

Involvement of Helicobacter Pylori in the Genesis of Precancerous Stomach Lesions: Epidemiological Aspects Based on an Algerian Study

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Received: 11 Oct 2022

Accepted: 20 Oct 2022

Published: 25 Oct 2022

J Short Name: AJSCCR

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Citation:

Did Loubna, Involvement of Helicobacter Pylori in the Genesis of Precancerous Stomach Lesions: Epidemiological Aspects Based on an Algerian Study. *Ame J Surg Clin Case Rep.* 2022; 5(13): 1-4

Keywords:

H. pylori; Prevalence; Chronic gastritis; Atrophy; Fundic antrum; Histological activity

1. Abstract

Helicobacter Pylori is a bacterium involved in the genesis of precancerous lesions of the stomach. This infection is universally prevalent but is higher in developing countries. A retrospective study spread over 3 years (2018-2019-2020) was conducted at the pathological anatomy laboratory of the Chadli Bendjedid Hospital in El Tarf, northeast Algeria. All patients were submitted to a fiberoptic, and the diagnosis was established by histological examination. The results show that of the 134 patients examined, 100% were diagnosed with chronic gastritis. The prevalence of H. pylori infection was 61.94%; 63.86% of women were affected, and the most susceptible age group was 50-59 years; 36.14% of men were affected, mainly in the age group 30-39 years. Locally, the fundic antrum area appeared to be the most affected (81.93%) with moderate chronic atrophic gastritis (78.57%) and silent activity (94.28%), which favors the development of cancer cells.

2. Introduction

In 1982, J. Robin Warren and Barry J. Marshall identified a bacterium colonizing the gastric mucosa and named it Helicobacter Pylori (*H. pylori*) [1]. Numerous studies have shown its etiopathogenic role in several gastric and duodenal diseases (gastritis, ulcer, lymphoma, gastric cancer) [2]. Other associations between H. pylori infection and certain extra digestive diseases have been reported [3]. It is estimated that about half of the world's population is infected with H. pylori, mainly in developing countries.

However, it affects 70-90% of the population in developing countries, making it a real public health problem [4]. In these countries, about 80% of the subjects are infected in childhood and remain so throughout their lives [5, 6]. In industrialized countries, the prevalence varies from 20 to 40%. The mode of transmission of H. pylori is still unclear. As H. pylori has been isolated from stool, saliva and dental plaque, this suggests that transmission is possible via the oral or fecal route [7]. Our work is based on an epidemiological study to determine the prevalence of detected chronic gastritis and the presence of H. pylori over the 3 years (2018- 2019- 2020) as well as demographic variables and site of infection, with the type of atrophy.

3. Patients and Method

A retrospective and descriptive study was conducted at the pathological anatomy laboratory of the Chadli Bendjedid Hospital of El Tarf. The study involved 134 patients with gastric pathologies and was spread over 3 years (2018-2019-2020). Our work consisted of a consultation of the department's analysis register, made available to us, and from which we proceeded to a census of gastritis cases diagnosed in the laboratory from biopsies sent by the attending physician. Demographic (age and sex) and clinical information was also collected.

4. Studied Variables

Five types of variables were recorded for this study: variability related to the prevalence of detected chronic gastritis and the pres-

ence of *H. pylori* over the study period, the demographic variable (age and sex), a variable related to the site of infection, the type of atrophy, and finally the histological activity.

5. Statistical Method

The study of demographic data (age and sex) of the 134 patients, as well as the clinical data, were collected and recorded, then processed by Excel 2013 software and described by their numbers and percentages.

6. Results

6.1. General characteristics of the sample studied

We studied the prevalence of detected chronic gastritis and the presence of *H. pylori* over the study period. The study population consisted of 134 biopsies from patients with gastric pathologies sent for anatomopathological examination. Of these, 100% were diagnosed as chronic gastritis. *H. pylori* was detected in 61.94% of patients with chronic gastritis (Table 1).

Table 1: Prevalence of *H. pylori*.

134	
Positive patients	Negative patients
83	51
61.94%	38.05%

6.2. Age and gender of patients

The 83 patients reported the presence of *H. pylori* with a mean age of 51 years, of which 53 women and 30 men, the rate of (63.86%) and (36.14%) were recorded, respectively. At the age of 30-39 years, men are affected by a rate of 26.66%, and a rate of 33.33% is recorded at the age of 50-59 years in women, constituting the largest rate of consultants. Table 2 shows the distribution of patients by age and gender.

Table 2: Description of the study sample by gender and age.

Type	Number		%	
Women	53		63.86%	
Men	30		36.14%	
Age group	Number		%	
	Woman	Men	Woman	Men
-20	4	1	7.54%	3.33%
20-29	11	4	20.37%	13.33%
30-39	4	8	7.54%	26.66%
40-49	7	6	12.96%	20%
50-59	18	7	33.33%	23.33%
60-69	7	3	12.96%	10%
70-79	1	0	1.85%	0%
80-89	1	1	1.85%	3.33%

6.3. Variability according to the location of the lesion

Among the three areas of the stomach, the fundic antrum appears to be the ideal location for *H. pylori* colonization, with a rate of (81.93%), and (9.64%) for the pyloric antrum and (8.43%) for the fundus (Figure 1).

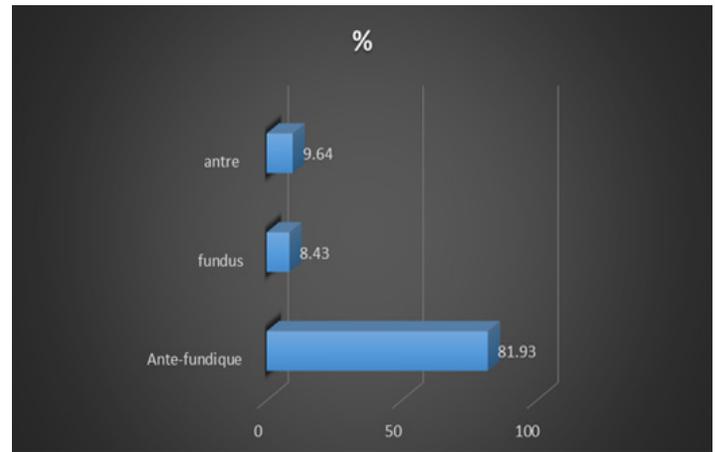


Figure 1: Prevalence of gastritis due to *H. pylori* infection by the lesion site.

6.4. Variability according to the presence and type of atrophy

The 83 patients with chronic gastritis with the presence of *H. pylori* (15.66%), superficial chronic gastritis, and (84.33%), moderate type atrophic cases (78.57%) with silent activity (not active) for a rate of (94.28%) presented as well as mild (4.29%) and severe atrophic (17.14%) and chronic atrophic gastritis with histologic activity (5.72%) (Figure 2 and Table 3).

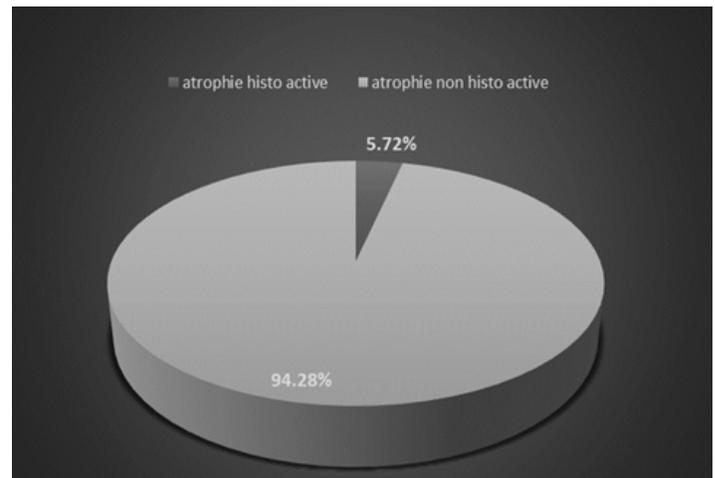


Figure 2: Overall prevalence of histological activity in *H. pylori*-infected chronic atrophic gastritis.

Table 3: The rate of different pathological types in the *H. pylori*-infected population.

Superficial chronic gastritis	Chronic atrophic gastritis		
15.66%	84.33%		
	Slight	Moderate	Severe
	17.14%	78.57%	4.29%

7. Discussion

Epidemiological studies on *H. pylori* infection in chronic gastritis are scarce in Algeria. This infection is widespread but is higher in developing countries (78% in Algeria, 71% in Morocco and 69% in Ivory Coast) [8]. It is frequently linked to the poor socio-economic status of families, determined mainly by the size of the dwelling and its sanitary level, the risk of infection increases when several people are in close contact, as in a large family [4]. However, these conditions are improved in industrialized countries.

Our study population included 134 patients. The *H. pylori* prevalence was estimated to be (61.94%). Another study on *H. pylori* infection in Morocco reported a rate of (69%). [9]. This frequency is within the limits of the values reported by several African studies, which vary from (56.4%) to (91.3%) and remains higher than in European data, where this frequency does not exceed (45%) [10-11]. In our epidemiological survey, a prevalence of the female sex in chronic gastritis and *H. pylori* infection was reported with rates of (60.44%) and (63.86%) respectively. Compared to the male sex, (39.55%) chronic gastritis and (36.14%) *H. pylori* infection were recorded. However, other studies noted the predominance of men [12]. Essedik et al. (2013) did not find a significant difference in prevalence between men [13].

In addition, *H. pylori* is found in our series if compared by age groups. At the age of 30-39 years, men are affected by a rate of 26.66%. A rate of 33.33% is recorded at the age of 50-59 years in women, constituting the highest rate among the consultants. Our results are close to those obtained in developed countries, where the highest prevalence (66%) is recorded at the age of 60 years [12]. The site of *H. pylori* proliferation is the fundic antrum par excellence. Indeed, the antrum is colonized by *H. pylori* in (81.93%) of our population. These results are in agreement with those of the study conducted in the Gharb Chrarda-Beni Hssen region in Morocco, which attributed (70.9%) of lesions to this site [14]. However, these values are still higher than those that Binan et al. (2006) reported. Seoane et al. (2005) attributed 40% and 48.1% of lesions to *H. pylori* at the antral site [15-16]. It should be noted that the high presence of *H. pylori* in the fundic antrum is since gastric biopsies are mainly performed at this level, which does not prevent the bacteria from colonizing the entire mucosa, including the pyloric region, hence its name.

On the other hand, *H. pylori* is the cause of several gastric pathologies. It is the cause of 80% of chronic atrophic gastritis [1]. These results are in agreement with those found in our study. Indeed, *H. pylori* is most frequently correlated with chronic atrophic gastritis, with a prevalence of (84.38%). Our results are also in agreement with those recorded in Morocco, where 91.8% of chronic atrophic gastritis is correlated with *H. pylori* [13]. In the study population, 78.57% of atrophies are of the moderate type with silent (non-active) activity for a rate of (94.28%).

The high rate of *H. pylori* infection reported in our survey and its pathogenicity are related to the presence of numerous proteins and cytotoxins (Cag A, Vag A). They generate cytotoxic power on the cell (inhibition of phagosome-lysosome fusion or alteration of the capacity to present antigens) [17]. This causes histopathological changes in the cell, leading to atrophy. The predominance of the moderate type of atrophy proves that the pathological process evolves beyond the stage of mild atrophy and may lead to severe atrophy with detrimental consequences for the gastric cell and the organism as a whole. Silent (non-active) atrophies are the most dangerous, as the disease potentially progresses silently to more severe stages.

8. Conclusion

The results of our study are similar to those of other epidemiological studies conducted in Morocco or several developing countries for the prevalence of *Helicobacter Pylori* infection. A female prevalence of *H. pylori* was reported at 63.86% and 36.14% in males. The results show that at the age of 30-39 years, men are affected by a rate of 26.66%, and a rate of 33.33% is recorded at the age of 50-59 years in women, constituting the largest rate of consultants.

The fundic antrum seems to be the ideal place for *H. pylori* colonization with a rate of (81.93%), as well as atrophic (84.33%), moderate (78.57%), and silent (non-active) cases with a rate of (94.28%). Since the discovery in 1982 by Marshall and Warren of the *H. pylori* bacterium in the pyloric antrum, numerous studies have shown its etiopathogenic role in several gastric and duodenal diseases (gastritis, ulcer, lymphoma, gastric cancer).

In our epidemiological survey, *H. pylori* was detected in 61.94% of the study population (134 patients). The most infected age group during these 3 years was between 21 and 59. Presence was reported in women more than in men, with 63.86% and 36.14%, respectively. *H. pylori* was most commonly correlated with chronic atrophic gastritis with a prevalence of 84.38%, and 78.57% of the atrophies were moderate, with low-grade dysplasia in 27% of cases.

References

1. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet*. 1984; 323(8390): 1311-5.
2. Mignon M. 2005 Nobel Prize in Medicine: Barry J. Marshall and J. Robin Warren. *Helicobacter pylori* crowned. *Medicine Sciences*. 2005; 21(11): 993-994.
3. Longo-Mbenza B, Nsenga JN, Mokondjimobe E, Gombet T, Assori IN, Ibara JR, et al. *Helicobacter pylori* infection identified as a cardiovascular risk factor in Central Africans. *Vasc Health Risk Manag*. 2012; 6:455-61.
4. Malaty HM. Epidemiology of *Helicobacter pylori* infection. *Best Practice Research Clinical Gastroenterology*. 2007; 21(2): 205-14.

5. Brown LM. Helicobacter pylori: epidemiology and routes of transmission. *Epidemiol Rev.* 2000; 22(2): 283-97.
6. Bommelaer G, Stef A. Peptic ulcer disease: before and after Helicobacter pylori. *Gastroenterol Clin Biol.* 2009; 33(8-9): 626-634.
7. Everhart JE. Recent developments in the epidemiology of Helicobacter pylori. *Gastroenterology Clinics of North America.* 2000; 29(3):78-559.
8. Faik M. Update on gastric infestation by Helicobacter pylori. *Médecine du Maghreb.* 2000; 79: 17-19.
9. Hjoutei HA, et al. Helicobacter pylori infection in 755 patients with digestive symptoms: Institut Pasteur du Maroc, 1998-2007 Eastern MediterraneanHealth Journal La Revue de Santé de la Méditerranée orient EMHJ. 2010; 16: 7.
10. Diomande MI, et al. Chronic gastritis and Helicobacter pylori infection in Côte d'Ivoire: a study of a series of 277 symptomatic patients. *Clinical and Biological Gastroenterology.* 1991; 15(10): 711-716.
11. Sobalah GM, et al. Screening dyspepsia by serology to Helicobacter pylori. *Lancet.* 1991; 338(13): 94-96
12. Ramanampamony RM, Randria MJD, Razafimahefa SH, Ratsiman-disa R, Rajaonarivelo P. Seroprevalence of Helicobacter pylori infection in a Malagasy population sample. *Bull Soc Pathol Exot.* 2007; 100(1): 57-60.
13. Amel Essedik, et al. Epidemiological and clinical aspects of Helicobacter pylori infection through a Moroccan study Hegel. 2013; 3: 3.
14. Attaf N, et al. Epidemiological profile of Helicobacter pylori infection in the Gharb-Chrarda-Beni Hssen region. *Biology & Health.* 2004; 4(1): 25-3.
15. Binan Y, et al. Gastric cancer and Helicobacter pylori: results from an endoscopy center in Abidjan. *International Journal of Medical Sciences.* 2006; 8(1): 23-27.
16. Seoane A, et al. Helicobacter pylori and gastric cancer: relationship to histologic subtype and tumor location. *Gastroenterology and Hepatology.* 2005; 28 (2): 4-60.
17. Brent Polk 1, Richard M Peek Jr. Helicobacter pylori: gastric cancer and beyond *Nat Rev Cancer.* June. 2010; 10(6): 403-14.