Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2022.112837.2443 Volume 29, Issue 4, Jully 2023
Manuscript ID ZUMJ-2112-2443 (R2)
DOI 10.21608/zumj.2022.112837.2443

ORIGINAL ARTICLE

Role of Endoscopy and Histopathology in Diagnosis and Follow up of Chronic Gastritis

Tawfeek Ibrahim Mohamed Ibrahim[1*], Gumie Mohamed Abd El Baset[2], Ismail Waleed Abd El Fattah[2], El Kashishy KamalAhmed[2]

[1]internal Medicine Department, Mataryea Teaching Hospital, Egypt.

[2]internal Medicine Department, Faculty of Medicine, Zagazig University, Egypt.

***Corresponding author:**
Tawfeek Ibrahim Mohamed Ibrahim
E-mail address:
Dr.mohameddaskora@yahoo.com

Submit Date 2021-12-28 01:36:59
Revise Date 2022-01-11 18:52:58
Accept Date 2022-01-17

ABSTRACT

Background: chronic gastritis is one of the most prevalent pathological entities in gastroenterology and digestive endoscopy conflicting patients' morbidity. This cohort study aims to study the role of upper gastro-intestinal tract (Upper GIT) endoscopy and histopathology in diagnosis, follow up and analyze the correlation between the endoscopic and histopathologic findings of chronic gastritis. **Methods:** In this cohort study, 85 patients of chronic gastritis were studied and followed up endoscopically and histopathologically within 3 to 6 months after treatment between January 2017 and January 2020. Six biopsies were taken from different areas of the stomach and comparing the endoscopic (modified Lanza score) with histopathologic findings according to Sydney System. **Results:** The longest duration of symptoms of chronic gastritis was (1 up to < 2 years), the most common cause was NSAIDS (65.9%) and H. Pylori induced gastritis by (50.6%). There was significant weak direct correlation between degree of gastritis (endoscope) and degree of gastritis (histopathology) before treatment (p- Value is 0.026) and after treatment (p- Value is 0.007) Also, there was highly significant (HS) improvement after treatment in degree and extent of chronic gastritis endoscopically and histopathologically as p- value <0.001 (HS). **Conclusions:** Chronic gastritis is a common disease that affects different age groups. NSAIDS induced chronic gastritis is the most common cause of chronic gastritis. H Pylori is the second most common cause of chronic gastritis so preventive measures and treatment regimens should be applied to eradicate H. Pylori.

Keywords: Chronic gastritis; Upper GIT Endoscopy; Histopathology; follow up; NSAID

INTRODUCTION

Chronic gastritis is a common condition worldwide which affects 50% of the world's population [1] and is defined as persistent gastric inflammation with "lymphocyte and plasma cell infiltration", intestinal metaplasia, dysplasia and /or glandular atrophy [2]. H. pylori prevalence in Egypt ranges from 13% to 72% in children, 26% up to 90% in adults reported by world gastroenterology organization and is considered one of the main causes of chronic gastritis [3, 4]. The underlying etiologies of chronic gastritis need to be clarified, as it is a predisposing factor for gastric adenocarcinoma and remains the second most frequent cause of cancer – related mortality in the world [5]. Most common causes of chronic gastritis are helicobacter pylori infection (50.6%) and chronic use of non-steroidal anti-inflammatory drugs (NSAIDS 65.9%) [6]. Other causes of chronic gastritis include bile reflux gastritis, radiation induced gastritis, lymphocytic gastritis, eosinophilic gastritis, autoimmune gastritis and idiopathic gastritis [1].

The correlation between the diagnosis of gastritis by endoscopic and histopathologic features is still confusing, most studies have concluded that H. pylori related gastritis cannot be diagnosed based on gastroscopic findings alone [7]. Using upper GI (Gastro-intestinal) endoscopy with gastric biopsy in the last twenty years has raised our minds to know the nature and importance of chronic gastritis and intestinal metaplasia which is more common in the

general population [5]. H. pylori chronic infection is believed to be the main cause of chronic gastritis and also it is related to acute gastric inflammation, dyspepsia, peptic ulcer disease, gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) Non-Hodgkin's lymphoma. "Histopathological examination remains the gold stander for diagnosis of chronic gastritis, which is mainly caused by H. pylori and can be identified by hematoxylin and eosin stain and Giemsa stain" [2]. Eosinophilic gastritis (EG) is characterized by eosinophilic infiltration of the stomach by twenty or more eosinophilic cells per high-power field [8] Lymphocytic gastritis (LG) is characterized by infiltration of the gastric foveolar epithelium by at least twenty-five lymphocytes per one hundred epithelial cells, regardless of the inflammation in the lamina propria [9, 10, 11]. "Treatment of H. pylori may improve gastric mucosal inflammation, atrophy, and prevent the progression of intestinal metaplasia" [12].

Aim of the work:

To assess types of chronic gastritis in our locality, correlate histopathological parameters of chronic gastritis according to the revised Sydney system with its endoscopic findings according to modified Lanza score and to assess the treatment effect on chronic gastritis according to its etiology on the endoscopic and histopathologic findings.

METHODS

This cohort study has been carried out in gastroenterology units at Zagazig University, Faculty of Medicine and Matareya Teaching hospital, General Organization of Teaching Hospitals and Institutes (GOTHI) during the period from January 2017 to January 2020. Net patients (85) on this study undergo upper Gastro-intestinal tract (GIT) endoscopy and biopsy (before and after treatment of chronic gastritis) after fulfillment of the inclusion and exclusion criteria.

All patients complaining of symptoms of gastritis in the form of dyspepsia, epigastric pain or heart burn for more than three months and patients with long-term therapy with Non-Steroidal Anti-Inflammatory Drugs (NSAIDS) were included in the study.

Patients with Dropped or refused re-endoscopy and follow up, patients received an H2- receptor blockers or proton pump inhibitor (PPI) during the 4 weeks prior to the endoscopic examination; patients with present or history of gastric surgery, neoplasms or H. pylori eradication therapy; Heart failure.; End stage renal disease (on hemodialysis); decompensated liver disease (liver cirrhosis) and pregnant women were excluded.

The study includes 85 patients as a comprehensive sample of chronic gastritis calculated by StatCalc sample size and power 80%, C.I. 95%. All patients were complaining of symptoms of chronic gastritis and fulfilled the inclusion criteria and agreed for follow up during treatment periods and re-endoscopy during the study period.

Explanation of the procedure to all patients participating in the study. A pre-endoscopy with written consent was taken from all patients and all patients followed the instructions and ethics of a local committee.

Protocol Approval by Ethical Committee: Before the beginning of the study and in accordance with the local regulation followed, the protocol and all corresponding documents were declared for Ethical and Research approval by Zagazig University Institutional Review Broad (IRB) number ZU-IRB#: 3190-20-11-2015 and IRB approved that the study is within the ethical guidelines as outlined in the Declaration of Helsinki.

Patients were subjected to the following: Complete history taking including age, any special habit of medical importance (drug history) etc. Clinical examination according to inclusion and exclusion criteria. Routine investigation: CBC- liver and kidney functions, fasting blood sugar, urine analysis and stool analysis, serology for H. pylori antibody, fecal stool antigen for H pylori, occult blood in stool, chest x-ray- ultrasound abdomen & pelvis – ECG routine investigations in hospital pre-endoscopy.

H Pylori Ab (Ag) combo rapid test CTK Biotech, Inc 13855 Stowe Drive Poway, CA 92064, USA MDSS GmbH Schiffgraben 41 – 30175 Hannover, Germany.

Endoscopic assessment of gastric mucosa (according to modified Lanza) [13] (Supplementary Table 1) before and after treatment by known conventional therapy according to specific etiology of chronic gastritis as Chronic gastritis appeared endoscopically as (hyperemic mucosa, erosion, ulcer, even normal mucosa) [2]. Upper gastrointestinal endoscopy using the video-endoscope pentax EG-3490K or Pentax EG-3890LK (Pentax, Tokyo, Japan) after overnight fast 6 hours, after local pharyngeal anesthesia was provided using lidocaine (xylocaine) and intravenous 3-4 mg of midazolam.

Histopathological assessment (according to standardized biopsy protocols and Sydney protocol) of gastric mucosa before and after treatment by six biopsies taken from: two antral biopsies put in bottle 1, two biopsies one from incisura angularis and the other from the body (greater curvature) put in bottle 2 and two biopsies one from the fundus and the cardia put in bottle 3 and the need to take any additional biopsy from any focal lesion.

Tissue sections stained by routine Hematoxylin & Eosin and examined according to the revised Sydney system [14] (Supplementary Table 2) and gastritis graded as (nil-mild-moderate-severe) for each histopathologic feature (Chronic inflammation by lymphocyte and plasma cells - Neutrophilic infiltration - atrophy - Intestinal metaplasia - H. pylori density detected by Giemsa stain).

Treatment according to specific cause of chronic gastritis:

Sequential therapy for H. pylori is encouraging, treatment in two-steps: 14-day program consisting of administration of a PPI (esomeprazole 20 mg twice daily) with amoxicillin (1 g twice daily) for the first 7 days, followed by triple therapy that includes a PPI, clarithromycin (500 mg twice daily) and tinidazole (500 mg twice daily) for another 7 days [15]

Stop NSAIDS and give antacids or PPI according to the severity of gastritis induced by NSAIDS.

Repeated upper endoscopy with biopsy (3 biopsies will be taken from (antrum –incisura & body - funds) after end of treatment within 6 months to follow up endoscopic and histopathologic findings.

Patient non-compliance (7 patients). Patient improved on treatment and refused follow up (20 patients).

STATISTICAL ANALYSIS

All data were collected, tabulated and statistically analyzed using SPSS 22.0 for windows (IBM Inc., Chicago, IL, USA). Categorical qualitative variables were expressed as absolute frequencies (number) & relative frequencies (percentage). Categorical data were compared using Chi-square test or Fisher's exact test when appropriate. Stuart-Maxwell test was used to test homogeneity in paired categorical data. All tests were two sided. P-value < 0.05 was considered statistically significant (S), p-value < 0.001 was considered highly statistically significant (HS), and p-value ≥ 0.05 was considered statistically insignificant (NS).

RESULTS

Regarding the different etiologies of chronic gastritis among the studied 85 patients, the most common cause was reactive gastropathy especially (NSAIDS accounted for 65.9%) and H. Pylori induced gastritis by 50.6% (Table 1).

There is highly significant relationship when comparing the degree of chronic gastritis in endoscopy before and after treatment: There was 83.6 % of the studied cases of chronic gastritis were improved, while 16.4% no change and no cases became severe (Table 2).

There is significant relationship between histopathological grading of gastritis (Sydney system) and degree of gastritis by endoscope among H. Pylori intensity and degree of gastritis but other parameters of Sydney system revealed no significant relationship (Table 3).

There is significant relationship between degree of chronic gastritis in endoscopy and histopathology before treatment as we found that there is 36.4% of the studied cases had the same degree of chronic gastritis, 42.3% are underestimated and 21.3% are overestimated. So, there is a significant weak direct correlation between degree of gastritis (endoscope) and degree of gastritis (histopathology) before treatment (Table 4).

There is a significant relationship between degree of chronic gastritis in endoscopy and histopathology after treatment as p- Value is 0.007. We found that 70.6% of the studied patients had the same degree of chronic gastritis by endoscopy and histopathology, 4.8 % of the studied cases were underestimated, and 24.8% were overestimated. So, there is a significant weak direct correlation between degree of gastritis (endoscope) after treatment and Degree of gastritis (histopathology) after treatment (Table 5).

Eosinophilic gastritis was detected in 2.4% of cases in this study (Figure 1 A, B).

Collagenous gastritis although it is rare but one case is detected in this study, also the same patient had collagenous colitis (Supplementary figure 1).

Table 1: Number and percentage of causes of chronic gastritis among the studied patients (N=85)

Etiology	The studied chronic gastritis patients (N=85)	
	No.	%
No apparent etiology	2	2.4%
H.pylori alone	22	25.9%
NSAID alone	33	38.8%
Bile reflux alone	3	3.5%
Autoimmune alone	1	1.2%
Eosinophilic alone	1	1.2%
NSAID + Collagenous	20	23.5%
H.pylori + NSAID + Bile reflux	1	1.2%
H.pylori induced gastritis	**43**	**50.6%**
Reactive gastropathy	**59**	**69.4%**
NSAID induced gastropathy	56	65.9%
Bile induced gastropathy	4	4.7%
Immune mediated gastritis	**3**	**3.5%**
Autoimmune mediated gastritis	1	1.2%
Eosinophilic gastritis	2	2.4%
Idiopathic gastritis	1	1.2%
Collagenous	1	1.2%

Table 2: Comparison between degree of chronic gastritis endoscopically according to Endoscopic scoring of gastric mucosal lesions modified Lanza by Shim et al.., 2019) before and after treatment

Degree of gastritis (endoscope) before Treatment	Total		Degree of gastritis (endoscope) after treatment							
	NO	%	Nil		Mild		Moderate		Severe	
			No	%	No	%	No	%	No	%
Nil	3	3.5 %	3	3.5%	0	0%	0	0%	0	0%
Mild	17	20%	9	10.6%	8	9.4%	0	0%	0	0%
Moderate	32	37.6%	14	16.5%	15	17.6%	3	3.5%	0	0%
Severe	33	38.8%	10	11.8%	22	25.9%	1	1.2%	0	0%

Degree of gastritis (endoscope) before	Total		Degree of gastritis (endoscope) after treatment							
Total	85	100%	36	42.4%	45	52.9%	4	4.7%	0	0%
Test[a]	64.687									
p-value (Sig.)	<0.001 (HS)									

Categorical variables were expressed as number (percentage); a: Stuart-Maxwell test; p-value < 0.05 is significant; Sig.: Significance.

Table 3: Histopathological grading (revised Sydney system by Aydin et al., 2003) according to severity of gastritis by endoscopy modified Lanza by Shim et al., 2019)

Histo-pathological grading before treatment	All patients (N=85)		Degree of gastritis before treatment (endoscope) Nil (N=3)		Mild (N=17)		Moderate (N=32)		Severe (N=33)		Test[a]	p-value (Sig.)
	No	%	No	%	No	%	No	%	No	%		
Chronic inflammation												
Nil	0	0%	0	0%	0	0%	0	0%	0	0%	6.622	0.357 (NS)
Mild	32	37.6%	2	66.7%	7	41.2%	15	46.9%	8	24.2%		
Moderate	40	47.1%	1	33.3%	6	35.3%	14	43.8%	19	57.6%		
Severe	13	15.3%	0	0%	4	23.5%	3	9.4%	6	18.2%		
Neutrophil activation												
Nil	10	11.8%	1	33.3%	2	11.8%	4	12.5%	3	9.1%	3.215	0.781 (NS)
Mild	62	72.9%	1	33.3%	13	76.5%	24	75%	24	72.7%		
Moderate	13	15.3%	1	33.3%	2	11.8%	4	12.5%	6	18.2%		
Severe	0	0%	0	0%	0	0%	0	0%	0	0%		
Surface epithelium damage												
Nil	5	5.9%	1	33.3%	1	5.9%	2	6.2%	1	3%	10.708	0.296 (NS)
Mild	37	43.5%	1	33.3%	10	58.8%	15	46.9%	11	33.3%		
Moderate	38	44.7%	1	33.3%	6	35.3%	14	43.8%	17	51.5%		
Severe	5	5.9%	0	0%	0	0%	1	3.1%	4	12.1%		
Glandular atrophy												
Nil	73	85.9%	3	100%	16	94.1%	27	84.4%	27	81.8%	3.197	0.784 (NS)
Mild	11	12.9%	0	0%	1	5.9%	5	15.6%	5	15.2%		
Moderate	1	1.2%	0	0%	0	0%	0	0%	1	3%		
Severe	0	0%	0	0%	0	0%	0	0%	0	0%		
Intestinal metaplasia												
Nil	76	89.4%	3	100%	16	94.1%	28	87.5%	29	87.9%	4.511	0.608 (NS)
Mild	7	8.2%	0	0%	1	5.9%	4	12.5%	2	6.1%		
Moderate	2	2.4%	0	0%	0	0%	0	0%	2	6.1%		
Severe	0	0%	0	0%	0	0%	0	0%	0	0%		
H. pylori intensity												

Histo-pathological grading before treatment	All patients (N=85)		Degree of gastritis before treatment (endoscope) Nil (N=3)		Mild (N=17)		Moderate (N=32)		Severe (N=33)		Test[a]	p-value (Sig.)
Nil	41	48.2%	3	100%	4	23.5%	12	37.5%	22	66.7%	20.364	0.016 (S)
Mild	24	28.2%	0	0%	5	29.4%	14	43.8%	5	15.2%		
Moderate	19	22.4%	0	0%	8	47.1%	5	15.6%	6	18.2%		
Severe	1	1.2%	0	0%	0	0%	1	3.1%	0	0%		

Categorical variables were expressed as number (percentage); a: Chi-square test; p-value < 0.05 is significant; Sig.: Significance.

Table 4: Comparison & correlation between degree of chronic gastritis in endoscopy (modified Lanza and histopathology (revised sydney system by Aydin)

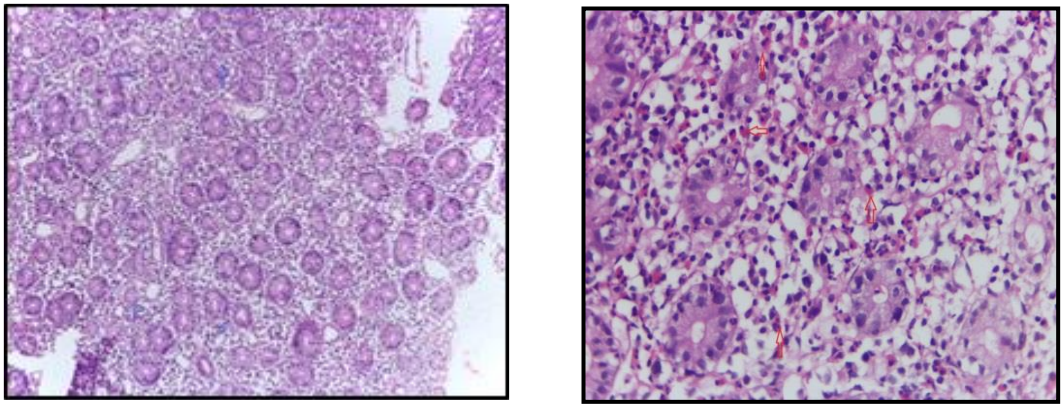
Degree of gastritis (endoscope) before treatment	Total NO	%	Degree of gastritis (histopathology) before treatment							
			Nil		Mild		Moderate		Severe	
			No	%	No	%	No	%	No	%
Nil	3	3.5%	0	0%	2	2.4%	0	0%	1	1.2%
Mild	17	20%	0	0%	7	8.2%	6	7.1%	4	4.7%
Moderate	32	37.6%	0	0%	15	17.6%	12	14.1%	5	5.9%
Severe	33	38.8%	0	0%	6	7.1%	15	17.6%	12	14.1%
Total	85	100%	0	0%	30	35.3%	33	38.8%	22	25.9%
Test[a]			9.362							
p-value (Sig.)			0.024 (S)							
tau[b]			+0.212							
Test			2.225							
p-value (Sig.)			0.026 (S)							

Categorical variables were expressed as number (percentage); a: Stuart-Maxwell test; b: Kendall's tau-c correlation coefficient; p-value < 0.05 is significant; Sig.: Significance.

Table 5: Comparison & correlation between degree of chronic gastritis in endoscopy (modified Lanza by Shim et al.., 2019) and histopathology (revised Sydney system by Aydin et al.., 2003) after treatment.

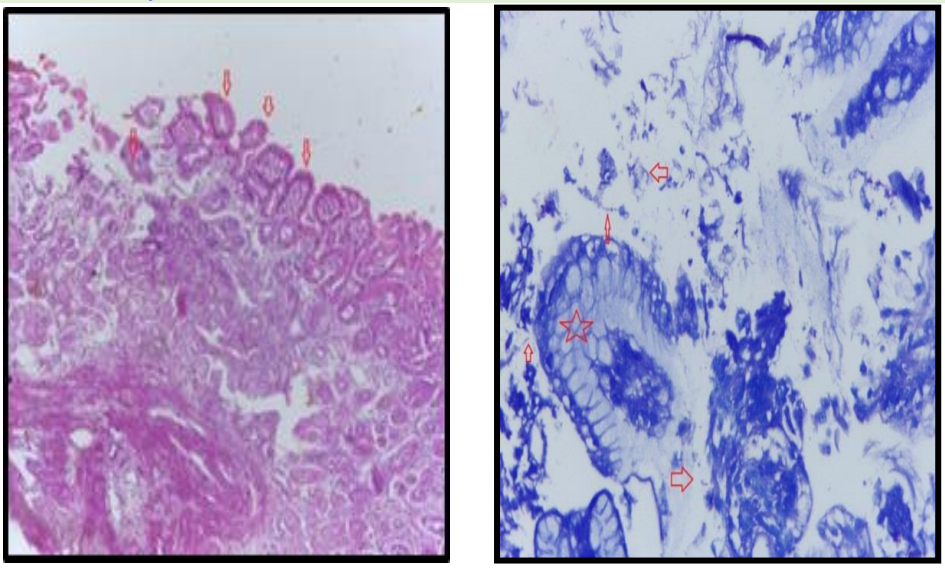
Degree of gastritis (endoscope) after treatment	Total		Degree of gastritis (histopathology) after treatment							
			Nil		Mild		Moderate		Severe	
	No	%	No	%	No	%	No	%	No	%
Nil	36	42.4%	19	22.4%	13	15.3%	4	4.7%	0	0%
Mild	45	52.9%	1	1.2%	41	48.2%	2	2.4%	1	1.2%
Moderate	4	4.7%	2	2.4%	1	1.2%	0	0%	1	1.2%
Severe	0	0%	0	0%	0	0%	0	0%	0	0%
Total	85	100%	22	25.9%	55	64.7%	6	7.1%	2	2.4%
Test[a]			11.987							
p-value (Sig.)			0.007 (S)							
tau[b]			+0.278							
Test			2.942							
p-value (Sig.)			0.003 (S)							

Categorical variables were expressed as number (percentage); a: Stuart-Maxwell test; b: Kendall's tau-c correlation coefficient; p-value < 0.05 is significant; Sig.: Significance.



(A) (H & E × 200). ***(B): High power view***

Figure 1: A case of eosinophilic gastritis showing mucous secreting glands surrounded by large number of eosinophils blue and red arrows



(A) (H & E × 200) *(B) (Gemisa stain × 400).*

Figure 2: A case of chronic gastritis showing **(A)** Intestinal metaplasia formed of numerous villi (red arrows) (Haematoxylin & Eosin × 200) and underlying gastric mucosa **(B)** Intestinal vilious (red star) and comma – shaped H. Pylori (red arrows) (Gemisa stain × 400).

DISCUSSION

This study of 85 patients with dyspeptic symptoms all were subjected to endoscopic examination & biopsies with follow up for at least 3 to 6 months.

Results of this study showed that there is:

A highly significant relationship between the degree of chronic gastritis in histopathology before and after treatment which revealed that 77.6% patients improved, 20% not changed; and 2.4% became severe.

A highly significant relationship between extent of chronic gastritis in histopathology before and after treatment which revealed that There was (63.4%) of 85 patients studied improved; 27.1% became the same (not changed); and only (9.5%) worsened.

High significant relationship between degree of gastritis by endoscopy (Modified Lanza) [12] before and after treatment among patients had a positive test for H. pylori in stool before treatment and became negative after treatment (N=36). It was noticed that 88.9% of cases improved; 11.1% not changed; and there were no cases became worsened.

Highly significant relationship between degree of gastritis (endoscopy) before and after treatment among patients had a positive H. pylorus in endoscopic biopsy before treatment and became negative after treatment (N=27). It was noticed that 81.4% of patients improved; 18.5% not changed; and there were no patients who became worsened.

A highly significant relationship between degree of gastritis (endoscopy) before and after treatment among patients had NSAID induced chronic gastritis (N=56). 84.1% of patients improved, 16.2% not changed, there were no cases became worsened.

chronic gastritis was noticed in a wide age group ranging from 18-77 years with a mean age of 41.7 years; most common age group was between (30 < 40). Female (65.9%) were more than males (34.1%) with ratio of 1.9:1 (Supplementary Table 3) This is agreed with Singh et al.., 2015 [16] who founded that the mean age was 42.9 years, patients ages ranged between 18 to 70 yrs. 31–40-year age group comprised of the maximum number of the cases which were 34.3% followed by 22.5% cases in 41-50year age group. Maximum number of cases both males and females were in the age group of 31-40 years and females: males= 1.6:1. However chronic gastritis was reported in an older age group in another previous study with a mean of 65.8 years and M: F ratio was 1:1.07 reported by Maharjan et al.., 2017 [17].

There is no significant relationship between symptoms of chronic gastritis and its degree by endoscopy. We found that the main symptoms were

epigastric pain present in 77 cases (90.6 %) followed by anemia present in 37 cases (43.5 %).

This study showed that the most common causes of chronic gastritis were reactive gastropathy especially (NSAIDS (65.9%) and H. Pylori induced gastritis (50.6% by serology and 51.8% by biopsy & histopathology) (Table 1) and this is similar to Wanjohi et al.., 2014 [6], at Kenyatta general hospital in Africa, who founded that the prevalence of H. Pylori in their study was 50%.

The present result showed that there is highly significant relationship between endoscopic mucosal abnormalities (97.6%) and erosion (44.7%) (P-value was <0.001) and significant relationship with presence of mucosal erythema (78.8%), (p-value 0.015) has been observed according to classification of severity of chronic gastritis endoscopically by Modified Lanza [13], furthermore; there was non-significant relationship between other endoscopic findings as nodular mucosa (15.3%); gastric ulcer (10.6%) and / or atrophic gastric mucosa (9.4%). However duodenal ulcer was present in 11.8%, duodenitis was detected in 48.24% of the studied populations.

Antral gastritis and antro-corpal gastritis present in (55.3%) which is near to the study done by Hussein, 2019 [18] where antral gastritis present in (66%), this is comparable to another study done by Hassan et al.., 2016 [19], where antral gastritis represented the main form of gastritis.

Histological examination of gastric mucosal biopsies before treatment showed histologic evidence of: Chronic inflammation present in (100%) of cases; mild mononuclear infilterate 37.6% cases; moderate 47.1% cases and severe 15.3% cases. This is agreement with Hussein, 2019 [16] and Garg et al.., 2012 [2] who reported that mononuclear cell infiltration formed the major histological variable in study, since it was encountered in all cases (100%). While neutrophil activation is present in (88.2%) cases. This agrees with Hussein, 2019 [16] who reported that neutrophilic infiltration in the lamina propria or inside the glandular lamina was observed in (84%) of cases and, our results are in line with the study conducted by Park et al, 1995 [18] in which the result was (78.7%). However, the result in the present study outnumbered that observed in a previous one which showed only (33.6%) of cases having neutrophilic activity by Maharjan et al.., 2017. [15] This difference is attributed to multiple etiologic factors of chronic gastritis in this study, recent endoscopic advances and better visualization of gastric mucosa, number of gastric biopsies between different studies Moreover, atrophic changes of the mucosa were found in (14.1%). This is relatively lower than Hussein, 2019 [18] study which showed atrophy in (22%) of cases and in agreement with study of Garg et al.., 2012 [2] which showed atrophy in (12.3%) of cases. This variability between studies is explained by the more gastric biopsies, the more detection of intestinal metaplasia and, because of recent advances of endoscopy and better endoscopic visualization, and the more chronicity(duration)of symptoms the more detection of atrophic changes.

Intestinal metaplasia (Figure 2A) was encountered in (10.6%) which is relatively lower than the study of Hassawi et al.., 2015 [21] study which recorded this change in 15% of cases and the study of Hussein, 2019 [18] who reported intestinal metaplasia in (14%) of cases. However, our result is higher than that recorded by Dhakhwa et al., 2012 [22] in which intestinal metaplasia was found in only 5% of cases. On the other hand, a higher rate was found in the study of Al- Nuaimy and Faisal, 2019 [23] in which 23% of cases showed intestinal metaplasia. This variability between studies is explained by the more gastric biopsies, the more detection of intestinal metaplasia cases.

Lymphoid follicle formation was founded in (20%) of subjects. Hussein, 2019 [15] reported Lymphoid aggregates in 25% and lymphoid follicles identified in 19% of subjects. This is attributed to H pylori infection which was presented in (51.8%).

Surface epithelium damage presented in (94.1%) which is in agreement with the study conducted by Hussein, 2019 [16] who observed surface epithelial damage in (98.67%).

H. pylori (Figure 2B) intensity presented in (51.8%) and there is highly significant relationship between degree of gastritis by endoscope and H. Pylori intensity (P value 0.016).

During this study follow up (Table: 2 in supplementary file) after treatment endoscopic and histopathologic biopsies showed that chronic inflammation is present in (74.1%) of cases. Neutrophil activation present in (69.4%) of cases. Surface epithelium damage present in (56.5%) of cases. Glandular atrophy present in (11.1%) of cases. Intestinal metaplasia present in (4.7%) of cases. H. pylori intensity present in (4.7%) of cases.

Treatment of chronic gastritis improves gastric mucosal inflammation, atrophy, and intestinal

metaplasia and prevents its progression, this agrees with Lu et al, 2005 [12] who founded that treatment of H. pylori may improve gastric mucosal inflammation, atrophy, and prevent the progression of intestinal metaplasia".

Limitations of the study:

This interventional study of chronic gastritis natural history, correlation of endoscopic and histopathologic findings and follow up the treatment effects are points of strength but some patients were non -compliance (7 patients) and patients improved on treatment and refused the follow up (20 patients).

CONCLUSION AND RECOMMENDATION

Chronic gastritis is a common disease that affects different age groups. NSAIDS induced chronic gastritis is the most common cause of chronic gastritis, so NSAIDS should always be used cautiously, for the shortest possible time and at the lowest effective dose. H Pylori is also a common cause of chronic gastritis so preventive measures and treatment regimens should be applied to eradicate H. Pylori.

Routine biopsies are recommended to disclose degree of gastritis, possible causes, any degree of dysplasia or mucosal atrophy. Further long term follows up, 5-6 large scale studies (multicenter 5: 10 years clinical trial) to observe the natural history of chronic gastritis. Rationalization of NSAIDS and recommend PPI or H2 receptor blockers when we are forced to use NSAIDS. All heavy smoker patients with chronic gastritis are liable to have intestinal metaplasia and need to be followed up to prevent stomach cancer.

Conflict of Interest: None.

Financial Disclosure: None.

ACKNOWLEDGEMENT

For all professors of internal medicine departments and hepatogasteroenterology units at both Zagazig University Hospitals and MTH (Mataryea Teaching Hospital, especially prof. Dr. Tarek Ahmad Fouad).

REFERENCES

1- **Rugge MA, Pennelli GI, Pilozzi EM, Fassan MA, Ingravallo GI, Russo VA, et. al.** Gastritis: The histology report. Digestive and liver disease, 2011; 43S:S373-S384.Garg BH, Sandhu VI and SOOD NE, Sood AJ, Malhotra VI. Histopathological analysis of chronic gastritis and correlation of pathological features with each other and with endoscopic findings. J Pathol. 2012; 3: 172- 8.

2- **Alboraie M, Elhossary W, Aly OA, Abbas B, Abdelsalam L, Ghaith D et al.,** Egyptian recommendations for management of Helicobacter pylori infection: 2018 report, Arab Journal of Gastroenterology.

3- **Wadea FM, El-Hawary AT.** Demographic, clinical and endoscopic characteristics of active antibiotic resistant H. pylori associated gastritis in Egyptian adults, Journal of clinical and diagnostic research 2018, V 12 (10) OC32-7.

4- **Zullo AN, Hassan CE and Romiti AD, Giusto MI, Guerriero GA, Lorenzetti RO et al.** Follow-up of intestinal metaplasia in the stomach: When, how and why World J Gastro-intest. Oncol. 2012;15; V :(4) I :(3): Pages: 30- 6.

4- **Wanjohi WH, Ogutu EM, Oyoo GO. Radia KH, Mutei.TM,** Prevalence of gastro-duodenal lesions in chronic non-steroidal anti-inflammatory drug users presenting with dyspepsia at the Kenyatta National Hospital. Afr J Rheumatol 2014; 2(1): 29-34.

5- **Tongtawee TA, Kaewpitoon SO and Kaewpitoon NA, Dechsukhum CH, Loyd RA, and Matrakool LI.** Correlation between Gastric Morphologic Patterns and Histopathological Severity of Helicobacter pylori Associated Gastritis Using Conventional Narrow Band Imaging Gastroscopy. BioMed Research International 2015; (v) Article ID 808505, 7 pages.

6- **Uppal VI, Kreiger PO and Kutsch ER.** Eosinophilic gastroenteritis and colitis: a comprehensive review. Clin Rev Allergy Immunol.2016; 50(2):175-88 (ISSN: 1559-0267).Vol. 19, No. 1, p: 47-55.

7- **Carmack SW, Lash RH, Gulizia JM& Genta RM.** Lymphocytic disorders of the gastrointestinal tract: a review for the practicing pathologist. Adv Anat Pathol. 2009; 16(5):290-306.

8- **Ribeiro VL, Barbosa AJ and Barros CA.** Varioliform gastritis: frequency and relationship with lymphocytic gastritis. Arq Gastroenterol. 2000; 37(1):7-12

9- **Andrews TI and Campbell FI. Types of Gastritis in R. Feakins (Ed.),** Non-Neoplastic Pathology of the Gastrointestinal Tract: A Practical Guide to Biopsy Diagnosis. Cambridge University Press 2020; Chapter 13 (pp. 186-205). doi:10.1017/9781316823026.014.

10- **Lu Ho, Yamaoka Yo and Graham DY.** Helicobacter pylori virulence factors: facts and fantasies. Curr Opin Gastroenterol ; 2005; 21: 653-9.

11- **Shim KN, Kim JI, Kim NA, Kim GS, Jo YJ, Hong SJ, et al.** The efficacy and safety of irsogladine maleate in nonsteroidal anti-inflammatory drug or aspirin-induced peptic ulcer and gastritis. Korean J Intern Med 2019; 34:1008-1021.

12- **Aydin OZ, Egilmez RE, Karabacak TU, and Kanik AR.** Inter-observer variation in histopathological assessment of Helicobacter pylori gastritis. World J Gastroenterol 2003; 9: 2232- 5.

13- Chey WD, Leontiadis GI, Howden CW, and Moss SV. ACG Clinical Guideline: Treatment of Helicobacter pylori Infection. Am J Gastroenterol 2017; V; 112: Page: 212–238.

14- Singh PR, Goswami KC and Gupta BB. Gastric mucosal biopsies in non ulcer dyspepsia: A histopathologic study. Asian journal of medical sciences 2016; Pages: Vol 7 | Issue 2 80- 4.

15- Maharjan S, Ranabhat SK and Tiwari M, Aryal G, Rauniyar SK, Shrestha HG. Helicobacter pylori associated chronic gastritis and application of visual analogue scale for the grading of the histopathological parameters in Nepal. Biomed J Sci & Tech Res 2017; 1: 28 –34.

16- Hussein HA. Evaluation of Chronic Gastritis in Endoscopic Antral Biopsies Using the Up-dated Sydney System. Ann Coll Med Mosul 2019; 41 (2):95-105).

17- Hassan TM, Al-Najjar SI, Al-Zahrani IH. Alanazi FIB, Alotibi MG. Helicobacter pylori chronic gastritis updated Sydney grading in relation to endoscopic findings and H. pylori IgG antibody: diagnostic methods. J Microsc Ultrastruct 2016; 4: 167 –74.

18- Park J, Kim MK and Park SM. Influence of Helicobacter pylori colonization on histological grading of chronic gastritis in Korean patients with peptic ulcer. Korean J Intern Med. 1995; 10: 125 – 9.

19- Hassawi BA, Khalil KH, Sharafadin ZA. Prevalence of intestinal metaplasia and dysplasia in infectious and non-infectious chronic gastritis. Int J Res Med Sci. 2015; 3: 2228 – 31.

20- Dhakhwa R, Acharya IL, Shrestha HG, Joshi DM, Lama S, Lakhey M. Histopathologic study of chronic antral gastritis. J Nepal Health Res Counc 2012; 10: 57 – 60.

21- Al- Nuaimy WM and Faisal HM. Endoscopical and histopathological interpretation of gastritis in Nineveh Province. Annals of the college of medicine, Mosul, 2019; Volume 41, Issue 1 , pages 28- 35.

To Cite:

Tawfeek Ibrahim, M., Gumie, M., Ismail, W., El Kashishy, K. Role of upper GIT endoscopy and histopathology in diagnosis and follow up of chronic gastritis. *Zagazig University Medical Journal*, 2023; (1018-1034): -. doi: 10.21608/zumj.2022.112837.2443

SUPPLEMENTARY TABLES AND FIGURES

Table S1: Endoscopic scoring of gastric mucosal lesion and mucosal injuries modified Lanza

Gastric mucosal lesion	
Modified Lanza score	
0	No visible lesion
1	Mucosal hemorrhage only
2	One or two erosions
3	Numerous (3-10) numbers of erosions
4	Large (>10) numbers of erosions
5	Ulcer
Gastric mucosal injuries	
Edema	
1	No edema
2	Mucosa is somewhat pale, the white prominence, and the hexagonal gastric pit becomes prominent.
Redness	
1	No redness
2	Mild reddish change
3	More prominent reddish color change
4	Beefy reddish color change
Hemorrhage	
1	No hemorrhage
2	Single hemorrhagic lesion
3	2-5 hemorrhagic lesions
4	6-10 hemorrhagic lesions
5	>10 hemorrhagic lesions or larger area of a Confluent hemorrhage

Table S2: Sydney system for grading of chronic gastritis

Feature	Definition	Grading guidelines
Chronic inflammation	• Increased lymphocytes and plasma cells in lamina propria	• Mild, moderate or severe increase in density
Activity	• Neutrophilic infiltrates of the lamina propria, pits or surface epithelium	• Mild: less than one-third of pits and surface infiltrated • Moderate: one-third to two-thirds • Severe: more than two-thirds
Atrophy	• Loss of specialized glands from either antrum or corpus	• Mild, moderate, or severe loss
Helicobacter pylori	• *H. pylori* density	• Mild colonization: scattered organisms covering less than one-third of the surface • Moderate colonization: intermediate numbers • Severe colonization: large clusters or a continuous layer over two-thirds of surface

Feature	Definition	Grading guidelines
Intestinal Metaplasia	• Intestinal metaplasia of the epithelium	• Mild: less than one-third of mucosa involved • Moderate: one-third to two-thirds • Severe: more than two-thirds

Table S3: Characteristics of the studied 85 population of chronic gastritis according to Age, gender and Duration of symptoms (subgroups)

	The studied chronic gastritis patients (N=85)	
	No.	%
Age		
<30 years	17	20%
30-<40 years	24	28.2%
40-<50 years	15	17.6%
50-60 years	16	18.8%
>60 years	13	15.3%
Gender		
Male	29	34.1%
Female	56	65.9%
Duration of symptoms		
<1 year	26	30.6%
1 - <2 years	34	40%
2- <3 years	10	11.8%
3-5 years	9	10.6%
>5 years	6	7.1%

Categorical variables were expressed as number (percentage).

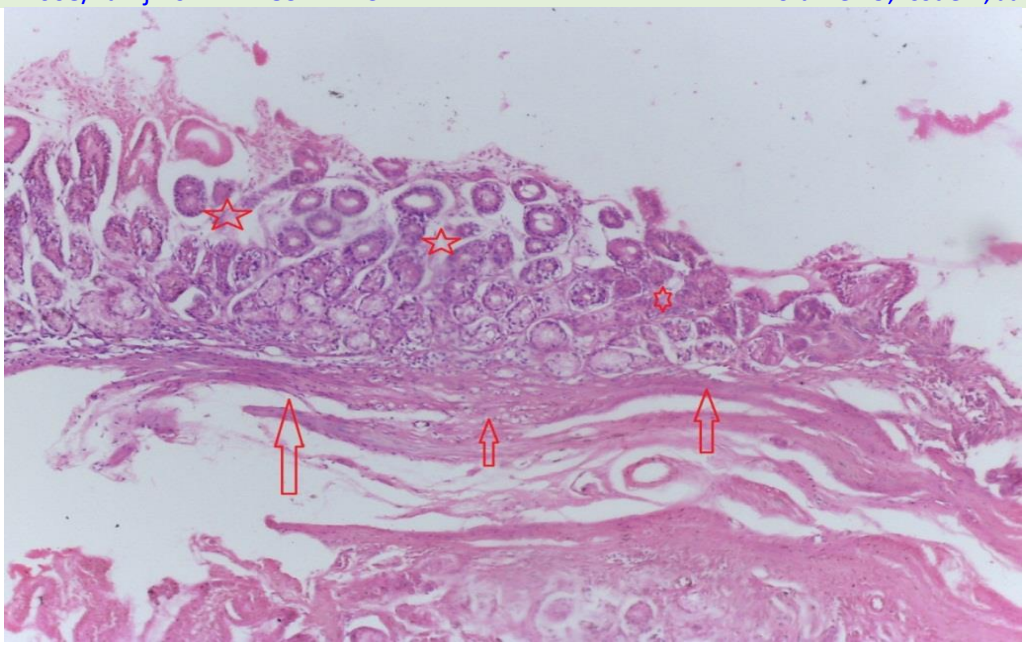


Figure S1: Collagenous gastritis showing atrophic mucosa (red star) with underlying bundles of collagen showed by red arrows (H & E × 200)

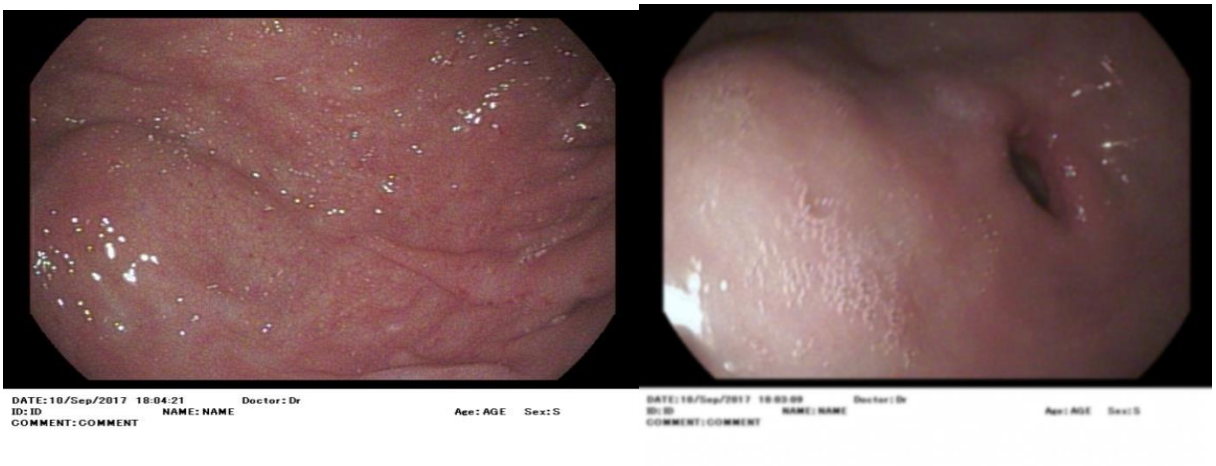


Figure: 2a **Figure: 2b**

Figure S2: NSAIDS induced gastritis: **(a)** Antro-corpal gastritis: Muco-erythema of the Antral canal with pre-pyloric superficial ulcer, mucosal edema **(b)** Muco-erythema with scattered hemorrhagic dots > 10), Modified Lanza score: severe gastritis

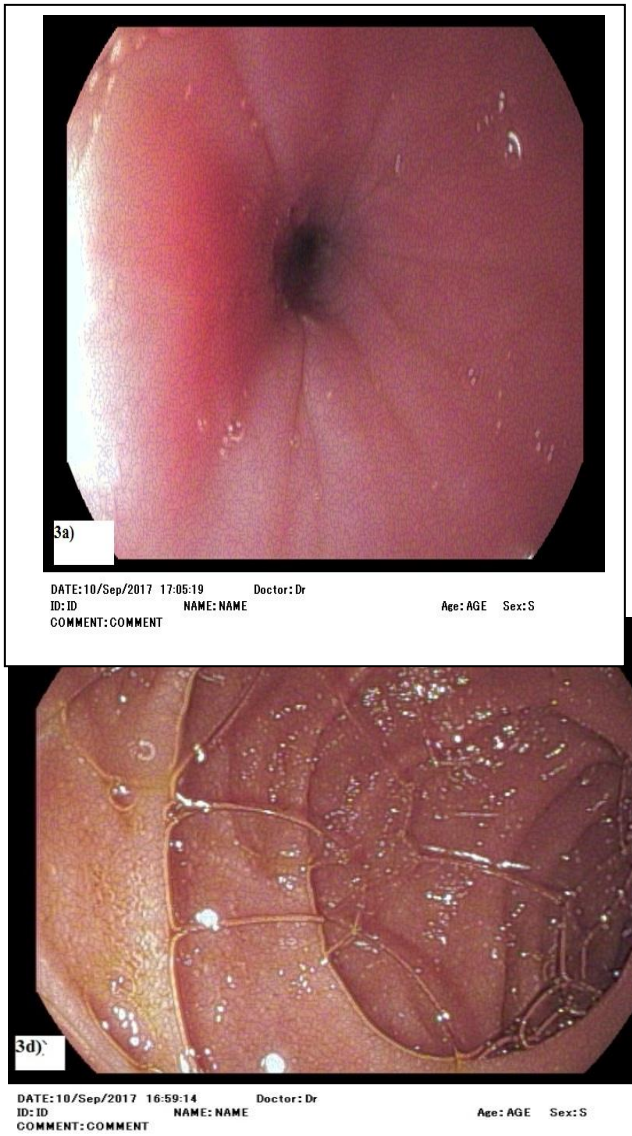


Figure S3C

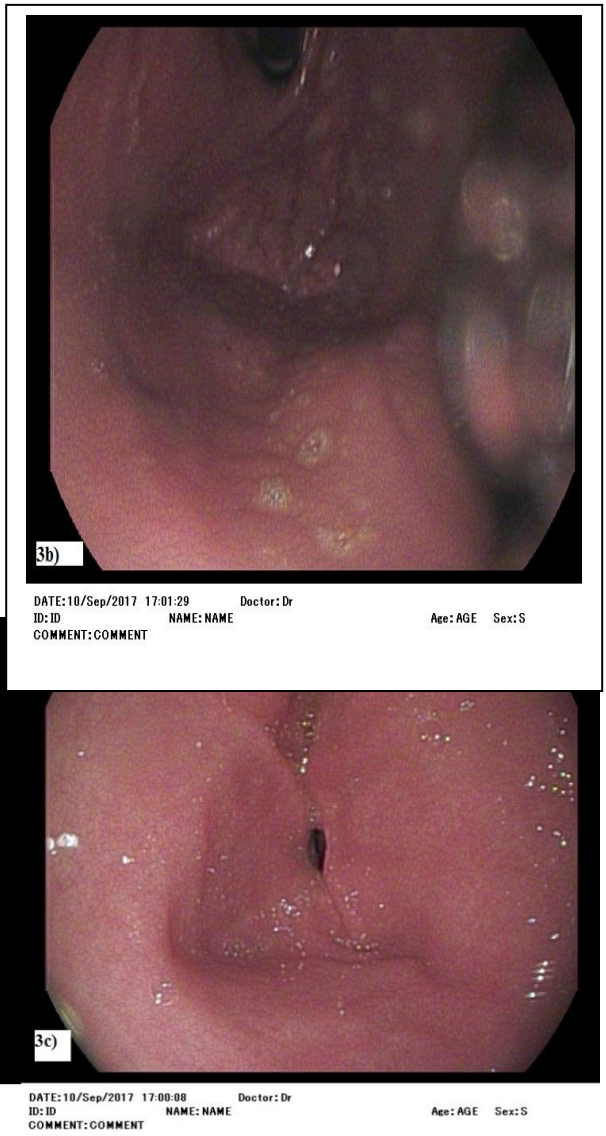


Figure S3d

Figure S3: Helicobacter pylori chronic gastritis **(a)** Esophagus was normal mucosa, no Hiatus Hernia or GERD (Gastero-Esophageal Reflux Disease) **(b)** Atrophic mucosa of the fundus of the stomach appearance of blood vessels in the fundus on endoscope retroversion. **(c):** Mild Muco- erythema of the antral canal and pre-pyloric region **(d):** Normal duodenum up to 2nd part. Modified Lanza score: Mild gastritis

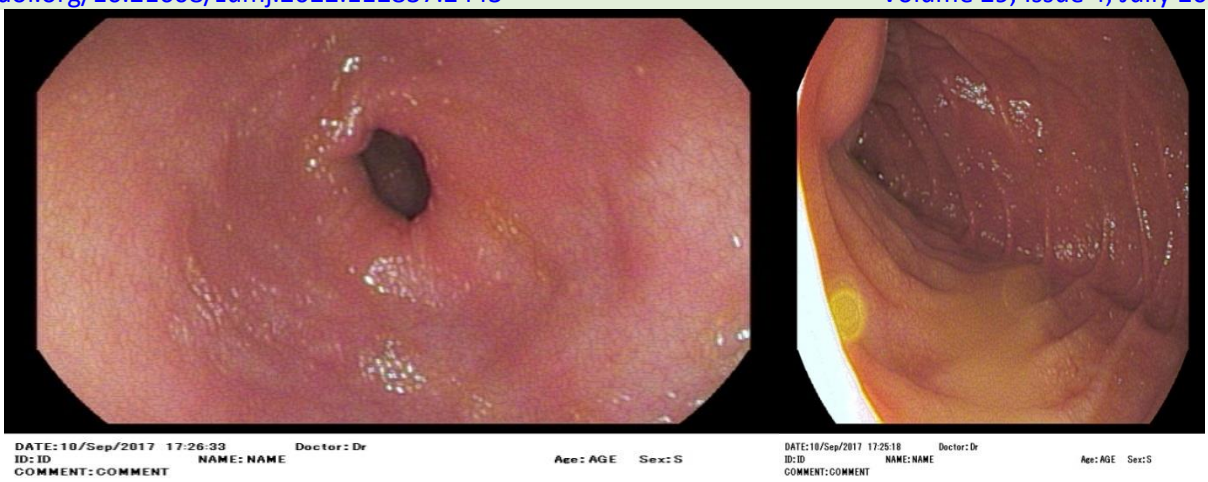


Figure: 4a ***Figure: 4b***

Figure S4: Bile reflux gastritis Antro-corpal gastritis): (a): duodenum 2nd part showing biliary juice (b): Antro-corpal billiry gastritis. Modified Lanza score: moderate gastritis

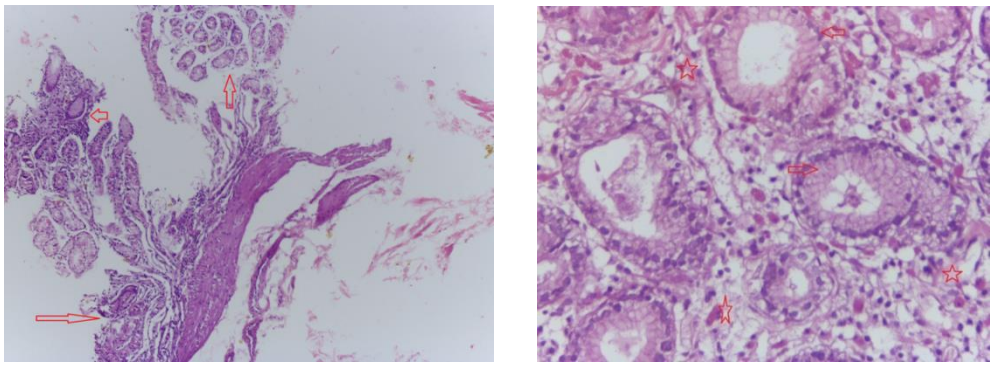
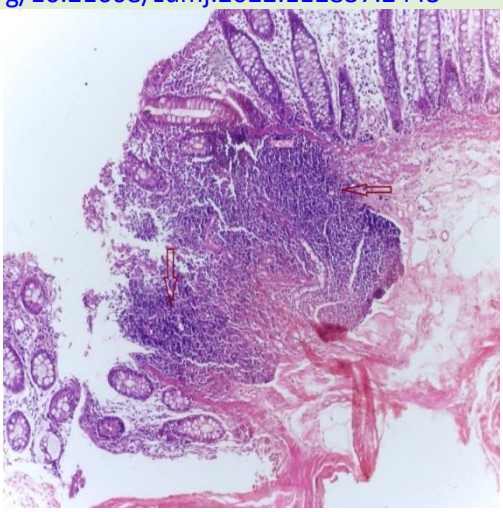


Figure a ***Figure b***

Figure S5: (a): A case of chronic atrophic gastritis showing few gastric mucosal glands (red arrow) surrounded by aggregates of inflammatory cells. (H & E × 200). **(b):** High Power view of the same case showing few mucus glands (red arrows) and relative increase in the stroma red stars containing mild inflammatory infiltrate (H & E × 400).

Parameters of Sydney system: Chronic inflammatory cells (mild); Neutrophilic activity (mild); H. pylori intensity (nil),) Atrophic mucosa (moderate) Intestinal metaplasia (nil), Surface epithelial erosion (mild) So, overall assessment mild gastritis



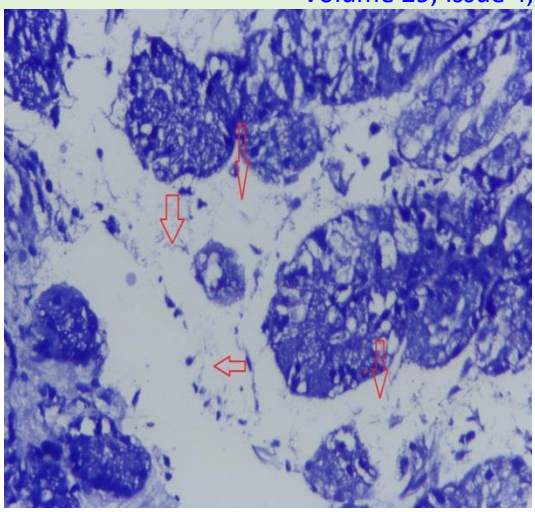
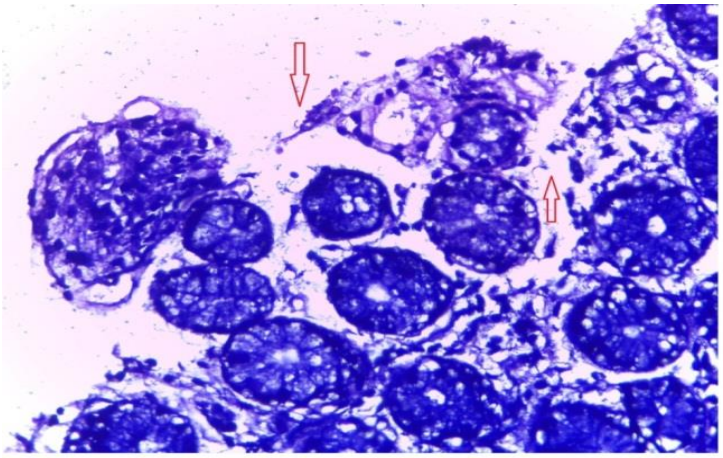


Figure: 6a ***Figure: 6b***

Figure S6: (a): A case (of chronic active gastritis showing ulcerated mucosa and enlarged lymphoid follicles (red arrows) in the lamina propria (H & E × 200), **(b):** the same case (b) stained by Geimsa stain showing numerous comma shaped H. Pylori red arrows on the mucosal surface (Geimsa stain × 400).

Sydney system parameters: Chronic inflammatory cells (severe), Neutrophilic infiltration (moderate), H. pylori intensity (severe), Surface epithelial erosion (moderate), Atrophy (nil), Intestinal metaplasia (nil) So, Overall assessment= Severe gastritis



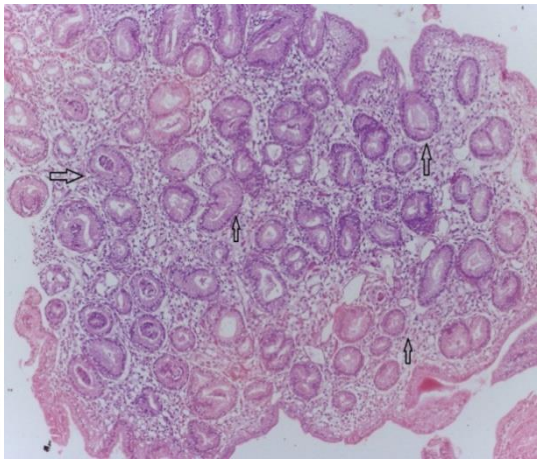


Figure: 7a ***Figure: 7b***

Figure S7: (a): A case of chronic superficial gastritis formed of uniform round mucus glands (black arrows) surrounded by aggregates of chronic inflammatory cells (Haematoxylin & Eosin × 200) **(b):** Case stained by Giemsa stain showing comma- shape H. pylori pylori ↑ red arrows on the mucosal surface (Giemsa stain × 400).

Sydney system parameters: Chronic inflammatory cells (moderate); Neutrophilic infiltration (moderate); H. pylori intensity (moderate); Atrophy (nil); Intestinal metaplasia (nil). Overall assessment = moderate gastritis