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

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**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.

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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 socio-economic 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. pylori*-related 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
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 hematoxylin–eosin 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 \( (p \leq 0.01) \) 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 H. pylori | 486    | 66.12          |
| Population not infected with H. pylori | 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          |

χ² = 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          |

χ² = 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          |
| Intestinal gastritis              | 43     | 63.24          |

χ² = 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 as 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 intestinal 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 intestinal 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.

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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] Kahir 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, Maaros H. 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 Tidazini ZC. Pathologies gastriques et infections à Helicobacter pylori: thèse 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, Soliman SSM, Burucoca 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, Kopáčová 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] Mbengué M, Diouf ML, Dangou JM, Ka MM, Ba-Seck A, Ndiaye MF, et al. Frequency of Helicobacter pylori infection in asymptomatic 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, Razafimahafea SH, Ratsimandisa R, Rajaonarivelo P, Rajaona HR. [Seroprevalence of Helicobacter pylori infection in Y aoundé: specificity 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, Ghsein 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] Iboudo 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 oil-field Community in Hebei, China. BMC Gastroenterol 2019;19;186.

[36] Attaf N, Chekaoui 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.