Helicobacter pylori prevalence in dentists in Japan: a seroepidemiological study

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Background. The infection mode of Helicobacter pylori is not well known. In order to prove that frequent exposure to saliva and dental plaque does not constitute a risk for acquiring H. pylori infection, we tested the hypothesis that the prevalence of H. pylori in dentists in Japan is the same as that in controls. We also studied factors associated with H. pylori prevalence by multivariate analysis. Methods. We examined serum anti-H. pylori-IgG in 232 Japanese subjects (116 dentists and 116 age- and sex-matched nonclinical controls). Participants were given a questionnaire that included demographic data, life style, past history, and gastrointestinal symptoms, and dental practice. Results. We analyzed the results for 111 dentists and 111 controls after exclusion of those who had an equivocal titer. The seroprevalence of H. pylori was 42.3% in dentists and 40.0% in controls. With multiple logistic regression, age was selected as the only independent variable correlated with seroprevalence (P = 0.0002; coefficient of determination 0.11). Factors associated with dental practice were not significant. Conclusions. We conclude that dental practice in Japan does not increase the risk of H. pylori infection for dentists.

Key words: dentists, ELISA, seroprevalence, epidemiology, Helicobacter pylori

Introduction

Infection with Helicobacter pylori is an important risk factor for type B gastritis, mucosa-associated lymphoid tissue (MALT) lymphoma, peptic ulcer, and gastric cancer.1–5 However, the exact mode of transmission of H. pylori is still unclear. The association of H. pylori infection with childhood socioeconomic status, crowded living conditions, and intrafamilial clustering6,7 indicates transmission by person-to-person contact. The absence of an environmental reservoir for H. pylori also suggests interpersonal transmission. H. pylori infection is typically acquired in childhood, and in developed countries, the frequency of acquiring the infection in adults is estimated to be about 0.5% a year.8 Fecal-oral and/or oral-oral routes are likely to be the cause of interpersonal infection by H. pylori.9–13 Some studies showed that the prevalence of H. pylori among endoscopy personnel was higher than that among nonendoscopy personnel.14–16 Exposure to gastric secretions must be the cause of this high prevalence. H. pylori can reach the mouth via the reflux of gastric contents, and it has been isolated from dental plaque and saliva.17–20 H. pylori in the oral cavity has mostly been isolated by polymerase chain reaction, but it has also been cultured on a small number of occasions.17,19,20 This discrepancy is considered to be caused by transformation of the rod-shaped H. pylori to coccoid forms. Coccoid forms can survive outside the stomach and are impossible to culture in vitro.10 If the oral cavity is also a reservoir of person-to-person infection of H. pylori, it can be hypothesized that frequent exposure to the oral cavity or the contents of the oral cavity may constitute a risk for acquiring H. pylori infection. In order to determine whether this hypothesis is true, four studies have been done.21–24 In these studies, the H. pylori prevalence of clinical dental personnel was compared with that of nonclinical controls. However, H. pylori prevalence was similar in clinical dental personnel and in controls. In Asian countries, including Japan, the prevalence of H. pylori in the general population is higher than that in Western countries.25 However, we hypothesized, according to the above previous studies, that the prevalence of H. pylori
in dentists would not be higher than that in nonclinical controls even in a country where the H. pylori prevalence in the general population is high. In the present study, we examined serum anti-H. pylori-antibody with an enzyme-linked immunosorbent assay (ELISA) to determine the prevalence of H. pylori. At the same time, we gave each participant a self-administered questionnaire asking about demographic data, life style, past history of gastrointestinal (GI) diseases, and GI symptoms. Dentists were also asked about their dental practice. We investigated the correlation between H. pylori infection and these explanatory variables with univariate and multivariate analysis.

Subjects

All participants were Japanese and were on the staff of Kanagawa Dental College Hospital, Yokosuka City, Japan. When the annual health checkup of all the personnel in the college was done on April 23–27, 1997, we recruited volunteers to donate blood samples for examination. A self-administered questionnaire was given to each participant.

From pervious studies, the prevalence of H. pylori in the Japanese general population was estimated to be about 40% at the age of 40, and it was known that the prevalence increases with age. The median age of dentists at Kanagawa Dental College Hospital was 39.0 years, and we estimated the prevalence of H. pylori in a population whose median age was 39.0 to be 40%. Assuming that the prevalence of H. pylori in dentists at Kanagawa Dental College Hospital was 40%, we calculated the sample size needed to prove equivalence of infection rates for dentists and nonclinical staff. We planned to recruit an age- and sex-matched group of nonclinical staff. We set the α level to 0.2, the power to 0.8, and the maximum allowable difference to 0.12 with a one-sided test. From these values, the sample size needed for each arm was estimated to be 95.27

A total of 488 subjects were sorted by age, sex, and name, and in each subgroup made, based on age and sex the same number of subjects were selected according to computer-generated random numbers. We studied 116 dentists, and randomly selected 116 age- and sex-matched nonclinical controls who were employees at the same college. Those who had received H. pylori eradication therapy previously or those who had taken antibiotics or a proton pump inhibitor or bismuth compounds during the preceding 2 months were not entered in this study. All participants gave their informed consent to the study.

Methods

ELISA

H. pylori infection was determined by the presence of serum IgG antibody to H. pylori, using an ELISA kit (HM-CAP; Enteric Products, Stony Brook, NY, USA), according to the manufacturer’s instructions. The sera were stored at −80°C before analysis. The results were expressed quantitatively as an ELISA value. The ELISA value was extrapolated from linear regression curves generated on the basis of the absorbance of the kit’s calibrators. A titer of less than 1.7 was regarded as negative. A titer of over 2.3 was regarded as positive. We excluded those who had an equivocal titer, of 1.8–2.2, from this study. All the assays were carried out at Mitsubishi-Kagaku Bio-Clinical Laboratory (Tokyo, Japan) as a cooperative study.

Questionnaire

The self-administered questionnaire contained questions on age, sex, occupation, past history of GI diseases, GI symptoms, regularly taken drugs, alcohol intake (ethanol above 55ml/week, or under 55ml/week), and smoking habits (none, less than one pack, more than one pack per day). The GI symptoms queried were nausea, epigastralgia, appetite loss, and dyspepsia. The GI diseases queried were gastric cancer, gastric ulcer, duodenal ulcer, and chronic gastritis, which had to be proved by endoscopic examination or by an upper GI series. Dentists were also asked about the number of patients seen per week (1–10, 11–20, 21–30, 31–40, 41–50, and 51 or more), whether they wore masks, whether they wore gloves, and whether they washed their hands before examining each patient during practice. All clinical dental personnel have worked at this hospital after graduating from Kanagawa Dental College, so that we could check their work history.

Statistical analysis

The JMP 3.2 (SAS software, Cary, NC, USA) was used for statistical analysis of data. The X² test, the Mann-Whitney U-test, and logistic regression were performed. Logistic regression was used to evaluate the association between occupation (dentists and controls) and the prevalence of H. pylori infection, with adjustment for the confounding variables. R²(coefficient of determination) was used to provide a measure of the strength of the association between suspected risk factors and the dependent variable (H. pylori infection).

Odds ratios and 95% confidence intervals (CIs) were calculated from the values of each cell in a 2 × 2 table (a, b, c, d) according to the formula: odds ratio = ad/bc; 95% CI = exponential [ln (odds ratio) ± square root