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Histopathological Pattern of Endoscopic Gastric Biopsy in a District Hospital in Nigeria: A Review of 118 Consecutive Cases

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Abstract Background: The use of endoscopes for visualization of gastric mucosa has improved over times and in taking biopsy for histology. The study sets out to determine the histopathological pattern of gastritis in dyspeptic patients and correlate the histological detection of H. pylori with that of urease breath test (UBT). Method: Prospective study of 118 consecutive patients with chronic dyspepsia who underwent upper gastrointestinal endoscopic examination and UBT using heliprobe. Biopsy of gastric antrum were taken at endoscopy and sent for histopathological analysis. Routine H&E and Giemsa stains were used. Results were recorded and analysed on the basis of sex, age, histology and UBT for H. pylori. Result: There were 118 patients who had endoscopy comprising 58 males and 60 females with male to female ratio of 1:1. Histology revealed varying degrees of chronic gastritis with or without H. pylori, activity, metaplasia, ulceration and dysplasia. Sixty eight (61%) of our patients were positive for H. pylori histologically. Of the first consecutive 66 patients, histology showed 38(57.6%) positive and 28(42.6%) negative; UBT, 46(69.6%) were positive for H. pylori and 20(30.4%) negative. There was a strong correlation between the true positive and true negative patients for the first 66 consecutive cases for both histology and UBT based diagnosis for detecting H. pylori. (Correlation coefficient=0.862, p=0.01). Conclusion: The study showed that histology and UBT are both useful for H. pylori detection. Large multi centre studies should be done to adopt the non-invasive UBT in resource poor economies for the eradication of H. pylori.

Keywords: *endoscopy, gastric mucosa, histology, UBT, H. pylori*

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1. Introduction

Helicobacter pylori infection can be diagnosed by invasive techniques requiring endoscopy and biopsy (e.g. histological examination, culture and rapid urease test) and by non-invasive techniques, such as serology, the urea breath test, urine/blood or detection of H. pylori antigen in stool specimen [1,2].

Injury to the gastric mucosa is associated with epithelial cell damage and regeneration and gastric lesions are frequent causes of clinical disease worldwide [3]. *H. pylori* is the principal agent that starts the cascade of histological events ranging from chronic gastritis to carcinoma through mucosal atrophy, intestinal metaplasia and epithelial dysplasia [4,5,6].

The choice of appropriate test depends on the pre-test probability of infection, the characteristics of the test being used and its cost-effectiveness [2]. Some non-invasive tests detect active infection e.g. the urea breath test and the stool antigen test while others are markers of exposure to H. pylori (e.g. serology or urine) [1].

With these in mind, the study sets out to determine the histopathological pattern of gastritis in dyspeptic patients and correlate the histological detection of *H. pylori* with that of urease breath test (UBT). This study is unique for this environment as it is one of the few studies on *H. pylori* gastritis correlating histological diagnosis with urease breath test (UBT).

2. Materials and Methods

This study was a prospective study of 118 consecutive patients who were suffering from chronic dyspepsia and underwent upper gastrointestinal endoscopic examination in a private facility and the samples sent to our laboratory at the Asokoro District Hospital, Abuja, Nigeria between July, 2010 and December 2011. The samples were taken from the gastric antrum and sent in 10% buffered formalin to the Pathology department of the hospital for the pattern

of gastritis and presence or otherwise of *H. pylori* using Haematoxylin & eosin (H&E) and Giemsa stains respectively.

The first consecutive 66 patients that had upper GI endoscopy also had UBT for *H. pylori* using Heliprobe® supplied by Biofem pharmaceuticals.

Results were recorded and analysed on the basis of sex, age, histopathological diagnosis and UBT for *H. pylori*. The results obtained from 66 patients who had UBT were correlated with histological method of detecting H. pylori using the Pearson correlation. The statistical analysis was done by SPSS version 17 and t-test and chi square used to compare means and significance set at $p < 0.05$.

3. Results

There were 118 patients who had endoscopy comprising 58 males and 60 females between July 2010 and December 2011. The age range of our patients was 23-82 years and mean of 42 years. This constitutes 7.8% of the total biopsies received from our laboratory during the study period. Thirty two (27.11%) were *H pylori* with chronic gastritis, 16(13.55%) were *H. pylori* with chronic active gastritis and ulceration and moderate chronic active gastritis each respectively. Moderate chronic gastritis constituted 14(11.86%) of cases and *H. pylori* with chronic gastritis, 6(5.08%), mild chronic gastritis and metaplasia constituting 8.47% of cases were seen and 4 cases of severe chronic gastritis (3.89%). Two cases constituting 1.69% each were seen for *H. pylori* with chronic gastritis with ulceration, H. pylori with chronic active gastritis with dysplasia, severe chronic gastritis with ulceration, severe chronic active gastritis with ulceration, ischaemic gastritis with ulceration, gastric adenocarcinoma and normal gastric histology (Table 1).

Table 1. Histopathological pattern of endoscopic gastric biopsies

Histopathological diagnosis	No of cases	Percentage
H. pylori with chronic gastritis	6	5.08
H. pylori with chronic active gastritis	32	27.11
H. pylori with chronic gastritis and ulceration	2	1.69
H. pylori with CAG and ulceration	16	13.55
H. pylori with CAG and metaplasia	10	8.47
H. pylori with CAG and dysplasia	2	1.69
Moderate chronic active gastritis	16	13.55
Mild chronic gastritis	6	5.08
Moderate chronic gastritis	14	11.86
Severe chronic gastritis	4	3.38
Severe chronic gastritis with ulceration	2	1.69
Severe CAG with ulceration	2	1.69
Ischaemic gastritis with ulceration	2	1.69
Adenocarcinoma	2	1.69
Normal	2	1.69
TOTAL	**118**	**100**

Of the first consecutive 66 patients, 38(57.6%) were positive for *H. pylori* histologically and 28(42.6%) were negative (Table 2). Sixty eight (57.6%) of our patients were positive for H. pylori of the total of 118 patients who had endoscopy for chronic dyspepsia (Table 3).

Table 2. The correlation of UBT with Histopathology in the detection of H. pylori for the 33 consecutive gastric biopsies

Histopathology			UBT		
Test	No of cases	%age	Test	No of cases	%age
Negative for H pylori	28	42.4	True negative	18	27.3
Positive for H. pylori	38	57.6	False negative	2	3.0
Total	**66**	**100**	False positive	4	6.1
			True positive	42	63.6
			Total	**66**	**100**

Pearson correlation value= .826** (significant at the 0.01level (2-tailed).

Table 3. Result of 66 consecutive tests for UBT compared with Histology

Histopathological diagnosis	Histology		UBT	
	No of cases	%age	Positive	%age
H pylori associated chronic gastritis	8	12.1	8	12.1
H pylori associated CAG	22	33.3	20	30.3
H pylori chronic gastritis with ulceration	2	3.0	2	3.0
H pylori CAG with ulceration	12	18.2	12	18.2
Moderate CAG	6	9.1	2	3.0
Moderate chronic gastritis	14	21.2	2	3.0
Severe chronic gastritis	2	3.0	0	0.0
TOTAL	**66**	**100**	**46**	**69.7**

Of the 66 patients tested using UBT, 46(69.6%) were positive for *H. pylori* while 20(30.4%) were negative (Table 2). When subjected to statistical correlation using the Pearson correlation coefficient, there was a strong correlation (0.862) between the true positive and true negative patients for the first 33 consecutive cases for both histology and UBT based diagnosis for detecting *H. pylori* (Table 2).

Table 4 shows the severity of acute inflammation (neutrophil polymorphs) which is a measure of activity associated with *H. pylori* gastritis: 42(61.8%) are associated with mild activity while 26(38.2) are associated with moderate activity. The degree of activity in non *H. pylori* associated gastritis is as shown in the same table.

Table 4. Degree of activity in H pylori associated gastritis

	H. pylori gastritis		Non H. pylori gastritis	
Degree of activity	**No of cases**	**%age**	**No of cases**	**%age**
Nil	-	-	14	31.8
Mild	42	61.8	10	22.7
Moderate	26	38.2	12	27.3
Severe	0	0.0	8	18.2
Total	**68**	**100**	**44**	**100**

Figure 1 and Figure 2 show the microscopic features of chronic active gastritis and H. pylori gastritis respectively.

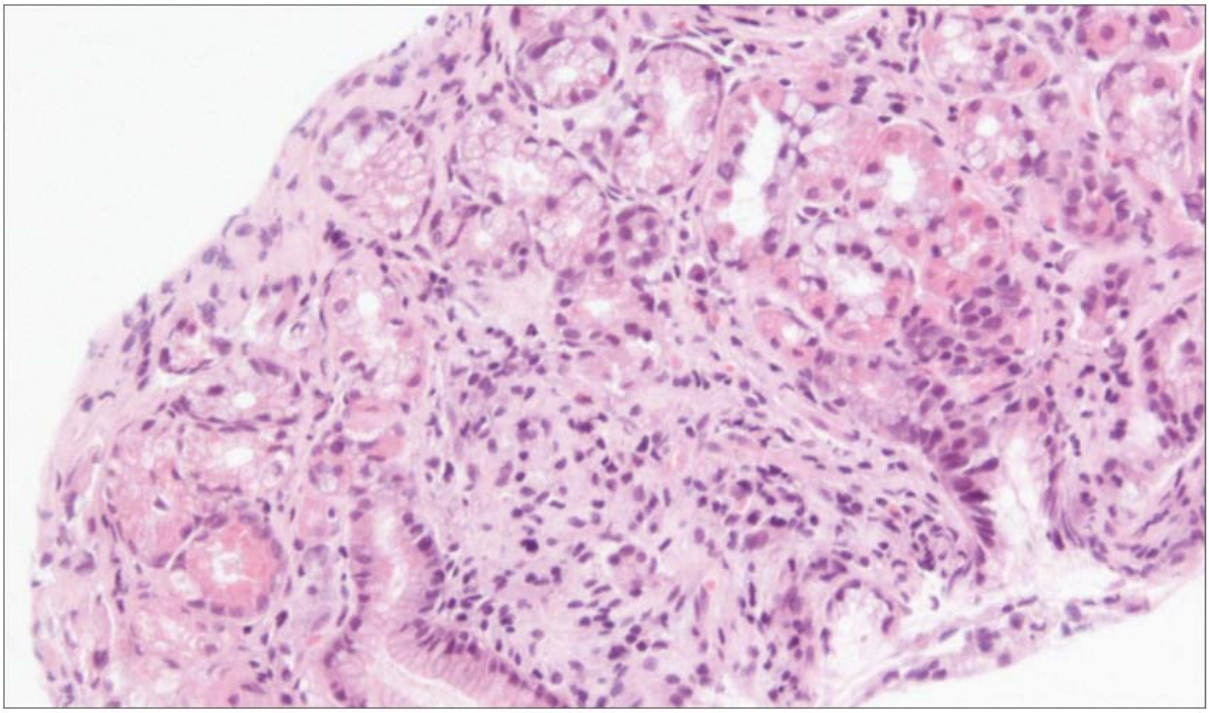

Figure 1. Section shows gastric antral mucosa biopsy with chronic active gastritis (H&E x100)

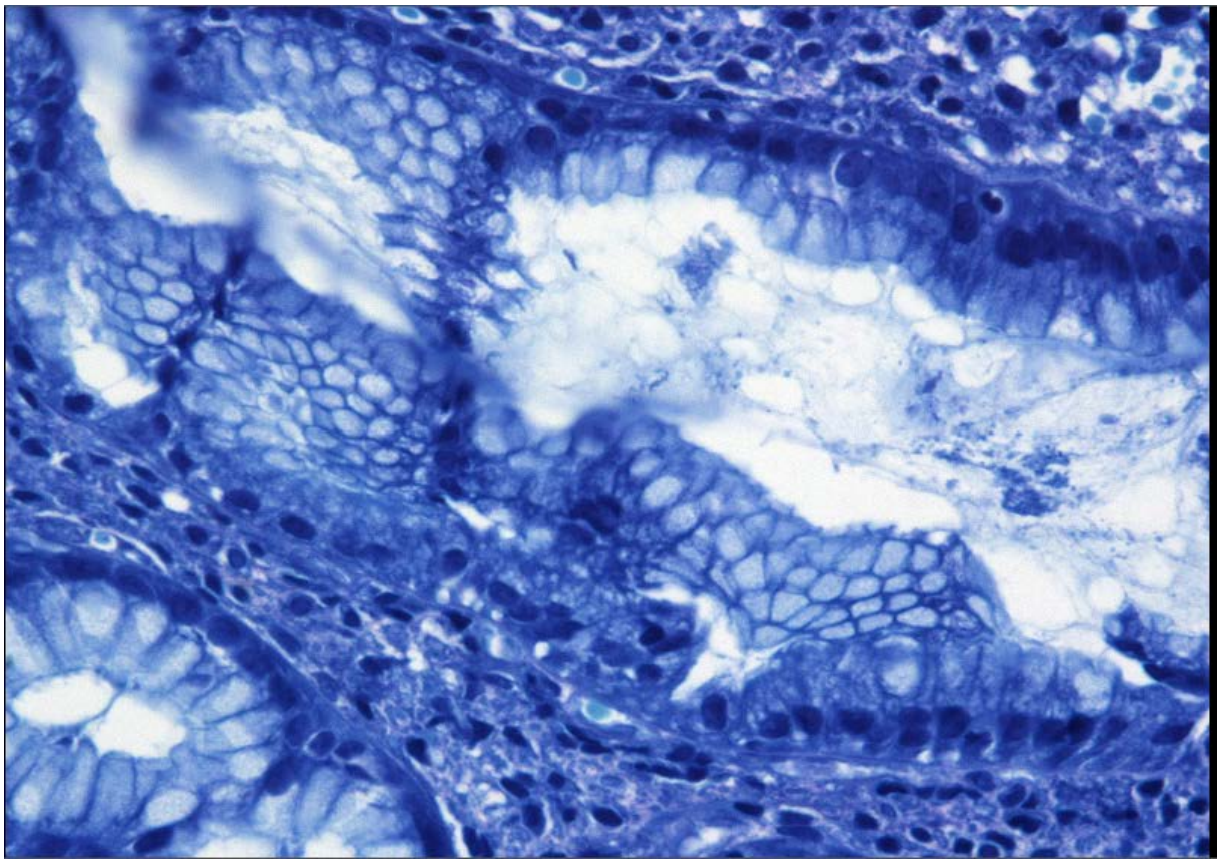

Figure 2. Section of gastric antral mucosa demonstrating H. pylori in the crypts of the gland. (Giemsa stain x400)

4. Discussion

This study shows a high rate of *H. pylori* associated gastritis (61.0%). The most important aetiologic association with chronic gastritis is chronic infection by the bacillus *Helicobacter pylori*. This link was discovered in 1983, when the bacterium was called *Campylobacter pyloridis* [7]. Compared with work done in a Sardjito General Hospital, Yogyakarta, Indonesia [8] and Ibadan, Nigeria [9] this is high compared with their 22.8% and 22.4% respectively. Our figure compared favourably with Shousha et al [10] among Yemins patients (94%); Rubio et al [11] among Mexicans (66%) and Holcombe et al (80%) in Maiduguri, Nigeria [12].

Also when compared with the Indonesia work, *H. pylori* with chronic active gastritis constituted the highest histopathological pattern 32 (27%) of all the gastric biopsy

and 47% of *H. pylori* associated gastritis. This is against chronic superficial gastritis constituting 60.87% recorded in Indonesia for all the biopsies as highest histopathological pattern and 8.90% of cases of chronic superficial gastritis as highest among *H. pylori* positive cases. Most classification systems for gastritis distinguish acute, short term from chronic, long term diseases [13]. The term acute and chronic were used to describe the type of inflammatory cell infiltrates. Acute (active) inflammation is usually associated with neutrophil polymorhs infiltration while chronic inflammation is characterised by mononuclear cells mainlylymphocytes, plasma cells and macrophages. A practical clinicopathologic framework for the classification of gastritis and gastropathy based on these factors can be seen in the work done by Dixon et al [14].

Outside chronic gastritis, *H. pylori* also plays a critical role in other major gastric and duodenal diseases. Peptic ulcer disease is now approached as an infectious disease that can be treated by antibiotics because of *H. pylori* involvement. It also increases the risk for developing gastric carcinoma by five to six folds [15]. It causes chronic gastritis followed by atrophy, intestinal metaplasia, dysplasia and carcinoma [5,16,17]. The sequential alterations depend on both the presence of bacterial proteins and the host immune response.

Two cases of gastric adenocarcinoma was reported in the series and it is not associated with *H pylori*. Not all *H. pylori* infections will cause cancer and the vast majority of individuals infected with this bacterium will not develop cancer. Environmental influences may be critical in gastric carcinogenesis [18].

A number of diagnostic tests, invasive and non invasive have been developed for *H. pylori* detection. Amongst the non invasive tests are serologic test for antibodies, faecal bacteria detection and urea breath test [19]. The urea breath test is based on the generation of ammonia by bacteria urease. Invasive test employs the identification of *H. pylori* in gastric tissue. Detection methods in gastric tissue include visualization of the bacteria in histologic sections, bacteria culture, rapid urease test and bacterial DNA detection by the polymerase chain reaction [14].

For this study, we compared urease breath test (non invasive) with visualization of the bacteria in histologic section using H&E and special stain Giemsa. Sixty eight (57.6%) patients of the total 118 cases were positive for *H. pylori*. When a correlation study was done for the first 66 consecutive samples using both urease breath test and histopathological diagnosis, a strong correlation was found between these two diagnostic methods. The import of this especially for this environment where access to invasive procedure like gastric endoscopy with biopsy is both not readily accessible and expensive lies in the fact that a non-invasive and relatively less expensive method can be used for *H. pylori* detection [1,2]. This is significant because chronic gastritis with *H. pylori* usually improves when treated but its relapses are associated with reappearance of the organism.

Current treatment modality includes antibiotics and proton pump inhibitors [20]. Prophylactic and therapeutic vaccine development is still in the early research stage.

5. Conclusion

The study supported the use of urease breath test and histological method for the detection of *H. pylori*. We also infer from the study that the non invasive and less expensive UBT could be adopted in the developing economies where endoscopists and pathologists may not be readily available. We recommend that a more comprehensive study comparing different detection methods should be done in the future with a view to adopting a cheap, sensitive and non-invasive method for the detection of *Helicobacter pylori*.

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