

Relationship between Helicobacter Pylori Infection and Endoscopic Findings among Patients with Dyspepsia in North Central, Nigeria

Matthew O Bojuwoye^{1*}, Abdulfatai B Olokoba¹, Olatunde O K Ibrahim²
Ayotunde O Ogunlaja³, Bababode J Bojuwoye⁴

ABSTRACT

Background: There is a dearth of reports on the prevalence rate of *Helicobacter pylori* from the North-Central zone of Nigeria. This study aimed to determine the prevalence of *H. pylori* infection and its association with gastro-duodenal pathologies in patients presenting with dyspepsia in a tertiary hospital in North-Central Nigeria.

Materials and Methods: One hundred and forty eight adult patients with dyspepsia underwent oesophagogastroduodenoscopy. Antral and corporal biopsy specimens were collected for histological examination. Diagnosis of *H. pylori* infection was made if Helicobacter-like organisms were identified in the processed specimens using Haematoxylin and Eosin, and Giemsa stains.

Results: Of the 148 subjects studied, 68 (46.0%) were males and 80 (54.0%) females. The age range was 18 to 80 years with a mean of 49.5 ± 15.1 years. *H. pylori* infection was found in 70 (47.3%) patients. Gastritis was the commonest endoscopic finding, 52 (35.1%). Others included: gastroduodenitis 42 (28.4%), normal findings 20 (13.5%), gastric cancer 11 (7.4%), duodenal ulcer 10 (6.8%), gastric ulcer 4 (2.7%) and gastroduodenal polyps 4 (2.7%). Twenty (38.5%) of the 52 patients with gastritis were infected with *H. pylori* compared to 27 (64.3%) of the 42 patients with gastroduodenitis. Seven (35.0%) of the 20 patients who had normal endoscopic findings tested positive for *H. pylori* infection.

Conclusion: *H. pylori* infection has a moderately high prevalence among dyspeptic patients in Ilorin, irrespective of the gastroduodenal pathology seen at endoscopy. The association between *H. pylori* infection and endoscopic gastroduodenitis and histologic gastritis was significant ($p=0.01$ and 0.0006 respectively).

Key words: *Helicobacter pylori*, Oesophagogastroduodenoscopy, Gastroduodenitis.

Dyspepsia is a symptom-complex that is often encountered among patients at Medical Out-patient Departments (MOPDs) and GI clinics worldwide. It is also a common indication for referral for upper Gastrointestinal (GI) endoscopy. *Helicobacter pylori* (*H. pylori*) is a gram negative, micro-aerophilic, spiral-shaped, flagellated, urease producing and highly motile bacillus, with

a cock-screw motion. It colonizes the mucous layer in the gastric pits of the human stomach in close proximity to the gastric epithelial cells where it causes damage to the cells of the gastric epithelium¹. This organism was first described by Robin Warren and Barry Marshall in 1983 and is now recognized as a major etiological factor in upper GI disorders such as chronic gastritis, peptic

1. Department of Medicine, University of Ilorin Teaching Hospital, Ilorin, Kwara State.

2. Department of Pathology, University of Ilorin Teaching Hospital, Ilorin, Kwara State.

3. Department of Obstetrics and Gynaecology, Bowen University Teaching Hospital, Ogbomosho, Oyo State.

4. Department of Medicine, Bowen University Teaching Hospital, Ogbomosho, Oyo State.

*Correspondence to: Matthew O Bojuwoye, Department of Medicine, University of Ilorin Teaching Hospital, P.M.B. 1459, Ilorin.
Telephone: +2348032631175
E-mail: bojuwoyem26@yahoo.com

ulcer disease (PUD), gastric carcinoma, and gastric mucosal associated lymphoid tissue (MALT) lymphoma^{2,3}. Many of these disorders may present initially or solely with dyspepsia, a common symptom in MOPDs and GI clinics.

The discovery of *H. pylori* was therefore a major breakthrough in the management of dyspepsia, and it revolutionized the management of PUD which is now viewed as an infectious disease since the eradication of *H. pylori* infection plus the suppression of acid production with histamine-2 receptor antagonists (H₂RAs) or proton pump inhibitors (PPIs) leads to cure. Various diagnostic tests for *H. pylori* have been developed and these can be broadly classified into invasive and non-invasive tests¹.

The invasive tests utilize endoscopic biopsy samples for histology, culture, rapid urease test (RUT), polymerase chain reaction (PCR) and fluorescent in-situ hybridization (FISH). All these tests have been found to have sensitivities and specificities that are well above 90%⁴. The non-invasive tests do not require endoscopy. These include urea breath test (UBT), immunoglobulin G and M (IgG and IgM) serology, stool antigen test (SAT), saliva antibody test and urinary antibody test¹. Upper GI endoscopy otherwise known as oesophagogastroduodenoscopy (OGD) is the preferred method of diagnosis of upper GI disorders which often present with dyspepsia.

Since the discovery of *H. pylori* as an important etiological agent in gastro-duodenal diseases, investigation for this organism using OGD has become a standard clinical practice.

This study was carried out to determine the prevalence of *H. pylori* among a cohort of patients with dyspepsia seen at a tertiary health facility located in the North-central zone of Nigeria.

MATERIALS AND METHODS:

Study Area and Subjects:

This was a hospital based cross-sectional study that was carried out at the Gastroenterology clinic and the Endoscopy suite of the University of Ilorin Teaching Hospital (UITH), Ilorin, the capital city of Kwara State from April 2012 to December 2012. The hospital is a tertiary health institution located in the North-Central region of the Nigeria and it provides services to patients from the entire state and neighbouring states.

Ethical approval was obtained from the Ethics and Research Committee of UITH, Ilorin. Consenting adult patients aged 18 years and above with dyspepsia were recruited consecutively over the study period.

Patients who had ingested PPIs in the preceding 2 weeks and/or bismuth-containing drugs and antibiotics in the preceding 4 weeks of OGD and patients who had contraindications to OGD such as an uncooperative patient, haemodynamic instability (blood pressure < 90/60mmHg), suspected perforation of hollow viscus, acute exacerbation of chronic obstructive pulmonary disease, severe cervical spine disorders, history of myocardial infarction in the preceding 3 months, coma and seizures were excluded from the study.

Bio-data:

A structured questionnaire was administered to each participant to obtain their bio-data, and to document the duration of dyspepsia, identifiable risk factors and the presence of alarm features.

Oesophagogastroduodenoscopy:

All the study subjects underwent OGD after an overnight fast using a flexible forward-viewing videoscope. Antral and corporal biopsy specimens were collected for histological examination. The endoscopic findings were recorded in the questionnaires.

Processing and histological evaluation of the gastric biopsies:

Each biopsy tissue was routinely processed, embedded in paraffin and cut into 3-5 micrometer slices. Diagnosis of *H. pylori* infection was made if Helicobacter-like organisms were identified in the processed specimens using Haematoxylin and Eosin, and Giemsa stains.

Data Analysis:

Data generated from the questionnaires were analyzed using the SPSS 16 software. Numerical data are presented as means \pm standard deviation (SD). Categorical variables are presented as proportions and compared using Chi-square test, with the level of significance set at $p < 0.05$.

RESULTS:

A total of 148 dyspeptic subjects comprising 68 (46.0%) males and 80 (54.0%) females were studied with a male to female ratio of 0.85:1. The age range of the subjects was 18 to 80 years with a mean age \pm SD of 49.5 ± 15.1 years. The 51-60 years age group had the highest frequency of 39 (26.3%); Table 1. The median duration of dyspepsia was 12 months with no significant gender difference (male 7 months vs female 12 months; p -value = 0.09).

Gastritis was the commonest endoscopic finding, 52 (35.1%). Other findings were gastroduodenitis 42 (28.4%), normal findings 20 (13.5%), gastric cancer 11 (7.4%), duodenal ulcer 10 (6.8%), gastric ulcer 4 (2.7%) and gastroduodenal polyps 4 (2.7%). The endoscopic findings were not mutually exclusive. Seventy study subjects tested positive for *H. pylori* infection giving a prevalence rate of 47.3% (Figure 1). The highest prevalence rate of 15 (21.4%) was observed in the 41-50 years and 51-60 years age groups. Thirty-six (51.4%) females compared to 34 (48.6%) males tested positive for *H. pylori*. ($X^2 = 0.37$; p -value = 0.54). Forty-

seven (50.0%) of the 94 patients with gastritis were infected with *H. pylori* compared to 8 (32.0%) of the 25 patients with other lesions (gastric ulcer, duodenal ulcer and gastric cancer). Seven (35.0%) of the 20 patients who had normal endoscopic findings tested positive for *H. pylori*. There was significant association between *H. pylori* infection and the

Table (1): Socio-demographics characteristics of the study subjects.

| Variables | n= 148 | % |
|---------------------|--------|------|
| Age Range | | |
| 18-20 | 2 | 1.3 |
| 21-30 | 20 | 13.5 |
| 31-40 | 24 | 16.2 |
| 41-50 | 28 | 19.0 |
| 51-60 | 39 | 26.3 |
| 61-70 | 22 | 14.9 |
| 71-80 | 13 | 8.8 |
| Sex | | |
| Female | 80 | 54.0 |
| Male | 68 | 46.0 |
| Marital Status | | |
| Single | 14 | 9.5 |
| Married | 121 | 81.7 |
| Divorced | 1 | 0.7 |
| Widowed | 12 | 8.1 |
| Educational Status | | |
| No formal education | 23 | 15.5 |
| Primary | 18 | 12.2 |
| Secondary | 25 | 16.9 |
| Tertiary | 82 | 55.4 |
| Religion | | |
| Islam | 80 | 54.1 |
| Christianity | 68 | 45.9 |
| Occupation | | |
| Civil servants | 65 | 44.0 |
| Traders | 44 | 29.7 |
| Farmers | 7 | 4.7 |
| Artisans | 4 | 2.7 |
| Others | 28 | 18.9 |
| Ethnicity | | |
| Yoruba | 133 | 89.9 |
| Igbo | 3 | 2.0 |
| Hausa | 2 | 1.3 |
| Others | 10 | 6.8 |

Table (2): Association between *H. pylori* infection and the endoscopic findings in the patients

| OGD findings | <i>H. pylori</i> Positive (70) N (%) | <i>H. pylori</i> Negative (78) N (%) | N= 148 N (%) | Chi-Square X ² | P-value |
|--------------------------------|--|--|-----------------|------------------------------|---------|
| Oesophagitis | 7 (36.8) | 12 (63.2) | 19 (12.8) | 0.96 | 0.33 |
| Gastritis only | 20 (38.5) | 32 (61.5) | 52 (35.1) | 2.51 | 0.11 |
| Gastroduodenitis | 27 (64.3) | 15 (35.7) | 42 (28.4) | 6.79 | 0.01 |
| Gastric and Duodenal Polyps | 0(0.0) | 0 (0.0) | 4 (2.7) | 4.58 | 0.04* |
| Gastric Ulcer | 3 (75.0) | 1 (25.0) | 4 (2.7) | 1.27 | 0.27* |
| Duodenal Ulcer | 5 (50.0) | 5 (50.0) | 10 (6.8) | 0.03 | 0.56* |
| Gastric Erosions | 3 (60.0) | 2 (40.0) | 5 (3.4) | 0.33 | 0.45* |
| Duodenitis only | 2 (100.0) | 0 (0.0) | 2 (1.3) | 2.26 | 0.22* |
| Duodenal Erosions | 1(100.0) | 0 (0.0) | 1(0.7) | 1.12 | 0.47* |
| Gastric mass lesion | 2 (15.4) | 11 (84.6) | 13 (8.8) | 5.82 | 0.02 |
| Normal Findings | 7 (35.0) | 13 (65.0) | 20 (13.5) | 1.40 | 0.24 |

*Fisher's exact test

Table 3: The association between *H. pylori* and chronic gastritis

| | <i>H. pylori</i> Positive N (%) | <i>H. pylori</i> Negative N (%) | TOTAL N (%) | X ² | P value |
|-------------------|------------------------------------|------------------------------------|----------------|----------------|---------|
| Chronic gastritis | | | | | |
| Yes | 70 (51.5) | 66 (48.5) | 136 (91.9) | | |
| No | 0 (0.0) | 12 (100.0) | 12 (8.1) | | |
| Total | 70 (45.8) | 78 (54.2) | 148 (100.0) | 11.72 | 0.0006 |

presence of gastroduodenitis and gastroduodenal polyps with p-values of 0.01 and 0.04 respectively (Table 2). Of the 148 subjects who had gastric biopsies, 1 (0.7%) had normal findings on histology, 11 (7.4%) had gastric adenocarcinoma whereas the remaining 136 (91.9%) had histological evidence of chronic gastritis.

Of the 136 patients with histological evidence of gastritis, 70 (51.5%) tested positive for *H. pylori*. The association between *H. pylori* infection and histologic gastritis was significant (p-value = 0.0006). Table 3.

DISCUSSION:

Dyspepsia is often a distressing GI symptom that may be associated with significant morbidity and reduction in the quality of life of those affected. It is

therefore not surprising that a lot of research work has been done and still ongoing, searching for improved and cost effective methods of evaluating and managing this condition. Dyspepsia often times points to the presence of an underlying upper GI disorder albeit structural or functional. Evaluation for the presence of *H. pylori* in the gastric mucosa of persons with dyspepsia is a key component of investigating the cause of dyspepsia. Furthermore, the presence or absence of this organism impacts on the approach to the management of patients with this symptom-complex.

In this study, the prevalence rate of *H. pylori* among the subjects was 47.3% (95% CI 39.4% -55.3%). It is consistent with the prevalence rates obtained from some recent studies conducted in Nigeria

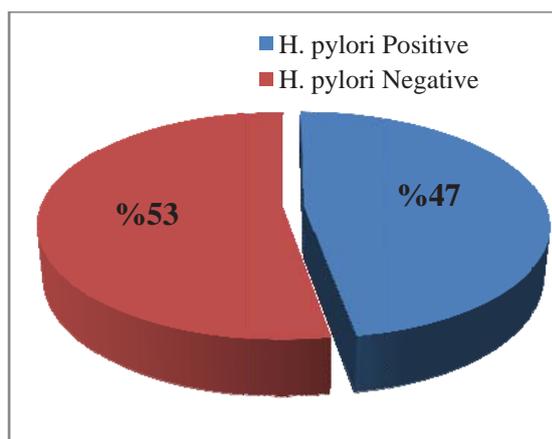


Figure (1): The prevalence of *H. pylori* in the study population.

with the use of biopsy based methods⁵⁻⁷. This rate is higher than the rates of 32.7% and 37.1% obtained with the use of histology of gastric biopsies and CLO test respectively and comparable with the rate of 48.7% obtained with the culture of gastric biopsies in a study of 167 dyspeptic patients recruited from two health centers in South-Western Nigeria⁵. Comparable prevalence rates of 52.35% and 52.38% were obtained with the use of histology of gastric biopsies and PCR respectively in Ibadan^{6,7}. A prevalence rate of 56.7% for *H. pylori* was obtained using histology in Yola, North-Eastern Nigeria⁸.

Higher prevalence rates ranging from 73.0% to 82.8% were reported in similar studies from other parts of the Nigeria and other countries in Africa⁹⁻¹⁵. The differences in the prevalence rates despite the use of similar detection methods may be due to the differences in time periods the studies were carried out, varying sample sizes, differences in geographical location, differences in the number of gastric biopsies and the regions of the stomach from which these were obtained, variations in staining techniques, prior antibiotic or anti-secretory agent use, and perhaps yet to be identified factors. Notably, the rate obtained from this study is moderately high but is similar to rates obtained from quite a number of recent

studies⁵⁻⁸. The widespread use of *H. pylori* eradication therapy probably resulting from an increase in the level of awareness of its etiologic role in gastroduodenal disorders and the growing problem of abuse of antibiotics in the populace may be responsible for the apparent decline over the years. Again, variation in the prevalence of *H. pylori* is dominated by the great differences between communities in the incidence of *H. pylori* infection during childhood¹⁶.

There was no significant difference in the prevalence of *H. pylori* among the various age groups. In the same vein, there was no significant difference in the prevalence of *H. pylori* infection between the males and females in this study. These findings are similar to those from previous studies in Nigeria^{6,8-11}. They are consistent with what has already been established about the epidemiology of this agent which affects all age groups and has not been reported to have a sex predilection. Similarly, there was no significant association between *H. pylori* infection and the marital status of the subjects. In Libya, there was a significant association between *H. pylori* infection and the education status of the patients¹⁷. No such association was observed in this study from Ilorin.

The association between *Helicobacter pylori* infection and the gross endoscopic findings:

Sixty-three (49.2%) of the 128 patients who had identifiable lesions at endoscopy tested positive for *H. pylori* compared to 7 (35%) of the remaining 20 patients with normal endoscopic findings. The difference in the prevalence of *H. pylori* between those who had lesions and those who had normal findings at endoscopy was not statistically significant. This is consistent with the findings in Ibadan, Kano and Maiduguri^{6,10,11}. The ubiquitous nature of *H. pylori* and the fact that not all those who are infected by the organism

develop the diseases linked with it may be the reason for this finding, and may also explain the high *H. pylori* prevalence rate of 35.0% even among the 20 dyspeptic patients with normal endoscopic findings. It may be important to treat this category of patients with *H. pylori* eradication therapy since the agent might be responsible for their dyspepsia. Fifty percent of patients with endoscopic gastritis had *H. pylori* infection, a finding comparable to findings from studies from Ibadan and Maiduguri^{6,11}. In this study, the association between *H. pylori* infection and endoscopic gastritis was not significant but its association with endoscopic gastroduodenitis was significant. It is important to test for the presence of the organism whenever gastritis or gastroduodenitis is seen at endoscopy. Fifty percent of patients with duodenal ulcer tested positive for *H. pylori* which is lower than the rates of 90-100% reported in previous studies^{9,10}. The fewer number of cases of duodenal ulcer (10) recorded in this study compared to the 323 and 48 reported in Ile-Ife⁹ and Kano¹⁰ respectively might provide an explanation for the relatively low rate of *H. pylori* infection among the patients with duodenal ulcer in this study. The *H. pylori* prevalence rate of 75.0% obtained among the cases of gastric ulcer is consistent with the reported rate of 60-90% in Ile-Ife and Kano^{9,10}. All four of the patients with gastric and/or duodenal polyps tested positive for *H. pylori*, a finding similar to that observed in Ile-Ife⁹ with the association between this organism and the presence of polyps found to be significant. The presence of polyps in the upper GI tract may thus indicate the presence of *H. pylori* and the need to test and treat for the organism. The lack of association between *H. pylori* infection and the other endoscopic findings in this study may be as a result of the sample size not being

large enough to bring out the association between the presence of this organism and these lesions and not necessarily that an association does not exist. Notably, a similar study from Ibadan did not observe any significant association between *H. pylori* and any of the endoscopic findings which may be due to the small size of the study population. With the moderately high rate of infection of 49.2% and 35% in patients with endoscopic abnormality and normal endoscopic findings respectively, it remains important to test and treat for *H. pylori* in Nigerians with dyspepsia.

The association between *H. pylori* infection and the histologic gastritis:

Helicobacter pylori causes continuous gastric inflammation in virtually all infected persons¹⁸. This inflammatory response consists of the recruitment of neutrophils, followed by T and B lymphocytes, plasma cells, and macrophages, along with epithelial-cell damage¹⁸. *Helicobacter pylori* associated histological gastritis was observed in 70 (51.5%) dyspeptic patients in Ilorin. This prevalence rate is similar but higher than the rate of 49.1% obtained from an earlier study of the histomorphology of *H. pylori* associated chronic gastritis in Ilorin¹⁹. It is comparable to the *H. pylori* associated gastritis rate of 57.2% reported in a retrospective study of 603 antral biopsies in Maiduguri²⁰. It is however lower than the prevalence rate of 89.1% observed in another study from Maiduguri¹¹, which may be due to the larger size of the study population.

The association between *H. pylori* infection and the presence of chronic gastritis on histology was significant which is consistent with findings in previous studies^{8,12,20}.

Helicobacter pylori infection was significantly associated with the presence of gastroduodenitis and gastroduodenal polyps at endoscopy as well as with

histologic gastritis among this cohort of Nigerians with dyspepsia.

Screening for *H. pylori* infection should remain a key component of the evaluation of patients with dyspepsia since the prevalence of this organism among these patients remains high.

REFERENCES:

1. Malfertheiner P, Megraud F, O'Morain C, Bazzoli F, El-Omar E, Graham D, *et al.* Current concepts in the management of *Helicobacter pylori* infection: the Maastricht III Consensus Report. *Gut*. 2007; 56 (6):772-781.
2. Tygat G, Langenberg W, Rauws E, Rietrap P. Campylobacter-like organism (CLO) in the human stomach. *Gastroenterology*. 1985; 88:1620-1624.
3. Suerbaum S, Michetti P. *Helicobacter pylori* infection. *N Engl J Med*. 2002; 347(15): 1175-1186.
4. Graham DY, Sung JY. Second Asia-Pacific Consensus Guidelines for *Helicobacter pylori* infection. In: Feldman M FL, Brandt LJ(editor). Sleisenger and Fordtran's gastrointestinal and liver disease: Pathophysiology, Diagnosis, Management. 9th ed: Philadelphia: Saunders/Elsevier; 2008. p. 990-998.
5. Smith SI, Omonigbehin EA, Goodluck HA, Abdulkareem FB, Onyekwere CA, Agomo C, *et al.* Diagnostic methods for the detection of *Helicobacter pylori* in Nigeria. *Trop Gastroenterol*. 2010; 31(2):113-115.
6. Jemilohun AC, Otegbayo JA, Ola SO, Oluwasola OA, Akere A. Prevalence of *H. pylori* among Nigerian patients with dyspepsia in Ibadan. *Pan Afr Med J*. 2011; 6:18.
7. Smith SI, Fowora MA, Otegbayo JA, Abdulkareem FB, Omonigbehin EA, Adegboyega A, *et al.* Comparison of PCR with other diagnostic techniques for the detection of *H. pylori* infection in patients presenting with gastroduodenal symptoms in Nigeria. *Int J MolEpidemiol Genet*. 2011; 2(2):178-184.
8. Olokoba AB, Apari E, Salawu FK, Nggada HA. *Helicobacter pylori* in dyspeptic Nigerians. *West Afr J Med*. 2013; 32(4):277-280.
9. Ndububa DA, Agbakwuru AE, Adebayo RA, Olasode BJ, Olaomi OO, Adeosun OA, *et al.* Upper gastrointestinal findings and incidence of *Helicobacter pylori* infection among Nigerian patients with dyspepsia. *West Afr J Med*. 2001; 20(2):140-145.
10. Tijjani BM, Borodo MM, Samaila AA, Mohammed AZ. *H. pylori* infection in dyspeptic patients at Kano, Nigeria. *Borno Medical Journal*. 2005; 2(1):14-17.
11. Mustapha SK, Ajayi NA, Nggada HA, Pindiga UH, Bolori MT, Ndahi A, *et al.* Endoscopic findings and the frequency of *H. pylori* among dyspeptic patients in Maiduguri, North-eastern Nigeria. *Highland Medical Research Journal*. 2007; 5(1):78-81.
12. Tanko MN, Manasseh AN, Echejoh GO, Mandong BM, Malu AO, Okeke EN, *et al.* Relation between *Helicobacter pylori*, inflammatory (neutrophil) activity, chronic gastritis, gastric atrophy and intestinal metaplasia. *Niger J ClinPract*. 2008; 11(3):270-274.
13. Olokoba AB, Gashau W, Bwala S, Salawu FK. *Helicobacter pylori* infection in Nigerians with dyspepsia. *Ghana Med J*. 2013; 47(2): 79-81.
14. Aduful HK, Naaeder SB, Darko R, Baako BN, Clegg-Lampsey JNA, Nkrumah KN, *et al.* Upper gastrointestinal endoscopy at Korle Bu Teaching Hospital, Accra, Ghana. *Ghana Med J*. 2007; 41(1): 12-16.
15. Mbengue M, Diouf ML, Dangou JM, Ka MM, Ba-Seck A, Ndiaye MF, *et al.* Frequency of *Helicobacter pylori* infection in symptomatic patients in Senegal. *Med Trop (Mars)*. 1997; 57 (3):256-258.
16. Pounder RE, Ng D. The prevalence of *Helicobacter pylori* infection in different countries. *Aliment PharmacolTher*. 1995; 9(2):33-39.
17. Bakka AS, El-Gariani AB, AbouGhrara FM, Salih BA. Frequency of *Helicobacter pylori* infection in dyspeptic patients in Libya. *Saudi Med J*. 2002; 23(10):1261-1265.
18. McColl KE. *Helicobacter pylori* infection. *N Engl J Med*. 2010; 1597: 604
19. Badmus SK, Olokoba AB, Ibrahim OOK, Abubakar-Akanbi SK. Histomorphology of *Helicobacter pylori*-associated chronic gastritis in Ilorin. *Afr J Med Sci*. 2010; 39(1):37-40.
20. Adisa J, Musa A, Yima U, Egbujo E. *Helicobacter Pylori* Associated Gastritis In North-Eastern Nigeria: A Histopathologic Study. *E-IntSci Research J*. 2011; 3(1):1-4.

