The prevalence of *Helicobacter pylori* infection in patients with gastric and duodenal ulcers – a 10-year, single-centre experience

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Abstract

Introduction: *Helicobacter pylori* (*H. pylori*) occurs throughout the world and causes gastroduodenal diseases. There is data indicating a change in the prevalence of *H. pylori* infection worldwide. The prevalence of *H. pylori* is 80% in Turkey, while it is higher in many developing countries, and the rate of infection varies throughout the world. In many developing countries, the prevalence of infection exceeds 90% by adulthood.

Aim: To determine the change in the rate of *H. pylori* infection in gastric ulcers and duodenal ulcers for a 10-year period in a single centre.

Material and methods: The study population included 550 patients (342 in 2004, 208 in 1994) with gastric and duodenal ulcers.

Results: In 2004 there were 125 (36.5%) patients with gastric ulcer and 217 patients with duodenal ulcer (64.5%). CLO test positivity was 39.2% in patients with gastric ulcers and 60% in patients with duodenal ulcers. In 1994 there were 208 patients (159 duodenal ulcers, 49 gastric ulcers). Urease test was positive in 74.2% of patients with duodenal ulcer and in 65.2% of patients with gastric ulcer. The decrease in the rate of urease positivity in patients with gastric ulcer was statistically significant (p = 0.01) during this 10-year period.

Conclusions: In the present study we found that the urease positivity decreased significantly in patients with gastric ulcer between 1994 and 2004.

Introduction

Helicobacter pylori (*H. pylori*) infection is a major cause of peptic diseases. Numerous studies have demonstrated that *H. pylori* is ubiquitous – approximately 50% of the world's population is infected with the organism [1, 2]. In many developing countries, the prevalence of infection exceeds 90% by adulthood [3]. While data on the prevalence of *H. pylori* in Turkey in 1994 is varied, it was reported as 71–80%. The most comprehensive study on the prevalence of *H. pylori* in adults in Turkey was the TURHEP study in 2003 [4]. For this study, 2504 households representing the generality of Turkey were chosen as the sample group, and 92% of them were reached. In total, 5555 persons over 18 years of age living in these households were found to be eligible for the study, and 99.9% of them (n = 5549) completed it. In this study, the prevalence of *H. pylori* in adults over 18 years of age found with the C13 urea breath test was 82.5%. The prevalence was determined to be 84% in men and 81% in women. The age group found to have the highest prevalence (86%) was 30–39, and the age group with the lowest prevalence (77%) was 70 years and older. When the findings were reviewed based on the geographical regions of Turkey, it was seen that the prevalence of *H. pylori* was highest (88%) in people living in the Eastern Anatolian Region, and lowest (79%) in people living in the South-

eastern Anatolian Region. In the studies conducted on asymptomatic adults in Turkey, which use the ELISA method to determine serum anti-HpIgG, the prevalence of *H. pylori* is found to be between 53% and 82%. In studies that have investigate the existence of *H. pylori* by invasive methods, the prevalence is reported to be between 41% and 96%. The prevalence of *H. pylori* is 80% in Turkey while it is higher in many developing countries, and the rate of infection varies throughout the world. This rate also increases with age and changes in relation to ethnicity [5]. A seroepidemiological study recruiting subjects while presenting to hospital in Germany found an overall seroprevalence of 13.1% in German citizens in Germany and 30.4% in Turkish-born people living in Germany [6].

Helicobacter pylori occurs throughout the world and causes gastroduodenal diseases. There is an association between the gastric colonization of *H. pylori* and chronic gastritis and peptic ulcer diseases [7, 8]. The rapid urease test, also known as the CLO test (Campylobacter-like organism test), is a rapid test for the diagnosis of *H. pylori* [9]. The basis of the test is the ability of *H. pylori* to secrete the urease enzyme, which catalyses the conversion of urea to ammonia and bicarbonate. The test is performed at the time of gastroscopy. A biopsy of mucosa is taken from the antrum of the stomach, and is placed into a medium containing urea and an indicator such as phenol red. The urease produced by *H. pylori* hydrolyses urea to ammonia, which raises the pH of the medium, and changes the colour of the specimen from yellow (negative) to red (positive).

Aim

The aim of this study was to determine the change in the rate of *H. pylori* infection in gastric ulcers and duodenal ulcers for a 10-year period in a single centre.

Material and methods

A total of 4560 endoscopies were performed in 2004 in our hospital, and the endoscopy reports of the patients diagnosed with gastric ulcer and duodenal ulcer were assessed. Three hundred and forty-two patients were found to have gastric ulcer and duodenal ulcer. One hundred and eighty-four of these patients (54%) were male, and 158 (46%) of them were female. One hundred and fifty-three patients (45%) had a history of use of nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin, and 189 (55%) patients did not have a history of use of NSAIDs and aspirin, etc. The report results of 208 patients who underwent endoscopy in 1994 and who were diagnosed with gastric ulcer and duodenal ulcer were found, and the data of those patients were compared with the results of the patients in 2004. All the data were assessed based on the endoscopy reports. No histopathological data were found.

The study population included 550 patients (342 patients in 2004, 208 patients in 1994). In 2004, peptic ulcer (218 duodenal ulcer, 125 gastric ulcer) was diagnosed by endoscopy initially – an endoscopic ulcer was arbitrarily defined as a circumscribed mucosal break with a diameter of least 5 mm and a perceptible depth [10, 11]. Urease test was performed at the time of gastroscopy. A biopsy of mucosa was taken from the antrum of the stomach and was placed intro a medium containing urea and indicator such as phenol red. With *H. pylori*-urease present, which turns the indicator from yellow to red (within 60 min), so it was considered negative unless devoid of colour for 12 h.

Exclusion criteria were as follows: previous gastric surgery, malignant ulcer, and gastrointestinal bleeding as in the present study. The results of studies conducted ten years apart in the same centre were compared.

Statistical analysis

Data were analysed by χ^2 test when the expected value was under 5 (SPSS 11.0 for Windows, SPSS inc. Chicago, IL, USA). A p-value < 0.05 was considered as statistically significant.

Results

During the 12-month study period in 2004, 342 cases were identified. One hundred and twenty-five (36.5%) patients had gastric ulcer, and 217 (64.5%) patients had duodenal ulcer (Table I). The mean age of patients with duodenal ulcer was 48 ±12 years, while it was 54 ±11 years in patients with gastric ulcer. The test positivity was 39.2% in patients with gastric ulcers and 60% in patients with duodenal ulcers.

In 1994, 208 patients (159 duodenal ulcers, 49 gastric ulcers) with similar test results were included in the study (Table I). Urease test was positive in 74.2% of patients with duodenal ulcer and 65.2% of patients with gastric ulcer.

The rate of urease positivity decreased in patients with duodenal ulcers, but this result was not statistically significant (Figure 1). However, the decrease in the rate of urease positivity in patients with gastric ulcers was statistically significant (p = 0.01) during this 10-year period (Figure 2).

Discussion

In the present study we found that the urease positivity decreased significantly in patients with gastric ulcers between 1994 and 2004, as shown in Figure 2. The rate of urease positivity was also found to reduced in patients

Table I. Characteristics of the patients

Parameter	1994 (n = 208)	2004 (n = 342)	Value of p
Ulcer location:			
Gastric ulcer	49 (24%)	125 (36%)	< 0.05
Duodenal ulcer	159 (76%)	217 (64%)	< 0.05
Age [years]:			
Gastric ulcer	55 ±15	54 ±12	> 0.05
Duodenal ulcer	47 ±14	48 ±11	> 0.05

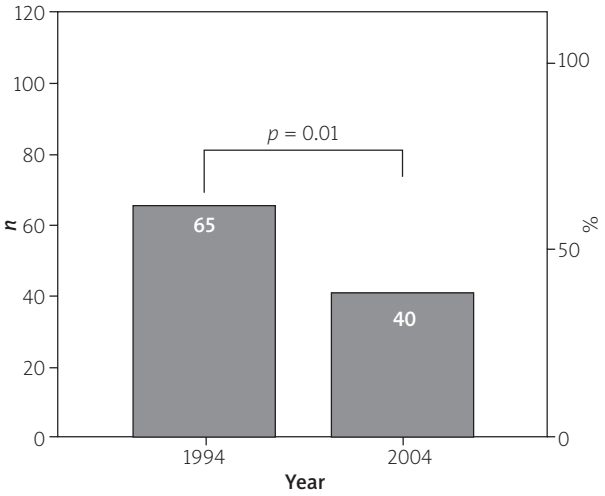

Figure 1. The urease test positivity in gastric ulcer patients in 1994 and 2004. Differences were assessed using χ^2 test, 51% male, 49% female in 1994, 54% male, 46% female in 2004

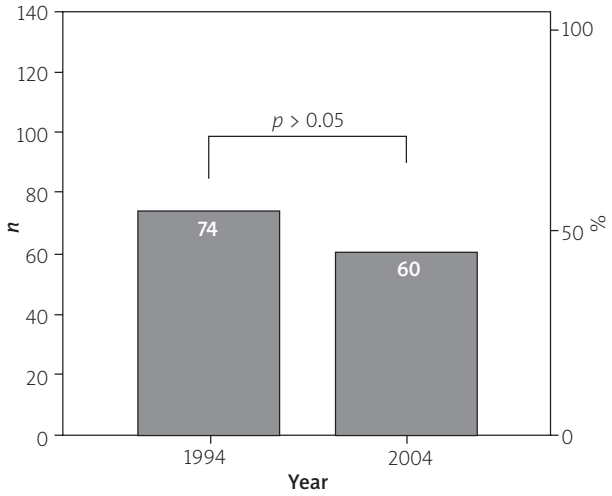

Figure 2. The urease test positivity in gastric ulcer patients in 1994 and 2004. Differences were assessed using χ^2 test, 54% male, 46% female in 1994, 52% male, 48% female in 2004

with duodenal ulcers, but the difference was not significant. We studied a hospital-based population, and our results do not demonstrate a decline in the prevalence of infection in the general population. However, this effect may have an impact on the results of our study.

For the past 20 years or so, peptic ulcer disease has been thought to be of multi-factorial aetiology. Acetylsalicylic acid (ASA) in particular, as well as most other NSAIDs, interacts with peptic ulcer disease in a number of ways. Some of the trends may be due to increased usage of ASA and NSAIDs in elderly persons.

There is conflicting data about the change in the prevalence of *H. pylori* infection worldwide. Chenn *et al.* found that the prevalence rate of *H. pylori* in residents of urban Shanghai, in a population aged over 30 years in 2001, was significantly greater than the rate found in 1990 ($p < 0.05$) [12]. However, recent studies have found a decreasing *H. pylori* infection rate in developed countries and those with rapidly improving socioeconomic conditions. Oona *et al.* found that the seroprevalence of *H. pylori* infection decreased significantly among hospitalised children from 1991 to 2002 (from 42.2% to 28.1%, respectively) [13]. Rehnberg-Laiho *et al.* compared the prevalence of *H. pylori* infection at different time points (1969–1973, 1983, and 1995) in females (age 20–34 years) from six different Finnish communities and showed a decrease from 38% to 12% [14]. Ozden *et al.* found that the overall prevalence of *H. pylori* antibodies was 78.5% in 1990 and 66.5% in 2000. The prevalence of *H. pylori* antibodies was found to decrease over a time span of 10 years ($p < 0.01$) [15]. Perez-Aisa *et al.* found a small decrease in the incidence of *H. pylori* infection: the inter-annual evolution was not associated with a significant decrease in total rates because the incidence remained high and was 76.3% in 1990, 75.6% in 1995, and 69.3% in 2000 [16]. In parallel with more effective eradication of *H. pylori*, the prevalence of this infection is changing, and the proportion of ulcers that are not associated with *H. pylori* infection seems to be increasing [7]. So the decline we observed in our study may be due to a decline in the prevalence in the general population.

One possible explanation for the increase in urease negative gastric ulcers and duodenal ulcers in our study may be due to the increase in the usage of NSAIDs in the general population. It is well known that NSAID-related GI complications are increasing worldwide [17]. However, we did not investigate the impact of this on the results in this study.

Conclusions

Urease positivity is decreasing in peptic ulcer disease especially in gastric ulcers. The cause of this decline should be revealed in further studies.

Conflict of interest

The authors declare no conflict of interest.

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