International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Index Copernicus Value (2016): 79.57 | Impact Factor (2017): 7.296

Histopatological Profile in Patient with Chronic Gastritis Who Underwent EGD in Wangaya Hospital Using Sydney System

Matius Daniel[1], Suwandi Mario[2]

General Practitioner, Wangaya Regional Public Hospital, Bali, Indonesia

Abstract: ***Gastritis is an inflammation of the lining of the gastric mucosa and submucosal. The Sydney system classified gastritis with three combination histological division : etiology, topography, and morphology. The aim of this study to show thehistopatologic profilein dyspepsia patient who went esophagogastroduodenoscopy in Wangaya Hospital with Sydney System.This descriptive study conducted on 200 patient with dyspepsia syndrome between January 2018 and June 2018 who doesn't respond with PPI therapy for 4 weeks. All patient underwent esophagogastroduodenoscopy and biopsies were taken on antrum, and corpus using Hematoxylin and Eosin, and Giemsa stain for grading of various parameters and for detection of HP. Our research show that H.Pylori can be one of the cause of dyspepsia in 27 patient (13.5%) ,which antrum is the most 24 patients (88%). The most common age group affected by H Pylori was 41- 50 years constituting 29,6 % of cases.The majority (67.56%) of the biopsies showed marked inflammation; 28.2% had moderate inflammation and there is no atrophy in this study (0%). In H Pylori infection correlated to marked inflammation, high activity of neutrophil and no atrophy process.The esophagogastroduodenoscopy needs to be done in patients with dyspepsia symptoms that untreatable with PPI therapy.***

Keywords: Esophagogastroduodenoscopy, Sydney System, Helicobacter Pylori, Dyspepsia, Histopatology

1. Introduction

Dyspepsia defined as any symptomps referable to the upper gastrointestinal tract such as epigastric fullness, nausea, vomiting or heartburn. Dyspepsia may be classified as: organic dyspepsia, functional dyspepsia, drug related dyspepsia and dyspepsia from extraintestinal systemic disease.[7] Dyspepsia can be related to chronic gastritis.

Gastritis is classified into acute and chronic gastritis. Chronic gastritis is divided into nonatrophic chronic gastritis, usually caused by Helicobacter pylori infection, and atrophic gastritis composed of autoimmune and multifocal atrophic gastritis caused by Hp or dietary factors, as well as special forms of gastritis composed of reactive (chemical, reflux), radiation, lymphocytic, non-infectious granulomatous, eosinophylic and other infectious gastritides.[18]

Chronic gastritis is an inflammatory condition of the gastric mucosa characterized by elementary lesions whose extent and distribution are related to their etiology and host responses. Infection with Helicobacter pylori is by far the most common cause of chronic active gastritis worldwide; chemical agents and autoimmune phenomena account for a small proportion of chronic, usually nonactivegastritides. Chronic gastritis is epidemiologically and biologically linked to the development of gastric cancerand H pylori is listed as a class I carcinogen .

2. Material and Method

2.1 Patient selection

The sample was taken on January 2018 to June 2018, 200 patients were selected from esophagogastroduodenoscopy clinic, Wangaya Hospital, Denpasar, Bali. These patients had symptomatic dyspepsia who doesn't respond with PPI therapy for 4 weeks. The patients were examined clinically, and various routine laboratory investigations were done, including complete blood count,,liver and renal function tests, and bleeding and coagulation assessment. Then, the patients underwent esophagogastroduodenoscopy . All the patients gave signed informed consent for biopsies.

2.2 Esophagogastroduodenoscopy

EGD was performed in all our patients in the esophagogastroduodenoscopy section using Fujifilm (Fujinon EPX - 2500, and colonoscope type EG – 530 NW). A minimum two gastric mucosal tissue biopsies (1 each from the antrum and corpus) were taken. All the patients were examined for findings suggestive of endoscopic gastritis, such as erythema, hyperemia, atrophy, and mucosal nodularity according to the criteria of the Sydney grading system. Additionally, they were examined for the presence of gastric erosion and ulceration. Also,they were evaluated for the anatomical location of gastritis,includingantrum, antrum predominant pangastritis, and corpus predominant gastritis.

2.3 Histopatology

Gastric biopsies from the antrum and corpus were paraffin embedded, sectioned at 2 mm and stained with haematoxylin and eosin. Giemsa stained slides used to see density of *H. pylori* colonizationThe grades of the major morphological variables [*H. pylori* density, neutrophilic activity, mononuclear cells (MNC) infiltration, IM, and glandular atrophy] were determined using the semi-quantitative method of scoring according scored 0-3 (absent, mild, moderate or marked) to each morphological variable. All biopsies was examined by one pathologist doctor.

The following histological features were examined on each slide: density of *H pylori*, neutrofil activity, chronic

inflammation, atrophy, and intestinal metaplasia. According to the histological criteria of the updated Sydney-system, activity of chronic inflammation were scored by using a scale from normal or absent (score 0), to slight (score 1), moderate (score 2) and severe (score 3) change or increase. Atrophy was scored as absent (score 0), slight (score 1), moderate (score 2) and severe (score 3). The presence of neutofil was scored as absent (score 0), slight (score 1), moderate (score 2) and severe (score 3). The presence and severity of intraepithelial neoplasia (dysplasia) were assessed as absent (score 0), mild (score 1), moderate (score 2) and severe (score 3)

H Pylori density were scored by using a scale as absent (score 0), mild (single cell dropouts present; score 1), moderate (dropouts of several cells; score 2) or true erosions (score 3)

2.4 Statisical Analysis

This study is using the descriptive study.

3. Results

3.1 Clinicoendoscopic finding

The patient's age ranged from 20 years to 80 years with an average of 48 years old, 93 patients were male and 107 were female. All patient had heartburn, nausea and epigastricpain, which did not respond to normal treatment. EGD revealed features of endoscopic gastritis in 200 patient. We analyzed 200 cases of gastric endoscopic biopsies.

The result was from 200 dyspepsia patient who underwent EGD, 27 person (13.5%) was infected by H.Pylori. There were 13 (48%) female and 14 (51%) male patients with only a slight predominance of male patients showing a ratio of 1.077 : 1.The most common age group affected by H Pylori was 41- 50 years constituting 29,6 % of cases, followed by 22,2% cases in 61-70 years, and 18,5 % in 51 – 60 years. Chronic gastritis was widely distributed among age group ranging from 20-82 years with a mean age of 50 years.

Table 1: Age of the patients and their H. pylori status in chronic gastritis

Age group(years)	*H.pylori*positive (n)	*H.pylori*negative	n (%)
11 – 20	-	7	0
21 – 30	2	13	7,4
31 – 40	4	32	14,8
41 – 50	8	46	29,6
51 – 60	5	35	18,5
61 – 70	6	21	22,2
71 – 80	2	15	7,4
81 – 90	-	4	0
Total	**27**	**173**	**200**

3.2 Histopatological finding

Table 2: Histopatological grading of chronic gastritis according Sydney Grading System

Histological Variables	None	Mild	Moderate	Severe
Chronic Inflammation	**26 (13%)**	**45 (22.5%)**	**92 (46%)**	**37 (18.5%)**
Neutrophilic infiltrate	**167 (83.5%)**	**23 (11.5%)**	**10 (5%)**	-
Atrophy	**200 (100%)**	-	-	-
Intestinal metaplasia	**198 (99%)**	**1 (0.5%)**	**1 (0.5%)**	-
HP Density	**173 (86.5%)**	**26 (13%)**	**1 (0.5%)**	-

Endoscopic findings H Pylori were available in 27 (13.5%) cases. Antrum was the most common site, comprising 23 (85.1%) cases among which 1 (3.7%) cases were affected in corpus. One (3.7%) lesions were found in antrum and corpus, 2 lesion (7.4%) cases had detected in gaster

Table 3: Association of HP and degree of chronic inflammation in chronic gastritis

Chronic Inflammation	No. of cases	HP positive	%
Mild	45	-	-
Moderate	92	2	2.17
Severe	37	25	67.56

Association HP with degree of chronic inflammation has found 2.17% of cases with moderate chronic inflammation, and 67.56% of cases with severe chronic inflammation.

Table 4: Association of HP and degree of neutrophil activity in chronic gastritis

Neutrophil Activity	No. of cases	HP positive	%
Mild	22	18	81.8
Moderate	10	9	90
Severe	-	-	

H. pylori was detected in 81.8% of cases with mild neutrophilic activity, and 90% of cases with moderate neutrophilic activity.

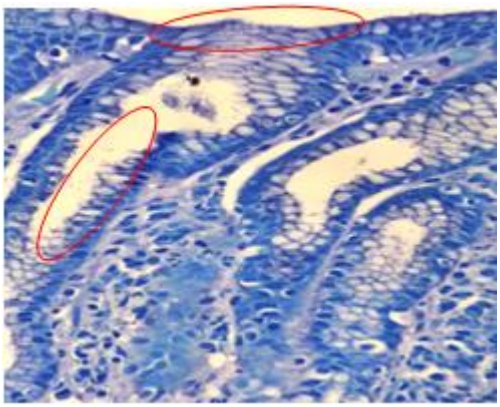

Figure 1: Photomicrograph of gastric biopsy showing Helicobacter pylori (Giemsa stain). Magnification 1000x

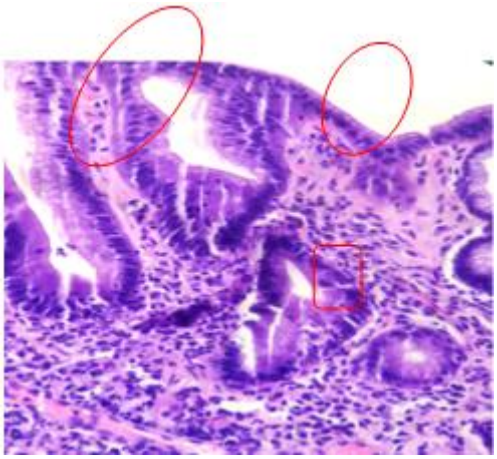

Figure 2: Photomicrograph of gastric biopsy showing lymphoid aggregates and polymorphonuclear intra epitel. Maginification 400X (H&E)

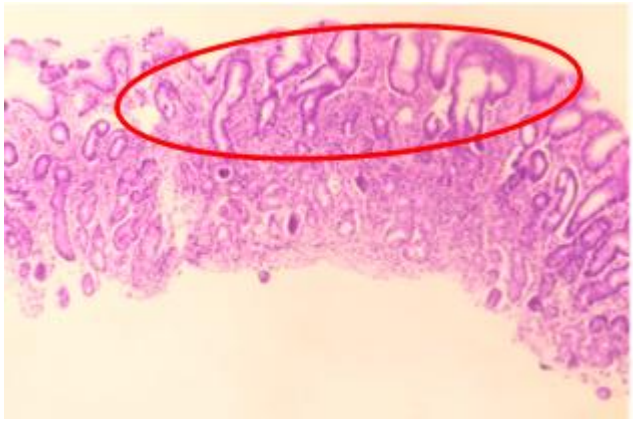

Figure 3: Photomicrograph of gastric biopsy showing no focal atrophy and intestinal metaplasia. Maginification 200X (H&E)

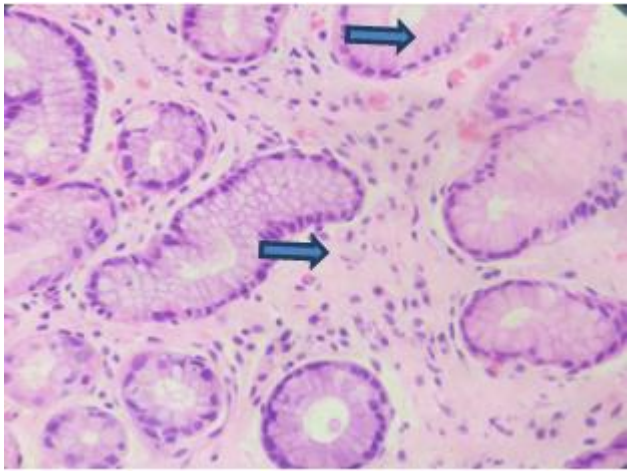

Figure 4: Photomicrograph of gastric biopsy showing aggregated lymphoid 200X (H&E)

4. Discussion

The phenotypes of gastritis[18]

Chronic gastritis can be atrophic or nonatrophic. Each of these two main categories encompasses several clinicopathologic entities with different patterns of inflammatory and epithelial alterations.

4.1. Non-atrophic gastritis

4.1.1. Antral-predominant non-atrophic gastritis

This pattern is the most common expression of H pylori gastritis. It is characterized by absence of atrophy, a moderately to severely inflamed antrum and a normal to mildly inflamed corpus. This condition is associated with either normal or increased acid secretion. Most patients with antrum-predominant gastritis experience no symptoms; they do, however, have an estimated lifetime risk of duodenal ulcer of ~20%, and possibly a minimally increased risk of adenocarcinoma of the distal stomach when compared to the noninfected population

4.1.2. Nonatrophicpangastritis

In some subjects infected with H pylori, marked inflammation is distributed throughout the stomach, with little or no difference between antrum and corpus. Particularly frequent in poorly sanitized areas where H pylori is highly endemic, pangastritis is widely believed to be the background on which atrophy eventually develops. Although this hypothesis is founded on reasonable grounds, we are not aware of any studies that substantiate this proposition.

4.2. Atrophic chronic gastritis

Gastric mucosal atrophy is defined as the loss of appropriate glands. This loss occurs when glands damaged by inflammation are replaced either by connective tissue (scarring) or by glandular structures inappropriate for location (metaplasia). Most often, the metaplastic transformation assumes the phenotype of the glands lined by intestinal-type epithelium (IM), but in the oxyntic mucosa, it may also take the form of mucin-secreting antral glands (pseudopyloric metaplasia). The histological criteria for scoring atrophic-metaplastic changes in both the antral and oxyntic mucosa have been extensively described, and visual analogue scales have been proposed as a reference standard.

4.2.1. Antrum-restricted atrophic gastritis

In the Western world, when atrophy is detected in biopsy specimens from dyspeptic patients, it is most frequently located in the antral biopsy samples. In such patients, atrophic-metaplastic changes are the consequence of current (or past) H pylori infection. The biopsy set will show patchy metaplastic atrophy restricted to the distal mucin-secreting mucosa (including the incisuraangularis) coexisting with moderate to severe inflammation and a normal or mildly inflamed corpus, with no atrophic changes.

4.2.2. Corpus-restricted (corpus-predominant) atrophic gastritis

In the oxyntic mucosa, atrophic-metaplastic changes can be detected in the absence of any coexisting atrophic changes of the distal stomach or in association with atrophic foci of the antral mucosa. The former condition is considered virtually pathognomonic of an autoimmune etiology and it is associated with an increased cancer risk.

4.2.3. Multifocal atrophic gastritis

Multifocal atrophic gastritis (MAG) is most prevalent in populations that are (or have been until recently) living in

suboptimal sanitary conditions, including much of southern and eastern Asia, Latin America, and several parts of central, eastern, and southern Europe. Socioeconomic factors may be a surrogate for other unknown agents or biological situations that modulate the evolution of gastritis, because there are notable epidemiological exceptions to this association. Atrophic gastritis is a risk factor for gastric noninvasive neoplasia (dysplasia) and intestinal-type adenocarcinoma. It also predisposes to gastric ulcer. In MAG, biopsy specimens show foci of atrophic-metaplastic changes in both antral and corpus mucosa. In contrast to antrumrestricted atrophic gastritis, MAG may display severe inflammation in the oxyntic mucosa, and acid secretion may be reduced, suggesting a more advanced disease.

4.2.4. Atrophic pangastritis

Atrophic pangastritis is likely to represent an advanced stage of MAG, with which it shares the epidemiological characteristics. Atrophic pangastritis is the most prevalent setting for both noninvasive and invasive gastric neoplasia.

The Sydney System

The Sydney system is a novel classification and grading of gastritis that was devised by a group of experts at the 9th World Congress of Gastroenterology in Sydney, Australia in 1990. This system is combining topographical, morphological and etiological information for the diagnostic evaluation of gastritis, [6]The Topography of the gastritis was considered the core of the classification. This classified gastritis restricted to the antrum, corpus, or a pangastritis. The etiology of gastritis, if known, was to be added as a prefix (e.g. *H. pylori*antral gastritis; autoimmune corpus gastritis, etc.). As suffix, phrases any of five key graded morphological variables were to be included. These were chronic inflammation (chronic gastritis) the activity of the gastritis measured by the presence of polymorphonuclear leucocytes alongside the mononuclear inflammatory infiltrate intestinal metaplasia (IM) atrophy manifest by the loss of the normal mucosal glands, and the presence of *H. pylori* organisms.

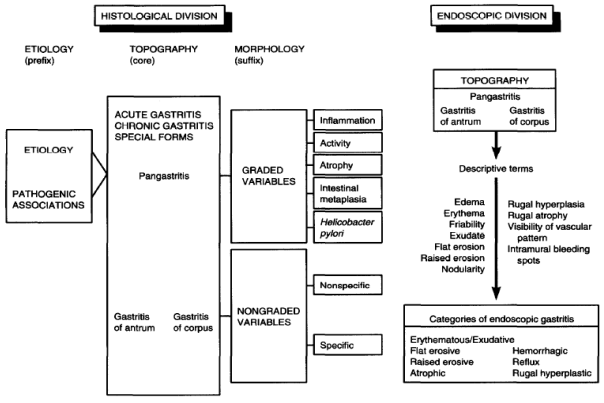

Figure 5: The chart of the histological division of the original Sydney System [6]

The guidelines recommended these five parameters were recorded separately for both antrum and corpus with at least two random biopsies to be taken from each site. Furthermore, it was recommended these parameters were to be semi-quantitatively graded as absent, mild, moderate or severe, each successive grade to represent an increase in severity of approximately one third.

The System provided a clear picture of the extent and topography of the gastritis and also its severity. In clinical diagnostic practice by adopting etiological prefix phrases, the core topography and morphological suffix phrases the histology report conveyed in a compact standard style the key data for that biopsy episode with a semi-quantitative format for future comparative episodes or studies

Table 5: Grading criteria of gastric antral biopsies according to Sydney System

Chronic inflammation	2-3 chronic inflammatory cells scattered randomly in the biopsy	nil
	10-15 chronic inflammatory cells/hpf	mild
	some areas with dense chronic inflammatory cells	moderate
	diffuse infiltration with dense chronic inflammatory cells	marked
Neutrophilic infiltrate	no neutrophils anywhere in the biopsy	nil
	scattered neutrophils in the biopsy	mild
	foci of dense neutrophilic infiltrate with scattered neutrophils in the rest of the biopsy	moderate
	several foci of dense inflammatory infiltrate in the biopsy with involvement of crypts	marked
Atrophy	no evidence of gastric gland loss	nil
	small areas where gastric glands have disappeared (< 25%)	mild
	25-50% of the biopsy shows loss of gastric glands	moderate
	> 50% of the biopsy shows loss of gastric glands	marked
Intestinal metaplasia	no intestinal metaplasia	nil
	focal areas of intestinal metaplasia (1-4 crytps)	mild
	multiple foci involving > 4 crypts but < 50% of the biopsy	moderate
	intestinal metaplasia involving > 50% of the biopsy specimen	marked
HP density	no HP identified	nil
	only a few HP seen in single or multiple foci	mild
	numerous HP seen in separate areas of foci	moderate
	> 50% of the surface area covered with HP	marked

Esofagogastroduodenoscopy

Esophagogastroduodenoscopy is a examination to evaluate upper gastrointestinal tract. It can be used to perform diagnostic & therapeutic procedures, like biopsy. Should be reserved for patients who have little or no response to therapy after 7-10 days or for patients whose symptoms have not resolved after 6-8 wk.[20]If upper GI esophagogastroduodenoscopy is unremarkable, patients w/ persistent symptoms or alarm features should be evaluated further for other diagnosis. For patients >55 yr (>40 yr in areas w/ high prevalence of gastric cancer), consider esophagogastroduodenoscopy when symptoms persist despite *H pylori* testing/treatment & acid suppression therapy and when patient has one or more of: Previous gastric ulcer or surgery, continuing need for NSAID treatment, raised risk of gastric cancer, anxiety about cancer

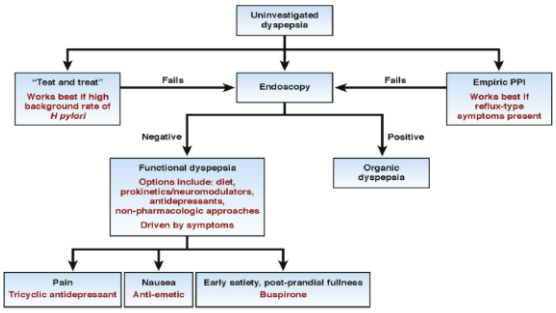

Figure 6: Suggested approach to the assessment and management of dyspepsia [12]

The most common age group affected by chronic gastritis in this study was 50 -60 years with a mean age of 50 years, which is not really different with Udoh et al. [23] also observed chronic gastritis in younger age group with mean age of 48.6 years.. However, Garg et al. [9], Aydin et al. [4] and Mustapha et al. [15]had reported chronic gastritis in relatively younger age group with a mean age of 47 years. We detected none cases of chronic gastritis below or at the age of 20 years and both

the cases were negative for *H. pylori* colonization. However, the study of chronic gastritis among children in Colombia showed *H. pylori* infection being common, affecting 59% of total children [2].

The rate of gastric colonization of *H. pylori* increased with increasing age in the present study, similar to the findings of other studies [10,3]. We observed a slight higher incidence rate of chronic gastritis in male with a M:F ratio of 1.07:1. Similarly, the other study from Nepal [8] observed a higher incidence in males with a M:F ratio of 1.6:1.Chen et al. [5], Garg et al. [9], and Pruthi et al. [17],and Park et al. [16] also had a higher incidence rate in males with a M:F ratio of 1.8:1, 2.1:1, 2.3:1 and 2.8:1 respectively. *H. pylori* colonization was detected more in male patients. [17,9,4,15,23]

Antrum (85.1%) was the most common site in our study. Similarly, common biopsy location was from antrum in other studies [17,9,4,15,23]. In the study by Dhakhwa et al. [8], Garg et al. [9] and Park et al. [16] tissue was obtained from antrum only. *H. pylori* colonization was more in the antrum than in the corpus [13].

Our study demonstrated *H. pylori* in 13.5% of cases of chronic gastritis. Dhakhwa et al. [8] and Pruthi et al. [17] had detected 44% and 47% of cases of chronic gastritis with *H. pylori* colonization respectively, which is more higher than in our study. The incidence rate of *H. pylori* in different studies is subject to the accuracy of biopsy techniques. Thus, multiple biopsies are to be taken to improve the yield. The lower incidence of *H. pylori* in our study could be due to the use of proton pump inhibitors or antimicrobial agents by the patients prior to endoscopic biopsies or not following the proper guidelines to obtain mucosal biopsies.

The majority (67.56%) of the biopsies showed marked inflammation; 28.2% had moderate inflammation. Likewise, Archila et al. [2], Park et al. [16], and Whitteman et al. [25]observed. However, Garg et al. [9] found mild inflammation in most of the cases.. The density of *H. pylori* colonization increased with severity of chronic inflammation in our study. This finding was compatible to other studies [9,2,24]. In contrast, there was no statistical significant relationship between the density of *H. pylor*i infection and the grade of chronic inflammation in the study conducted by Udoh et al. [23]

In the present study neutrophilic activity was present in 16% of cases. Dhakwa et al. [8] and Park et al. [16] had higher incidence of neutrophilic activity than in our study comprising 50% and 78.7% of cases, respectively. Neutrophilic activity was present in all *H. pylori* positive cases [16]. *H. pylor*i was not demonstrated in 86.5% of cases. 90% of cases with moderate neutrophilic activity have H.Pylori infection. There was a significant association between neutrophilic activity with *H. pylori* concentration in the present study as discussed elsewhere [9,8,26]. However, Park et al. [16] observed no statistical significance between neutrophilic activity and *H. pylori* concentration.

Atrophy was absent in our studies .Similarity had reported rare incidence of atrophic gastritis [11,22]. Other studies had shown increase of atrophy with increasing age [17,19]. The other study from Nepal [8] and Garg et al. [9]observed atrophy in 10% and 12.33% of cases respectively which is higher than in the present study.

5. Conclusion

The present study found that*H. pylori* infection has more found in male than female patient with the most case at age41 - 50. H Pylori infection more be found in gastric biopsies in antrum with neutrophilic activity mononuclear cell infiltrate, and no atrophy process. The esophagogastroduodenoscopy needs to be done in a population with a severe form of gastritis to find accurate treatment for the type of gastritis.

We agreed with other authors that histology is the goal standard and mandatory for accurate diagnosis of gastritis in all cases.

References

[1] Ajayi, AkandeOladimeji et al.Corelation between the Endoscopic and Histologic Diagnosis of Gastritis at the Ekiti State University Teaching Hospital, Ado Ekiti, Nigeria. 2015 : International Journal of Internal Medicine
[2] Archilap, Tovarl, (and) Ruiz M Histological characteristics of chronic gastritis reported in gastric biopsies from children aged 1 to 16 years at the Hospital Infantil de San José .2010 .September Revcol l27 (2): 74- 78.
[3] Asaka M, Kimura T, KudoM,Takeda H, Mitani S, Miyazaki T et al. Relationship of Helicobacter pylori to serum pepsinogens in an asymptomatic Japanese population.1992 Gastroenterology 102(3): 760-766.
[4] Aydino, Egilmezr, Karabacakt, KanikaInterobserver variation in histopathological assessment of Helicobacter pylori gastritis.2003 World J Gastroenterol9(10): 2232-2235.
[5] Chen XY, Liu WZ, Shi Y, Zhang DZ, Xiao SD, Tytgat GNJ. Helicobacter pylori associated gastric diseases and lymphoid tissue hyperplasia in gastric antral mucosa. 2002. JCP 55(2); 133-137.
[6] Correa P, Yardley J.H. Grading and Classification of Chronic Gastritis: One American Response to the Sydney System.1992Gastroenterology vol. 1 102. no. 102:355-359
[7] Desai, HG. Dyspepsia.SupplementTo JAPI.2012: Vol 60.
[8] Dhakhwa R, Acharya IL, Shrestha HG, Joshi DM, Lama S, Lakhey M.Histopathologic Study of Chronic Antral Gastritis.2012. J Nepal Health Res Counc 10(1): 57-60.
[9] Garg B, Sandhu V, Sood N, Sood S, Malhotra V.Histopathological analysis of chronic gastritis and correlation of pathological features with each other and with endoscopic findings.2012. Pol J Pathol63(3): 172-178.
[10] Graham DY, Malaty HM, Evans DJ, Evans DG, Klein PD, Adam E. Epidemiology of Helicobacter pylori in an asymptomatic population in the United States: Effect of age, race, and socioeconomic status.1991 Gastroenterology 100(6): 1495-1501.

[11] Hussien NR, Napaki SM, Atherton JC .Study of Helicobacter pylori associated gastritis patterns in Iraq and their association with strain virulence.2009. Saudi J Gastroenterol 15(2): 125-127.
[12] Jayanthi C, Lavanya M et al. Histopathological analysis of chronic gastritis and correlation of pathological features with helicobacter pylori. Indian Journal of Pathology and Oncology, October-December 2017;4(4):495-500
[13] Koduru, Pramoda , et al. Definition, Pathogenesis, and Management of That Cursed Dyspepsia.2018. Clinical Gastroenterology and Hepatology;16:467–479
[14] Manxhuka-Kerliu S, Telaku S, Devolli-Disha E, Ahmetaj H, Sahatciu-Meka V, Kerliu A et al. Helicobacter pylori gastritis updated Sydney classification applied in our material.2009.Prilozi 30(1): 45–60.
[15] Mustaphask, Bolorim, Ajayin, Nggadah, Pindigau, W gashau et al. Endoscopic findings and the frequency of Helicobacter pylori among dyspeptic patients in North-Eastern Nigeria.2007. The Internet Journal of Gastroenterology 6(1): 1528-1532.
[16] Park J, Kim MK, Park SM .Influence of Helicobacter pylori colonization on histological grading of chronic gastritis in Korean patients with peptic ulcer.1995. Korean J Intern Med 10(2): 125-129.
[17] Pruthi S, Nirupama M, Chakraborti S .Evaluation of gastric biopsies in chronic gastritis: Grading of inflammation by Visual Analogue Scale.2014. Med J DY PatilUniv 7(4): 463-467.
[18] Rugge Massimo, Genta Robert .Staging and grading of chronic gastritis. 2005. Elsevier Human Pathology (2005) 36, 228– 233
[19] Schlemper RJ, van der Werf SDJ, Vandenbroucke JP, Biemond I, Lamers CBHW Seroepidemiology of gastritis in Japanese and Dutch working populations: Evidence for the development of atrophic gastritis that is not related to Helicobacter pylori.1995. Gut 37(2): 199-204.
[20] Sudoyo A W, Setyohadi B, Alwi I dkk. Buku Ajar IlmuPenyakitDalam.Jilid II Edisi VI, Jakarta: Interna Publishing PusatPenerbitanIlmuPenyakitDalam.2009 : 371 – 373,1768 - 1771, 1772, 1805
[21] SuzanaManxhukaKerliu, SkenderTelaku et al.Helicobacter Pylori Gastritis Updated Sydney Classification Applied in Our Material.2009: Biol. Med. Sci., MASA : 45–60.
[22] Turkay C, Erbayrak M, Bavbek N, YendunyaS, Eraslani E, Kasapolu B .Helicobacter pylori and histopathological findings in cases with dyspepsia.2011. Turk J Gastroenetrol22(2): 122-127.
[23] Udohmo, Obasekde (2012) Histopathological evaluation of H. pylori associated gastric lesions in Benin City, Nigeria.2012.Eastafrmedj 89(12): 408-413
[24] Uhlig HH, Tannapfel A, Mössner J, Jedwilayties S, Deutscher J, Müller DM et al. Histopathological parameters of Helicobacter pylori-associated gastritis in children and adolescents: comparison with findings in adults.2003.Scand J Gastroenterol38(7): 701-706.
[25] Witteman EM, Mravunac M, Becx MJ, Hopman WP, Verschoor JS, Tytgat GN et al. Improvement of gastric inflammation and resolution of epithelial damage one year after eradication of Helicobacter pylori. 1995. JCP 48(3): 250-256.
[26] Zhang C, Yamada N, Wu YL, Wen M, Matsuhisa T, Matsukura N .Comparison of Helicobacter pylori infection and gastric mucosa histological features of gastric ulcer patients with chronic gastritis patients.2005. World J Gastroenterol 11(7): 976-981.