IBOM MEDICAL JOURNAL
Vol.15 No.3 September, 2022. Pages 209 - 214
www.ibommedicaljournal.org

Histopathological pattern of endoscopic gastric biopsies in dyspeptic patients in a Nigerian population

Amadi D. C[1], *Raphael S*[2]

[1]*Department of Histopathology, Federal Medical Center, Owerri, Imo State, Nigeria.*
[2]*Department of Pathology and Forensic Medicine, University of Abuja and University of Abuja Teaching Hospital, Gwagwalada, Abuja, Nigeria.*

Abstract

Background and Objective: Dyspepsia is one of the most common complaints encountered in the general outpatient and gastroenterology clinics in Nigeria. Histopathological assessment of endoscopic gastric mucosa biopsy is crucial to delineate the exact cause of dyspepsia to guide patients' management. This study aimed to determine and document the histopathological basis of dyspepsia among dyspeptic patients at our facility.
Material sand Methods: This was a three year descriptive retrospective study and the materials consisted of all gastric endoscopic biopsies taken from clinically diagnosed dyspeptic patients sent to the Department of Histopathology of the Federal Medical Centre, Owerri, Nigeria.
Results: The biopsies were from 64 (53.2%) male patients and 56 (46.8%) female patients, giving a male to female ratio of 1.14:1. The age range of the patients was 28-82 years with a mean of 56 years at presentation. Helicobacter Pylori (H. Pylori) bacilli were identified in the samples of 42 (35%) patients but absent in samples of 78 (65%) patients. The histopathological pattern of the aetiological basis of dyspepsia in this study consisted of gastritis (96, 80%), functional (17, 14.2%), adenocarcinoma (4, 3.3%) and polyps (3, 2.5%). H. Pylori bacilli were seen only in patients with gastritis (42/96, 43.8%), and it affected 19 (45.2%) male patients and 23 (54.8%) female patients. Chronic active H. Pylori associated gastritis (24, 25%) was the most common form of gastritis seen during the study period.
Conclusion: The main organic cause of dyspepsia in our setting was chronic gastric followed in the distant by gastric adenocarcinoma and polyp. Dyspepsia and H. Pylori associated gastritis did not show a significant gender predilection.

Keywords: Dyspepsia, Endoscopic biopsy, histopathological pattern, gastritis, functional dyspepsia, adenocarcinoma.

Introduction

Postprandial fullness, early satiety, and epigastric pain or burning occurring singly or in combination for three months within the initial six months of symptom onset constitute dyspepsia according to the Rome III criteria.[1] Dyspepsia can be functional or organic, the former which is more commonly encountered is not associated with an identifiable underlying disease process after thorough investigation, while the latter is caused by organic diseases in the stomach, oesophagus, duodenum, and occasionally systemic diseases.[1] Infection of the stomach by Helicobacter Pylori (H. pylori), a gram negative spiral microaerophilic bacilli and its sequelae; chronic gastritis, peptic ulcer, gastric carcinoma and gastric B-cell lymphoma are the major organic causes of dyspepsia.[1,2] Others include

Corresponding Author: Dr. Solomon Raphael

Department of Pathology and Forensic Medicine,
University of Abuja and University of Abuja Teaching Hospital
Gwagwalada, Abuja, Nigeria.
E-mail: soji2000solo@yahoo.com, Phone: +2348179958396

gastro esophageal reflux disease (GERD), gastric polyps, side effects of nonsteroidal anti-inflammatory medications, alcohol, radiation, and systemic diseases (inflammatory bowel disease, amyloidosis and graft vs. host disease).[2,3]

Dyspepsia is a common problem globally, affecting 40% of the general population and it is one of the most common complaints encountered in the general outpatient and gastroenterology clinics in Nigeria.[3,4] It causes reduction in quality of life and loss of man hours leading to economic loss.[3] H. Pylori infection rates exhibit national and regional variations, predicated on poverty levels, educational status, household settings, ethnicity and rural-urban population distribution.[2] Consequently, dyspeptic symptoms associated with H. Pylori infection vary from one region to another within the same country.

Empirical triple drug treatment of suspected organic dyspepsia is therefore based on the central role of H. Pylori in the aetiopathogenesis of this type of dyspepsia. However, persistent of symptoms warrants careful evaluation to ascertain the cause of the dyspepsia.[3] Upper gastrointestinal (GI) or gastric endoscopy is the recommended first line procedure in the work up of patients with dyspepsia.[1,5] Upper GI or gastric endoscopy allows visualization of the upper GI or stomach to identify and localize a lesion, deliver direct treatment and biopsy mucosa tissue for histopathological assessment. The histopathological assessment of gastric mucosa biopsies is crucial to classify the dyspepsia as functional or organic, and in the latter case, delineate the exact aetiology to guide patients management.[1,2,5] To increase the diagnostic yield of sampled gastric mucosa, it is advised that sampling and histopathology reporting should conform to the updated Sydney System agreed by the consensus of expert Gastroenterologists and Pathologists.[6,7] This system advocated that two sets of biopsy (tissue cores) should be taken from the gastric antrum and corpus in addition to specimen (s) from any other part of the stomach, especially the incisura angularis with tissues from each site put in the same labelled specimen containers.[7] It emphasized providing reproducible and clinically useful histological diagnoses that combine topographical, morphological, and etiological information.[2,7,8] Such diagnoses should differentiate H. Pylori associated gastritis from non H. Pylori associated (chemical, autoimmune, etc) gastritis in addition to providing morphological information of chronic inflammation, neutrophil infiltrates (activity), glandular/mucosa atrophy, ulcer and intestinal metaplasia indicated against the site of the stomach from which the biopsy was taken.[2,7,8]

Endoscopes have been available at our facility for about five years now and the number of upper GI, especially gastric endoscopy and biopsy performed on clinically diagnosed dyspeptic patients has increased over the years. However, there are no data on the histopathological changes and pattern of lesions of endoscopic mucosal biopsies from dyspeptic patients in our facility. This study evaluated gastric endoscopic mucosal biopsies from dyspeptic patients at our facility and documented the histopathological pattern and spectrum of gastric lesions underpinning dyspepsia, their relative frequencies and compared the findings with similar studies within and outside Nigeria.

Materials and methods

This was a three year descriptive retrospective study from January 1, 2015 to December 31, 2017. The materials for the study consists of all gastric endoscopic biopsies taken from clinically diagnosed dyspeptic patients sent to the Department of Histopathology of Federal Medical Centre, Owerri, Nigeria. This included any number of gastric biopsies whether or not they met the revised Sydney System guideline for sampling gastric mucosal tissue. The biopsies were fixed in ten percent (10%) neutral buffered formalin and processed using automatic tissue processor, from which formalin-fixed paraffin embedded (FFPE) tissue blocks were made. Two sets of slide sections (3-4μm) were made from the FFPE tissue blocks for each sample; one of the slides were stained with Haematoxylin and Eosin (H&E), and the other stained with modified Giemsa for H. pylori. These slides were reviewed and histological diagnoses rendered. The presence of metaplasia was confirmed by special stains such as PAS and Alcian blue. For each patient, we collated the age, sex, clinical presentation and histopathological diagnoses from the departmental registers, request forms and duplicate pathology reports. We excluded cases where the paraffin embedded blocks were not

available for reprocessing, are not properly preserved, the slides were not accessible or have been tampered with.

The ensuing data was entered into and analysed using IBM statistical package for social sciences version 20.21 (IBM SPSS version 20.21). Continuous variables were summarized using range and mean ± standard deviation, while categorical variables presented as percentages were determined using descriptive statistics. Data were displayed in form of frequency tables while photomicrographs of representative lesions were included. Statistical significance was set at p-value < 0.05. Ethical clearance for this study was obtained from the Research Ethical Committee of our centre.

Results

Endoscopic gastric mucosal biopsies taken from 120 dyspeptic patients qualified for inclusion during the study period. All of the samples were stained with H&E and modified Giemsa stains while samples from twenty one (21) patients were stained with special stains (PAS and Alcian blue) to confirm metaplasia. The biopsies were from 64 (53.2%) male patients and 56 (46.8%) female patients, giving a male to female ratio of 1.14:1. The age range of the patients was 28-82 years with a mean of 56 years at presentation.

Helicobacter Pylori (H. Pylori) bacilli were identified morphologically in the samples of 42 (35%) patients but absent in samples of 78 (65%) patients. The histopathological pattern of the aetiological basis of dyspepsia in this study depicted in figure 1 consist of gastritis (96, 80%), functional (17, 14.2%), adenocarcinoma (4, 3.3%) and polyps (3, 2.5%). Helicobacter Pylori bacilli were seen only in patients with gastritis (42/96, 43.8%), and the gender distribution show that it affected 19 (45.2%) males patients and 23 (54.8%) female patients.

Classification of the histopathological diagnoses according to the updated Sydney System showed there were 24 (20%) cases of chronic active H. Pylori associated gastritis (CAG), 10 (8.3%) cases of chronic H. Pylori associated gastritis with ulceration, 4 (3.3%) cases each of chronic active H. Pylori associated gastritis with metaplasia and chronic H. Pylori associated gastritis. There were 17 (14.2%) cases of chronic active non-H. Pylori

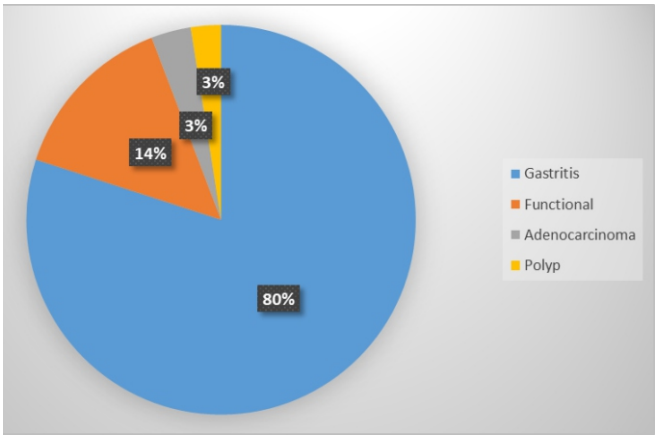

Figure 1: Frequency distribution of histopathological causes of dyspepsia

Table 1: Frequency distribution of histological pattern of gastric biopsy pathologies

Sydney histopathological diagnoses	Frequency, *n* (%)
Chronic active H. Pylori associated gastritis	24 (20.0%)
Chronic H. Pylori associated gastritis with ulceration	10 (8.3%)
Chronic active H. Pylori associated gastritis with metaplasia	4 (3.3%)
Chronic H. Pylori associated gastritis	4 (3.3%)
Chronic active non H. Pylori associated gastritis	17 (14.2%)
Chronic non H. Pylori associated gastritis	7 (5.8%)
Chronic non H. Pylori associated gastritis with metaplasia	9 (7.5%)
Chronic non H. Pylori associated gastritis with atrophy and metaplasia	6 (5.0%)
Chronic non H. Pylori associated gastritis with ulcer, dysplasia and metaplasia	2 (1.7%)
Chemical gastritis with erosion and ulceration	13 (10.8%)
Adenocarcinoma	4 (3.3%)
Polyps	3 (2.5%)
Normal gastric tissues/Functional dyspepsia	17(14.2%)
Total	**120 (100%)**

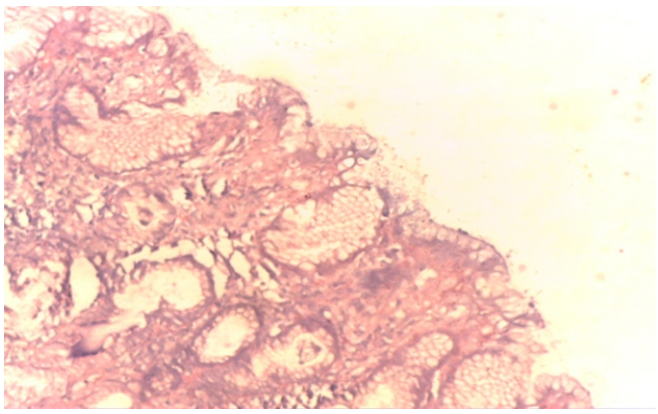

Figure 2: Photomicrograph of body of the stomach showing chronic active gastritis with Helicobacter Pylori in the mucosal lining on Giemsa stain, X200.

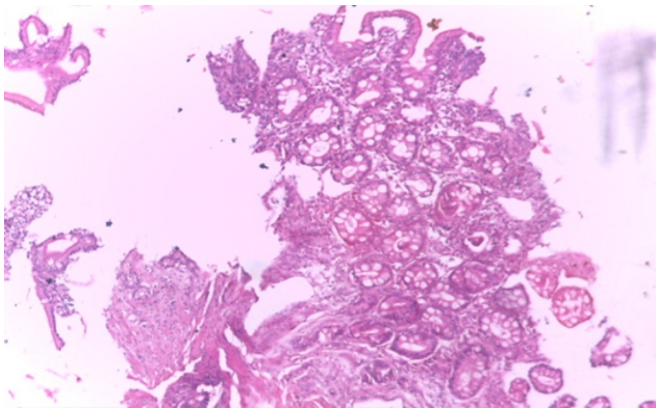

Figure 3: Chronic Gastritis affecting the gastric body, highlighting intestinal metaplasia characterized by glands lined by goblet cells, X100.

associated gastritis, 7(5.8%) cases of chronic non H. Pylori associated gastritis, 9 (7.5%) cases of chronic non H. Pylori associated gastritis with metaplasia, 6 (5%) cases of chronic non H. Pylori associated gastritis with atrophy and metaplasia, 2 (1.7%) cases of chronic non H. Pylori associated gastritis with ulcer, dysplasia and metaplasia. In addition, there were also 13 (10.8%) cases of chemical gastritis with erosion and ulceration, 4 (3.3%) cases of adenocarcinoma, 3 (2.5%) cases of polyps, while 17(14.2%) of the cases were essentially normal gastric tissues (table 1).

Discussion

Dyspepsia affectation in this study did not show a sex predilection as demonstrated by a male to female ratio of 1.14:1. This lack of significant influence of gender on the occurrence of dyspepsia has been recorded in other series in Nigeria and Africa.[4,8-10] Tanko et al.[4] documented a male to female ratio of 1:1 in Jos, Duduyemi et al.[8] recorded a male to female ratio of 1.0:1 in Abuja, and Mustapha et al.[9] recorded a ratio of 1.1:1 in Maiduguri, Nigeria while Oling et al.[10] recorded 1:1.4 in Kamplaa, Uganda. It must be stated though that Imam et al[11] in Kano, Nigeria and Zaltman et al[12] in Brazil reported a slightly higher male preponderance with a male to female ratio of 1.8: 1 and 2.2:1 respectively. While these studies like ours are hospital based histopathological analyses, and so may not be representative of the gender distribution of dyspepsia in our population, there is no known definitive relationship between gender and Helicobacter Pylori (H. Pylori) infection in the literature.[13]

Morphology aided by special stain in form of modified Giemsa stain showed that H. Pylori was associated with 35% of all the lesions in dyspeptic patients and with 44% of all the gastritis related dyspepsia in our study. This rate of H. Pylori association with dyspepsia and gastritis mirrored the report in Uyo, Nigeria (33.3%)[14] and Kampala, Uganda (36%).[10] Our rate is higher than the 16% and 25% reported in Kano, Nigeria[11] and Kharian, Parkistan[15] respectively but it is relatively low when compared with similar histopathological reports in Maiduguri (89.1%),[9] Iran (86.8%),[16] Abuja (61%),[8] Benin City (55.6%)[17] and Ibadan (52.4%).[18] Far lower figures were reported in Yogyakarta, Indonesia (22.4%).[19] Our relatively low rate of H. Pylori association with dyspepsia and gastritis in this study is not in keeping with the known relationship between the epidemiological factors of high H. Pylori infection in developing countries and chronic gastritis.[2] While the exact reason for this finding is not known, we propose that the rampant indiscriminate use of antibiotics and painkillers among our people to self-manage pain related medical conditions like dyspepsia before seeking medical help in decolonizing the gastric mucosa may be a factor in our low rate.

Chronic gastritis (80%, figure 1) is the most common histopathological basis of dyspepsia in our study consistent with most previous studies in Nigeria and Africa.[4,8,10,11,17,18,20,21] The reported range

of relative frequencies of gastritis in these series was 60.5 -95%.[4,8,11,14,17,18,18,20] Oesophagitis was the most common lesion reported in the study in Maiduguri and Benin as reported by Mustapha et al.[9] and Ugiagbe et al.[22] respectively. The distribution of the gastritis shows that chronic active H. Pylori associated gastritis (depicted in figure 2), representing 20% was the most prevalent subtype of gastritis seen in our study. This finding is consistent with previous studies in Nigeria,[8,14,17,18,20] Africa[10] and Indonesia.[19] The series from Abuja8 reported that chronic active H. Pylori associated gastritis constituted 33.3% while it represented 27% in the Indonesian work.[19]

Dyspepsia had no histomorphologically identified aetiology, that is, it was functional in 14.2% in our study. This rate is similar to two previous endoscopy based works in Benin City, where functional dyspepsia were reported to represent 15.4% and 12.4% respectively.[22,23] Another such study in Ibadan found functional dyspepsia accounted for 17.2% of all the cases of dyspepsia studied.[21] Histopathology based studies in Nigeria reported variable functional dyspepsia findings. Duduyemi et al.[8] in Abuja found they constituted only 1.69% while Nwokediuko et al.[20] in Enugu recorded that they accounted for 49.3% of all the dyspeptic patients whose biopsies were examined histologically. Higher prevalence rates were reported in western population (60%)[3,24] and the mixed population in Eastern Cape province in South Africa (33.3%).[25] The reason for the relatively low rates of functional dyspepsia in our work and other histopathology based studies in the country relative to the western world is not known. It has been speculated that non biopsy of patients with "normal findings" at endoscopy to save them the cost associated with histology in our setting may be a factor.[22]

Gastric adenocarcinoma (4/120, 3.3%) and polyp (3/120, 2.5%) were the relatively distant causes of dyspepsia in our study, and understandably were absent in most of the referenced works. In the Jos series[4] gastric carcinoma made up 3% of all the patients studied while they accounted for 1.69% of Abuja study.[8] Only three cases (2.7%) of gastric adenocarcinoma were seen among dyspeptic patients in the Kampala series.[10] This paucity of dyspepsia due to gastric adenocarcinoma is understandable given the general rarity of gastric adenocarcinoma in our setting and the fact that it presents more commonly in advanced stage with outlet obstruction, weight loss and anaemia.[2]

The limitations of this study include that it is a retrospective single institution study instead of a community study which may be more representative. Also, some dyspeptic patients may have refused endoscopy and biopsy because of the cost implications.

Conclusion

The main organic cause of dyspepsia in our setting was chronic gastric followed in the distant by gastric adenocarcinoma and polyp. Dyspepsia and H. Pylori associated gastritis did not show a significant gender predilection. The gender profile and histopathological pattern of causes of dyspepsia in our study is broadly congruent with previous studies in Nigeria and Africa, but in our centre, Helicobacter Pylori frequency rate is relatively lower in comparison.

Financial support and sponsorship

No financial support was received for this work

Conflicts of interest

None declared

References

1. Shaukat A, Wang A, Acosta RD, David H, Chandrasekhara BV, Krishnavel V. et al. The role of endoscopy in dyspepsia. American society of Gastroenterologists guideline. 2015; 82 (2): 227-232.
2. Jerrold RT. The Stomach. In: Kumar V, Abbas AK, Aster JC, editors. Robbins and Cotran Pathologic Basis of Diseases. 9th ed. Philadephia: Elsevier Saunder; 2015. p. 1474-1506.
3. Ford AC, Moayyedi P. Dyspepsia. Curr Opin Gastroenterol. 2013; 29(6): 662-8.
4. Tanko MN, Echejoh GO, Mandong BM, Manasseh AN, Malu AO. Gastric histopathological findings in mucosal biopsies of symptomatic patients in Jos central Nigeria. Nigerian J Med. 2007; 16 (2):113-8.
5. Tytgat GNJ. Role of endoscopy and biopsy in the work up of dyspepsia. Gut. 2002; Suppl 4

(50): 13.
6. Stolte M, Meining A. The updated sydney system: Classification and grading of gastritis as the basis of diagnosis and treatment. Canadian J Gastroenterol: 2001; 15(9): 591-8.
7. Lash JG, Genta RM. Adherence to the Sydney System guidelines increases the detection of Helicobacter gastritis and intestinal metaplasia in a 400738 sets of gastric biopsies. Alimentary Pharmacology and Therapeutics. 2013; 4 (38): 424-431.
8. Duduyemi BM, Ojo BA, Olaomi OO, Atiba AS. Histological pattern of endoscopic biopsy in a district in Nigeria a review of 118 consecutive cases. Am J Med Bio Res. 2014; 2(3):83-86.
9. Mustapha YS, Bolori M, Ajayi N, Nggada H, Pindiga U, Gashau W, Khalil M. Endoscopic findings and frequency of Helicobacter Pylori Infection Among Dyspeptic Patients in Northern-Eastern Nigeria. The Internet Journal of Infectious Diseases.2006; 1: (6):1-5.
10. Oling M, Odongo J, Kituuka O, Galukande M. Prevalence of Helicobacter pylori in dyspeptic patients at a tertiary hospital in a low resource setting. BMC Research Notes. 2015; 8:2.
11. Imam MI, Raphael S. Histopathological Pattern of Gastric Biopsies: Experience in a Tertiary Institution in Northern Nigeria and Review of Literature. J Med & Bas Sci Res. 2022; 3(1):47-52.
12. Zaltman C, Souza HS, Castro ME, Sobral MDE, Dias PC, Lemos VJ. Upper gastrointestinal bleeding in a Brazillian hospital: a retrospective study of endoscopic records. Arc Gastroenterol 2002; 39: 74-80.
13. Repogle ML, Glaser SL, Hiatt RA, Parsonnel J. Biologic sex as a risk factor for Helicobacter Pylori infection in healthy young adults. Am J Epidemiol. 1995; 142: 856-63.
14. Nwafor CC, Nwafor NN, Etuk EB, Kanu O. Histopathological spectrum of gastrointestinal lesions seen in the University of Uyo Teaching Hospital, South –South Nigeria. Ann Trop Pathol.2019; 10:27-33.
15. Tariq S, Muhammad H, Nighat S, Humaira T, Muhammad A, Kamran NA, et al. Histopathological Analysis of Gastric Mucosal Biopsies in Non Ulcer Dyspepsia. Park Armed Forces Med J. 2016: 1-5.
16. Shokrzadeh L, Baghaei K, Yamaoka Y, et al. Prevalence of Helicobacter pylori infection in dyspeptic patients in Iran. Gastroenterol Insights. 2012; 4:24–7.
17. Udoh MO, Obaseki DE. Histopathological evaluation of H. Pylori associated gastric lesions in Benin City, Nigeria. Eat Afri Med J. 2012; 89 (12): 408-13.
18. Jemilohun AC, Otegbayo JA, Ola SO, Oluwasola AO, Akere A. Prevalence of Helicobacter pylori among Nigerian patients with dyspepsia in Ibadan. Pan Afr Med J. 2010; 6: 18.
19. Nurdjanah S, Bayupurnama P. Histopathological pattern of gastric biopsies of Helicobacter pylori positive patients in Sardjito General Hospital, Yogyakarta, Indonesia. Journal of Gastroenterology & Hepatology, 2000; 12(15): 1440-1746.
20. Nwokediuko SC, Okafor OC. Gastric Mucosa in Non-ulcer Dyspepsia: A Histopathological Study of Nigerian Patients. The Internet J Gastroenterol. 2006; 5(2):1-8.
21. Adeniyi BA, Otegbayo JA, Lawal TO, Oluwasola AO, Odaibo GN, Okolo C, et al. Prevalence of Helicobacter pylori infection among dyspepsia patients in Ibadan, South West Nigeria. African Journal of Microbiology Research. 2012; 6 (14): 3398-3402.
22. Ugiagbe RA, Umuemu CE. Non-ulcer dyspepsia: An endoscopic review. Afr J Med Health Sci 2013; 12:6-9.
23. Ugiagbe EE, Ugiagbe RA. Endoscopic and histological evaluation of upper gastrointestinal lesions in a tertiary health institution in Benin City. J Med Biomed Res 2011; 10:52-6.
24. Richter JE. Dyspepsia: Organic causes and differential characteristics from functional dyspepsia. Scand J Gastroenterol Suppl 1991; 182:11-6.
25. Tanih NF, Okeleye BI, Ndip RN (2010) Helicobacter pylori prevalence in dyspeptic patients in the Eastern Cape province—race and disease status. Afr Med J 100:734–737.