Helicobacter pylori Infection in Asymptomatic Elderly Subjects living at Home or in a Nursing Home: Effects on Gastric Function and Nutritional Status

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Summary
Age and close living conditions are known to be risk factors for the acquisition of Helicobacter pylori (HP) infection. It is unknown whether institutionalization of asymptomatic, elderly subjects is an additional risk factor and whether gastric function and nutritional status are affected by the HP infection. The study sample comprised 102 subjects over 65 years of age: 52 living in a nursing home and 50 at home. No subject had symptoms or previous pathology related to the upper digestive tract. In all subjects, serum levels of specific anti-HP antibodies were determined. Gastric function was evaluated by levels of pepsinogen A (PGA), pepsinogen C (PGC) and gastrin. The nutritional status of the subject was evaluated by measuring: albumin, haemoglobin, iron, ferritin, transferrin, vitamin B₁₂, and folic acid in blood, and body mass index and mid-arm muscle area. The prevalence of anti-HP antibodies was 86.5% in institutionalized subjects (men: 100%; women: 76.6%, p < 0.05) and 82.0% in subjects living at home (men: 86.3%; women: 76.3%). No differences between the two groups were observed in levels of serum anti-HP antibodies, PGA, PGC or gastrin. In institutionalized subjects, a significant correlation between anti-HP antibodies and PGC was identified. In neither group were differences observed between serum positive (HP + ve) and negative (HP − ve) subjects with respect to the biohumoral and anthropometric indices of nutritional status. We conclude: (1) the seroprevalence of the HP infection was high (82—86%) in asymptomatic elderly patients living either at home or in an institution; (2) the presence of specific IgG anti-HP antibodies in asymptomatic elderly individuals, at home or in a nursing home, was not associated with changes in PGA, PGC or gastrin; however, there was a correlation between anti-HP antibody titre and PGC levels in institutionalized subjects; (3) nutritional indices were not influenced by the presence of anti-HP antibodies.

Introduction
Helicobacter pylori (HP), a Gram-negative curved bacteria, is currently known to be the aetiological agent of type B antral gastritis [1, 2] and is associated with gastric and duodenal ulcer disease [3], as well as being a risk factor for gastric neoplasia [4, 5].

HP infection, diffuse throughout the world, has been shown in the most recent epidemiological studies screening for the serum anti-HP antibody to have a greater prevalence in underdeveloped countries [6], with notable associations with race [7] and socioeconomic conditions, particularly when contracted in childhood [8].

The seroprevalence of the infection increases with age [9, 10], but it is not known whether this depends on a gradual acquisition of the infection by the adult population, or on a cohort effect, since the HP infection is, in the majority of cases, contracted in childhood [11].

High percentages of HP infection have been reported in subjects in institutions [12, 13] and in close-contact living conditions [14]. Identical bacterial strains have been isolated from infected persons living together [15], indicating that, although the mechanism of transmission has not yet been defined as faecal–oral, oral–oral, or both, high density living conditions are a potential risk factor for person-to-person transmission of the infection [16].

It was demonstrated recently that asymptomatic carriers of anti-HP antibodies have levels of pepsinogen A (PGA) and pepsinogen C (PGC) higher than those of serum-negative subjects, suggesting higher prevalence of chronic gastritis [17].
The objectives of the present work were (1) to study the prevalence of HP infection in asymptomatic elderly subjects living at home or in a nursing home; (2) to evaluate if HP infection induced changes in indices of gastric function (gastrin and pepsinogen A and C) and nutritional status.

Methods

The study was conducted in 102 subjects over 65 years of age, of whom 52 (men 22; women 30; mean age 82.9 years; range 66–101) lived in a nursing home for at least 5 years (mean period of institutionalization 10.9 years; range 5–61) and 50 (men 22; women 28; mean age 78.7 years; range 65–103) lived at home. No subject at the time of testing had symptomatology suggestive of an upper digestive tract disease or a history of pathology of the digestive system.

A blood sample was collected from all subjects for the determination of serum anti-HP antibodies using an established enzyme-linked immunosorbent assay (ELISA, Biolife, Milan, Italy) [18]. The levels of specific anti-HP antibodies were derived from a standard curve of IgG mass against optical density at 450 nm and the results were expressed in monoclonal units/ml (MU/ml): a cut-off point of greater than 20 MU/ml indicated positivity. Serum pepsinogens A and C (Sorin Biomedica, Saluggia, Italy; μg/ml) and gastrin (pg/ml) were measured by radioimmunoassay. In all subjects the following nutritional indices were measured in blood or serum: albumin (g/dl), haemoglobin (g/dl), iron (μg/ml), transferrin (mg/dl), ferritin (ng/ml), vitamin B12 (pg/ml) and folic acid (ng/ml). Anthropometric nutritional indices measured were: Body Mass Index [BMI = weight (kg)/height (m)] and the mid-arm muscle area (MAMA), the latter derived from arm circumference and triceps skin-fold [19].

Statistical analyses were conducted using the Student's t test for non-paired parametric data, the Mann–Whitney test for non-parametric data, and linear regression for the evaluation of correlation between variables.

Results

In the institutionalized group, the prevalence of specific, anti-HP antibodies in serum was 86.5% (7/52 subjects were HP negative), with a significantly greater number of infected men than women (100% versus 76.6%, p < 0.05). In subjects living at home, the prevalence was 82.0%: again, with a greater number of infected men than women (86.3% versus 78.5%, p NS) (Figure 1). No statistically significant differences were obtained between the two groups studied with respect to anti-HP antibody titre, levels of PGA, PGC or gastrin (Table I). When the subjects were grouped by positivity to HP, again, no statistically significant differences in the gastric function indices were found (Table II).

A highly significant correlation (r = 0.455; p < 0.0001) was found in the institutionalized group between titre of anti-HP antibodies and levels of PGC (Figure 2).

In Tables III and IV, results are reported on the

Table I. Levels of IgG anti-HP antibodies, pepsinogen A, pepsinogen C and gastrin in asymptomatic elderly subjects living at home or in a nursing home

<table>
<thead>
<tr>
<th>Variable</th>
<th>Institutionalized subjects [Mean (SEM)]</th>
<th>Subjects living at home [Mean (SEM)]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-HP antibodies (MU/ml)</td>
<td>77.07 (6.93)</td>
<td>78.48 (13.23)</td>
</tr>
<tr>
<td>Pepsinogen A (μg/ml)</td>
<td>98.02 (7.7)</td>
<td>96.5 (2.18)</td>
</tr>
<tr>
<td>Pepsinogen C (μg/ml)</td>
<td>12 (0.70)</td>
<td>15.4 (1.84)</td>
</tr>
<tr>
<td>Gastrin (pg/ml)</td>
<td>89.9 (13.52)</td>
<td>113.2 (23.59)</td>
</tr>
</tbody>
</table>

p = NS.
**Table II.** Pepsinogen A (PGA), pepsinogen C (PGC), PGA/PGC ratio and gastrin levels in asymptomatic, HP positive and negative elderly subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>HP-positive subjects [Mean (SEM)]</th>
<th>HP-negative subjects [Mean (SEM)]</th>
</tr>
</thead>
<tbody>
<tr>
<td>PGA (µg/ml)</td>
<td>97.72 (6.28)</td>
<td>95.37 (17.83)</td>
</tr>
<tr>
<td>PGC (µg/ml)</td>
<td>14.12 (1.09)</td>
<td>11.54 (2.01)</td>
</tr>
<tr>
<td>PGA/PGC ratio</td>
<td>7.91 (0.39)</td>
<td>8.35 (0.83)</td>
</tr>
<tr>
<td>Gastrin (pg/ml)</td>
<td>92.21 (8.98)</td>
<td>79.73 (13.49)</td>
</tr>
</tbody>
</table>

p = NS.

The nutritional status of the two groups studied. No significant effect of HP infection was demonstrable in either the institutionalized or home subjects.

**Discussion**

The prevalence of HP infection is known to increase with age [9—11] and close-living conditions [12—16]. It was unknown, however, if the institutionalization of an elderly subject involved a greater risk of acquiring the HP infection and its eventual pathological sequelae, both gastric and nutritional. The present study, having reported an almost equal prevalence of HP infection in institutionalized and at home asymptomatic elderly subjects, has shown that institutionalization does not seem to be a risk factor for HP infection. The exceptionally high percentage of serum positivity (82–86%) identified in both elderly groups should, however, be noted. These results are similar to those found in asymptomatic elderly subjects in Japan [17] and Switzerland [20], while a lower prevalence (50–60%) was reported in Canada [21] and the United States [22].

It is impossible to identify from this study whether the high prevalence observed in these two elderly groups was due to a continual and progressive acquisition of the HP infection over time [21] or to a cohort effect [23, 24], having isolated a population of subjects who had contracted the infection in childhood, the period of greatest risk of infection by *H. pylori* [8, 13]. The second hypothesis may be more plausible for the groups we studied, since all subjects born between the years 1891 and 1930, grew up during historical periods in which hygienic living conditions were greatly impaired in Italy owing to war (1914–1918 and 1939–1945). The argument for a cohort effect is further strengthened by the finding of a significantly greater prevalence in men than women. Men are more exposed in wartime

**Table III.** Nutrition-related variables in asymptomatic HP positive and negative elderly subjects living at home or in a nursing home

<table>
<thead>
<tr>
<th>Variables</th>
<th>HP-positive subjects [Mean (SEM)]</th>
<th>HP-negative subjects [Mean (SEM)]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Institutionalized</td>
<td>At home</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>3.55 (0.07)</td>
<td>3.9 (0.09)</td>
</tr>
<tr>
<td>Iron (µg/ml)</td>
<td>72.6 (3.62)</td>
<td>67.3 (5.31)</td>
</tr>
<tr>
<td>Ferritin (ng/ml)</td>
<td>160 (20.01)</td>
<td>154 (21.08)</td>
</tr>
<tr>
<td>Transferrin (mg/dl)</td>
<td>230 (5.81)</td>
<td>252 (9.37)</td>
</tr>
<tr>
<td>Vitamin B12 (pg/ml)</td>
<td>492 (60.2)</td>
<td>631 (77.77)</td>
</tr>
<tr>
<td>Folic acid (ng/ml)</td>
<td>4.92 (0.82)</td>
<td>4.2 (0.27)</td>
</tr>
<tr>
<td>Haemoglobin (g/ml)</td>
<td>13.10 (0.24)</td>
<td>12.8 (0.31)</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>24.0 (0.75)</td>
<td>25.0 (0.47)</td>
</tr>
<tr>
<td>Mid-arm muscle area</td>
<td>39.4 (1.91)</td>
<td>40.2 (1.41)</td>
</tr>
</tbody>
</table>

p = NS.
to inadequate hygiene and sanitation, close living conditions, and a promiscuous life-style.

In the present study, PGA, PGC, and the PGA/PGC ratio were used as the indices of gastric function. Since they are produced in different sites in the stomach (body and fundus for PGA and the antral region for PGC), the pepsinogens are indirect indices of gastric mucosal inflammation. Elevated levels of PGA, indicative of gastric mucosal atrophy [26], are commonly found in geographical areas with a high prevalence of HP infection and with a high rate of gastric cancer [27]. Conversely, PGC levels have been shown to increase in subjects with HP-related superficial gastritis and to decrease after eradication therapy of the bacillus from the gastric mucosa of elderly HP-positive subjects [28].

In this study, institutionalization significantly did not affect anti-HP antibody titres and PGA, PGC or gastrin levels, although HP-positive subjects did tend to have higher PGC and gastrin levels, and a lower PGA/PGC ratio than HP-negative subjects. This result may, in part, have been an effect of the non-homogeneity of the population studied (86 HP-positive and 16 HP-negative subjects); however, it is in agreement with a study by Asaka et al. in which the PGC and PGA/PGC ratio differed significantly between HP-positive and HP-negative subjects aged from 10 to 69 years, while no differences were found in subjects over 70 years old [17].

It is difficult to interpret the finding of a highly significant correlation between PGC levels and anti-HP antibody titre only in institutionalized subjects. It is possible that in a closed community such as a nursing home relatively recent HP infections explained the elevated anti-HP titres. A subsequent gastric inflammation limited to the antral region (without the gastric fundus involvement observed in successive periods of time) would have induced a rise in PGC levels only. Conversely, in the group living at home, the lack of correlation between PGC levels and anti-HP titre may reflect old infections. Nevertheless, in neither group did possible gastric involvement lead to a modification in nutritional status, as evaluated by humoral and anthropometric indices.

In conclusion, the results of the present study demonstrated: (1) the seroprevalence of HP infection was high (82–86%) in asymptomatic elderly subjects, living at home or in institutions; (2) the presence of specific IgG anti-HP antibodies in asymptomatic elderly subjects was not associated with alterations in PGA, PGC or gastrin; however, there was a correlation between HP antibody titres and PGC levels in institutionalized subjects; and (3) the nutritional status of asymptomatic elderly subjects living at home or in a nursing home was not influenced by the presence of anti-HP antibodies.

References
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