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EPIDEMIOLOGY OF GASTRIC HISTOLOGICAL LESIONS INDUCED BY HELICOBACTER PYLORI IN THE HOSPITAL OF SIKASSO.

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Abstract

Introduction: Helicobacter pylori infection is a ubiquitous infection, but is most prevalent in developing countries. Its relationship with gastric cancer has been known since the early 1990s, Helicobacterpylori having been classified as a gastric carcinogen by the WHO since 1994.

The aim of this study is to assess the prevalence of gastric histological lesions induced by H.P.

Methodology: This was a descriptive study of patients treated in the hepathogastroenterology department of Sikasso hospital.

We included in this study, which took place from May 2020 to December 2021, patients developing chronic Helicobacter pylori gastritis, diagnosed on gastric biopsies performed during FOGD with their informed consent. The data collected by means of a questionnaire from the results of the anatomopathological examination of the gastric biopsies were entered into Excel and then analysed with Epi-info version 7 software.

Results: A total of 77 patients were included. The mean age was 45.41 years (16-83) and the gender was 1.026.

In the OLGA classification, low risk stages represented 87.01% and 79.22% in the gastric and antral body respectively and high risk stages (0%). In the OLGIM classification, low-risk stages accounted for 87.01% and 89.61% in the gastric and antral body respectively, and high-risk stages for 0%.

Mild HP density was mild in 37.66% and 40.25%; moderate in 40.25% and 31.16% and severe in 5.19% and 2.59% in the gastric and antral body respectively.

Glandular atrophy was mild in 16.88% and 23.37% moderate in 2.59% at the fundic and antral level respectively. Intestinal metaplasia was present in 3.89%,

Cellular activity was present in 89.61% and 80.51% at the fundial and antral levels respectively. The chronicity of the lesions was mild in 12.98% and 10.38%, moderate in 64.93% and 37.66% and severe in 0% at the fundic and antral level respectively.

Follicular density was mild in 23.37% and 16.88%, moderate in 24.67% and 18.18%, and severe in 6.49% and 2.59% at the fundic and antral level respectively.Low grade dysplasia was present in 1.29%. Poorly differentiated adenocarcinoma, moderately differentiated adenocarcinoma and independent cell adenocarcinoma were present in 1.29%

each.adenomatousfibroinflammatory polyp of fundus HP associated reactive gastritis were present in 1.29% each.

CONCLUSION:

The results of our study reveal various gastric histological lesions induced by Helicobacter pylori (HP).It reinforces the studies already carried out in this field throughout the world because the notion of susceptibility of gastric cancer related to HP is a reality and especially the variability of other factors is according to the regions of the world make that the mystery which surrounds this bacterium still deserves to be elucidated.

Key words: Epidemiology, gastric histological lesions, Helicobacter pylori, Sikasso hospital.

Introduction

Helicobacter pylori infection is a ubiquitous infection, but it is more prevalent in developing countries due to low income and poor sanitation.1 Gastric infestation leads to acute gastritis, which is rapidly followed, if the infection persists, by chronic active gastritis, which may persist for several decades and evolve in a variable manner under the influence of host, bacterial and environmental factors. The relationship with gastric cancer has been known since the early 1990s, with Helicobacter pylori being classified as a gastric carcinogen by the WHO as early as 1994 [1, 2].Its role in peptic ulcer disease has been well defined, leading to consensus recommendations for routine investigation and eradication of Helicobacter pylori gastric infection [3, 4].

Worldwide, gastric cancer remains a major public health problem, constituting the second leading cause of cancer mortality. In 2000, the number of new cases in France was approximately 8000 and the number of deaths was 6323, representing 3.1% and 4.2% of all cancers respectively [5].

The association between H. pylori infection and gastric cancer has also been supported by 12 prospective cohort studies conducted in 2001 [6].

Available statistics on the incidence of gastric cancer show remarkable differences, probably related to geographical conditions, despite high prevalences of H. pylori infection in some regions [7, 8-12]. This is the case in Africa and South Asia, where cancer incidence remains low despite the high prevalence of H. pylori infection [10, 13, 14]. This paradoxical situation is reported to be the African and Asian enigma [7,15].One explanation for this phenomenon is the difference in virulence of the bacterial strains, as described by Yamaoka et al in 2010 [16]. These authors reported a higher virulence in strains carrying Cag A, Vac A, Oip A, or Dup A genes. Molecular genetic studies have also illustrated the oncogenic role of the number of EPIYA A sequences within the same gene in the occurrence of gastric cancer [17]. Other

environmental factors have been implicated in carcinogenesis, in particular high salt and nitrate consumption, alcoholism and smoking, in contrast to refrigeration and increased consumption of fresh produce (fruit, vegetables), which are thought to play a protective role [18, 16, 19].

A few rare studies have been conducted on gastric cancer in DR Congo, suggesting its low incidence. In 2004, Wani, in his final thesis (unpublished work) noted 81 cases of gastric adenocarcinoma over a period of 36 years in the digestive surgery department of the CUK, i.e. an annual average of 2.3 cases [20].

There is increasing evidence to support the prevention of gastric adenocarcinoma by eradication of H. pylori, mainly in countries with a high incidence of cancer and in the absence of pre-neoplastic lesions [21,22], the presence of which requires gastroscopic surveillance of patients [23,24].

The recent consensus meeting in Kyoto considers that Helicobacter pylori gastritis is an infectious disease, even in symptomatic patients and independently of the development of associated diseases (peptic ulcers, gastric cancer, etc.) [25].

Five histological parameters are investigated and graded according to the "Sydney system" classification published in 1991 for the evaluation of histological data from gastric biopsies [26,27]. These are :

o density of inflammatory infiltrate in the chorion

o gastritis activity defined by the existence of neutrophils in the mucosa

o Glandular atrophy;

o intestinal metaplasia which represents the transformation of the gastric type epithelium into an intestinal type epithelium. This metaplasia can be either "complete", small intestine type, or "incomplete", colonic type. The accuracy of this typing was not recommended in the revised Sydney classification but it was recommended to determine the presence and extent of intestinal metaplasia.

o HP density: This bacterium is visible on the standard Haemathein Eosin (HE) stain but it was recommended that a special stain be used to better identify and quantify it in cases of gastritis without identifiable bacteria on the HE stain.

As for the prognostic or predictive value of the malignancy of the lesions, two classification systems have recently been proposed by Rugge, Capelle and a group of expert pathologists in order to allow, by a semi-quantitative and topographical approach, to type gastritis at the level of gastric biopsies and to give an indication of the evolutionary risk towards dysplasia and cancer [28,29].

These classifications are called OLGA (Operative Link for GastritisAssessment) and OLGIM (Operative Link on Intestinal Metaplasia) and focus respectively on intestinal atrophy and metaplasia lesions which are pre-neoplastic lesions, favouring the development of intestinal-type gastric adenocarcinoma [28,29].

The high rate of HP infection in the population through a finding of our daily practices, the delay in the diagnosis of cancer at the early stage, the irregularity of patients in the endoscopic follow-up of histological lesions, the limited financial means of our populations, the insufficiency or even the absence of national support on gastric cancer, the derisory equipment of digestive endoscopy in our hospital structures, the lack of anatomopathologists in almost all our hospitals The absence of a real policy to fight cancer, self-medication, the exponential and uncontrolled proliferation of the illicit sale of "street" drugs and above all the lack of real information for the population on the risks of gastric cancer recently established as a result of infection by Helicobacter pylori, constitute factors that are highly conducive to the development of gastric lesions to stages that are difficult to treat, hence the interest of this study.

The objective of this study is to assess the overall prevalence of all gastric histological lesions induced by H.P. in adults in the hospital of Sikasso.

METHODOLOGY :

Design of the study: this is a two-component study:

o A retrospective descriptive study on the files of patients treated at the digestive endoscopy unit of the hepatogastroenterology department of Sikasso hospital and who met the inclusion criteria.

o A cross-sectional study of symptomatic patients who underwent FOGD+biopsies with positive histology for Helicobacter pylori.

Study setting and population: the Sikasso region, which is the 5th administrative region of Mali, has 10 health districts and one regional hospital. Patients were collected over a period from May 2020 to December 2021 in the digestive endoscopy unit of the Sikasso hospital.The study population was composed of symptomatic patients who performed FOGD plus biopsies with positive histology for Helicobacter pylori.

Inclusion and exclusion criteria:we included patients developing chronic Helicobacter pylori gastritis, diagnosed on gastric biopsies performed during FOGD with their informed consent. Other forms of gastritis without Helicobacter Pylori infection were not included as well as patients who had already received proton pump inhibitors (PPI) treatment and the poor quality biopsies.

Data sources and means of collection: the data sources were the digestive endoscopy and anatomo-pathological examinations registers, the results of the anatomopathology of gastric biopsiesand medical records. A standardized questionnaire was used for data collection.

Information collected : the variables collected mainly included age, sex and the various histological lesions associated to helicobacter pylori, diagnosed on pathological examination.

Sample size :as the study was descriptive, there was no sample size calculation. A total of 158 files were used for this study, of which 77 met the inclusion criteria.

Description of the procedure for the biopsies performed : the samples concerned biopsies of the antral mucosa, of the gastric body (fundus) and of the angulus. Each patient had an indication for FOGD during which two (02) antral, two (02) fundic and one (01) angulus biopsies were performed and immediately immersed in separate and labeled vials, containing formalin solution diluted to 10%. They were then referred for an anatomopathologic examination.

Histological classification used: In our study, we based ourselves on the classifications of OLGA and OLGIM which are more recent and better detailed.

- OLGA classification: is based on the semi-quantitative evaluation of the intensity of the atrophic lesions of the fundic and antral mucosa with a staging ranging from 0 to IV. Stages 0, I and II represent stages with low progressive risk.Stages III and IV define the stages with high progressive risk (Table 1).
- OLGIM classification: consisted in subdividing the patients into five classes (from 0 to IV) according to the grade and site of the intestinal metaplasia (IM), thus defining the global score of the intestinal metaplasia (Table 2).

For these two classifications, stages 0, I and II represent stages with low progressive risk. Stages III and IV define the stages with high evolutionary risk.

Data collection procedure and statistical analysis: the data collected through a questionnaire were entered in Excel, then analyzed using Epi-info 7. In, given the purpose of the study and the sample size, the data analysis was based on descriptive analysis. The quantitative variables were described as mean plus extreme and qualitative variables in terms of percentage.

Ethical aspect: the verbal agreement of patients on the use of their medical data in order to contribute to the dissemination of scientific data has been obtained.

Tables

Table 1: OLGA classification according to Rugge and Al.

Table 2: OLGIM classification according to CapelleandAl

RESULTS :

Socio-demographiccharacteristics: a total of 77 patients were included in this study according to the inclusion criteria. The mean age of our patients was 45.41 years (range 16-83) and the gender was 1.026.

Histological features on pathological examination: the epidemiological study included all histological lesions diagnosed on pathology associated with HelicobacterPylori. The lesion assessment allowed us to use the OLGA and OLGIM classifications, which allow us to establish a score of evolution according to the degrees of atrophy and intestinal metaplasia, and also all the other histological lesions listed at pathology in order to globally appreciate the consequences of Helicobacter Pylori infection on the gastroduodenal mucosa, allowing us to plan a better management of patients according to the current recommendations

OLGA classification: according to this classification, low risk stages (0, I and II) represented 87.01% in the gastric body and high risk cases represented 0%; in the antrum, low risk stages represented 79.22% and high risk stages represented 0%.

OLGIM classification: in the latter, low risk stages in the gastric body represented 87.01% and high risk stages 1.29%.

At the antral level, the low risk stages (0, I and II) represented 89.61% and the high risk stages represented 0%.

HelicobacterPylori density:

At the gastric body level, HP density was mild in 37.66%, moderate in 40.25% and severe in 5.19%.At the antral level, it was mild in 40.25%, moderate in 31.16% and severe in 2.59%.Glandular atrophy:

Atrophy was present in 45.45%, mild in 40.25%, moderate in 5.19% and severe in 0%. This distribution was according to the location of the atrophy. Thus, in the fundus and antrum, it was mild in 16.88% and 23.37% respectively. Atrophy was moderate in 2.59% of the fundus and antrum respectively. Intestinal metaplasia (IM):

Intestinal metaplasia was present in 3.89%, mild in 2.59% and severe in 1.29%.

Depending on the site, it was mild and severe in 1.29% at the fundic level respectively.

However, it should be noted that independently of the lesions falling within the OLGA and OLGIM classifications, the anapath results revealed other abnormalities in the gastric tract related to the presence of HP, namely

Cellular activity:

It was present in 89.61% at the fundic level, mild in 83.11%, moderate in 6.49% and severe in 0%.At the antral level the activity was present in 80.51%, mild in 79.22%, moderate in 1.29% and severe in 0%.

Chronicity of lesions:

At fundic level it was mild in 12.98%, moderate in 64.93% and severe in 0%.

At the antral level, chronicity was mild in 10.38%, moderate in 37.66% and severe in 0%.

Follicular density:

At the fundic level it was mild in 23.37%; moderate in 24.67% and severe in 6.49%.

At the antral level, follicular density was mild in 16.88%, moderate in 18.18% and severe in 2.59%.

Low grade dysplasia was present at the antral level in 1.29%.

In addition, poorly differentiated adenocarcinoma with a focus of mucosecretion on a focus of intestinal metaplasia at the antral level was present in 1.29%, moderately differentiated adenocarcinoma of the stomach accounted for 1.29% and independent cell adenocarcinoma on intestinal metaplasia associated with HP of the stomach was present in 1.29%.

Adenomatous-fibro-inflammatory polyp of the fundus was present in 1.29%.

Reactive gastritis associated with HP was present in 1.29%.

DISCUSSIONS:

Between May 2020 and December 2021, we conducted this study at the regional hospital of Sikasso with the aim of describing the epidemiology of gastric histological legions related to Helicobacter pylori. Different histological classification methods were used to classify and describe the lesions found. The different histological lesions found have allowed us to know that there is an evolution of the inflammation according to the anatomical site.

In this study, the average age of the patients was 45.41 years (range 16-83) and the sex ratio was 1.026.

Regarding gender, the literature reports a varied predominance. Some studies find a similar tendency in our case [30,31], while others note a female predominance [32,33,34], and this difference was not significant in other authors [35,36,37].

Gender does not appear to be a risk factor. Indeed, H. pylori infection is not related to individual characteristics but mainly to hygiene and socio-economic conditions [35].

The majority of epidemiological studies show that H. pylori infection starts early in childhood and persists throughout life [38]. However, it should be noted that there is little data available on the age of onset, rate and mode of infection.

However, some studies have shown that contamination occurs early in childhood, and before the age of 10 more than 50% of children in developing countries are already contaminated [39,40].

In our study, the low risk stages of lesions (0, I and II) represented the majority of lesions (87.01%) in the gastric body and the antrum (79.22%). In the literature, the low risk stages represent more than 90% of chronic gastritis with a clear predominance of stage 0 [41]. The trend is similar between what we reported and the data in the literature, but the discrepancy observed can be explained by the limited size of our sample.

In addition, we did not find any cases of high-risk stages in the present study, whereas in a reported Italian series, high-risk stages represent 6.4% with a clear predominance of stage III (44%) with a very low rate of stage IV (<1%) [42]. High risk gastritis according to OLGA is characterised by a marked atrophy of the mucosa, which would lead to environmental variations with a disturbance of the ecosystem becoming inadequate for bacterial multiplication [41].

In terms of Helicobacter pylori density, we found a mild density in the gastric body in 37.66%, moderate in 40.25% and severe in 5.19%. Our data agree with those of Sana Ben Slama from the Internal Security Force (ISF) hospital in La Marsa, Tunisia, who reported a mild density in 37% (n=37). In contrast, in his study, the density of HP was moderate in 26% while our series found 40.25%.As for the severe density of HP, our data are much lower than those (37%) found by Sana Ben Slama [43].

In our series, this disparity in data can be explained on the one hand by the small size of our sample and on the other hand by environmental differences.

The variable anatomical and clinical expressions of the infection depend on the interaction between the virulence of the bacteria, the inflammatory and immune response of the host and environmental factors [23, 43].

As for glandular atrophy (45.45%) in our series, it was mild in 40.25%, moderate in 5.19% and severe in 0%. According to the distribution according to the site, the antral region predominated with a mild intensity in 23.37%, moderate in 2.59%, severe in zero cases. The same trend of predominance according to antral site was reported in the study by Sana Ben Slama from the FSI hospital in La Marsa, Tunisia [43] and that of Chen et al [44]. However, our population was much smaller than those of these two authors in terms of intensity, hence the absence of severe cases in our series. Environmental factors and the limited number of patients in our study could explain the few differences observed between our data and those reported by Sana Ben Slama and Chen et al.

The study of glandular atrophy according to the OLGIM classification shows a clear correlation between glandular atrophy and high risk stages [45].

Thus, in our series, low risk stages represented 89.01% of both OLGA and OLGIM classifications. Our data are consistent with and support those reported in the literature [40].

High risk stages represented 1.29% at the fundic level. These data in the OLGIM classification are slightly lower than those found in the literature but reflect their low occurrence [46].

However, the presence of intestinal metaplasia at the gastric level (IMG), regardless of its rate, is a sign of a definite evolution of gastric HP lesions towards neoplasia and therefore imposes a fairly thorough surveillance and regular follow-up.

A distinction can be made between complete GIM (type 1) and incomplete GIM (type 2), which is a less well-differentiated form and is associated with a higher neoplastic risk [47].

Other histological lesions associated with HP were considered in our study in order to know their impact on the evolution of the lesions considered at risk of gastric adenocarcinoma. 89.61% of the cellular activity reflecting the quantity of neutrophilic polynuclear cells (NPC) in the gastric mucosa was present at the fundic level and 80.51% at the antral level.

Several studies have shown that cell activity correlates with the presence and number of Helicobacter Pylori on the mucosal surface [26, 48, 49].

In the study, the presence of cellular activity was more prevalent at the antral (94.5%) than at the fundic (91.8%) level. This means de facto that there is a greater effect in the antrum than in the fundus in our patients, which needs to be taken into account in the follow-up treatment.

The chronicity of gastric lesions is a chronic inflammation of the gastric mucosa attributed in most cases to the presence of Helicobacter pylori. The different classifications of chronic gastritis include two distinct frameworks [53]:

o "Classic" chronic atrophic gastritis, which progresses to progressive atrophy of the gastric mucosa, regardless of the aetiology.

o Chronic gastritis, because of its prolonged evolution in successive attacks. This group includes bile reflux gastritis, varioliform-lymphocytic gastritis, eosinophilic gastritis and granulomatous gastritis.

Depending on the site, our study reported chronic mild gastritis in 12.98%, moderate in 64.93% at the fundic level, while at the antral level it was mild in 10.38%, moderate in 37.66%.

Other studies have shown that the prevalence of chronic Helicobacter pylori gastritis varies from 72-91.3% in Africa and 64-100% in Europe and North America [54, 55].Our data are

lower than those found in the literature and the difference observed reflects the small size of our sample.

When it comes to follicular density at the fundic level, it was mild in 23.37%; moderate in 24.67% and severe in 6.49% while at the antral level it was mild in 16.88%; moderate in 18.18% and severe in 2.59%. Garg et al. also identified lymphoid follicles in 19% of subjects, and suggest that the presence of lymphoid follicles was strongly associated with mucosal inflammation, activity and Helicobacter Pylori infection. [56]

In our study, low-grade dysplasia was present at the antral level in 1.29%.

In the literature review, the presence of Helicobacter Pylori and the carcinogenic risk is well established.In China a cohort study revealed that Helicobacter Pylori infection is one of the risk factors for the progression of precursor lesions (superficial gastritis, chronic atrophic gastritis and intestinal metaplasia) to gastric dysplasia and cancer[57].

Our data corroborate this finding of fact.

Three cases of gastric cancer were noted in our series, namely

o poorly differentiated adenocarcinoma with a focus of mucosecretion on a focus of intestinal metaplasia at the antral level was present in 1.29%.

o moderately differentiated adenocarcinoma of the stomach accounted for 1.29%.

o Independent cell adenocarcinoma on intestinal metaplasia associated with HP of the stomach was present in 1.29%.

Epidemiological and pathophysiological arguments demonstrate a strong association between distal gastric cancer and Helicobacter pylori. In a prospective study of Japanese patients with H. pylori gastritis, the rate of development of gastric adenocarcinoma was 2.9% after 8 years of follow-up [58].

Our results reflect and are consistent with those in the literature, although our patients were not followed up.

Adenomatous-fibroinflammatory polyps of the fundus were present in 1.29% of our study. A gastric polyp is a raised lesion protruding into the lumen of the stomach. This very simple macroscopic and endoscopic definition covers a very wide spectrum of lesions, epithelial or non-epithelial, neoplastic or non-neoplastic [59,60].

The histological series do not allow us to know the real prevalence of gastric polyps, but give a good assessment of the relative frequency of the different histological types [61].

In our series, HP-associated reactive gastritis was present in 1.29%.

Reactive gastropathy, formerly called chemical gastritis, is the most common gastritis after H. pylori gastritis. It is not a chronic gastritis, but a gastric reaction to auxiliary salts (especially after gastric surgery) or to certain drugs (NSAIDs) [62].

CONCLUSION:

The results of our study reveal various gastric histological lesions induced by Helicobacter pylori.It reinforces the studies already carried out in this field throughout the world because the notion of susceptibility of gastric cancer related to HP is a reality and especially the variability of other factors is according to the regions of the world make that the mystery that surrounds this bacterium still deserves to be elucidated.

There is therefore an urgent need to treat Helicobacter Pylori and to closely monitor histological lesions caused by this bacterium according to the updated recommendations for the prevention of gastric cancers.

What is known about this topic

Previous studies on the histological lesions caused by HP infection in the stomach have led to classifications to assess their evolution over time. These are mainly :

o the "sydney system" classification published in 1991 for the evaluation of histological data from gastric biopsies

o OLGA and OLGIM classifications of intestinal atrophy and metaplasia lesions which are pre-neoplastic lesions

Our study reinforces the studies already carried out in this field throughout the world because the notion of susceptibility to gastric cancer linked to HP is a reality and above all the variability of other factors according to the regions of the world make the mystery surrounding this bacterium still deserve to be clarified.

Whatthisstudyadds

Our study adds value by taking into account all gastric lesions diagnosed in histology in order to appreciate their evolution as well as their minimal repercussions on the gastric mucosa and in the occurrence of gastric cancer, unlike previous studies based essentially on the lesions that enter and define these classifications.

Conflicts of interest

The authors declare no competing interests

Authors' contributions

OumarTraore: study design, data collection, edition of the final report and edition of this manuscript;

Abdoul Salam Diarra: study design, data statistical analysis, edition of the final report and edition of this manuscript;

Tawfiq Abu: reviewing and translating the manuscript in English;

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All the authors have read and agreed to the final manuscript.

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Liste de Tableaux

Tableau 1: classification OLGA d'après Rugge et Al.				
Score de l'atrophie	**Corps Gastrique**			
	Absente	**Légère (glandes atrophiques<30%)**	**Modérée (glandes atrophiques entre 30-60%)**	**Sévère (glandes atrophiques>60%**

Antre	Absente	Classe 0	Classe I	Classe II	Classe III
	Légère Glandes atrohiques<30%	Classe I	Classe I	Classe II	Classe III
	Modérée Glandes atrophiques entre 30-60%	Classe II	Classe II	Classe III	Classe IV
	Sévère Glandes atrophiques>60 %	Classe III	Classe III	Classe IV	Classe IV

Tableau 2 : classification OLGIM d'après Capelle et Al.

Score de Métaplasie intestinale		**Corps Gastrique**			
		Absente	**Légère (glandes en métaplasie int.<30%)**	**Modérée (glandes en métaplasie int.30-60%)**	**Sévère (glandes en métaplasie int.>60%**
Antre	Absente	Classe 0	Classe I	Classe II	Classe III
	Légère Glandes atrohiques<30%	Classe I	Classe I	Classe II	Classe III
	Modérée Glandes atrophiques entre 30-60%	Classe II	Classe II	Classe III	Classe IV
	Sévère Glandes atrophiques>60%	Classe III	Classe III	Classe IV	Classe IV