Seroprevalence of *Helicobacter pylori* among Tunisian blood donors (outpatients), symptomatic patients and control subjects

Séroprévalence de *Helicobacter pylori* chez l’adulte donneur de sang, l’adulte symptomatique et une population témoin

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Available online 29 October 2009

Summary

Objectives. — *Helicobacter pylori* is a worldwide infection, although little data are available in the Tunisian population. The aims of our study were to detect the prevalence of *H. pylori* in a blood-donor population (*n* = 250) and in another population of hospital-consulting patients comprising 87 symptomatic patients and 59 controls, and to determine the factors that influence the prevalence.

Materials and methods. — Study subjects answered a standardized questionnaire, and IgG anti-*H. pylori* and anti-*cag* were detected by ELISA. In the second population, culture and *cag*A polymerase chain reaction were performed.

Results. — The seroprevalence of *H. pylori* in blood donors was 64%, and 11% had anti-*cag*. All patients positive for anti-*cag* were also positive for anti-*H. pylori* antibodies. The seroprevalence of *H. pylori* was 99.3% in the hospital-consulting patients, of whom 55.5% were positive for anti-*cag*. The difference between the anti-*cag* and symptomatic patients (66.7%) and controls (39%) was significant. Symptomatic patients had a higher rate of anti-*cag* (66.7%) compared with the controls (39%) and blood donors (11%).

This multicentre study was performed in Microbiology laboratory—Rabta University Hospital, Tunis, Tunisia in research unit UR04SP08.

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doi:10.1016/j.gcb.2009.06.015
Conclusion. — H. pylori seroprevalence in blood donors is low (64%) compared with symptomatic patients (99.3%), and anti-cag was statistically associated with symptomatic patients and pathology. Also, some environmental factors were correlated with H. pylori seroprevalence.

Materials and methods

Study subjects

We evaluated the prevalence of H. pylori infection in a total of 396 Tunisian subjects, of whom 284 (71.7%) were male and 112 (28.3%) were female, from November 2006 to April 2007. Three groups of subjects, who consented to provide venous blood, were recruited. One group comprised 250 blood donors (212 male/38 female; gender ratio 0.18; mean age 33.5 years, range 25–55) seen as outpatients, who had no history of gastroduodenal disease and had come to the National Blood Transfusion Center in Tunis to make a blood donation. All had tested negative for HBsAg, HCV Ab and HIV Ab by ELISA testing. The other group included 87 patients (54 male/33 female; gender ratio 1.63; mean age 48.6 years, range 16–81), who were being seen for gastroduodenal disorders and clinically diagnosed with gastritis (n = 35; 40.2%), gastric ulcer (n = 8, 9.2%), duodenal ulcer (n = 35, 40.2%), duodenal ulcer and gastritis (n = 5, 5.7%), smoking, alcohol consumption, plate-sharing and rural or urban origin;

• to evaluate the prevalence of H. pylori infection in asymptomatic Tunisian subjects (n = 59) consulting for other pathologies (such as hernia or hepatitis);

• to determine the rate of infection in symptomatic Tunisian patients (n = 87) with gastroduodenal disorders who were being seen by the gastroenterology unit in the Rabta Hospital and Charles Nicolle Hospital (both in Tunis) and the Menzel Bourguiba Hospital; and

• to determine the cagA seroprevalence and cagA status among asymptomatic and symptomatic subjects.

Introduction

Since Marshall and Warren first isolated and described their novel gastric bacterium in a pure culture in 1982 [1], Helicobacter pylori has been an intensively studied subject worldwide, as it inhabits the hostile environment of the human stomach [2] and induces chronic gastritis [3]. Several diagnostic methods for detecting H. pylori are available, and can be divided into invasive [rapid urease test, histology, culture and polymerase chain reaction (PCR), all of which require endoscopy] and non-invasive (mainly, the urea breath test, antigen detection in stools and serological detection of antibodies) tests [4,5]. Serological tests are recommended for epidemiological surveys and for antimicrobial therapy [6,7], and represent the most rapid and convenient means of obtaining a snapshot of the prevalence of H. pylori in a given population. The seroprevalence of H. pylori has been studied in symptomatic and asymptomatic populations and healthy volunteers in both the developed and developing countries [6,8–10]. In European epidemiological studies, blood donors are often used to represent the general population, as all European countries have unpaid donors [11,12]. The majority of serological studies are now conducted using commercial kits and enzyme-linked immunosorbent assay (ELISA) methods.

In Tunisia, because of the limited data on the epidemiology of H. pylori and its associated risk factors, the objectives of the present prospective study were:

• to detect the seroprevalence of H. pylori in blood donors (outpatients) who had no history of gastric pathology (such as duodenal or gastric ulcer, or gastritis) and to correlate the prevalence of H. pylori infection with height,
Seroprevalence of *Helicobacter pylori*

Serum samples

Venous blood (5–10 mL) was collected from all of the enrolled subjects. Serum samples were obtained from the blood by centrifugation (3000 rpm for 10 min), then aliquoted and immediately stored at −20 °C until serology testing. Lipemic and icteric samples were eliminated.

Diagnosis of *H. pylori* infection in symptomatic patients and controls

Two antrum and two corpus biopsy specimens were collected for urease testing, Gram-staining and culture, using the standard protocols. Genomic DNA was extracted, using the QIAamp DNA Mini Kit (QIAGEN, Hilden, Germany) according to the manufacturer’s instructions, to determine the presence of the *cagA* gene by PCR reaction (primers: F1/5′GATAACAGCCGCTTTTGAGG3′ and B1/5′CTGCAAAGATGTTTTGGCAAG3′). The objective of this investigation was to affirm the symptomatic or asymptomatic presence of *H. pylori* in the two groups and to compare their *cag* antigen prevalences.

Enzyme immunoassay

To compare the three groups according to their serological data, all of the enlisted subjects underwent a serological test for the presence of IgG antibodies against *H. pylori* (ELISA, PLATELIA, Bio-Rad) and an anti-*cag* IgG (ELISA, EUROIMMUN, Germany) in accordance with the manufacturers’ guidelines.

For the detection of IgG antibodies against *H. pylori cagA*, diluted patient samples were incubated in wells coated with recombinant *H. pylori cagA* antigen. In positive samples, specific IgG antibodies bind to the antigens. Negative and positive controls and three calibrators were used for this assay, and the results can be evaluated as: ratio = extinction of the control or patient sample/extinction of calibrator 2. The recommendations for interpreting the results were: ratio < 0.8 = negative; ratio ≥ 1.1 = positive; ratio ≤ 0.8 and < 1.1 = borderline.

Statistical analysis

Data were analyzed using the $X^2$ test, and a $P$ value of less than 0.05 was considered to be statistically significant.
Table 1  Distribution of anti-\textit{H. pylori} antibodies in blood donors and symptomatic patients.

<table>
<thead>
<tr>
<th>Studied groups</th>
<th>Anti-\textit{H. pylori}-positive</th>
<th>Anti-\textit{H. pylori}-negative</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood donors ((n))</td>
<td>158</td>
<td>92</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Symptomatic patients ((n))</td>
<td>87</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Total ((n))</td>
<td>245</td>
<td>92</td>
<td></td>
</tr>
</tbody>
</table>

Table 2  Distribution of anti-\textit{H. pylori} antibodies in the symptomatic and control groups.

<table>
<thead>
<tr>
<th>Studied groups</th>
<th>Anti-\textit{H. pylori}-positive</th>
<th>Anti-\textit{H. pylori}-negative</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic patients ((n))</td>
<td>87</td>
<td>0</td>
<td>0.3</td>
</tr>
<tr>
<td>Controls ((n))</td>
<td>58</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Total ((n))</td>
<td>145</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Table 3  Distribution of anti-\textit{cag} antibodies in blood donors and symptomatic patients.

<table>
<thead>
<tr>
<th>Studied groups</th>
<th>Anti-\textit{cag}-positive</th>
<th>Anti-\textit{cag}-negative</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood donors ((n))</td>
<td>27</td>
<td>223</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Symptomatic patients ((n))</td>
<td>53</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>Total ((n))</td>
<td>80</td>
<td>257</td>
<td></td>
</tr>
</tbody>
</table>

Results

\textit{H. pylori} culture, Gram-staining and urease testing were positive in all biopsies taken from both the symptomatic patients and control subjects. The 349-pb PCR product (Fig. 1), indicating the presence of the \textit{cagA} gene, was obtained in 57 symptomatic isolates (65.5\%) and in 35 isolates (59.3\%) from the controls. There was no statistically significant difference in the distribution of the \textit{cagA} gene between the two groups (\(P = 0.6\)).

As for the serological tests, the IgG anti-\textit{H. pylori} antibodies were detected by ELISA in 158 (64\%) of the 250 blood donors, and in 100 and 98.3\% of the symptomatic patients and control subjects, respectively. There was a statistically significant difference in the distribution of IgG anti-\textit{H. pylori} antibodies detected between blood donors and symptomatic patients (\(P < 0.001\); Table 1), but not between the symptomatic patients and controls (\(P = 0.3\); Table 2). Anti-\textit{cagA} antibodies were present in 27 (11\%) of the blood donors, and in 53 (66.7\%) and 23 (39\%) of the symptomatic patients and controls, respectively. The differences in distribution of anti-\textit{cagA} antibodies were statistically significant between blood donors and symptomatic patients (\(P < 0.001\)) and controls (\(P = 0.01\); Tables 3 and 4).

Table 4  Distribution of anti-\textit{cag} antibodies in the controls and symptomatic patients.

<table>
<thead>
<tr>
<th>Studied groups</th>
<th>Anti-\textit{cag}-positive</th>
<th>Anti-\textit{cag}-negative</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group ((n))</td>
<td>23</td>
<td>36</td>
<td></td>
</tr>
<tr>
<td>Symptomatic patients ((n))</td>
<td>53</td>
<td>34</td>
<td>0.01</td>
</tr>
<tr>
<td>Total ((n))</td>
<td>76</td>
<td>70</td>
<td></td>
</tr>
</tbody>
</table>

The distribution of cases and controls according to age and other characteristics is shown in Tables 5 and 6. The overall rate of infection was found to increase with age. In the first age-range group (< 30 years), positive anti-\textit{H. pylori} antibody results were observed in 8.2\% of the blood donors, 17.2\% of the symptomatic patients and 20.6\% of the controls. The highest prevalence of anti-\textit{H. pylori} was found in the 30—49 age range. Differences in the distribution of anti-\textit{H. pylori} according to age were statistically significant between blood donors and symptomatic patients (\(P < 0.001\)) and controls (\(P = 0.005\)). However, the distribution of anti-\textit{cag} according to age was not significant (Tables 7 and 8).

No differences in alcohol intake and height were seen in terms of having the \textit{H. pylori} infection. However, a significant association was noted between \textit{H. pylori} infection and smoking (Tables 5 and 6). As for the eating habits investigated, there was a significant difference between the blood donors and symptomatic patients (\(P < 0.001\)), but not between symptomatic patients and controls (\(P = 0.9\)). In terms of rural or urban origin, the difference between blood donors and symptomatic patients was statistically significant (\(P < 0.001\)), but not between the symptomatic and control groups (\(P = 0.9\)).

Among the blood donors who had anti-\textit{H. pylori} antibodies, it did not appear to be obligatory to also have anti-\textit{cag
Table 5  Prevalence of anti-\textit{H. pylori} antibodies, and characteristics of the blood donors and symptomatic patients.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Blood donors ((n = 250))</th>
<th>Symptomatic patients ((n = 87))</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HP-positive ((n = 158))</td>
<td>HP-negative ((n = 92))</td>
<td>HP-positive ((n = 87))</td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 30</td>
<td>13 (8.2%)</td>
<td>23</td>
<td>15 (17.2%)</td>
</tr>
<tr>
<td>30–39</td>
<td>15 (9.4%)</td>
<td>20</td>
<td>11 (12.6%)</td>
</tr>
<tr>
<td>40–49</td>
<td>65 (41.1%)</td>
<td>17</td>
<td>18 (20.6%)</td>
</tr>
<tr>
<td>50–59</td>
<td>55 (34.8%)</td>
<td>18</td>
<td>17 (19.5%)</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>10 (6.3%)</td>
<td>14</td>
<td>26 (29.8%)</td>
</tr>
<tr>
<td>Smoker</td>
<td>88 (55.6%)</td>
<td>45</td>
<td>28 (32.2%)</td>
</tr>
<tr>
<td>Non-smoker</td>
<td>70 (44.4%)</td>
<td>47</td>
<td>59 (67.8%)</td>
</tr>
<tr>
<td><strong>Alcohol drinker</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>29 (18.3%)</td>
<td>26</td>
<td>11 (12.6%)</td>
</tr>
<tr>
<td>No</td>
<td>129 (81.7%)</td>
<td>66</td>
<td>76 (83.4%)</td>
</tr>
<tr>
<td><strong>Plate-sharing</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>67 (42.4%)</td>
<td>43</td>
<td>83 (95.4%)</td>
</tr>
<tr>
<td>No</td>
<td>91 (57.6%)</td>
<td>49</td>
<td>5 (4.6%)</td>
</tr>
<tr>
<td><strong>Height</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1.71 m</td>
<td>95 (62.5%)</td>
<td>57</td>
<td>56 (64.3%)</td>
</tr>
<tr>
<td>&gt; 1.71 m</td>
<td>63 (43.4%)</td>
<td>35</td>
<td>31 (35.7%)</td>
</tr>
<tr>
<td><strong>Origin</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>47 (29.7%)</td>
<td>26</td>
<td>44 (50.5%)</td>
</tr>
<tr>
<td>Urban</td>
<td>111 (70.3%)</td>
<td>66</td>
<td>43 (49.5%)</td>
</tr>
</tbody>
</table>

HP: \textit{Helicobacter pylori} (\textit{H. pylori}).

Table 6  Prevalence of anti-\textit{H. pylori} antibodies, and characteristics of the symptomatic and control groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Symptomatic patients ((n = 87))</th>
<th>Control subjects ((n = 59))</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HP-positive ((n = 87))</td>
<td>HP-negative ((n = 58))</td>
<td>HP-negative ((n = 1))</td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 30</td>
<td>15 (17.2%)</td>
<td>12 (20.6%)</td>
<td></td>
</tr>
<tr>
<td>30–39</td>
<td>11 (12.6%)</td>
<td>17 (29.3%)</td>
<td></td>
</tr>
<tr>
<td>40–49</td>
<td>18 (20.6%)</td>
<td>8 (13.7%)</td>
<td>0.005</td>
</tr>
<tr>
<td>50–59</td>
<td>17 (19.5%)</td>
<td>11 (18.9%)</td>
<td>1</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>26 (29.8%)</td>
<td>10 (17.2%)</td>
<td></td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>53 (60.9%)</td>
<td>18 (31%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Female</td>
<td>34 (39.1%)</td>
<td>40 (69%)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Smoker</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoker</td>
<td>28 (32.2%)</td>
<td>2 (3.4%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Non-smoker</td>
<td>59 (67.8%)</td>
<td>56 (96.6%)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Alcohol drinker</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>11 (12.6%)</td>
<td>2 (3.4%)</td>
<td>0.1</td>
</tr>
<tr>
<td>No</td>
<td>76 (83.4%)</td>
<td>56 (96.6%)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Plate-sharing</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>83 (95.4%)</td>
<td>57 (98.2%)</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>5 (4.6%)</td>
<td>1 (1.8%)</td>
<td></td>
</tr>
<tr>
<td><strong>Height</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1.71 m</td>
<td>56 (64.3%)</td>
<td>42 (72.4%)</td>
<td>0.9</td>
</tr>
<tr>
<td>&gt; 1.71 m</td>
<td>31 (35.7%)</td>
<td>16 (27.6%)</td>
<td></td>
</tr>
<tr>
<td><strong>Origin</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>44 (50.5%)</td>
<td>37 (63.8%)</td>
<td>0.9</td>
</tr>
<tr>
<td>Urban</td>
<td>43 (49.5%)</td>
<td>21 (36.2%)</td>
<td>1</td>
</tr>
</tbody>
</table>

HP: \textit{Helicobacter pylori} (\textit{H. pylori}).
antibodies, and we found that subjects who were anti-cag antibody-positive were also *H. pylori*-positive. The difference was statistically significant (*P* = 0.001).

As for the association of *H. pylori* infection with a clinical diagnosis in symptomatic patients, the prevalence of anti-cag antibodies is shown in Table 9. Also, the prevalence of anti-cag antibodies in patients with duodenal ulcer (71.4%) was higher than seen in the other patients, although the distribution was not statistically significant (*P* = 0.5).

### Discussion

The present study was the first to focus on the seroprevalence of *H. pylori* infection in blood donors, and in symptomatic and control populations. We were interested in blood donors (outpatients) because, first, they represent the general population and, second, they allow comparisons with the symptomatic population. In our study, the serological prevalence of *H. pylori* infection in blood donors was 64%, a result that is similar to the rate reported by Zaterka et al. [13] in Brazilian blood donors (65.7%), but lower than that in developing countries, reported to be 84.2% in South Africa, 75% in Peru and 80—90% in the Ivory Coast. However, it is higher than that of many developed countries, reported to be 37% in the USA, 38% in Canada, 35% in France and 20% in Belgium [14—16]. On comparing the three study groups (blood donors with symptomatic patients, and symptomatic patients with control subjects), we observed an increased prevalence of *H. pylori* according to age. This result is similar to those of previous published studies from both developed and developing countries [17—19]. In Tunisia, the data indicate that *H. pylori* is common in childhood and peaks at ages 30—49 years, suggesting exposure of the population to the organism and the cumulative risk of infection. Mahrez et al. and Fendri et al. [20,21] detected *H. pylori*-specific antibodies in 30 and 32%, respectively, in asymptomatic Tunisian children. In addition, Mégraud et al. [9] suggested that there is a higher risk of infection with *H. pylori* at any age in the developing versus developed countries, but it is inconceivable that all these individuals had or will develop a duodenal ulcer.

As for smoking, the differences between our three studied groups were statistically significant (*P* < 0.001). Russo et al. [22] found that current smokers of more than 20 cigarettes per day also had a greater risk of developing complete metaplasia in *H. pylori*-positive subjects. However, no relationship between alcohol intake and *H. pylori* infection was found in our three study populations. Several studies have suggested that alcohol consumption might facilitate elimination of *H. pylori* infection, increase acid secretion and protect against active *H. pylori* infection [23—25]. Regarding eating habits, the seroprevalence was more frequent in subjects who shared their plate (98.2%), and the difference was significant (*P* < 0.001) between the blood donors and symptomatic patients, but not between the symptomatic and control populations. Indeed, sharing plates is a risk factor, as it favors oral transmission. Our results are in accord with other Tunisian studies aiming to identify the risk factors that predispose children in Cap Bon (northeastern Tunisia) [26]. In terms of height across our three groups, the results showed that the difference between subjects of short stature (< 1.71 m) who were positive for *H. pylori* and those over 1.71 m was not significant. These results are in agreement with other reports that show that the relationship between short stature and

### Table 7 Distribution of anti-cag antibodies by age in the symptomatic and control groups.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Symptomatic patients (n)</th>
<th>Controls (n)</th>
<th><em>P</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 30</td>
<td>10</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>30—39</td>
<td>6</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>40—49</td>
<td>13</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>50—59</td>
<td>10</td>
<td>5</td>
<td>0.05</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>14</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Total (n)</td>
<td>53</td>
<td>23</td>
<td></td>
</tr>
</tbody>
</table>

### Table 8 Distribution of anti-cag antibodies by age in blood donors and symptomatic patients.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Blood donors (n)</th>
<th>Symptomatic patients (n)</th>
<th><em>P</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 30</td>
<td>2</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>30—39</td>
<td>3</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>40—49</td>
<td>9</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>50—59</td>
<td>8</td>
<td>10</td>
<td>0.1</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>5</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Total (n)</td>
<td>27</td>
<td>53</td>
<td></td>
</tr>
</tbody>
</table>

### Table 9 Association between *Helicobacter pylori* infection and clinical diagnosis of symptomatic patients.

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Anti-cag antibody-positive [n (%)]</th>
<th>Anti-cag antibody-negative (n)</th>
<th><em>P</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Duodenal ulcer</td>
<td>25 (71.4)</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Gastric ulcer</td>
<td>4 (50)</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Gastritis</td>
<td>20 (57.14)</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Duodenal ulcer &amp; gastritis</td>
<td>3 (60)</td>
<td>2</td>
<td>0.5</td>
</tr>
<tr>
<td>Adenocarcinoma</td>
<td>1 (33.33)</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>MALT lymphoma</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Total (n)</td>
<td>53</td>
<td>34</td>
<td></td>
</tr>
</tbody>
</table>

MALT: mucosa-associated lymphatic tissue.
Seroprevalence of Helicobacter pylori

H. pylori infection requires further clarification, and that H. pylori is not a risk factor for short stature [27–29]. The seroprevalence of H. pylori infection was not similar for the studied urban and rural populations, and the difference was statistically significant between blood donors and symptomatic patients, but not between the controls and symptomatic populations. These results are in accordance with those of Nascimento et al. [30], but not so for the blood donors (which had a predominance of women due to the smaller number of women who give blood in Tunisia). Our finding does not accord with that of Ching et al. [33], who found no significant difference in H. pylori seroprevalence between men and women. However, several other studies have found a higher prevalence of H. pylori infection in men, which may be related to their greater exposure to potential environmental sources of infection [34,35]. Comparison across the three groups showed that the risk factors were more strongly associated with the symptomatic patients than with blood donors or the control population, thereby suggesting that the symptoms are influenced by the presence of H. pylori infection.

Among the symptomatic and control groups, the percentages who were positive for anti-H. pylori IgG reached 99.3% (145/146 subjects), among whom anti-cagA antibodies were seen in 55.5% (81 subjects), a rate that was clearly higher than in blood donors (11%). However, the presence of anti-H. pylori antibodies does not appear to be an obligatory condition for having anti-cag antibodies. Indeed, gene carriage rather than inflammatory status accompanies the cagA island of pathogenicity, although we have found that those who were anti-cag antibody-positive were also H. pylori-positive, and the relationship was statistically significant (P = 0.001).

In the present study, the presence of the cagA gene was strongly correlated with the presence of anti-cag antibodies: the prevalence of the cagA gene was 65.5% in symptomatic patients and 59.3% in the control group. Jenks et al. [36] found that 87% of symptomatic patients were cagA-positive compared with 47% of asymptomatic patients.

In the present study, the prevalence of anti-cag antibodies in patients with duodenal ulcer was higher (71.4%) than in other patients, but the difference was not significant. In other reports, anti-cag antibodies were prevalent regardless of the presence of gastroduodenal disease [37]. However, in Mexico and Thailand, there is an association of anti-cag antibodies with peptic ulcer disease and duodenal ulcer, respectively [38,39].

Our study provides the first data in Tunisia on the seroprevalence of H. pylori infection in outpatients, and in symptomatic and control populations. H. pylori infection was prevalent in the studied subjects, and the infection starts early in childhood and increases with age. The present study also showed a strong link between a high prevalence of H. pylori infection and lifestyle habits. For this reason, preventative measures should be undertaken to prevent the risk factors for infection by this microorganism. Another problem that needs to be resolved in the Tunisian population is the eradication of asymptomatic infection at an early age to avoid the later development of gastric disorders in adulthood such as stomach cancer.

References


