

Prevalence of *Helicobacter pylori* infection and gastric preneoplastic lesions in patients admitted for upper gastro-intestinal endoscopy in Cotonou (Benin Republic)

Abstract

Infection with *Helicobacter pylori* (*H. pylori*) is common in developing countries such as Benin. This germ can cause several gastroduodenal diseases such as gastritis, ulcer, adenocarcinoma or gastric MALT lymphoma. This study aimed to determine the prevalence of *H. pylori* infection and gastric pre-neoplastic histological lesions in patients admitted for upper gastrointestinal endoscopy (UGE) and to identify factors associated with this infection.

Methods: This was a cross-sectional descriptive and analytical study, with prospective data collection, conducted from October 2014 to December 2015. We included all patients admitted to Menontin Hospital for UGE in whom a gastric biopsy has been done. *H. pylori* research was conducted in anatomy-pathology, either in a medical laboratory in Cotonou or at the CERBA laboratory in Paris, France, depending on the patient's choice. The microscopic study was carried out after staining with Haematoxyline-Eosine-Safran (HES) and the search for *H. pylori* using Giemsa staining.

Results: *H. pylori* was investigated in 137 patients, 67 men (48.9%) and 70 women (51.1%). The mean age was 48.3±14.6 years with extremes of 10 and 83 years. Some patients had had at least one previous *H. pylori* eradication treatment (63 cases or 46%). For the general population, the test was positive in 98 cases, i.e. a prevalence of 71.5%. Depending on whether or not patients had been pre-treated for *H. pylori*, the test was positive in 40 out of 63 (63.5%) pre-treated patients, versus 58 out of 74 (78.4%) patients who were naïve to any eradication treatment ($p=0.057$). The pre-neoplastic lesions noted were gastric atrophy in 35 patients (including 25 *H. pylori* positive), intestinal metaplasia in 13 patients (including 10 *H. pylori* positive) and low grade dysplasia in 14 patients (including 12 *H. pylori* positive); high grade dysplasia was found in one patient who was *H. pylori* negative. Only the type of prior eradication treatment appeared to be associated with *H. pylori* infection ($p=0.182$).

Conclusion: *H. pylori* infection is common in our patients admitted for upper gastrointestinal endoscopy. Gastric atrophy was the most common pre-neoplastic lesion.

Keywords: *Helicobacter pylori*, upper gastrointestinal endoscopy, gastric preneoplastic lesions, cotonou

Volume 11 Issue 6 - 2020

Aboudou Raïmi Kpoussou,¹ Benoît Kouwakanou,² Falilatou Séidou,³ Khadidjatou Saké Alassane,⁴ Rodolph Koffi Vignon,¹ Comlan N'déhougèa Martin Sokpon,¹ Carin Ahouada,⁵ Vincent Zoundjiekpon,⁶ Fadel Sourokou,⁷ Nicolas Kodjoh,⁸ Jean Séhonou¹

¹Department of Hepato-Gastroenterology, National and University Hospital Hubert Koutoukou Maga (CNHU-HKM), Benin Republic

²Department of Hepato-Gastroenterology, Mohammed VI University Hospital of Marrakech, Morocco

³Department of Anatomopathology and Cytopathology, Faculty of Health Sciences, Benin Republic

⁴Department of internal medicine, Borgou-Alibori University Hospital, Benin Republic

⁵Medicine Department, Allada Zone Hospital, Benin Republic

⁶Department of Hepato-Gastroenterology and Geriatrics, Olomouc University Hospital, Czech Republic

⁷International Clinic of Cotonou Aupiais (CICA), Benin Republic

⁸Archangel Clinic, Agblangandan - Sèmè kpodji, Benin Republic

Correspondence: Dr Aboudou Raïmi Kpoussou, Department of Hepato-Gastroenterology, National and University Hospital Hubert Koutoukou Maga (CNHU-HKM), Benin Republic, Tel 0022966181939, Email kpossou.raimi@yahoo.fr

Received: October 27, 2020 | **Published:** November 13, 2020

Introduction

Infection with *Helicobacter pylori* (*H. pylori*) occurs in half of the world's population.¹ Its prevalence varies considerably according to geographic location, ethnicity, age, and socio-economic factors; it is high in developing countries and lower in developed countries.^{1,2} It is 20-30% in industrialized countries and 70-90% in developing countries where it is a real public health problem.¹ This is confirmed by most recent studies carried out around the world and particularly in Africa, notably in Nigeria with a prevalence of 80% in 2020,³ 88% in Ghana in 2017,⁴ 70.41% and 93.1% in 2015 respectively in Togo⁵ and Congo Brazzaville,⁶ 70.8% in Burundi in 2014,⁷ 66.12% in Egypt in 2019,⁸ 63.8% in Morocco in 2016⁹ and 71.43% in Algeria.¹⁰ In contrast, the prevalence of *H. pylori* is declining in developed countries, around 24-32% in Central and Northern Europe excluding non-European immigrants.^{1,11} The high prevalence of *H. pylori* infection in developing countries has been associated with overcrowding, poor housing, poor sanitation and lack of safe drinking water.^{4,12} *H. pylori* infection occurs in childhood, mainly in the first

five years of life, and is transmitted primarily by the fecal-oral and/or oral-oral route.¹³ It usually goes unnoticed, asymptomatic and can persist throughout life if not eradicated by adequate therapy. Its role in the genesis of various gastroduodenal pathologies such as gastritis, dyspeptic syndromes, gastric or duodenal ulcer, mucosa-associated lymphoid tissue lymphoma (MALT) and gastric adenocarcinoma is currently proven.¹⁴ It is the only bacterium currently recognized as a human carcinogen and classified as such since 1994 by the International Agency for Research on Cancer, i.e. the World Health Organization.¹⁵

Several methods exist to detect *H. pylori*. Some are invasive, such as methods requiring gastric biopsies taken during gastroscopy (pathological anatomy, culture, polymerase chain reaction, rapid urease test). Others are non-invasive (labeled urea breath test, antigen detection in stool, serology).^{14,16} Anatomico-pathological examination of gastric biopsies, apart from staining for *H. pylori*, offers the advantage of good sensitivity and specificity, and also allows the detection of pre-neoplastic lesions (intestinal metaplasia or dysplasia)

and the typing of gastritis.^{14,17} In Benin, the search for *H. pylori* by anatomico-pathological examination was not common before the year 2012, and little data exists on this subject.¹⁸ Two prospective studies of *H. pylori* testing in Cotonou in patients admitted for UGE reported a prevalence of 56.41% in 1991¹⁹ and 66.5% in 1996,²⁰ respectively, and a retrospective study from 2007 to 2016 reported a prevalence of 56% in Cotonou for *H. pylori* gastritis.¹⁸

The aim of this study was to determine the prevalence of *H. pylori* infection and gastric pre-neoplastic histological lesions in patients admitted for upper gastrointestinal endoscopy (UGE) and to identify factors associated with this infection.

Methods

This was a descriptive and analytical cross-sectional study with prospective data collection during the period from October 2014 to December 2015. We included all patients admitted to Menontin Hospital for UGE and in whom gastric biopsies could be taken. Patients were either naïve to any *H. pylori* eradication treatment, or may have already had one or more eradication treatments. The *H. pylori* research was performed by anatomico-pathological examination, either in a medical laboratory in Cotonou (Benin Republic) or at the CERBA laboratory in Paris (France), depending on the patient's choice. The microscopic study was done after hematoxylin-eosin-safran (HES) staining and the search for *H. pylori* using Giemsa staining.

A standardized survey form was used for data collection. The main dependent variable was *H. pylori* infection on the anatomico-

pathological examination of the gastric biopsy results. The other dependent variable was gastric pre-neoplastic lesions (atrophy, intestinal metaplasia and low or high-grade dysplasia). The independent variables were sociodemographic, clinical, endoscopic and therapeutic.

Statistical analysis was performed using SPSS 18.0. The analysis of the qualitative variables was performed by Chi2 and Fischer exact tests. A $p \leq 0.05$ was considered statistically significant. The association between *H. pylori* infection and the different variables was studied by logistic regression in univariate and multivariate analysis to confirm the involvement of the studied variables in *H. pylori* infection.

Results

Characteristics of the study population

Our study involved 137 patients who all underwent upper gastrointestinal endoscopy with gastric biopsy for pathological examination, including 67 men (48.9%) and 70 women (51.1%). The mean age was 48.3 ± 14.6 years with extremes of 10 years and 83 years. One hundred and sixteen patients (84.7%) lived as a couple compared to 14 (10.2%) who were single. Seventy-seven patients (56.6%) had a university education compared to 6 (4.4%) who had no schooling. More than half of the study population (74 patients, i.e. 54.4%) was employees. And more than $\frac{3}{4}$ were Christians (122 patients or 89.1%) (Table 1). As co-morbidities, 31 patients (22.6%) were hypertensive, 29 patients (21.2%) were obese; diabetes and history of gastric ulcer in first-degree relatives were found in 5 patients (3.7%) (Table 2).

Table 1 Distribution of socio-demographic characteristics of the study population according to whether or not they carry *H. pylori* infection

Socio-demographic characteristics	<i>H. pylori</i>		P
	Yes [n = 98(71,5%)]	No [n = 39(28,5%)]	
Age (Average in years)	47.3	50.9	0.1901
Gender			0.7089
Male	49(35.8%)	18(13.1%)	
Female	49(35.8%)	21(26.9%)	
Marital Status			0.6392
Single	11(8.0%)	3(2.2%)	
As A Couple	83(60.6%)	33(24.1%)	
Divorced	1(0.7%)	00	
Widow(Er)	3(2.2%)	3(2.2%)	
Level of Education			0.5039
Primary	16(11.8%)	6(4.4%)	
Secondary	19(14.0%)	12(8.8%)	
Superior	58(42.7%)	19(14.0%)	
Unschoolled	4(2.9%)	2(1.5%)	
Profession			0.6326
Liberal	31(22.8%)	13(9.6%)	
Reseller	10(7.4%)	1(0.7%)	
Employee	51(37.5%)	23(16.9%)	
Pupil/Student	5(3.7%)	2(1.5%)	

Table Continued...

Socio-demographic characteristics	<i>H. pylori</i>		P
	Yes [n = 98(71,5%)]	No [n = 39(28,5%)]	
Religion			0.2336
Islam	9(6.6%)	5(3.7%)	
Christianity	89(64.9%)	33(24.1%)	
Animism	00	1(0.7%)	

Table 2 Distribution of medical history according to whether or not *H. pylori* infection was carried

Background	<i>H. pylori</i>		P
	Yes [n = 98(71,5%)]	NO [n = 39(28,5%)]	
Obesity			0.8173
Yes	20(14.6%)	9(6.6%)	
No	78(56.9%)	30(21.9%)	
Diabetes			1.000
Yes	4(2.9%)	1(0.7%)	
No	94(68.6%)	38(27.7%)	
Hta			0.3680
Yes	20(14.6%)	11(8.0%)	
No	78(56.9%)	28(20.4%)	
Cirrhosis			
Yes	00	00	
No	98(71.5%)	39(28.5%)	
Hiv			
Yes	00	00	
No	98(71.5%)	39(28.5%)	
Alcoholism			0.2847
Yes	00	01(0.7%)	
No	98(71.5%)	38(27.7%)	
Gastric Ulcer Grade I			0.6229
Yes	3(2.2%)	2(1.5%)	
No	95(69.3%)	37(27.0%)	
Gastric Cancer Grade I			1.000
Yes	2(1.5%)	1(0.7%)	
No	96(70.1%)	38(27.7%)	
Other Background			0.4146
Yes	5(3.7%)	3(2.2%)	
No	92(64.7%)	36(26.5%)	

The main clinical manifestations that motivated the UGE were diverse but dominated by epigastric disorders in 92 patients (67.2%), painful dyspepsia in 25 patients (18.3%), painless dyspepsia in 8 patients (5.8%), non-specific abdominal pain in 5 patients (3.7%), and upper GI hemorrhage in 14 patients (10.2%).

Prevalence of *H. pylori* infection and gastric pre-neoplastic lesions

The prevalence of *H. pylori* in the study population was 71.5% (98/137). Among the 98 patients with *H. pylori*, 58 (59.2%) were naïve to any eradication treatment, while 40 (40.8%) had previously received at least one *H. pylori* eradication treatment (Table III). Depending on whether or not the patients had been pre-treated for *H. pylori*, the search for *H. pylori* was positive in 40 pre-treated patients out of 63 (63.5%) compared to 58 patients out of 74 (78.4%) for patients naïve to any eradication treatment, with a $p=0.057$ (non-significant difference). According to the place of analysis, for samples sent to France, the search for *H. pylori* was positive in 31 cases (73.8%), and for those tested in Benin it was positive in 67 cases (70.5%), with a $p=0.693$ (non-significant difference).

Histologically, the pre-neoplastic lesions noted were gastric atrophy in 35 patients (including 25 *H. pylori* positive), intestinal

metaplasia in 13 patients (including 10 *H. pylori* positive) and low grade dysplasia in 14 patients (including 12 *H. pylori* positive); high grade dysplasia was found in one patient who was *H. pylori* negative. One case of gastric adenocarcinoma was noted.

Factors associated with *H. pylori* infection

H. pylori were present in patients with an average age of 47.26 years. However, neither age ($p=0.1901$) nor sex ($p=0.7089$) was associated with *H. pylori* infection (Table 1). Furthermore, in our study, other socio-demographic factors such as marital status ($p=0.6392$), religion (0.2336), occupation ($p=0.6326$), and education ($p=0.5039$) were not associated with *H. pylori* infection (Table 2). Similarly, none of the co-morbidities studied were associated with *H. pylori* infection (Table 2). Furthermore, endoscopically, the prevalence of *H. pylori* appeared to be higher in cases of pangastritis (65.69%), but this association was not statistically significant ($p=0.7243$) (Tables 3&4). Previous treatment was not associated with *H. pylori* infection ($p=0.057$). However, the type of previous treatment appears to be statistically associated ($p=0.0182$), with triple therapy including Amoxicillin-metronidazole being associated with more positive cases of *H. pylori* infection (30.16%) (Table 3).

Table 3 Distribution of *H. pylori* infection by prior treatment status

Previous <i>H. pylori</i> eradication treatment	<i>H. pylori</i>		P
	Yes [n = 98(71.5%)]	No [n = 39(28,5%)]	
Previous Treatment For <i>H. Pylori</i>			0.0602
Yes	40(29.2%)	23(16.8%)	
No	58(42.3%)	16(11.7%)	
Type of Previous Treatment			0.0182
Amoxicillin-Metronidazol	19(30.2%)	4(6.4%)	
Amoxicillin-Clarithromycin	7(11.1%)	11(17.5%)	
Clarithromycin-Metronidazol	3(4.8%)	00	
Sequential	10(4.8%)	7(11.1%)	
Amoxicillin-Levofloxacin	00	00	
Bismuth Therapy	2(3.2%)	00	

Table 4 Distribution of *H. pylori* infection by endoscopic appearance

Endoscopic data	<i>H. pylori</i>		P
	Yes [n = 98(71.5%)]	No [n = 39(28.5%)]	
Antral Gastritis			0.4453
Yes	8(5.8%)	1(0.7%)	
No	90(65.7%)	38(27.7%)	
Fundic Gastritis			0.4898
Yes	1(0.7%)	1(0.7%)	
No	97(70.8%)	38(27.7%)	
Pangastritis			0.7243
Yes	90(65.7%)	37(27.0%)	
No	8(5.8%)	2(1.5%)	

Table Continued...

Endoscopic data	<i>H. pylori</i>		P
	Yes [n = 98(71.5%)]	No [n = 39(28.5%)]	
Gastric Ulcer			0.5404
Yes	9(6.6%)	5(3.7%)	
No	89(65.0%)	34(24.8%)	
Duodenal Ulcer			0.6743
Yes	5(3.7%)	1(0.7%)	
No	93(67.9%)	38(27.7%)	
Gastric Tumor			1.000
Yes	2(1.5%)	1(0.7%)	
No	96(70.1%)	38(27.7%)	
Other Lesion			0.2314
Yes	13(9.5%)	2(1.5%)	
No	85(62.0%)	37(27.0%)	

Discussion

The prevalence of *H. pylori* in our series is 71.5% and confirms that Benin is a developing country like most African countries, some South American and West Asian countries.^{1,2} There was no significant variation in the presence of *H. pylori* by age and socio-economic factors in our series, contrary to the results of Hunt et al.¹ Sokpon et al.⁹ and Odigie et al.²¹ reported a significant association with female sex, whereas most African studies did not find a statistically significant relationship between sex and *H. pylori*.^{4,5,7,9,22} *H. pylori* infection is frequently associated with certain socio-demographic factors such as living conditions (promiscuity, access to drinking water), level of education, lifestyle, and occupation.^{1,4,7,12,21,23} None of these factors classically associated with *H. pylori* infection were found in our series. This could be explained by the fact that most of the patients included were living in urban areas in Cotonou.

Approximately 1/3 of the previously treated patients still had *H. pylori*, with the highest rate in the batch of those who used amoxicillin and metronidazole in combination with PPI. Indeed, the only effective treatment currently available for *H. pylori* infection involves the use of antibiotics. This observed result could be related to resistance or recontamination. The main mechanisms for the development of antibiotic resistance in *H. pylori* include mutations that alter the ability of antibiotics to bind to ribosomes and interfere with protein synthesis; mutations that affect DNA replication and transcription and modify penicillin-binding proteins involved in peptidoglycan biosynthesis.²⁴ This resistance is therefore multifactorial and depends not only on the possibility of the germ to mutate but also on the free access to antibiotics allowing an abusive and uncontrolled use that would nest the mutation at the gene level.⁷ Resistance to metronidazole has been reported in most African countries^{1,23} and may be due to open access and misuse. This study did not assess the level of antibiotic resistance of *H. pylori*.

The endoscopic aspects of the lesions were dominated by the pangastritis aspect in our study. This finding corroborates most of the studies carried out in Africa on this subject.^{9,20,22} Gastritis is thought to result from an inflammatory and immunological response induced by *H. pylori*.^{1,25} In patients infected with *H. pylori* in our series, pre-

neoplastic lesions were noted such as gastric atrophy in 7.1% of cases, intestinal metaplasia in 7.69% of cases and low grade dysplasia in 8.57% of cases. These values are slightly lower than those reported by Darré et al.²⁶ in Togo, where 99% of *H. pylori* positive patients had glandular atrophy and 85% had intestinal metaplasia.²⁶ Sokpon found a statistically significant relationship between these lesions (fundal and anal atrophy, fundal metaplasia) and *H. pylori*.⁹ The near-constancy of chronic gastritis in southern countries is probably related to the early onset of *H. pylori* infection, which occurs most often in childhood.¹³

Our study has the interest to evaluate the prevalence of *H. pylori* in Benin using histology which offers the advantage of good sensitivity and specificity, but also to type the associated gastritis for a good patient follow-up (therapeutic and endoscopic evaluation of the response to treatment). However, its accessibility poses a problem due to its cost, which is often not accessible to everyone. The main limitation of this study is the small number of patients included.

Conclusion

The prevalence of *H. pylori* is high in our series, at 71.5%. It is not significantly associated with any of the socio-demographic factors studied, nor with any co-morbidity. Gastric atrophy was the most common pre-neoplastic lesion. The lack of difference between pre-treated patients and those naïve to any eradication treatment raises the suspicion of either a high level of antibiotic resistance or frequent re-infections in the study population. It is desirable to conduct studies to measure the sensitivity of *H. pylori* infection to antibiotics in our country.

Acknowledgments

None.

Conflicts of interest

Author declares that there are no conflicts of interest.

Funding

None.

References

1. Hunt RH, Xiao SD, Megraud F, et al. *Helicobacter Pylori* in developing countries. World Gastroenterology Organisation Global Guideline. *J Gastrointestin Liv Dis*. 2011;20(3):299–304.
2. Hooi JKY, Lai WY, Ng WK, et al. Global prevalence of *Helicobacter pylori* infection: systematic review and meta-analysis. *Gastroenterology*. 2017;153(2):420–429.
3. Gashau W, Adamu AS. Blind *Helicobacter pylori* treatment in dyspeptics in a high prevalence area. *Int J Cur Res Rev*. 2020;12(4):2–7.
4. Awuku YA, Simpong DL, Alhassan IK, et al. Prevalence of *Helicobacter pylori* infection among children living in a rural setting in sub-Saharan Africa. *BMC Public Health*. 2017;17(1):360.
5. Lawson-Ananissou LM, Bouglouga O, Bagny A, et al. Epidemiological profile of peptic ulcers at the Lomé campus hospital and university center (Togo). *J Afr Hépatol Gastroentérol*. 2015;9(3):99–103.
6. Bossali F, Deby G, Ahoui-Apendi CR et al. Study of the management of *Helicobacter pylori* infection in the cities of pointe-noire and brazzaville in 2015. *Ann Univ M Nguabi*. 2017;17(1):1–9.
7. Ntagirabiri R, Harerimana S, Makuraza F, et al. *Helicobacter pylori* in Burundi: first assessment of endoscopic prevalence and eradication. *J Afr Hépatol Gastroentérol*. 2014;8:217–222.
8. Alborai M, Elhossary W, Aly OA, et al. Egyptian recommendations for management of *Helicobacter pylori* infection: 2018 report. *Arab J Gastroenterol*. 2019;20(3):175–179.
9. Sokpon M, Salihoun M, Lahlou L, et al. Predictors of *Helicobacter pylori* (Hp) infection in chronic gastritis: about a Moroccan study. *J Afr Hépatol Gastroentérol*. 2016;10(4):203–207.
10. Kasmi H, Doukani K, Ali A, et al. Epidemiological Profile of *Helicobacter pylori* Infection in Patients with Digestive Symptoms in Algeria. *Journal of Epidemiology and Global Health*. 2020.
11. van Blankenstein M, van Vuuren AJ, Looman CW, et al. The prevalence of *Helicobacter pylori* infection in the Netherlands. *Scand J Gastroenterol*. 2013;48(7):794–800.
12. Mégraud F. When and how do you get infected with *Helicobacter pylori*? *Gastroenterol Clin Biol*. 2003;27:374–379.
13. Calvet X, Ramirez Lázaro MJ, Lehours P, et al. Diagnosis and epidemiology of *Helicobacter pylori* infection. *Helicobacter*. 2013;18:5–11.
14. De Korwin JD. Nouvelles recommandations pour le diagnostic et le traitement de l'infection à *Helicobacter pylori* New recommendations for the diagnosis and the treatment of *Helicobacter pylori* infection. *La Presse Médicale*. 2013;42(3):309–317.
15. IARC. Working Group on the Evaluation of Carcinogenic Risks to Humans. *Helicobacter pylori*. In: Schistosomes, liver flukes, and *Helicobacter pylori*. Lyon: IARC; 1994. p. 177–240.
16. Pohl D, Keller PM, Bordier V, et al. Review of current diagnostic methods and advances in *Helicobacter pylori* diagnostics in the era of next generation sequencing. *World J Gastroenterol*. 2019;25(32):4629–4660.
17. Michaud L, Gottrand F. Infection à *Helicobacter pylori*: Quelle prise en charge en 2013. *Réalités pédiatriques*. 2013;181:35–38.
18. Seidou F, Kpoussou R, Akpo W, et al. Gastrites à *Helicobacter pylori*: about 159 cases in two laboratory of anatomopathology of Cotonou. *J Afr Chir Digest*. 2018;18(1):2332–2337.
19. Kodjoh N, Hountondji A, Addra B. Apport de l'endoscopie au diagnostic des affections oeso-gastro-duodénales en milieu tropical: Expérience béninoise à propos de 930 examens. *Ann Gastroenterol Hepatol*. 1991;27(6):261–267.
20. Hountondji A, Addra B, Kodjoh N, et al. *Helicobacter pylori* and gastric diseases in Benin. *Journal of the Society of Clinical Biology of Benin*. 1996;3:71–76.
21. Odigie AO, Adewole AJ, Ekunwe AA. Prevalence and factors associated with *Helicobacter pylori* infection among treatment naïve dyspeptic adults in University of Benin Teaching Hospital, Benin City, Nigeria. *Afr J Clin Exper Microbiol*. 2020;21(2):97–105.
22. Hafidi R, Oubaha S, El Gamrani Y, et al. *Helicobacter pylori* infection: epidemiological, clinical and endoscopic aspects. *J Afr Hépatol Gastroentérol*. 2013;7:74–77.
23. Smith S, Fowora M, Pellicano R. Infections with *Helicobacter pylori* and challenges encountered in Africa. *World J Gastroenterol*. 2019;25(25):3183–3195.
24. Zanotti G, Cendron L. Structural Aspects of *Helicobacter pylori* Antibiotic Resistance. *Adv Exp Med Biol*. 2019;1149:227–241.
25. Sibony M, Jones NL. Recent advances in *Helicobacter pylori* pathogenesis. *Curr Opin Gastroenterol*. 2012;28(1):30–35.
26. Darré T, Amégbor K, Bagny A, et al. Histo-epidemiologic profile of chronic gastritis and infection with *Helicobacter pylori*: about 296 cases of biopsies in Togo. *J Afr Chir Digest*. 2013;13(1):1426–1430.