

## Research Article

# Prevalence of Helicobacter Pylori Among Patients with Chronic Gastritis in Nigeria

Adamu Mohammad Ewa<sup>1</sup>, Yussuf Maisuna Abdulkadir<sup>2,\*</sup>, Yusuf Ibrahim<sup>3</sup>

<sup>1</sup>Department of Medicine, Dalhatu Araf Specialist Hospital, Lafia, Nigeria

<sup>2</sup>Department of Medicine, Aminu Kano Teaching Hospital, Kano, Nigeria

<sup>3</sup>Department of Histopathology, Aminu Kano Teaching Hospital, Kano, Nigeria

## Abstract

**Introduction:** The stomach wall is made up of smooth muscles which are arranged in three layers: outer longitudinal, inner circular and innermost oblique layers. The mucosa is made up of simple columnar epithelium and is made up of several cell types which include: mucous cells which secrete alkaline mucous that protects the epithelium against acid and shear stress. Gastritis can involve the whole of the stomach, or a specific part of the stomach such as the fundus, body or the antrum and the distribution of gastritis may be related to an aetiological factor. *H. pylori* causes passive inflammation inside the gastric epithelium and alters signal transduction pathways that serve as a platform for the pathogenesis, but it also develops antimicrobial resistance via genetic changes and biofilm development. The bacterium floats in the direction of the epithelial membrane when it enters the stomach, taking advantage of areas of the stomach wall that are injured. **Method:** The study was conducted in Kano, Nigeria from April 2021 to October 2021 and it is a Hospital-based Cross-sectional Descriptive study. Patients with dyspepsia referred for oesophagogastroduodenoscopy (OGD) at the endoscopy suite of the hospital constitute the study population, 197 patients that met the inclusion criteria were recruited and underwent OGD. Each patient were biopsied in accordance with the updated Sydney protocol. Data were collected and the report of both endoscopy and histological assessment for the presence of gastritis and *H. pylori* were documented and analyzed using the SPSS 20. **Result:** *H. pylori* was detected in 137 patients out of the 197 gastric biopsies sample of gastritis, the prevalence was 69.5%. Also, There was a statistically significant relationship between *H. pylori* and those aged 31-40years ( $p = 0.025$ ), however, the relationship was not significant for the other age groups and no significant relationship was found between sex ( $p = 0.512$ ). The most common site affected by histologic gastritis in this study was the antrum (99.4%) and the high predominance of antral involvement is probably due to the high rate of *H. pylori* infection in the study subjects which is known to be associated with antral affectation. **Conclusion:** The most predominant pattern of histologic gastritis was chronic *H. pylori* gastritis located in the antrum. The prevalence of *H. pylori* infection was 69.5%, and it had significant association with both endoscopic and histologic gastritis. Therefore, prevention and eradication program for the *H. pylori* infection are necessary in our environment.

## Keywords

Oesophagogastroduodenoscopy, Endoscopic Gastritis, Histologic Gastritis, Helicobacter Pylori, Proton Pump Inhibitors, Potassium-competitive Acid Blocker

\*Corresponding author: ymadoc@yahoo.com (Yussuf Maisuna Abdulkadir)

**Received:** 12 November 2024; **Accepted:** 23 November 2024; **Published:** 12 December 2024



Copyright: © The Author(s), 2024. Published by Science Publishing Group. This is an **Open Access** article, distributed under the terms of the Creative Commons Attribution 4.0 License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution and reproduction in any medium, provided the original work is properly cited.

## 1. Introduction

The stomach is a J-shaped organ located in the upper abdomen distal to the oesophagus. It is continuous with the oesophagus at its upper part and with the duodenum distally. It is divided into four parts: cardia, fundus, body (corpus), and antrum. The gastroesophageal sphincter separates the distal end of the oesophagus from the stomach while the pyloric sphincter separates the pyloric antrum from the duodenum. The cardia is a small ill-defined area about 1-3cm distal to the lower end of the oesophagus while the fundus refers to the part of the stomach above the gastroesophageal junction just below the left hemidiaphragm. The antrum is the distal 1/3 of the stomach proximal to the pyloric sphincter while the body or corpus refers to the remaining part between the antrum and the fundus. The stomach has two curvatures: the greater curvature at the inferior-lateral border and the lesser curvature at the superior-medial border. The stomach has many rugal folds when empty that flattens out when it is distended [1, 2].

Gastritis can involve the whole of the stomach, or a specific part of the stomach such as the fundus, body or the antrum and the distribution of gastritis may be related to an aetiological factor.

The stomach wall is made up of smooth muscles which are arranged in three layers: outer longitudinal, inner circular and innermost oblique layers [1]. The mucosa is made up of simple columnar epithelium and is made up of several cell types which include: mucous cells which secrete alkaline mucous that protects the epithelium against acid and shear stress [1], parietal cells which secrete hydrochloric acid and intrinsic factor [2, 3], chief (zymogenic) cells that secrete pepsinogen [4], G cells which secrete gastrin [1], D cells which secrete somatostatin and amylin [5], enterochromaffin cells contain histamine [2]. Enterochromaffin cells, enterochromaffin-like cells, D cells and A-like cells constitute neuroendocrine cells of the stomach [2]. The upper 2/3 of the mucosa contains parietal cells and chief cells predominantly while G cells and D cells predominate in the antrum [1].

The stomach is innervated by the enteric nervous system (a third division of the autonomic nervous system in addition to sympathetic and parasympathetic systems) which sends afferent and receives efferent impulses via the vagus nerve [2].

The stomach has several important functions in humans among which hydrochloric acid (HCL) production is central. It is produced by the parietal cells at a concentration of about 160 mmol/L or pH 0.8 [4]. The rate of acid production varies little with ageing except in the presence of diseases such as *H. pylori* infection.

*Helicobacter pylori* (*H. pylori*) infection, which affects approximately half of the world's population, remains a serious public health problem. It is a highly mobile gram-negative, distinctively twisted bacterium [6]. *H. pylori* is typically associated with chronic active gastritis, and the bacteria lives in the glands beneath the mucosal surface. There is no clear way to explain how this bacterium is spread, but

oral or faecal exposure leading to person-to-person transfer is thought to be the dominant method [7]. *H. pylori* is more commonly found in Asia, Latin America, and Africa than in North America and Oceania,

*H. pylori* causes passive inflammation inside the gastric epithelium and alters signal transduction pathways that serve as a platform for the pathogenesis. The bacterium floats in the direction of the epithelial membrane when it enters the stomach, taking advantage of areas of the stomach wall that are injured [8, 9]. It uses Tlp receptors, mainly TlpB, to regulate flagellar motion based on chemical messengers in the cell environment [10]. Reactive oxygen species, as well as urea, gastric acid, lactate, and gastric acid, serve as signals for these receptors; urea is a key factor in microbial invasion [10]. There are also unknown molecules that may play a role in this mechanism [10]. *H. pylori* uses urease to defend itself against the acidic medium around it. Urea is converted into ammonia and other beneficial compounds by urease, which raises the pH of the microenvironment while protecting the bacterium from the acid in the stomach. In the presence of this barrier, the mucosal gel lining the stomach wall becomes less viscous, allowing the bacteria to travel through the mucus towards the gastric pits in which they will eventually colonize [10, 11].

The study aim to determine the clinical profile and *H. pylori* as detected in histological sample of patients with gastritis among patients with dyspepsia in Aminu Kano Teaching Hospital.

## 2. Method

The study was conducted in Kano, Nigeria from April 2021 to October 2021 and it is a Hospital-based Cross-sectional Descriptive study. Patients with dyspepsia referred for oesophagogastroduodenoscopy (OGD) during the study period constitute the study population.

Of the 454 endoscopy procedures performed during the study period only 197 patients met the inclusion criteria and were enrolled into the study. The endoscopy findings were assessed and documented, each patient was biopsied for histologic confirmation of *H. pylori*. Five biopsy samples were taken, two each from the corpus and antrum (anterior and posterior walls respectively), and one from incisura angularis in accordance with the updated Sydney protocol. The biopsy samples were placed in three specimen bottles containing Bouin's solution, one each for biopsies taken from the corpus, antrum, and incisura angularis respectively.

The samples were processed through surgical cut-off, then placed in automated tissue processor containing different strength of alcohol (ascending grade 75-100%) followed by embedding with paraffin wax, and then sectioned with the microtome. The processed tissue was then placed on a clean glass slide and DPX mountant was added before covering with a cover slip and then examined under the microscope.

The final report was then written in accordance with the Sydney system with the full participation of the researcher.

Data were collected and the report of the histological assessment were documented and analyzed using the SPSS 20 (SPSS inc, Chicago IL, USA). Frequencies and percentages were used to describe categorical variables while mean, standard deviation and median were used for continuous variables. Tests for association between gastritis and categorical variables were done using Chi-square test (or Fisher's exact test where applicable) while continuous variables were analyzed using student t-test. The level of statistical significance was set at  $p < 0.05$ . Data were presented in tables and charts.

Ethical clearance was obtained from the Ethical Committee of AKTH, Kano REF No. NHREC/28/01/2020/AKTH/EC/3002 dated 16<sup>th</sup> March, 2021, before the commencement of the study. The provisions of the Helsinki declaration were respected. Participants were adequately informed about the study in the language they understand, before obtaining written informed consent. All data collected from the participants were kept confidential.

### 3. Results

#### 3.1. The Prevalence of *H. Pylori* Among Patients with Dyspepsia

*H. pylori* was detected in 137 patients out of the 197 gastric biopsies taken and the prevalence was 69.5%.

#### 3.2. Relationship Between Socio-demographic Characteristics and *H. Pylori*

Table 1 shows the relationship between socio-demographic characteristics and *H. pylori*.

There was a statistically significant relationship between *H. pylori* and those aged 31-40years ( $p = 0.025$ ), however, the relationship was not significant for the other age groups. No significant relationship was found between sex ( $p = 0.512$ ), marital status ( $p = 0.084$ ), Types of education ( $p = 0.338$ ), level of education ( $p = 0.297$ ), occupation ( $p = 0.223$ ) and *H. pylori*.

**Table 1.** Relationship between socio-demographic characteristics and *H. pylori*.

Characteristics	<i>H. pylori</i>	No <i>H. pylori</i>	p-value
Age group (years)			0.025
18-20	12(70.6)	5(29.4)	

Characteristics	<i>H. pylori</i>	No <i>H. pylori</i>	p-value
21-30	36(69.2)	16(30.8)	
31-40	41(83.7)	8(16.3)	
41-50	29(72.5)	11(27.5)	
51-60	11(47.8)	12(52.2)	
>60	8(50.0)	8(50.0)	
Mean $\pm$ SD	37.7 $\pm$ 14.1	43.5 $\pm$ 15.2	0.070
Sex			0.512
Male	64(71.9)	25(28.1)	
Female	73(67.6)	35(32.4)	
Type of education			0.338
Formal	111(71.2)	45(28.8)	
Non-formal	26(63.4)	15(36.6)	
Level of education			0.297
Nil	26(63.4)	15(36.6)	
Primary	5(62.5)	3(37.5)	
Secondary	30(62.5)	18(37.5)	
Tertiary	76(76.0)	24(24.0)	

#### 3.3. Relationship Between *H. Pylori* and Histologic Gastritis

There was a statistically significant relationship between *H. pylori* ( $P < 0.001$ ) and histologic gastritis. (Table 2).

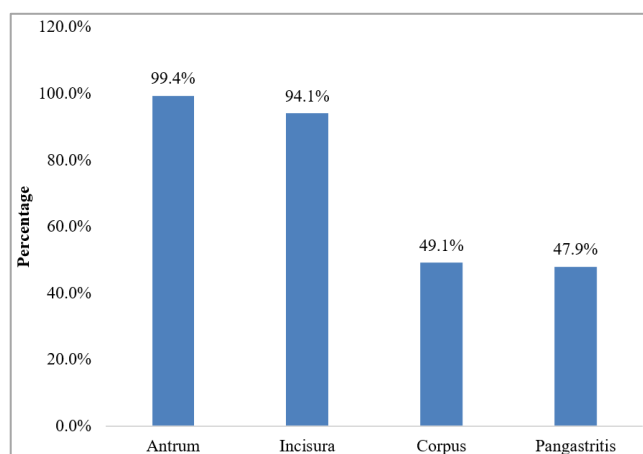
**Table 2.** Relationship between *H. pylori* and histologic gastritis.

Characteristics	Gastritis	No gastritis	p-value
<i>H. pylori</i>			<0.001
Yes	137(100)	0(0)	
No	32(53.3)	28(46.7)	

#### 3.4. Histologic Gastritis and Site of Biopsy

Figure 1 shows the prevalence of histologic gastritis in relation to sites of biopsy.

Out of the 169 participants with histologic gastritis, 168 (99.4%) had antral gastritis, 159 (94.1%) had gastritis affecting the incisura, 83 (49.1%) had gastritis affecting the corpus and 81 (47.9%) had pangastritis. Most of the participants had gastritis at more than one site as depicted in figure 1.



**Figure 1.** Histologic gastritis and site of biopsy.

## 4. Discussion

### 4.1. Socio-demographic Characteristics

#### 4.1.1. Age

In this study, most of the study participants 158 (80%) are aged 50 years and below with *H. pylori* seen in 118/158 and the majority 52 (26%) being in the 21-30 age group with *H. pylori* in 36/52. This is similar to the finding reported by Feyisa et al, from Ethiopia in which most of the participants are within the same age group. The age group with most cases of histologic gastritis 49 (94.2%) was 21-30years [12].

#### 4.1.2. Gender

Males accounted for 45% (89) while females accounted for 55% (108) with male to female ratio of 1:1.2 and *H. pylori* M:F 64:73. The Females also accounted for most of the study participants in other studies for gastritis [12-15]. The slight predominance of females in the study may be due to high health-seeking behavior among females when compared to males. However, no statistically significant association was found between sex and either endoscopic gastritis or histologic gastritis (p-values of 0.256 and 0.886 respectively) in this study.

#### Educational status:

Most of the study participants had formal education (64%), however, neither type of education nor level of education had any significant association with histologic gastritis. This may be due to the endemic nature of *H. pylori* which is the most predominant risk factor for gastritis in our study. The finding from this study is similar to what was reported among patients with dyspepsia in north-central Nigeria [16]. This finding is contrary to what was reported by Harun et al where low level of education had significant association with gastritis [17] and a study by Genta et al, which reported decreasing prevalence of gastritis with an increasing level of education and income [18].

### 4.2. Prevalence of *H. Pylori*

The overall prevalence of *H. pylori* in this study was 69.5% and there was a statistically significant association between it and the presence of histologic gastritis (P-value <0.001 at 95% CI). This prevalence is similar to reports from studies in this part of the world [19, 20] but slightly lower than the 81% previously reported in AKTH [21, 22], and that reported by Olokoba et al [23]. This may be due to the increasing widespread use of antibiotics among dyspeptic patients before presentation.

Among the studied risk factors for gastritis in this study, *H. pylori* (47.5%) was found to have a statistically significant relationship with histologic gastritis (P-value <0.001 at 95% CI). This is similar to a finding by Olokoba et al where *H. pylori* was shown to have strong association with chronic gastritis [24]. It is also similar to the finding by Bojuwoye et al where it was found to have significant association with histologic gastritis [16].

### 4.3. Histopathologic Characteristics of Gastritis

Most of the participants had moderate grade inflammation and mild activity with prevalence of chronic active gastritis of 63%. Chronic atrophic gastritis had a prevalence of 34% with the majority having mild atrophy while Chronic *H. pylori* gastritis had a prevalence of 69.5% in this study. These findings are comparable to those reported by other studies within the country [19, 20, 23, 25]. Another study by Tariq et al from Pakistan also reported the prevalence of histologic gastritis to be 70% but with significantly lower activity, atrophy and *H. pylori* association, which was attributed to widespread use of antibiotics and acid suppressing medications [26].

The most common site affected by histologic gastritis in this study was the antrum seen in almost all the study participants (99.4%) and the high predominance of antral involvement is probably due to the high rate of *H. pylori* infection in the study subjects which is known to be associated with antral affectation. In fact, all the study participants who had *H. pylori* had histologic gastritis, which goes to show that *H. pylori* is highly associated with the development of gastritis even though it's not the only cause of the condition. The findings in this study are similar to the finding by Margarita et al where all the patients with *H. pylori* had histologic gastritis [27]. Although *H. pylori* gastritis can affect all regions of the stomach, it tends to predominate in the antrum [28].

## 5. Conclusions

Chronic Gastritis accounted for 99.4% and predominantly located in Antrum 94.1%. *H. pylori* was confirmed only among the patients with Chronic Gastritis patients with prevalence of 69.5%. This high prevalence of *H. pylori* infection (69.5%), had significant association with both endoscopic and histologic gastritis.



This findings further emphasize the importance of thorough evaluation of all patients with dyspepsia for the presence of *H. pylori* infection especially if they have either endoscopic or histological gastritis due to its significant association with Gastric Cancer.

## 6. Recommendation

Importantly, eradication program for the *H. pylori* infection should be intensive in our environment and from the few available studies, newer regimen with Potassium-competitive acid blocker (Vonoprazan) is better at *H. pylori* eradication than the convention Proton-pump inhibitors.

## Abbreviations

OGD	Oesophagogastroduodenscopy
<i>H. pylori</i>	<i>Helicobacter pylori</i>
HCL	Hydrochloric Acid
AKTH	Aminu Kano Teaching Hospital
DPX	Dibutylphthalate Polystyrene Xylene
SPSS	Statistical Package for the Social Science
M:F	Male:Female Ratio
CI	Confidence Interval

## Acknowledgments

All praise is to Almighty who made it possible for me to complete this work and profound gratitude to Professor MM Borodo and Professor AA Samaila for their guidance and advice. I sincerely appreciate the support of my colleagues and family.

## Authors Contributions

**Adamu Mohammad Ewa:** Development of the concept, Seeking of Ethical Approval, Questionnaire administration.

**Yussuf Maisuna Abdulkadir:** Concept, Manuscript design and Data analysis, interpretation and presentation

**Yusuf Ibrahim:** Histo-pathological analysis of the gastric tissues

## Conflicts of Interest

The Authors declare no conflicts of interest.

## References

- [1] James Lindsay, Louise Langmead SLP. Clinical Medicine. 9th ed. Parveen Kumar MC, editor. Edinburgh: elsevier; 2017. 376–381 p.
- [2] Schubert ML, Peura DA. Control of Gastric Acid Secretion in Health and Disease. *Gastroenterology* 2008; 134(7): 1842–60.
- [3] Cinti S, De Matteis R, Picó C, Ceresi E, Obrador A, Maffei C, et al. Secretory granules of endocrine and chief cells of human stomach mucosa contain leptin. *Int J Obes Relat Metab Disord*. 2000 Jun; 24(6): 789–93. <https://doi.org/10.1038/sj.ijo.0801228>
- [4] Bado A, Levasseur S, Attoub S, Kermorgant S, Laigneau JP, Bortoluzzi MN, et al. The stomach is a source of leptin. *Nature*. 1998 Aug 20; 394(6695): 790–3. <https://doi.org/10.1038/29547>
- [5] Zaki M, Koduru S, McCuen R, Vuyyuru L, Schubert ML. Amylin, released from the gastric fundus, stimulates somatostatin and thus inhibits histamine and acid secretion in mice. *Gastroenterology*. 2002 Jul; 123(1): 247–55. <https://doi.org/10.1053/gast.2002.34176>
- [6] Bizzozzero, G.; der Eidechsen, D. Ueber die schlauchförmigen Drüsen des Magendarmkanals und die Beziehungen ihres. *Arch. Mikrosk. Anat.* 1893, 42, 82.
- [7] Stefano, K.; Marco, M.; Federica, G.; Laura, B.; Barbara, B.; Gioacchino, L.; Gian, L. d. A. *Helicobacter pylori*, transmission routes and recurrence of infection: State of the art. *Acta Biomed*. 2018 Dec 17; 89(8-S): 72–76. <https://doi.org/10.23750/abm.v89i8-S.7947>
- [8] Camilo, V.; Sugiyama, T.; Touati, E. Pathogenesis of *Helicobacter pylori* infection. *Helicobacter* 2017, 22, e12405.
- [9] Denic, M.; Touati, E.; De Reuse, H. Pathogenesis of *Helicobacter pylori* infection. *Helicobacter* 2020, 25, e12736.
- [10] Hanyu, H.; Engevik, K. A.; Matthis, A. L.; Ottemann, K. M.; Montrose, M. H.; Aihara, E. *Helicobacter pylori* uses the TlpB receptor to sense sites of gastric injury. *Infect. Immun*. 2019, 87, e00202–e00219.
- [11] Idowu, S.; Bertrand, P. P.; Walduck, A. K. Gastric organoids: Advancing the study of *H. pylori* pathogenesis and inflammation. *Helicobacter* 2022, 27, e12891.
- [12] Feyisa ZT, Woldeamanuel BT. Prevalence and associated risk factors of gastritis among patients visiting Saint Paul Hospital Millennium Medical College, Addis Ababa, Ethiopia. *PLoS One*. 2021 Feb 9; 16(2): e0246619. <https://doi.org/10.1371/journal.pone.0246619>
- [13] Jemilohun A, Otegbayo J, Ola S, Oluwasola A, Akere A. Correlation Between Endoscopic and Histological Gastritis in South-Western Nigerians With Dyspepsia. *Niger J Gastroenterol Hepatol* 2010; 2(2): 73–6.
- [14] Ajayi AO, Ajayi EA, Solomon OA, Duduyemi B, Omonisi EA, Taiwo OJ. Correlation between the Endoscopic and Histologic Diagnosis of Gastritis at the Ekiti State University Teaching Hospital, Ado Ekiti, Nigeria. *Int J Intern Med* 2015; 4(1): 9–13.
- [15] Mustapha SK, Kida IM, Dayar A, Gundiri LB. Indications for upper gastrointestinal endoscopy in maiduguri. *BOMJ*. 2010; 7(2): 2008–11.
- [16] Bojuwoye MO, Olokoba AB, Ibrahim OOK, Ogunlaja AO, Bojuwoye BJ. Relationship between *helicobacter pylori* infection and endoscopic findings among patients with dyspepsia in north central, Nigeria. *Sjms* 2016; 11(4): 56–9.

- [17] Harun DO, Kes M, Hakim AF, Kep S, Suwandiana W, Kep S. The Correlation Of Knowledge And Education Level Of The Patients With The Gastritis Incident At Sindangbarang Public Health Center Cianjur. *Int J Sci Technol Res* 2015; 4(8): 6–13.
- [18] Genta RM, Turner KO, Sonnenberg A. Demographic and socioeconomic influences on *Helicobacter pylori* gastritis and its pre-neoplastic lesions amongst US residents. *Aliment Pharmacol Ther.* 2017 Aug; 46(3): 322-330. <https://doi.org/10.1111/apt.14162>
- [19] Udoh MO, Obaseki DE. Histopathological evaluation of *H. Pylori* associated gastric lesions in benin city, Nigeria. *East Afr Med J* 2012; 89(12): 408–13.
- [20] Duduyemi B, Ojo B, Olaomi O, Atiba A. Histopathological Pattern of Endoscopic Gastric Biopsy in a District Hospital in Nigeria: A Review of 118 Consecutive Cases. *Am J Med Biol Res* 2014; 2(3): 83–6.
- [21] Bello AK, Umar AB, Borodo MM. Prevalence and Risk Factors for *Helicobacter pylori* Infection in Gastroduodenal Diseases in Kano, Nigeria. *African J Med Heal Sci* 2017; 16: 12–8.
- [22] Tijjani B, Umar A. Peptic ulcer disease and *Helicobacter pylori* infection. *Curr Opin Gastroenterol* 1994; 10(1): 98–104.
- [23] Olokoba AB, Gashau W, Bwala S, Adamu A, Salawu FK. *Helicobacter Pylori* Infection in Nigerians with Dyspepsia. *Ghana Med J* 2013; 47(2): 79-81.
- [24] Olokoba AB, Apari E, Salawu FK, Nggada HA. *Helicobacter pylori* in dyspeptic Nigerians. *West Afr J Med* 2013; 32(4): 277–80.
- [25] Bello U, S Maiyaki A. Morphologic Pattern of Diseases in Gastric Biopsies and Role of *Helicobacter Pylori* in Chronic Gastritis at a Nigerian Teaching Hospital. *Saudi J Pathol Microbiol* 2021; 6(2): 71–5.
- [26] Sarfras T, Hafeez M, Shafiq H, Azhar M, Ahmed K, Jamal N. Histopathological analysis of gastric mucosal biopsies in non-ulcer dyspepsia. *PAMJ* 2016; 66(6) 875-6.
- [27] Dehesa M, Dooley CP, Cohen H, Fitzgibbons PL, Perez-perez GI, Blaser MJ. High Prevalence of *Helicobacter pylori* Infection and Histologic Gastritis in Asymptomatic Hispanics. *J Clin Microbiol.* 1991 Jun; 29(6): 1128-31. <https://doi.org/10.1128/jcm.29.6.1128-1131.1991>
- [28] Dixon MF, O'Connor HJ, Axon AT. Reflux gastritis: distinct histopathological entity? *J Clin Pathol.* 1986 May; 39(5): 524-30. <https://doi.org/10.1136/jcp.39.5.524>