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HELICOBACTER PYLORI (HP) INFECTION AMONG HEALTHY BLOOD DONORS AND ENDOSCOPED PATIENTS IN NORTHERN PENINSULAR MALAYSIA

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Abstract

Over a 9-month period, sera from 5370 healthy blood donors were investigated for anti-HP IgG and anti-HP IgA antibodies. Seroprevalence for HP infection was 14.2%. There was no significant difference between the prevalence rate across all races and both sexes. Over a 36-month period, prevalence rate of HP infection among endoscoped patient was 14.6% but with Malays significantly lower compared to non-Malays. Generally significantly higher number of males were infected, but for a particular race such a trend was observed only among the Malays. Further prevalence of HP infection among Malay males or females was significantly lower compared to those of non-Malays. Infection by HP is equally distributed among all the major races in Malaysia, but how this primary infection predisposes significantly higher non-Malays to develop gastrointestinal related symptoms later remains unknown.

INTRODUCTION

Helicobacter pylori (HP) was first isolated in Australia by Warren and Marshall (11) from gastric antrum of patients with active chronic gastritis. The organism is now accepted as an etiologic agent of active chronic gastritis and peptic ulcer disease and a risk factor for pathogenesis of gastric cancer in humans (1,3). The prevalence of HP infection in populations varies with geographic area, age, race, ethnicity, and socio-economic status and is higher in developing than in developed countries, with most of the infections occuring during childhood (2).The present study, carried out on healthy blood donors and endoscoped patients, was aimed at determining the prevalence of HP infection between the major races in Malaysia.

MATERIALS AND METHODS

Serum samples from 5370 healthy blood donors were collected from various blood donation centres around the Penang Island from May 2000 till January 2001. In-house ELISA was used to detect the presence of anti-HP IgG and IgA antibodies in serum samples. The ELISA was performed as previously described (9) with slight modification in that excretory antigen was used instead of acid glycine extract. The antigen was prepared from an isolate of HP isolated from a 60 year old Indian female endoscopically confirmed with antral gastritis. In-house ELISA was evaluated against sera from 101 confirmed HP-positive and 519 HP-negative patients undergoing endoscopy. Culture was the gold standard. Cut-off point for positivity was established at $\geq$1.0 for IgG and $\geq$0.1 for IgA. Adsorption of pooled positive sera with *Escherichia coli, Pseudomonas aeruginosa, Pseudomonas stutzeri, Proteus vulgaris, Campylobacter jejuni, Campylobacter coli, Campylobacter fetus, Salmonella typhi, Shigella dysenteriae, Vibrio cholerae* and *Vibrio parahaemolyticus* did not result in a decrease in optical density below the cut-off point indicating no cross-reactivity with these organisms. In another study, the prevalence of HP infection was determined among 697 patients undergoing routine gastroduodenoscopy for gastrointestinal related problems at the Hospital Seberang Jaya, Penang from June 1998 to May 2001. Antral biopsies from patients were transported in Stuart transport medium (Oxoid, Basingstoke UK), inoculated on Eugon agar (BBL) plus 10% human blood and incubated in a 10% CO_2-air atmosphere at 37°C. Presence of HP was confirmed by rapid urease test and microscopy. Statistical analysis was carried out using Fischer's exact test and level of significance was taken at $p<0.05$.

RESULTS AND DISCUSSION

By taking seropositivity as positive by both IgG and IgA, sensitivity, specificity, positive predictive values and negative predictive values of in-house ELISA were 86.1%, 97.1%, 85.3% 97.7% for the detection of HP. Therefore the performance of in-house ELISA is comparable to commercial ELISA kits as reported (6,7). Mean age for all races or for both sexes was 32 years. Overall the seroprevalence was 14.2% which was higher than previously reported in Kelantan and lower compared to a recent report from a prospective study on 2381 samples in West and East Malaysia (4,10). There were 3588 Chinese (504 or 14% seropositive), 556 Indians (71 or 12.8% seropositive), 1114 Malays (169 or 15.2% seropositive) and 112 other races (16 or 14.3% seropositive), 3677 males (530 or 16.8% seropositive) and 1693 females (230 or 13.6% seropositive). There was no significant difference between the prevalence rate across all the races and both sexes. The donors were divided into five age groups and prevalence did not increase with age across all ethnic groups. Overall the present study among blood donors did not reveal the same significant

distribution of HP seropositivity with respect to race (Malays significantly lower) and increasing age as previously reported (4). Blood group antigens traditionally have been associated with a risk of developing peptic ulcer and gastric cancer. Since HP is an etiologic agent for active chronic gastritis and ulcer disease and a risk factor for carcinogenesis, studies were also carried out to assess the prevalence of anti-HP antibodies among donors with different blood group antigens. We found no significant association of seropositivity with ABO blood group, results which are in agreement with earlier reports (5,8). In a study on endoscoped patients, prevalence of HP infection was 14.6% a result which is in agreement with the present seroepidemiological study. Contrary to that, but in agreement with the generally held view (4), prevalence of HP infection among the Malays were significantly lower compared to non-Malays. Contrary to present seroepidemiological study also, significantly higher number of males were infected (75/396) compared to females (27/301): for a particular race, such a significant difference was observed only among the Malays and prevalence of HP infection among Malay males or females was significantly lower compared to those of non-Malays. The present seroepidemiological study revealed that infection by HP is equally distributed among all the major races in Malaysia, but how this primary infection predisposes significantly higher non-Malays to develop gastrointestinal related symptoms later remains unknown. Seropositivity may remains for long but other factors may have changed since primary infection. Genetic predisposition of a particular race with a particular HP phenotypes could also be a possibility.

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