

Journal of Epidemiology and Global Health
Vol. **10(4)**; December (2020), pp. 293–297
DOI: https://doi.org/10.2991/jegh.k.200527.001; ISSN 2210-6006; eISSN 2210-6014
https://www.atlantis-press.com/journals/jegh



Research Article

Epidemiological Profile of *Helicobacter pylori* **Infection in Patients with Digestive Symptoms in Algeria**

Houria Kasmi¹, Koula Doukani^{1,5,10}, Ahmad Ali², Souhila Tabak¹, Hasna Bouhenni¹

¹Department of Nature and Life Sciences, University of Ibn Khadoun, Tiaret, Algeria

ARTICLE INFO

Article History

Received 10 August 2019 Accepted 18 May 2020

Keywords

Helicobacter pylori infection digestive symptoms gastric pathology Algeria

ABSTRACT

Background: The purpose of this study was to assess the prevalence of *Helicobacter pylori* infection in Algerian patients with peptic disorders and evaluate the impact of different epidemiological factors (age, sex, sampling site, presence or absence of *H. pylori*, and type of pathology related to this bacterium).

Methods: We undertook a retrospective and descriptive study on a series of 735 symptomatic patients identified in the laboratory of pathological anatomy at Hassani Abdelkader University Hospital Center of Sidi Bel Abbes, Algeria, over a period of 16 years from January 2002 to December 2017. All patients had benefited from a high gastroscopic fibroscopy and the diagnosis was made by histological examination (hematoxylin–eosin staining). The epidemiological factors, as well as the main gastric diseases related to this bacterium, were studied.

Results: The prevalence of H. pylori infection was 66.12%. The infection was more important in the age group 60–69 years (71.43%). The prevalence of H. pylori infection was statistically higher in women than in men (69.3% vs. 60.7%, p < 0.01). The antral region was most colonized by H. pylori (71.73%). In addition, the infection was associated mainly with atrophic gastritis (69.65%).

Conclusion: In this context, the identification of epidemiological data would be of great value in guiding strategies to control the spread of this bacterium.

© 2020 Atlantis Press International B.V.

This is an open access article distributed under the CC BY-NC 4.0 license (http://creativecommons.org/licenses/by-nc/4.0/).

1. INTRODUCTION

Helicobacter pylori is the only bacterium that colonizes the human stomach where it can cause gastric ulcers, gastritis and potentially lead to stomach cancer [1]. Indeed, WHO recognized H. pylori as the only bacterial carcinogen in 1994 [2]. It infects more than half of the world's population; its prevalence varies according to the populations, even within the same country [3]. The prevalence of H. pylori infection is influenced by several factors such as age, socio-economic status, ethnicity, and inter-family relations [4]. Pathways of transmission of H. pylori infection have been suggested [5]. It has been detected in saliva [6], vomit [7], gastric reflux product, and stool [8]. The most likely mode of transmission is from person to person by either the oral-oral route or perhaps the fecal-oral route [9]. Currently, to detect the presence of *H. pylori* infection, several methods are available (rapid urease test, bacterial culture, molecular tests involving PCR, histology, breath test, looking for antibodies in the blood or urine, and looking for antigens in the stool, etc.) [10]. Treatments for *H. pylori* infection consist of the

combined administration of antibiotics and gastric anti-secretory drugs, mainly Proton Pump Inhibitors (PPIs). Today, PPI-based triple therapy is the most commonly used method worldwide [11]. In Algeria, the high prevalence of *H. pylori* and the spread of this bacterium are alarming; moreover, its association with serious gastric pathologies requiring deep research in epidemiology by studying the impact of epidemiological factors includes socioeconomic status, nutritional status, hygiene, urbanization, and methods of diagnosis [12]. In addition, investigations have to be involved concerning the gastric pathologies associated with H. pylori and, much more, gastric cancer which is generally related to this bacterium [13]. In terms of diagnosis, it should be applied bacterial culture and characterization of different strains of H. pylori that allow the determination of its sensitivity to antibiotics (antimicrobial resistance) and its virulence factors to apply therapeutic tests for the purpose of diagnosis and eradication of H. pylori to reduce the risk of developing gastric cancer [14]. In Algeria, in particular, it requires the improvement of sanitary conditions and therapeutic strategies of patients suffering from H. pylorirelated gastric diseases to decrease the prevalence of this infection. Since 2000 there were no any studies in the field of epidemiology. However, the prevalence of this infection was very high. Through this study, our objective was the estimation of prevalence of

²Department of Life Sciences, University of Mumbai, Mumbai, India

[&]quot;Corresponding author. Email: kouladoukani@gmail.com
Data availability statement: The authors confirm that the data supporting the findings of this study are available within the article.

H. pylori infection in Algerian patients with peptic disorders collected over a period of 16 years from January 2002 to December 2017 at the University Hospital Center in Sidi Bel Abbes, Algeria, and evaluation of the impact of different epidemiological factors (age, sex, sampling site, presence or absence of *H. pylori*, and type of pathology related to this bacterium).

2. MATERIALS AND METHODS

2.1. Study Site

The city of Sidi Bel Abbes is located in the west of Algeria at the crossroads between the big cities of this part of the country. Its population is estimated at 212,935 inhabitants (year 2010) and covers an area of 9,150.63 km². It has two specialized hospitals, three public hospitals, seven public health facilities of proximity, and one University Hospital Center. The latter was selected for data collection because there is an anatomopathological investigation unit and a gastro-enterology department that provides fibroscopy. Both services are located in the same setting (collaboration between the two services), which makes it very easy for the patient to follow-up.

2.2. Study Population

It is a retrospective and descriptive study on 735 patients (463 women and 272 men) who are between 6 and 87 years old, over a period of 16 years from January 2002 to December 2017. They presented digestive symptoms (abdominal pain, vomiting, nausea, gastric burn) and have benefited from a high digestive fibroscopy. The medical form mentioned the name, age, sex, nature of the sampling site, and clinical diagnosis.

2.3. Inclusion Criteria

- 1. Patients of origin from the region of Sidi Bel Abbes.
- 2. Patients with gastroduodenal diseases.
- 3. Each patient was undergoing a gastric biopsy.

2.4. Study Design

The gastroenterologist had performed a high digestive endoscopy, which had allowed biopsy sampling, often at the level of the antral regions, sometimes at the antro-fundic or pyloric. These biopsies were often fixed with formalin and they constitute the biological material object of histopathological examination. Then they were sent to the Laboratory of Anatomo-cyto-pathology for a histological study.

The histological examination of gastric biopsies by hematoxylineosin staining which makes it possible to determine the type of histological lesions caused by *H. pylori*, has always been supplemented by slow Giemsa staining, which gives a better contrast for the bacterium. This examination makes it possible to obtain a good morphological quality of the abnormalities of the gastric mucosa, notably an inflammation or an atrophic process.

2.5. Data Collection

- 1. The first step was to look for cases of gastroduodenal diseases from the histopathological examinations on gastric biopsies.
- 2. The second step was to determine the cases of gastroduodenal diseases caused by *H. pylori* or not.
- 3. The third step was to study the distribution of gastroduodenal diseases due to *H. pylori* by sex, age groups, sampling site, and type of gastric pathology associated with this bacterium.

2.6. Statistical Analysis

Descriptive statistics, count and percentage, were calculated using SPSS version 24.0 (IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Armonk, NY, USA). A non-parametric one-sample Chi-square test ($p \le 0.01$) was performed to assess differences among proportions of the population in terms of test (negative and positive), age, sex, sampling site, and type of gastric pathology associated with H. pylori.

3. RESULTS

Among the 735 patients with peptic diseases, we found 486 *H. pylori* infected patients who give an infection rate of 66.12% (Table 1).

Table 2 shows the prevalence of *H. pylori* infection by age group. The difference of values between ages gave a maximum rate of 71.43% in patients aged between 60 and 69 years. Indeed, we have noted a frequency of 55.56%, 61.18%, 66.43%, 67.53%, 66.40%, 63.04%, and 66.67% of *H. pylori* infection in the age group and ranging successively for '0–19', '20–29', '30–39', '40–49', '50–59', '70–79' and '80–89' years.

Table 1 Percentage of patients infected or not with *H. pylori*

Population	Number	Percentage (%)
Population infected with <i>H. pylori</i>	486	66.12
Population not infected with <i>H. pylori</i>	249	33.88
Total	735	100

 $\chi^2 = 76.42, p < 0.01.$

Table 2 Prevalence of *H. pylori* infection by age group

Age group	Number	Prevalence (%)
0–19	10	55.56
20-29	52	61.18
30-39	93	66.43
40-49	104	67.53
50-59	83	66.40
60-69	80	71.43
70-79	58	63.04
80-89	6	66.67

 $\chi^2 = 3.77, p < 0.01.$

Table 3 Prevalence of *H. pylori* infection by sex of patients

Sex	Number	Prevalence (%)
Female	321	69.33
Male	165	60.66

 $\chi^2 = 5.75, p < 0.01.$

Table 4 Prevalence of *H. pylori* infection by sampling site

Sampling site	Number	Prevalence (%)
Antrum	439	71.73
Fundus	33	32.35
Antrum-Fundus	14	66.67

 $\chi^2 = 60.53, p < 0.01.$

Table 5 | Rates of different gastric pathologies in the population associated with *H. pylori*

Pathology associated with infection	Number	Prevalence (%)
Atrophic gastritis	358	69.65
Superficial gastritis	85	55.56
Interstitial gastritis	43	63.24

 $\chi^2 = 10.73, p < 0.01.$

A one-sample Chi-square test was conducted to assess whether women are more susceptible to the disease incidence than men. Results showed that the prevalence was 69.3% in female, which is statistically greater than in male (60.7%) (Table 3).

The obtained results, with regard to the distribution of *H. pylori* according to the sampling site, showed that the proliferation site of *H. pylori* was the antrum with excellence. Indeed, 71.73% of the lesions are attributed to the antrum and 66.67% was observed at the antrum–fundus level. On the other hand, the fundus remained the least infected site with 32.35% (Table 4).

All these patients had gastric diseases at different levels of the stomach. Among the patients infected with *H. pylori*, 69.65% suffered from atrophic gastritis, 55.56% are carriers of superficial gastritis, and 63.24% had interstitial gastritis (Table 5).

4. DISCUSSION

Helicobacter pylori prevalence of symptomatic patients ranges between 70% and 95% in developing countries and between 30% and 50% in developed countries [15]. In Morocco, the prevalence of *H. pylori* infection is 69.2% corresponds well to the prevalence noted in developing countries [16]. While in our studied population, the prevalence rate was 66.12%. Although our results are those of a study limited to the population attending the specialized endoscopy care center, they corroborate with other work done in Algeria. In particular, the study based on the histological analysis (Pathology department of the Mustapha Pacha University Hospital Center in Algiers over a 5-years period from January 1996 to December 2000) on a series of 3411 patients suffering from a gastric pathology, revealed a prevalence of 75% infection [12]. Serological studies of Raaf et al. [17] reported a prevalence of *H. pylori* in the Algerian

population; 56%. According to Faik [18], the prevalence was 71% in Algeria in 2000. In comparison with developed countries like France 46.9%, England 35.5% and Belgium 32.7% [19], 15% in Spain, 10% in Sweden and 1.8% in Japan [17] and Switzerland (18.9%) [15], our results are much higher. In developing countries, the prevalence remains the highest; 90% in Egypt [20], 94% in Libya [21], 80% in Saudi Arabia [22], 69% in Ivory Coast [23], 82.8% in Senegal [24], 87% in Nigeria, 97% in Gambia and 75.4% in Ghana [25], 82% in Madagascar [26], and 68.3% in Cameroon [27]. This frequency is thus related to socio-economic status and levels of hygiene [28].

The results of this study indicate that females were the most infected by *H. pylori* compared with the males. However, Malaty [29], reported that it is generally accepted that men and women have the same risk of becoming infected at any age. Elmanama et al. [30] demonstrated that both sex are infected by *H. pylori*, confirming the results of our series. In contrast, other studies have found a male predominance [26,31].

Concerning the prevalence of *H. pylori* infection by age, we obtained a peak rate (71.43%) affecting the '60–69' age group. This average is higher than those found by Joutei et al. [32], which revealed a rate of 11% among subjects in the '60–70' age groups. For Ivorian, no significant difference has been reported for age groups [33]. It is argued that in Africa, every adult, regardless of socio-economic status, had a childhood in an environment that is conducive to contamination [34]. Our results are similar to those in European countries, which are around 60 years old [25]. It has been reported that the prevalence of *H. pylori* infection increased by age manner [35].

The antrum is colonized by *H. pylori* in 71.73% of our population. The same results were found by Attaf et al. [36] which attributed 70.9% of lesions to this site, and Joutei et al. [32] with 73%. In another study, these values remain higher than those reported by Binan et al. [37] who attributed 40.0% of *H. pylori* lesions to the antral site.

Helicobacter pylori is at the origin of the occurrence of several gastric pathologies. It is at the origin of 80% of chronic atrophic gastritis resulting from the inflammatory and immunological response induced by this bacterium [38], 1–10% gastroduodenal ulcers, and 1–3% gastric cancers [39]. These results are consistent with those found in our study. Indeed, *H. pylori* is most often linked to atrophic gastritis (69.65%). Concerning superficial and interstitial gastritis, the evidence of the involvement of *H. pylori* in these pathologies remains less documented. It should be noted that a population suffering from gastritis caused by *H. pylori* would constitute a risk for the occurrence of cancer [13].

5. CONCLUSION

The results of this research indicate a high prevalence of patients infected with *H. pylori* in the study population. This prevalence varied according to age, sex, sample site, and gastric pathology associated with this infection. Further studies are needed for a better understanding of the epidemiological aspects in order to guarantee an improvement in the treatment of patients infected with *H. pylori*.

CONFLICTS OF INTEREST

The authors declare they have no conflicts of interest.

AUTHORS' CONTRIBUTION

KH and DK were responsible for study conceptualization and writing (review and editing) the manuscript. KH and BH wrote (original draft) the manuscript. AA analyzed the data. TS contributed in results interpretation. All authors revised it critically for important intellectual content and approved the final version to be published.

FUNDING

The authors received no financial support for this research.

ACKNOWLEDGMENTS

The authors would like to sincerely thank the personnel of the laboratory of pathological anatomy at Hassani Abdelkader University Hospital Center of Sidi Bel Abbes, Algeria for their contribution in this study especially Dr. Bakhta Bouzouira. We also would like to express our thanks to Dr. Maad Ravan for his statistical analysis and Dr. Md Sahab Uddin for his continuous help.

REFERENCES

- [1] Sweeney EG, Henderson JN, Goers J, Wreden C, Hicks KG, Foster JK, et al. Structure and proposed mechanism for the pH-sensing *Helicobacter pylori* chemoreceptor TlpB. Structure 2012;20;1177–88.
- [2] International Agency for Research on Cancer. IARC monographs on the evaluation of carcinogenic risks to humans, vol. 61: schistosomes, liver flukes and *Helicobacter pylori*. Lyon, France: International Agency for Research on Cancer; 1994, pp. 177–240.
- [3] Peleteiro B, Bastos A, Ferro A, Lunet N. Prevalence of *Helicobacter pylori* infection worldwide: a systematic review of studies with national coverage. Dig Dis Sci 2014;59;1698–709.
- [4] Miendje Deyi VY, Vanderpas J, Bontems P, Van Den Borre C, De Koster E, Cadranel S, et al. Marching cohort of *Helicobacter pylori* infection over two decades (1988-2007): combined effects of secular trend and population migration. Epidemiol Infect 2011;139;572–80.
- [5] Jamkhande PG, Gattani SG, Farhat SA. *Helicobacter pylori* and cardiovascular complications: a mechanism based review on role of *Helicobacter pylori* in cardiovascular diseases. Integr Med Res 2016;5;244–9.
- [6] Allaker RP, Young KA, Hardie JM, Domizio P, Meadows NJ. Prevalence of *Helicobacter pylori* at oral and gastrointestinal sites in children: evidence for possible oral-to-oral transmission. J Med Microbiol 2002;51;312–7.
- [7] Kabir S. Detection of *Helicobacter pylori* DNA in feces and saliva by polymerase chain reaction: a review. Helicobacter 2004;9;115–23.
- [8] Sinha SK, Martin B, Gold BD, Song Q, Sargent M, Bernstein CN. The incidence of *Helicobacter pylori* acquisition in children of a Canadian First Nations community and the potential for parent-to-child transmission. Helicobacter 2004;9;59–68.
- [9] Everhart JE. Recent developments in the epidemiology of *Helicobacter pylori*. Gastroenterol Clin North Am 2000;29;559–78.

- [10] Oona M, Utt M, Nilsson I, Uibo O, Vorobjova T, Maaroos HI. Helicobacter pylori infection in children in Estonia: decreasing seroprevalence during the 11-year period of profound socioeconomic changes. Helicobacter 2004;9;233–41.
- [11] Malfertheiner P, Megraud F, O'Morain CA, Atherton J, Axon AT, Bazzoli F, et al. Management of *Helicobacter pylori* infection—the Maastricht IV/Florence Consensus Report. Gut 2012;61;646–64.
- [12] Amir Tidadini ZC. Pathologies gastriques et infections à *Helicobacter pylori*: thése pour l'obtention du diplôme de doctorat d'état en sciences médicales à la faculté de Médecine d'Alger. 2003, pp. 13–34 [Article in French].
- [13] Delchier JC. [Gastric MALT lymphoma, a malignancy potentially curable by eradication of *Helicobacter pylori*]. Gastroenterol Clin Biol 2003;27;453–8 [Article in French].
- [14] Ayala G, Galván-Portillo M, Chihu L, Fierros G, Sánchez A, Carrillo B, et al. Resistance to antibiotics and characterization of *Helicobacter pylori* strains isolated from antrum and body from adults in Mexico. Microb Drug Resist 2011;17;149–55.
- [15] Khoder G, Muhammad JS, Mahmoud I, Solmian SSM, Burucoa C. Prevalence of *Helicobacter pylori* and its associated factors among healthy asymptomatic residents in the United Arab Emirates. Pathogens 2019;8;44.
- [16] Essadik A, Benomar H, Rafik I, Hamza M, Guemouri L, Kettani A, et al. Aspects épidémiologiques et cliniques de l'infection à *Helicobacter pylori* à travers une étude marocaine. Hegel 2013;3;163–9 [Article in French].
- [17] Raaf N, Amhis W, Saoula H, Abid A, Nakmouche M, Balamane A, et al. Prevalence, antibiotic resistance, and MLST typing of *Helicobacter pylori* in Algiers, Algeria. Helicobacter 2017;22;e12446.
- [18] Faik M. Mise au point sur l'infestation gastrique par l'*Helicobacter pylori*. Med Maghreb 2000;79;17–19 [Article in French].
- [19] Hooi JKY, Lai WY, Ng WK, Suen MMY, Underwood FE, Tanyingoh D, et al. Global prevalence of *Helicobacter pylori* infection: systematic review and meta-analysis. Gastroenterol 2017;153;420–9.
- [20] Niv Y. H pylori recurrence after successful eradication. World J Gastroenterol 2008;14;1477–8.
- [21] Bures J, Kopácová M, Skodová Fendrichová M, Rejchrt S. Epidemiology of *Helicobacter pylori* infection. Vnitr Lek 2011;57;993–9 [Article in Czech].
- [22] Suerbaum S, Michetti P. *Helicobacter pylori* infection. N Engl J Med 2002;347;1175–86.
- [23] Bardhan PK. Epidemiological features of *Helicobacter pylori* infection in developing countries. Clin Infec Dis 1997;25;973–8.
- [24] Mbengue M, Diouf ML, Dangou JM, Ka MM, Ba-Seck A, Ndiaye MF, et al. Frequency of *Helicobacter pylori* infection in symptomatic patients in Senegal. Med Trop 1997;57;256–8 [Article in French].
- [25] Bello AK, Umar AB, Borodo MM. Prevalence and risk factors for *Helicobacter pylori* infection in garstoduodenal diseases in Kano, Nigeria. Afr J Med Health Sci 2018;17;41–6.
- [26] Ramanampamonjy RM, Randria MJD, Razafimahefa SH, Ratsimandisa R, Rajaonarivelo P, Rajaona HR. [Seroprevalence of *Helicobacter pylori* infection in Malagasy population]. Bull Soc Pathol Exot 2007;100;57–60 [Article in French].
- [27] Andoulo FA, Noah DN, Tagni-Sartre M, Ndam EC, Blackett KN. [Epidemiology of infection *Helicobacter pylori* in Yaoundé: specificity of the African enigma]. Pan Afr Med J 2013;16;115 [Article in French].
- [28] Smith S, Fowora M, Pellicano R. Infections with *Helicobacter pylori* and challenges encountered in Africa. World J Gastroenterol 2019;25;3183–95.

- [29] Malaty HM. Epidemiology of *Helicobacter pylori* infection. Best Pract Res Clin Gastroenterol 2007;21;205–14.
- [30] Elmanama A, Mokhallalati M, Abu-Mugesieb R. Risk factors associated with *Helicobacter pylori* infection in Gaza, Palestine. Islam Univ J 2008;16;97–110.
- [31] Khalife H, Khalife H, Khodor HH, Ghssein G, El Rashed Z, Abdel-Sater F. Epidemiology of *Helicobacter pylori* infection among the healthy population in Lebanon. World J Pharm Pharm Sci 2017;6;363–72.
- [32] Joutei HAH, Hilali A, Fechtali T, Rhallabi N, Benomar H. [*Helicobacter pylori* infection in 755 patients with digestive complaints Pasteur Institute, Morocco, 1998-2007]. East Mediterr Health J 2010;16;778–82 [Article in French].
- [33] Vilaichone RK, Mahachai V, Shiota S, Uchida T, Ratanachu-ek T, Tshering L, et al. Extremely high prevalence of *Helicobacter pylori* infection in Bhutan. World J Gastroenterol 2013;19;2806–10.
- [34] lboudo D, Sangare L, Sanou J, Bougouma A, Diomande I. Aspects épidémiologiques et cliniques de l'infection à *Helicobacter pylori*

- en zone tropicale: à propos de 150 patients à l'hôpital national d'Ouagadougou (Burkina Faso). Méd Afr Noire 1997;44;24–8 [Article in French].
- [35] Wang W, Jiang W, Zhu S, Sun X, Li P, Liu K, et al. Assessment of prevalence and risk factors of *Helicobacter pylori* infection in an oilfield Community in Hebei, China. BMC Gastroenterol 2019;19;186.
- [36] Attaf N, Cherkaoui N, Choulli MK, Ghazali L, Mokhtari A, Soulaymani A. Profil épidémiologique de l'infection à *Helicobacter pylori* dans la région du Gharb-Chrarda-Beni Hssen. Biol Santé 2004;4;25–34 [Article in French].
- [37] Binan Y, Adom H, Tanon A, Yao H, Toutou T. Cancer gastrique et Helicobacter pylori: résultats d'un centre d'endoscopie à Abidjan. Rev Int Sci Méd 2006;8;23–7 [Article in French].
- [38] Bravo D, Hoare A, Soto C, Valenzuela MA, Quest AF. Helicobacter pylori in human health and disease: mechanisms for local gastric and systemic effects. World J Gastroenterol 2018;24;3071–89.
- [39] Brown LM. *Helicobacter pylori*: epidemiology and routes of transmission. Epidemiol Rev 2000;22;283–97.