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Comparative Prevalence of Asymptomatic Intestinal Bleeding and *Helicobacter pylori* Infection among Adolescents in South Eastern Nigeria.

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ABSTRACT

Helicobacter pylori has been deeply implicated in diseases of the gastrointestinal tract and duodenal ulcer. Proper diagnosis and determination of risk factors for acquiring this infection is pertinent in containing it. The study was aimed at determining the prevalence of asymptomatic intestinal bleeding and *Helicobacter pylori* infection among adolescents of Ebonyi State, Nigeria. The research was a cross sectional study, carried out among students of Ebonyi State University. Samples were randomly collected from 420 students. The blood samples were analyzed for the presence of *Helicopter pylori* antibody by chromatographic immunoassay while the faecal samples were analyzed for the presence of blood using faecal occult blood test kit. Out of the 420 subjects, 349 were positive of either *H. pylori* antibody, intestinal bleeding or both while 71 were negative, 303 (72.1%) were *Helicopter Pylori* seropositive and 134 (31.9%) were positive of faecal occult blood test, whereas, 88(21.0%) were positive of both *Helicopter pylori* and faecal occult blood. The males were found to be significantly more exposed to *Helicopter pylori* (p<0.05). Participants within the ages of 20 years and below were found to be significantly more susceptible to *Helicobacter pylori* infection (p<0.05). A significant association was found between sources of drinking water as risk factor and seropositivity of *Helicopter pylori* antibody (p<0.05). In conclusion, the high prevalence of positivity recorded in this study constitute a problem of public health concern. The need for proper screening and diagnosis are therefore advocated for better management of intestinal bleeding.

Keywords: Duodenal Ulcer Disease, Ebonyi State, Faecal occult blood, *Helicobacter pylori*, Intestinal Bleeding, Prevalence.

INTRODUCTION

Peptic ulcer is a term commonly used to refer to both stomach and duodenal ulcers. It occurs due to breakdown of the epithelial lining of the stomach and the first part of the small intestine (duodenum) by stomach acids [1] when there is an imbalance between the amount of acid that is produced and the mucous defense barrier, in favor of the acid [2], [3].

A peptic ulcer within the stomach is called a gastric ulcer while one that occurs in the duodenum is called a duodenal ulcer [4]. It affects people of all ages but mostly people above 60 years, and more in males than in females [5]. A person may develop bothgastric and duodenal ulcers at the same time and can also develop peptic ulcer more than once in a lifetime [6]. *Helicobacter pylori* has been implicated in the development of peptic ulcer as well as other diseases of the gastrointestinal tract [7], [8].

The virulence of *Helicobacter pylori* is potentiated by its ability to provoke an inflammatory process by allowing the gastric acid to get through sensitive lining beneath the stomach and duodenum and the consequent weakening of the protective mucous coating. Occasionally, *Helicobacter pylori* inhabit the ectopic gastric tissues in the duodenum but usually reside in the mucous layer of the antral and fundal gastric epithelium [9]. *Helicobacter pylori,* gram negative *bacilli*, is able to survive in stomach acid because of its ability to secrete the enzyme; urease that breaks down urea into ammonium salts that buffer gastric acidity [10], [11]. This mechanism allows *Helicobacter pylori* to make its way to the "safe area" which is the protective mucous lining. Once there, the bacterium's spiral shape helps it burrow through the lining [12].

Helicobacter pylori have been reported to have infected about 50% of the world population with a higher rate of prevalence in underdeveloped countries due to low socioeconomic and poor sanitation conditions [13], [14]. Although the exact route of transmission is not well understood, *Helicopter pylori* infection has been reported to be contagious [15] and mostly acquired during childhood. A prevalence of 73% in the South-West, 81% in Kano and 87% in Jos have been reported [16], [17], [18]. Similarly, a prevalence of 75.4% in Ghana, 91.7% in Egypt, 97% in Gambia, 92% in Bangladesh and 62% in China have also been reported [19], [20], [21], [22], [23].

Once one is infected, unless treated, the infection usually stays for the rest of life [24]. In many people, *Helicopter pylori* live as a commensal and cause no harm. However, progressively as they inhabit the region, they multiply and cause the defense mucous barrier to be disrupted which allows the acid to cause inflammation [25]. Previous studies showed that *Helicobacter pylori* causes more than 90% of duodenal ulcer and up to 80% of gastric ulcer as such, plays a significant role in pathogenesis of duodenal ulcer [26], [27].

Other factors that can cause inflammation in the stomach and as such result in peptic ulcer include prolonged used of anti-inflammatory medicines particularly ibuprofen and aspirin, which are sometimes called non-steroidal anti-inflammatory drugs (NSAIDs). This medicines affects the mucous barrier lining of the duodenum and allow acid to cause an ulcer [28]. About 1 in 20 duodenal ulcers are caused by anti-inflammatory medicines [29], socio-economic status, poor hygiene, smoking, family history of gastric disease may possibly increase one's chances of having a duodenal ulcer [30], [31]. There is a paucity of information on the prevalence of *Helicobacter pylori* infection and its role in gastritis and peptic ulcer diseases especially in the area of study. This study aimed to determine the comparative prevalence of duodenal ulcer and *Helicobacter pylori* infection among adolescents of Ebonyi State, Nigeria.

MATERIALS AND METHODS

Study Site

The study was carried out at the Ultramodern Diagnostic and Research Laboratory, Department of Medical Laboratory Science, Ebonyi State University, Nigeria. The University is made up of four campuses, all within Abakaliki metropolis, with an estimated population of 20,000 students. Over 80% of the students are indigenes of the state.

Study design and Sample size

A cross sectional study was carried out among students in the different campuses of the University who consented to the study. Blood samples were randomly collected aseptically from 420 students comprising of 242 males and 178 females. The sample size was calculated using the formula below [32].

$$n = \frac{Z^2pq}{d^2}$$

where n is the desired sample size when the population is more than 10,000, z is the standard variation, usually set at 1.96 (which corresponds to 95% confidence interval), p is the proportion in the target population estimated to have a particular characteristics (Prevalence of 81.7% (0.817) for *H. pylori* and 60.0% (0.6) for Duodenal ulcer recorded in Kano State and Delta State respectively) [10, 23], q is 1.0 – p, d is the degree of accuracy desired; set at 0.05.

Using the formula, the minimum sample size was 229 and 368 for *H.pylori* and duodenal ulcer respectively. However, with uncertainty about the true prevalence, a 10% attrition rate was anticipated, therefore adjusting the minimum sample size to 252 and 405 respectively.

Sample Analysis

Determination of *Helicobacter pylori* infection was done by chromatographic immunoassay using a *Helicobacter pylori* antibody cassette manufactured by CLIA Waived Inc. Ohio, USA. Faecal occult blood test kit manufactured by QUICK VUE CLIA Waived Inc. Ohio, USA, was used for the determining the presence of blood in the faecal sample. The researcher adhered strictly to manufacturer's instruction in the processing and analysis of the samples. Assessment of respondents' exposure to risk factors was done by the use of standard structured questionnaires.

Data analysis

The data generated in this study were analyzed using Statistical Package for Social Sciences, version 20.0 (SPSS Inc. Chicago Illinois). Results were presented in frequency and percentages. Categorical variables were compared with Pearson's chi-square. Significant P-value was taken as <0.05.

Ethical approval

The study was approved by the Ethical Research Committee of the University and all subjects gave their consent before participating in the study.

RESULTS

A total of 420 samples collected from participants comprising of 242 males and 178 females were tested for *H. pylori* antibodies and intestinal bleeding. Out of the 420 subjects, 349 were positive of either *H. pylori* antibody, intestinal bleeding or both while 71 were negative.

Percentage prevalence of *H. pylori* seropositivity and Faecal Occult Blood (FOB)

The prevalence of *H. pylori* in the studied population was 303 (72.1%) and that of intestinal bleeding was 134 (31.9%) (figure 1, table 1). The overall prevalence positivity of both *H. pylori* antibody and faecal occult blood was 88 (21.0%) (figure 2).

Prevalence of *H. pylori* and Faecal Occult Blood (FOB) among the participants by Gender

Out of the 303 *H. pylori* seropositivity found in this study, the males made up 186 (61.4%) while females were 178 (38.6%). Also, the prevalence of FOB positivity for males and females were 67 (50.0%) and 67 (50.0%) respectively. A strong significant association was observed between gender difference and prevalence of *H. pylori* seropositivity and FOB positivity (P<0.05) (Table 1).

Prevalence of *H. pylori* and faecal occult blood among the participants by age

The participants in the age group ≤ 20 years were found to be more prone to *H. pylori* infection (34.7%) while the least was recorded amongst participants aged ≥ 35 years (13.2%). Participants within the age group 21-25 and 26-30 years had prevalence of 30.7% and 21.4% respectively. The highest prevalence of FOB positivity was observed amongst participants in the age group 21-25 years (44.0%) while the least was recorded among participants in the age group ≤ 20 years (12.7%). Participants within the ages of 26-30 and ≥ 35 years had FOB prevalence of 27.6% and 15.7% respectively. The association between age and prevalence of *H. pylori*, as well as FOB were statically significant (P<0.05) (table 2)

Risk factor assessment

Out of the 48 participants whose source of drinking water was well water, 39 (81.3%) were *H. pylori* seropositive while 9 (18.7%) were seronegative. Out of 79 participants whose source was borehole water, 64 (81.0%) were seropositive while 15 (19.0%) were negative. Also, 89 (73.6%) of participants who drinks pipe born water were seropositive while 32 (26.4%) were negative and out of the 172 participants who drinks portable (sachet) water, 111 (64.5%) were seropositive while 61 (35.5%) were seronegative (table 3).

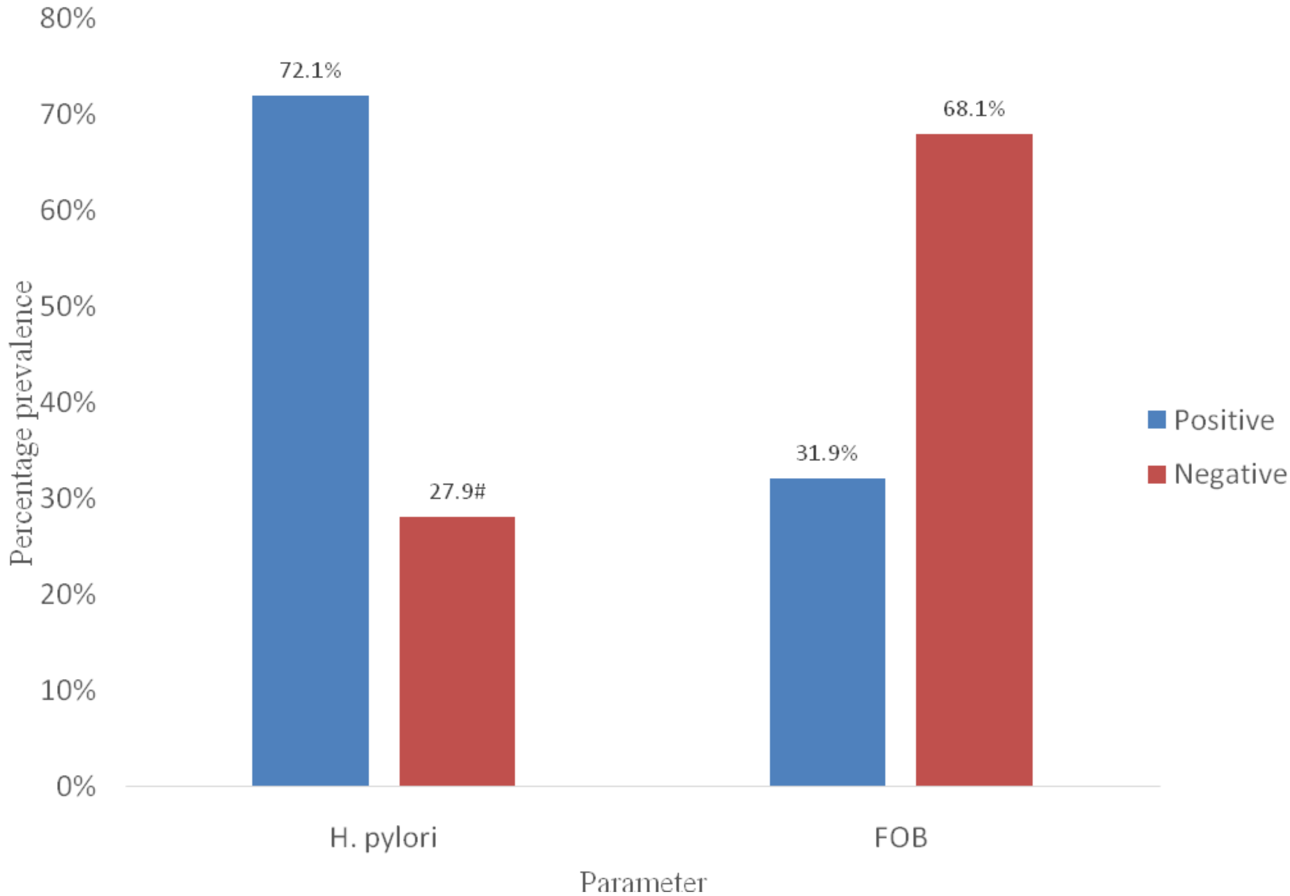

Figure 1: A Bar Chart Showing the Percentage Prevalence of *H. pylori* and Faecal Occult Blood among the participants

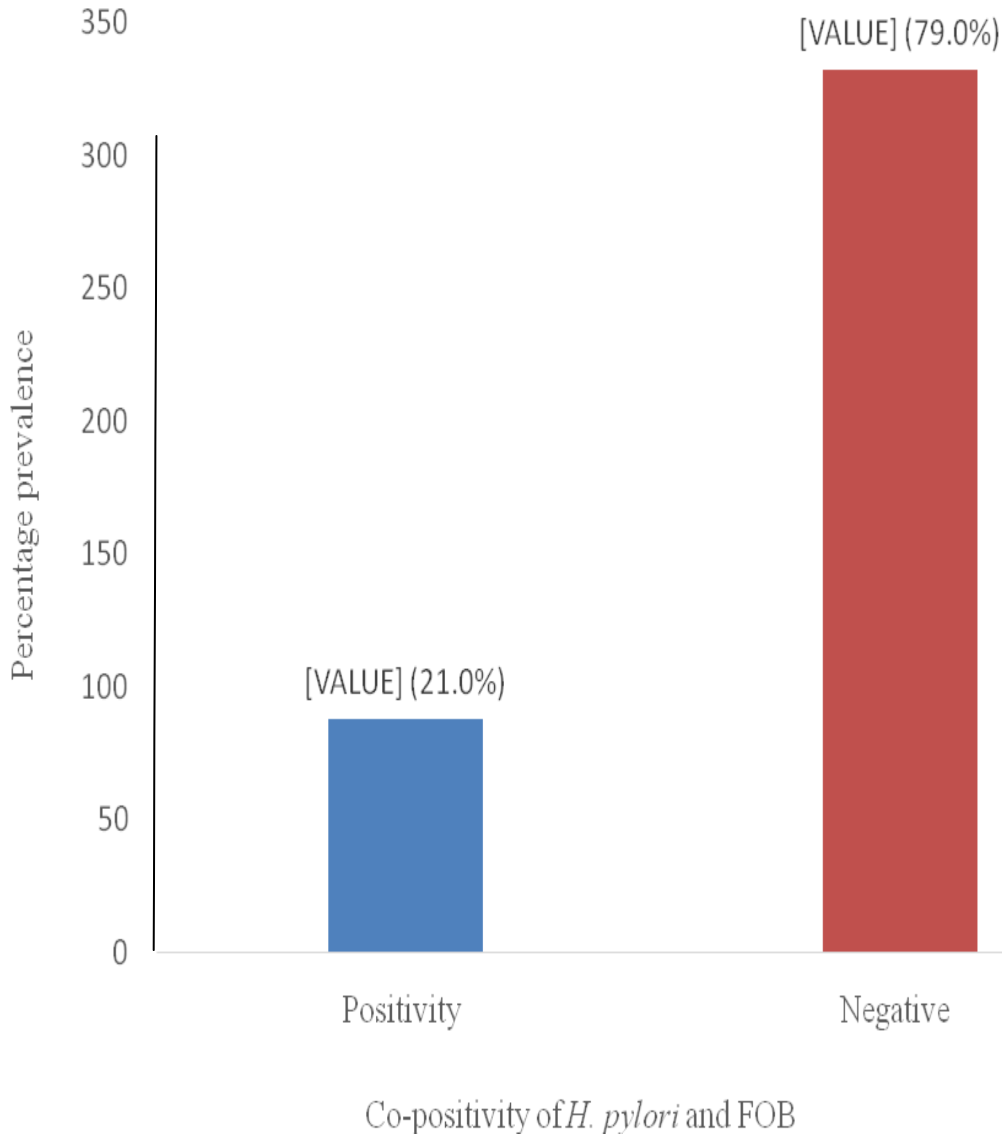

Figure. 2: A bar Chart Showing the Percentage of the subjects Positive for both Faecal Occult Blood and *H. pylori*

Table 1: Gender Prevalence of *H. pylori* and Faecal Occult Blood (FOB) among the Participants

Gender	*H. pylori*				Faecal Occult Blood			
	Total No. of participants	No. positive (%)	X^2-value	p-value	Total No. of participants	No. Positive (%)	X^2-value	p-value
Male	242	186 (61.4)	6.321	0.012*	242	67 (50.0)	4.678	0.031*
Female	178	117 (38.6)			178	67 (50.0)		
Total	**420**	**303 (72.1)**			**420**	**134 (31.9)**		

* Statistically significant (P<0.05)

Table 2: Prevalence of *H. pylori* and Faecal Occult Blood among the participants by age group

Age (years)	*H. pylori*				Faecal Occult Blood			
	Total No. of participants	No. positive (%)	X^2 value	p-value	Total No. of participants	No. Positive (%)	X^2 value	p-value
≤ 20	145	105 (34.7)	11.293	0.010*	145	17 (12.7)	50.546	0.000*
21-25	114	93 (30.7)			114	59 (44.0)		
26-30	93	65 (21.4)			93	37 (27.6)		
≥ 31	68	40 (13.2)			68	21 (15.7)		
Total	**420**	**303**			**420**	**134**		

* Statistically significant (P<0.05)

Table 3: Source of drinking water and prevalence of *H. pylori* infection

Source	Number of subjects examined	Number positive (%)	Number negative (%)	X^2-value	p-value
Pipe borne water	121	89 (73.6)	32 (26.4)	10.147	0.017*
Portable (sachet) water	172	111 (64.5)	61 (35.5)		
Borehole water	79	64 (81.0)	15 (19.0)		
Well water	48	39 (81.3)	9 (18.7)		

* Statistically significant (P<0.05)

DISCUSSION

Out of the 420 participants recruited for the study, 349 were found to be either positive of *H. pylori* antibody, FOB or both. The prevalence rate of *H. Pylori* recorded was 72.1%, that of intestinal bleeding was 31.9% and prevalence of both was 21.0%. Making the prevalence of *H. pylori* higher than that of intestinal bleeding. The occurrence of *H. pylori* antibody and FOB positivity recorded in the study, underlines the role played by these conditions in intestinal bleedings as seen mainly in duodenal ulcerations and in other diverse conditions from inflammatory, infectious to neoplastic conditions. It has been reported that *H. pylori* infection is acquired by oral-oral or faecal-oral route of transmission amongst others and from drinking contaminated water [24]. Several studies have reported varying prevalence of *H. pylori* infection across the globe [25], [26]. The study agreed, though higher than the findings of [27] and Moradi and Rashidi [28] who reported a prevalence rate of 61.6 % and 64.2% respectively. The variation in prevalence may be attributed to the difference in socioeconomic status of the studied population, as well as the difference in demographic characteristics. Our findings also support the report of Malaty and Grahamin in USA who reported that the prevalence of *H. pylori* infection was 82% in the lower social class, 52% in the middle class and 11% in the higher social class [29]. In Denmark, similar finding was reported by [30], that the risk of chronic *H. pylori* infection increases with decreasing socioeconomic status.

Our findings showed a strong association between gender and *H. pylori* infection. Ahigher prevalence of *H. pylori* infection among male participants than their female counterparts was found in the study. This is in agreement with previous studies which also reported a higher prevalence in males than females [5], [28], [31], [32]. However, in the study of Bahamahmoodi *et al*. [33], a higher prevalence was recorded among females. The researchers believed that the higher prevalence in males may have been influenced by numerous factors which includes their supposed involvement in rigorous physical exercise with the consequent intake of NSAIDs as pain relievers, gastroesophageal reflux, less hygienic than the females, increased involvement in sexual activities in urban areas and occupational exposure to the bacterium through kissing, source of drinking water and improperly prepared food [28], [32].

Regarding age differences, the highest prevalence of *H. pylori* infection was recorded among the age group 20 years and belowwith a consistent decrease with increasing ageas participants aged 35 years and above had the least prevalence.Conversely, the highest prevalence of faecal occult blood by age was found among participants in the age group 21-25 years while the least was found among age group 20years and below. The findings supports the literature that gastrointestinal ulcer is more prevalent among people aged 20 years and below and the elderly above the age of 60 years [5, 28, 31, 32]. However, it disagreed with Laure *et al*. [5] and Eniojukan *et al*. [23] who reported a higher prevalence among participants aged 35 years and above. Although, the participants recruited in their study was predominated with older people. In other studies reported elsewhere, the frequency of *H. pylori* infection increased with advance in age [31, 33]. In a study in Iran, the frequency of infection was not age dependent [32]. The current study showed that an infection with *H. pylori*, follows the pattern observedin underdeveloped and developing parts of the world where households are overcrowded and hygiene poorly maintained [8].

In the assessment of source of drinking water as risk factor for *H. pylori* infection, more risks were arrogated to well water, borehole water, pipe borne water and portable (sachet) water in decreasing order.With 81.3% and 81.0% of respondents who drinks from wells and boreholes respectively, being positive of *H. pylori* antibody out of 303 respondents is not surprising as wells and boreholes are found in almost every compound and serve as readily and cheap source of water for the inhabitant of Abakaliki.

CONCLUSION

In conclusion, although, researches have been carried out on the epidemiology of the *H. pylori*, the precise mode of transmission of *H. pylori* infection has not been firmly established. The study found a *H. pylori* infection prevalence of 72.1% and FOB positivity of 31.9% and 21.0% being positive of both. The high prevalence rate in this study is of public health significance. This study therefore recommends that education about proper oral hygiene and adoption of good hygiene practices generally should be encouraged.

REFERENCES

1. Abdel-salam, O., Czimmer, J., Dabreceni, A., Szolcsanyi, J. andMozsik G. (2001). Gastric mucosal integrity. *Journal of Physiology Paris*,95(6):105-127.
2. Ramakrishinan, K. and Salinas, R. (2007). Peptic ulcer disease. *American Family Physician,*76 (7):1005-1012.
3. WebMd. Understanding Ulcers. http://www.webmd.com/digestive disorders/ understanding-ulcers-basic-information 2017.
4. Najm, W.(2011). Peptic ulcer disease. *Primary care,*38(3):383-394.
5. Laure, B.K.M., Michelle, L.N.N. and Hubert, L. (2018). Potential risk factors and prevalence of *Helicobacter pylori* infection among adult patents with dyspepsia symptoms in Cameron. *BMC Infectious Disease*.doi.org/10.1186/s12879-018-3146-1
6. Brown, L.M. (2000).*Helicobacter pylori* epidemiology and routes of transmission. *Epidemiology review,*22(2):283-297.
7. Fock, K.M. andAng, T.L. (2010). Epidemiology of *Helicobacter pylori* infection and gastric cancer in *Asia. J GastroenterolHepatol,*25:479–86.
8. Saad, R. and Chey, W.D. (2008). Persistent *Helicobacter pylori* infection after a course of antimicrobial therapy-what's next? *ClinGastroenterolHepatol,*6:1086-1090.
9. Aboderin, O.A., Abdu, A.R., Odetoyin, B.,Okeke, I.N., Lawal, O.O., Ndububa, D.A., Augustine, E.A. and Adebayo, L. (2007). Antibiotic resistance of *Helicobacter pylori* from patients in Ile-Ife, South-west, Nigeria. *Afr Health Sci,*7:143-147.
10. Bashir, M.T. and Ali, B.U. (2009). Peptic ulcer disease and *Helicobacter pylori* infection at Kano, Nigeria. *Internet J Gastroenterol,*8:1-3.
11. Malu, A.O., Ani, A.E. and Bello, S.S. (2010). The prevalence of *Helicobacter pylori* in dyspeptic patients from the Jos Plateau.*Nigeria. Niger Med J,*41:1-3.
12. Baako, B.N. and Darko, R. (1996). Incidence of *Helicobacter pylori* infection in Ghanaian patients with dyspeptic symptoms referred for upper gastrointestinal endoscopy. *West Afr J Med.*15:223-227.
13. El Dine, S.S., Mubarak, M., Salama, R., El Raziky, M., El Sherbily, E. andZakzria, S. (2008).Low seroprevalence of anti-CagA antibodies inspite of high seroprevalence of anti-*H pylori* antibodies in rural Egyptian community. *Res J Med MedSci,*3:118-123.
14. Secka O, Antonio M, Tapgun M, *et al.* PCR-based genotyping of *Helicobacter pylori* of Gambian children and adults directly from biopsy specimens and bacterial cultures. *Gut Pathog* 2011; 3(5): doi:10.1186/1757-4749-3-5.
15. Ahmad, M.M., Rahman, M., Rumi, A.K., Islam, S., Hug, F., Chowdhury, M.F., Jinnah, F., Morshed, M.G., Hassan, M.S., Khan, A.K. and Hasan, M. (1997). Prevalence

of *Helicobacter pylori* in asymptomatic population – A pilot serological study in Bangladesh. *J Epidemiol,7*:251-254.
16. Shi, R., Xu, S., Zhang H., Ding, Y., Sun, G., Huang, X., Chen, X., Li, X., Yan, Z. and Zhang, G. (2008). Prevalence and risk factors for *Helicobacter pylori* infection in Chinese populations. *Helicobacter,*13:157-165.
17. Eusebi, L.H., Zagari, R.M. and Bazzoli, F. (2014).Epidemiology of *Helicobacter pylori* infection. *Helicobacter,*19:1–5.
18. Salih, B.M., Fatih, A., Nizamettin, B. and Ersan, S. (2007).*H. pylori* infection and other risk factors associated with peptic ulcers in Turkish patients: a retrospective study. *World Journal of Gastroenterology,*13(23): 3245-3248.
19. Center for Disease Control and Prevention (2017).*Helicobacter pylori* and peptic ulcer disease.www.cdc.goov/ulcer/economic.htm.Accessed 12/5/2020.
20. Martin, D., Montgomery, E., Dobek, A., Patrissi, G. and Peura, D. (2008).*Helicobacter pylori*, NSAIDS, and smoking: Risk factors for peptic ulcer disease. *American Journal of Gastroenterology*84(10):1268-1272.
21. Moayyedi, P., Axon, A. andFeltbower, R. (2002).Relation of adult lifestyle and socioeconomic factors to the prevalence of *Helicobacter pylori* infection. *International Journal of Epidemiology,*31:624-631.
22. Araoye, M.O. (2004).Sample size determination in research methodology with statistics for health and social sciences. Ilorin: Nathadox publishers,pp:115-122.
23. Eniojukan, J.F., Okonkwo, O.C. and Adje, U.D. (2017). Risk factors, management and other correlates of peptic ulcer disease in a university community in South-South, Nigeria. *UK Journal of Pharmaceutical and Biosciences,*5(6):7-15.
24. Ahmed, K.S., Khan, A.A., Ahmed, I., Tiwari, S.K., Habecb, A. and Ahi, J.D. (2007).Impact of household hygiene and water source on the prevalence and transmission of *H. pylori*: A southern Indian perspective. *Singapore Medical Journal,*48(6):543-549.
25. Levin, A.D., Watermeyer, G., Mohamed, N., Epstein, P.D., Hlatshwayo, J.S. and Metz, C.D. (2007).Evaluation of a locally produced rapid urease test for the diagnosis of *Helicobacter pylori* infections. *African Medical Journal,*97(12):1281-1284.
26. Ndip, R.N., Malrnge, T.A.E., Ojongokpolo, J.E.A., Luma, H.N., Malongue, A. and Akoachere, J.F.K. (2008).*Helicobacter pylori* isolates recovered from the gastric biopsies of patients with gastro duodenal pathologies in Cameroon. Current status of antibiogram. *Tropical Medicine of Internal Health,*13(6):102-104.
27. Zahedi, M.J., Darvish, M.S., Atapour, M. and Jahanbakhsh, A.M. (2000). Relative frequency of *Helicobacter pylori* in patients referring to health centers in 2000, in kerman, Iran. *Journal of Medical University of kerman,* 3:140-144.
28. Moradi, A. and Rashidi, P.A. (1999).Seroepidemiologic study of *Helicobacter pylori* infection in Semnan, Iran in 1999.*Journal of Medical University of Semnan,*1:45-49.
29. Malaty, H.M. and Graham, D.Y. (2004). Importance of childhood socioeconomic status on the current prevalence of Helicobacter pylori infection. *Gut,*35:742-745.
30. Steffen, J.R., Leif, P.A., Charlotte, V.R., Olaf, B. and Torben, J. (1996). Helicobacter pylori stool antigen in asymptomatic children in Lagos State, Nigeria. *Am J. Pub. Health.*86:156-162.
31. Lin, D.B. (1999). Sero-epidemiology of *Helicobacter pylori* infection

among pre-school children in Taiwan. *American Journal of Tropical Medicine and Hygiene*,61:554-558.
32. Farhadi, A., Bahar, A., Kowsarian, M. and Mahdavi, M. (2000). Seroepidemoilogic study of *Helicobacter pylori* infection in children aged 7-18 in Sari, iran in 1999. *Journal of Mazandaran University of Medical Sciences*,10:9-25.
33. Babamahmoodi, F., Ajemi, A., Kalhor, M., Shfiei, G.R. and Khalilian, A.R. (2003). Seroepidemiologic study of infection with *Helicobacter pylori* in Sari, Iran in 2001-2002. *Journal of Mazandaran University of Medical Sciences*,14:39-47.
34. Gerald, M. and Lawrence, W. (2006). Current Surgical diagnosis and treatment, Raw-Hill Companies, New York. 12th ed.pp:513-515.
35. Roshana, S., Kamal, K., Shiv Raj, K.C. and Kabita, H.B. (2014). Heliacobacter pylori infection among patients with upper gastrointestinal symptoms: prevalence and relation to endoscopy diagnosis and histopathology. *Journal of Family Medicine and Primary Care*,3(2): 154-158.