronic inflammatory infiltrate	mild – 145	mild – 11	mild – 4	mild – 50
	moderate – 51	moderate – 10	moderate –11	moderate – 10
	severe – 8	severe – 0	severe – 0	severe – 0
activity	66 (32%)	12 (57%)	9 (60%)	13 (22%)
surface epithelial damage	200 (98%)	21 (100%)	15 (100%)	60 (100%)
lymphoid follicles	27 (13%)	12 (57%)	14 (93%)	8 (13%)
glandular atrophy	25 (12%)	2 (10%)	1 (7%)	9 (15%)
intestinal metaplasia	18 (9%)	0 (0%)	0 (0%)	3 (5%)
H. pylori density	91 (45%)	12 (57%)	14 (93%)	9 (0%)

Table VIII. Summary of intercorrelation among various histological parameters with each other
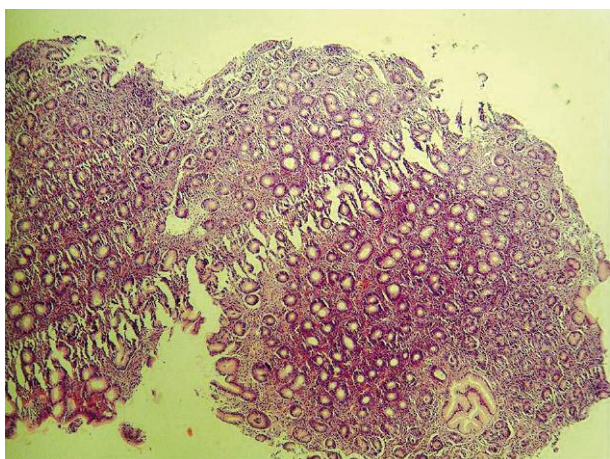
PARAMETERS	CHRONIC INFILTRATE	ACTIVITY	*H.PYLORI*	GLANDULAR ATROPHY	SURFACE EPITHELIAL DAMAGE	INTESTINAL METAPLASIA	LYMPHOID FOLLICLES
chronic infiltrate	–	p = 0.00	p = 0.00	p = 0.12	p = 0.01	p = 0.15	p = 0.00
activity	p = 0.00	–	p = 0.00	p = 0.58	p = 0.02	p = 0.99	p = 0.00
H. pylori	p = 0.00	p = 0.00	–	p = 0.53	p = 0.01	p = 0.09	p = 0.00
glandular atrophy	p = 0.10	p = 0.58	p = 0.53	–	p = 0.19	p = 0.00	p = 0.30
surface epithelial damage	p = 0.01	p = 0.02	p = 0.01	p = 0.19	–	p = 0.13	p = 0.00
intestinal metaplasia	p = 0.15	p = 0.99	p = 0.09	p = 0.13	p = 0.13	–	p = 0.10
lymphoid follicles	p = 0.00	p = 0.00	p = 0.00	p = 0.30	p = 0.10	p = 0.10	–

The association between the histological parameters was assesses by means of chi square test. A probability value of p value = 0.05 was considered statistically significant.
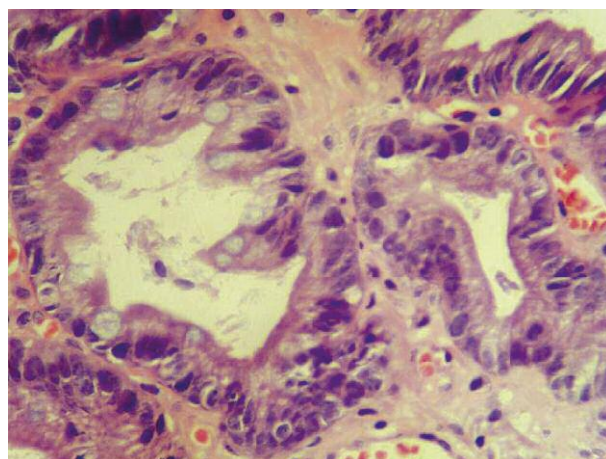** P value equal or less than 0.05 is statistically significant*
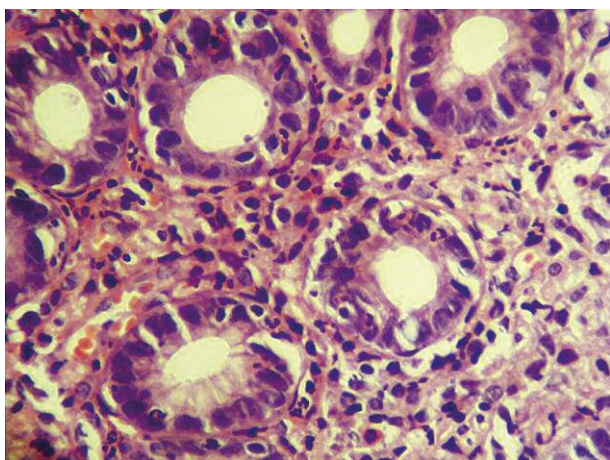** P value equal 0.00 is highly significant*
* P value greater than 0.05 is not statistically significant
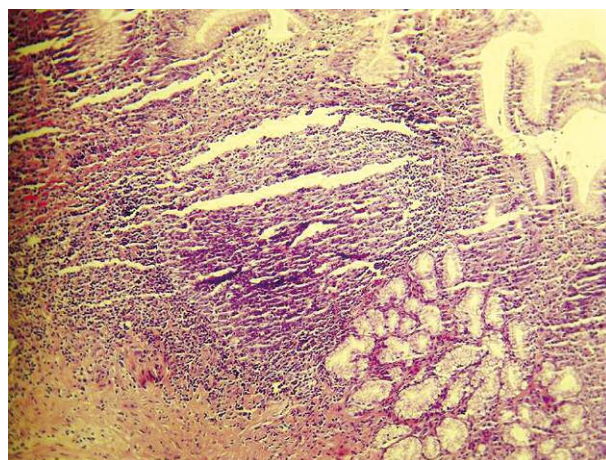

Fig. 1. Gastric biopsy showing surface epithelial erosion. Magnification 40×

Fig. 2. Gastric biopsy showing intestinal metaplasia. Magnification 400×

Fig. 3. Gastric biopsy showing intraepithelial neutrophils. Magnification 400×

Fig. 4. Gastric biopsy showing lymphoid follicle. Magnification 100×

inflammatory activity of *H. pylori*, surface epithelial damage and lymphoid follicles.

E. **Correlation of surface epithelial damage and other parameters**

Chronic infiltrates, activity, *H. pylori* density and lymphoid follicles had a strong correlation with surface epithelial damage, while a glandular atrophy and intestinal metaplasia had no association.

F. **Correlation of intestinal metaplasia with other parameters**

Only glandular atrophy was associated with intestinal metaplasia. Other parameters were not statistically significant.

G. **Correlation of lymphoid follicles with other parameters**

There was a statistically significant association between lymphoid follicles and chronic infiltrate, activity, *H. pylori* and surface epithelial damage; whereas glandular atrophy and intestinal metaplasia had no association with lymphoid follicles.

Discussion

In the present analysis, the most common age group affected by chronic gastritis was 46-60 years with a mean age of 47 years, which is consistent with studies done by Aydin *et al.* [3] and Mustapha *et al.* [6] who reported a mean age of 47.2 years. A higher incidence was seen in males with an M : F ratio of 2.1 : 1. Chen *et al.* [7] also found a M : F ratio of 1.8 : 1.

In this study we concentrated only on antral biopsies because most of the studies have reported that antrum is the most likely site of histopathological findings in gastritis [8]. The criteria laid down by Aydin *et al.* [3] are extremely useful in analyzing the gastric biopsies for suspected gastritis.

The endoscopic findings in our study revealed the presence of erythema in 68% of cases, antral erosion in 7%, duodenal ulcer in 5% and normal gastric mucosa in 20% of cases. These findings were also noted by Khakoo *et al.* [9] and Calabrese *et al.* [10] who found erythema in 44% and 43% of cases, respectively. Chronic inflammatory infiltrate was found in the majority of the cases (70%). However, in 20% of cases who were endoscopically normal, chronic inflammation was also revealed on histology; which was also observed by Khan *et al.* [11] who had 32% of patients with chronic gastritis histologically and normal endoscopic findings, hence emphasizing the role of biopsy even in normal endoscopic cases. Frequency of *H. pylori* in the present study in patients with hyperema and erosion was 37% and 57%, respectively. In patients with duodenal ulcer, colonization by *H. pylori* was seen in 93% of cases; which is extremely significant. Also the majority of patients with duodenal ulcer had more severe gastritis on histology as compared to subjects having antral hyperemia and erosions. Chronic in-
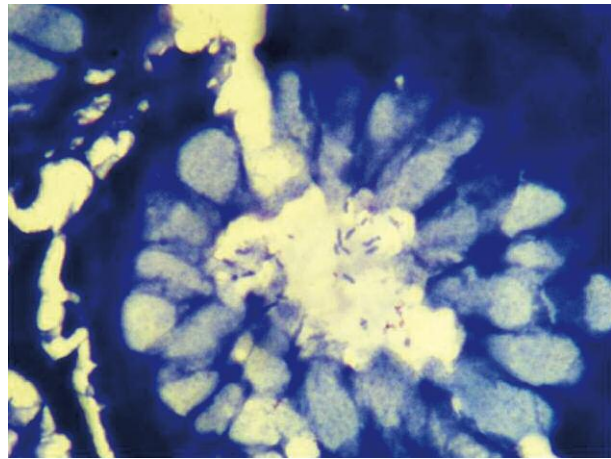

Fig. 5. Gastric biopsy showing *H. pylori*. Magnification 1000×

flammatory infiltrates were present in all the cases with the majority (70%) having only mild inflammation while 27% had moderate inflammation. A study done by Witteman *et al.* [12] observed chronic infiltrate in all biopsies but the majority had moderate inflammation. In 33% of our cases, an activity was seen, which is in accordance with a study published by Misra *et al.* [13]. Also in cases with a significant activity, *H. pylori* density was very high. Glandular atrophy was seen in only 12.33% of cases and intestinal metaplasia again in only 7% of cases. This was also observed by Atisook *et al.* [14] and Nawfal *et al.* [15]. *Helicobacter pylori* was observed in 43% of all the cases only in our study while studies done by Kumar *et al.* [16] and Gill *et al.* [17] showed positivity in 78% and 65% of cases, respectively. This low prevalence of *H. pylori* could be attributed to the use of proton pump inhibitors by the patients and also some patients were on antibiotics which are known to suppress *H. pylori*.

In 98.67% of antral biopsies, we observed surface epithelial damage. Lymphoid aggregates and lymphoid follicles were identified in 25% and 19% of subjects, respectively. Two cases were reported as low grade B cell lymphoma of the MALT type. The paradox that lymphoid follicles are absent in normal stomach mucosa is explained by the observation that mucosa-associated lymphoid tissue (MALT) appears in stomach in response to *H. pylori* infection. Hyperactive follicles may then develop into lymphoma. The presence of lymphoid follicles was strongly associated with severe mucosal inflammation, activity and *H. pylori* infection.

We observed that chronic inflammatory infiltrate correlated strongly with acute inflammatory activity, surface epithelial damage, lymphoid follicle and density of *H. pylori*. There was no correlation between chronic inflammation and intestinal metaplasia and glandular atrophy. This was also seen by Witteman *et al.* [12].

Again presence of neutrophils meant a higher density of *H. pylori*, chronic inflammatory infiltrate, more surface epithelial damage and lymphoid follicles. Khalid *et al.* [19] also reported that neutrophil activity is very commonly noted in *H. pylori* infection. A greater density of organisms was noted in higher grades of inflammation, surface epithelial damage and lymphoid follicles. Mysorekar *et al.* [18] also substantiated this finding. However, in our study there was no association among *H. pylori*, intestinal metaplasia and glandular atrophy. In fact, intestinal metaplasia is considered a negative indicator for *H. pylori* colonization; meaning that *H. pylori* does not colonize epithelium that has undergone intestinal metaplasia. This was also seen by Witteman *et al.* [12]. Lymphoid follicles also correlated strongly with mucosal inflammation activity, epithelial damage and density of *H. pylori*. It is widely believed that presence of lymphoid follicles and aggregates in gastric mucosa is a conspicuous feature of *H. pylori*-associated chronic gastritis as lymphoid tissue hyperplasia is a specific immunological reaction to *H. pylori* infection. Studies have shown that a proliferation of T cells and macrophages is induced by *H. pylori*, which release several cytokines (interleukin 2 and 6) which leads to proliferation of B cells and development of lymphoid follicles.

In conclusion, our study has highlighted the role of histopathology in all cases of gastritis and shown that *H. pylori* is strongly associated with an acute and chronic inflammatory infiltrate. Hence, we suggest that a search for *H. pylori* should be initiated if neutrophils and lymphoid follicles are seen in the antral biopsies. Also the modified Sydney classification is extremely useful in analyzing antral biopsies. It was also observed that cases of duodenal ulcer had much severer antral gastritis and stronger positivity for *H. pylori*. Intestinal metaplasia and glandular atrophy is not correlated with other histological parameters and *H. pylori* infection. Meanwhile, histological features like lymphoid follicles, activity, *H. pylori* density and surface epithelial damage are associated with one another.

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