

Detection of *H. pylori* infection and cagA strains seropositivity in adult dyspeptic patients in east Azerbaijan, northwest of Iran

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ABSTRACT

Background: *Helicobacter pylori* (*H. Pylori*) bacteria colonize human stomach mucosa and may establish acute or chronic gastric inflammation. Cytotoxic-associated gene A (cag A) is associated with higher grades of gastric inflammation and carcinoma. In the present study we determine cag A seropositive strain in dyspeptic patients with *H. pylori* infection.

Patients and methods: Six hundred adult dyspeptic patients examined for anti *H. Pylori* and anti-cag A antibodies by enzyme-linked immunoassay (ELISA) method. All cases resided in east Azarbijan in northwest of Iran and were enrolled in a 5-year period (2003–2008).

Results: A total of 85.5% of dyspeptic patients were positive for *H. pylori* infection. Anti-cag A antibody was detected in 35.6% of patients with *H. pylori* infection.

Conclusion: Screening of *H. pylori* infection by ELISA method revealed that the vast majority of (85.5%) dyspeptic patients are seropositive for *H. pylori*. Determining of photogenic strains of *H. pylori* by anti-cag A antibody could be diagnostic in severe gastric infections.

Keywords: *Helicobacter pylori*; Cytotoxic-associated gene A (Cag A); Gastric inflammation.
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INTRODUCTION

Helicobacter pylori (*H. pylori*) colonizes human stomach mucosa where it induces acute or chronic gastric inflammation. *H. pylori* infection increases the risk of peptic ulcers, stomach adenocarcinoma and lymphoproliferative disease of stomach mucosa (1,2).

H. pylori heterogenic are associated with a variety of clinical manifestations. Cytotoxic associated gene A (cag-A), one of the critical

pathogenic genes of *H. pylori*, has close relationship with peptic ulcers. This gene encodes a toxic vocoulizing protein, Vag A, in about 50% of *H. pylori* infections (1,2). P₃₀ and P₃₃ are the other pathogenic genes which participate in *H. pylori* pathogenicity. Cag A gene's length is 40 KD and most of the ulcerogenic strains of *H. pylori* have this gene which increases the risk of peptic ulcers, gastric atrophia and gastric adenocarcinoma (3).

In the present study, cag-A positive *H. pylori* strains have been evaluated in adult dyspeptic patients.

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PATIENTS and METHODS

Six hundred dyspeptic patients for whom gastric disorder has been confirmed by clinical examinations and endoscopy, enrolled in this study during a five year period (2003-2008). Anti-*H. pylori* IgG and anti-cag A antibody were analyzed by ELISA method (Padten and Diapro Kits) using six standards: 0, 15, 30, 60 and 100 unit (arbu/ml).

Data were entered and analyzed using SPSS software (version 14, SPSS Inc., Chicago, USA).

RESULTS

Six hundred dyspeptic adults (280 females and 320 males) aged 25-75 years old (mean: 43.2±12.1 years) and 200 controls with no signs of dyspepsia (mean: 42.1±15.2) were enrolled.

Anti-*H. pylori* IgG was positive in 85.5% (n=513) of patients and 81% (n=162) of controls. Totally, anti-cag A was positive in 30.5% of patients and 28% of controls. The difference did not reach a statistically significant level. Meanwhile, cag A-IgG was positive in 183 cases (30.5%) and 56 controls (28%).

Of 513 *H. pylori*-positive patients, 183 (35.6%) were also positive for cag A-IgG, however, this figure was 34.6% (56 of 162) in controls.

All *H. pylori* seronegative patients and controls were negative for cag A.

DISCUSSION

Frequency of cag A positive *H. pylori* strains was 35.6% and 34.6% among cases and controls, respectively.

Invasive and noninvasive methods have been developed for diagnosis of *H. pylori* infection. Serologic tests are among the best noninvasive methods. Because of the long half-life of *H. pylori* IgG it could be a good indicator of infection severity. Serologic detection of cag A could be very important in prognosis of *H. pylori* infection (4). Cag A strain is a pathologic marker that is

associated with strong immunologic response (5,6), hence, detection of this gene not only in *H. pylori* infected patients but also in asymptomatic cases, plays a critical role in treatment follow up and prevention of gastric ulcers and carcinomas. Frequency of cag A seropositivity has been variable in different societies: 50% in Turkish population (2), 40.2% in Iranian GERD patients and 43% in normal cases (7). In a study in Italy, frequency of cag A seropositivity was 86.1% in patients with duodenal ulcers, 96.4% in peptic ulcers and 52.4% in normal controls (8). In a study from China, *H. pylori* IgG in patients and controls was 88.9% and 45%, and cag A seropositivity was 78.1% and 31.3%, respectively (9).

European and American studies showed that 50% of *H. pylori* strains contain vag A gene and cag A (1).

Meta analysis of 21 study reports showed that specificity and sensitivity of ELISA testes were 79% and 85%, respectively (2). New commercial kits have higher sensitivity and specificity (98%) therefore, they could be more suitable to diagnose *H. pylori* infection. On the other hand, detection of pathogenic strains, cag A and vag A, with ELISA method and other genes which are associated with *H. pylori* pathogenicity, P₁₂₀ (cag A), P₃₀ (OMP) and P₃₃, could be helpful.

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REFERENCES

1. Lamarque D, Peek Jr RM. Pathogenesis of *Helicobacter pylori* infection. *Helicobacter*. 2003;8(suppl. 1):21-30.
2. Nazime OY, Ahmet S, Ali K, Iikay S. Detection of *H. pylori* infection by ELISA and Western blot techniques and evaluation of anti cagA seropositivity in adult

Turkish dyspeptic patients. *World Gastroenterol* 2006; 12(33):5375-78.

3. Sicinschi LA, Correa P, Bravo LE, Schneider BG. A positive assay for identification of *cagA* negative strains *Helicobacter pylori*. *J Microbial Methods* 2003; 55:625-33.

4. Peters TM, Owen RJ, Slater E, Varea R, Teare E, Saverymutu S. Genetic diversity in the *Helicobacter pylori* *cag* pathogenicity island and effect on expression of anti-*cagA* serum antibody in UK patient with dyspepsia. *J Clin Pathol*. 2001;54:219-23.

5. Herbrink P, van Doorn LJ. Serological methods for diagnosis of *Helicobacter pylori* infection and monitoring of eradication therapy. *Eur J Clin Microbiol Infect Dis*. 2000;19:164-73.

6. Monteiro L, Bergey B, Gras N, Megraud F. Evaluation of the performance of the Helico Blot 2.1 as a tool to investigate the virulence properties of *Helicobacter pylori*. *Clin Microbiol Infect*. 2002;8:676-79.

7. Somi MH, Fattahi E, Fouladi R, Karimi M, Bonyadi R, Baballou Z. An inverse relation between *CagA*+ strains of *Helicobacter pylori* infection and risk of erosive GERD. *Saudi Medical Journal*. 2008; 29(3):393-96.

8. Orsini B, Ciancio G, Surrenti E, Macrí G, Biagini MR, Milani S, et al. Serologic detection of *cagA* positive *Helicobacter pylori* infection in a northern Italian population. *Helicobacter*. 1998;3(1):15-20.

9. Abraham MY, Guillermo I, Lee J. Relation between *Helicobacter pylori* *cagA* status and risk of peptic ulcer disease. *Chinese Medical Journal* 2004;117(2):301-2.