the Journal of CONTEMPORARY DENTAL PRACTICE at www.thejcdp.com
Volume 10 Number 1 January 1, 2009

A Study of the Relationship between *Helicobacter pylori* and Recurrent Aphthous Stomatitis Using a Urea Breath Test

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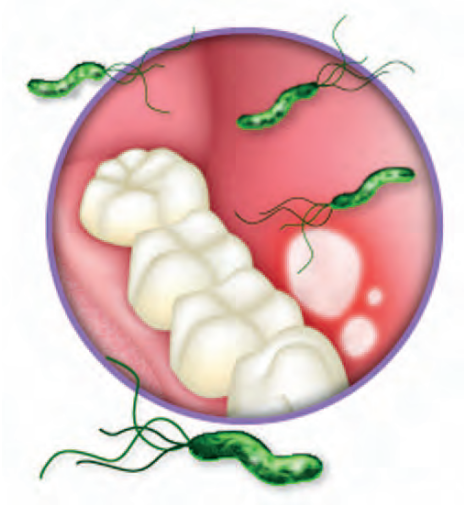


Abstract

Aim: *Helicobacter pylori (H. pylori)* is one of the most common, well-known pathogenic agents in the development of peptic ulcers. Some investigators have shown a relationship between *H. pylori* and recurrent aphthous stomatitis (RAS). However, this relationship is controversial. The aim of this study was to determine the association between *H. pylori* and RAS using the urea breath test (UBT).

Methods and Materials: Forty-three patients with RAS and 44 non-RAS controls were evaluated. There were no differences in gender or age in the two groups. The UBT was used to detect *H. pylori* infection. Data were analyzed using the Chi Square Test.

Results: Sixteen individuals in the RAS patients (37.2%) and 14 individuals in the control group (31.8%) had a positive breath test. The difference was not considered statistically significant (p=0.597).

Conclusion: In the present study no statistically significant difference was found between frequency of a positive UBT in the RAS patients and the control group.

Clinical Significance: Since the probability of a positive test was higher in the more severe cases this factor needs to be considered in the diagnosis and treatment of RAS.

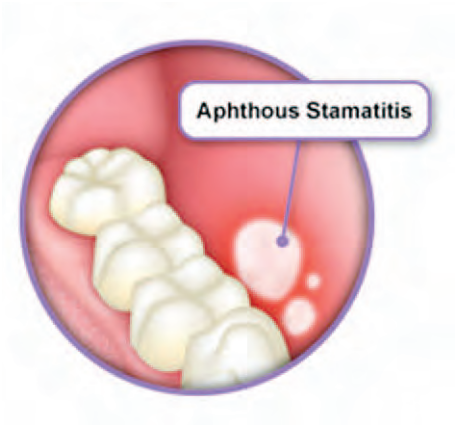
Keywords: *Helicobacter pylori*, *H. pylori*, recurrent aphthous stomatitis, RAS, urea breath test, UBT

Citation: Maleki Z, Sayyari AA, Alvavi K, Sayyari L, Baharvand M. A Study on the Relationship between *Helicobacter pylori* and Recurrent Aphthous Stomatitis Using a Urea Breath Test. J Contemp Dent Pract 2009 January; (10)1:009-016.

Introduction

Recurrent aphthous stomatitis (RAS) is a common disease, affecting about 20% of the general population at any time.[1] RAS is characterized by periodical painful solitary or multiple mucosal ulcerations. Etiology of RAS is unknown, but it has been suggested aphthous lesions are multifactorial.[2] Some factors, such as stress, trauma, iron deficiency, vitamin B12 and/or folate deficiency, food hypersensitivity, hormonal or immunological patterns, and family history, are known to be associated with RAS.[3-5] On the molecular level, stimulation of mucosal T cells by endogenous (autoimmune) or exogenous (hyperimmune) factors are shown be associated with RAS.[6] However, there is no single specific factor shown to be the causative factor. Therefore, there is no eradicating treatment for RAS and available ones are commonly palliative in nature.

It is assumed several viruses such as human herpes virus (HSV), varicella zoster virus (VZV), cytomegalovirus (CMV), human herpes virus 6 (HHV-6), and bacteria, including coagulase-negative staphylococci, α-hemolytic streptococci, and *Neisseria*, have a possible role in the pathogenesis of RAS.[7] *Helicobacter pylori*



(H. pylori), firstly named as *Campylobacter pylori*, is a gram-negative, microaerophilic bacterium which is strongly associated with gastritis and peptic ulcers.[8-11] Several investigations have shown *H. pylori* to be associated with RAS. For example, *H. pylori* DNA was detected in oral lesions in six of 29 patients with oral ulcerations.[12] However, there was no control group in this study. Riggio et al.[7] showed *H. pylori* DNA was present in 11% of biopsies from RAS lesions but not found in any oral lichen planus or normal samples. In a study on a group of patients with RAS, using polymerase chain reaction (PCR), *H. pylori* was detected in the oral cavity of 11.3% patients and 5.8% of controls, although the difference was not significant.[2] Porter et al.[5] found IgG anti-*H. pylori* antibodies in 30.6% of patients suffering from RAS compared to 24% of healthy controls, but the difference was not statistically significant.[5] In another study *H. pylori* DNA was found in 38.9% of patients with RAS and in 33.3% of controls, but this was not significant with a p value more than 0.05.[13] Using a three-medicine antibacterial regimen to eradicate *H. pylori*, Albanidou-Farmaki, et al.[14] observed both the severity and frequency of aphthous lesions decreased significantly in *H. pylori*-infected patients with RAS. Unfortunately, this study did not include a control group. In addition, in a survey in the United States the presence of *H. pylori* was associated with periodontal involvement.[15] It seems as though the oral colonization of H. pylori is not substantial and may involve dental plaque.[16-18]

The aim of the present study was to determine the relationship between *H. pylori* and RAS, based on a urea breath test (UBT), in a group of Iranian patients. UBT is a common, safe, and non-invasive test used in gastroenterology based on the presence of *H. pylori* urease.[19,20]

Methods and Materials

In this study patients with RAS lesions referred from three dental and medical centers in Tehran, Iran were evaluated. The centers included the Department of Oral Medicine at Shaheed Beheshti Dental School, Dermatology Department of Shohada-ye-Tajrish Hospital, and the Department of Gastroenterology of Mofid Hospital, all part of Shaheed Beheshti University of Medical Sciences and Health Services. The study was conducted from June 2006 to March 2007.

RAS was defined using Scully and Porter's criteria.[21] Patients with systemic conditions and aphthous-like lesions were excluded. These conditions included Bechet's disease, Crohn's disease, Reiter's disease or MAGIC (Mouth and Genital ulcers with Inflamed Cartilage) syndrome, celiac disease, nutritional deficiencies, cyclic neutropenia, HIV infection, FAPA (periodic fever, aphthous ulcers, pharyngitis, and cervical adenitis) syndrome, Sweet's syndrome, and drug reactions.[22] In addition, patients were excluded if they had received medical therapy for a *H. pylori* infection during the month prior to the test. However, patients with gastrointestinal diseases, such as peptic ulcers or gastritis but without antibacterial treatment, were not excluded. The study protocol was approved by the local ethics committee and informed consent was obtained from all participants.

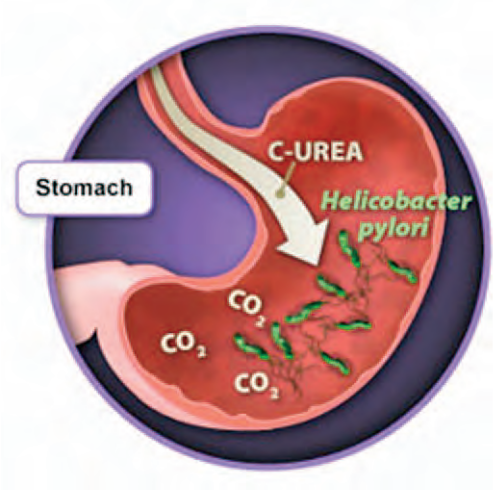
Sampling was done as a simple non-random method. As reported by Porter et al.,[5] the prevalence of infection with *H. pylori* is about 30% in the RAS population. Forty-three RAS patients and 44 volunteer controls were evaluated. With an α level of 5% and power of 80%, a statistically significant difference of approximately 30% between groups should be detectable. The control group included patients who were referred to the same centers and who had no oral lesions.

Potential participants were evaluated by a dentist who specialized in oral medicine, a pediatrician, and a dermatologist to confirm the inclusion criteria. The severity (scale) of aphthous lesion was recorded as described by Albanidou-Farmaki et al.:[14]

- 0 = No symptoms
- 1 = Mild symptoms; recorded as the presence of one or two lesions, with duration of 4-7 days and frequency of recurrence of every 2-3 months
- 2 = Moderate symptoms; recorded as the presence of two to five lesions with duration of 10-15 days and frequency recurrence of every one month
- 3 = Severe symptoms; recorded as more than five lesions with duration of more than 15 days and continuation in most of the days

In intermediate cases, the higher scale was recorded.

The UBT was conducted by first asking participants to fast overnight. Each participant was asked to hold his or her breath for 10 seconds and then exhale into a special bag marked "before" which was then closed. The participant was then asked to ingest 100 mg of non-radioactive isotopic urea (^{13}C). Labeled urea comes into contact with the mucosa and diffuses through the mucus. Here, urea hydrolysis by *H. pylori* produces ammonia and labeled carbon dioxide. Urea rapidly passes down its concentration gradient into the epithelial blood supply and within minutes appears in the breath.[19,20] After 30 minutes, the participant was asked to exhale into a second bag labeled "after" which was then securely closed. Then the two bags were attached to an ISOMAX 2000 mass spectrometer which looked for labeled ^{13}C and displayed the results as positive or negative. ISOMAX 2000 (Isodiagnostika Inc., Alberta, Canada) has a sensitivity of 100% and is 93% specific.



Data analysis was done using SPSS 14.0 software (SPSS Inc., Chicago, IL, USA). For quantitative data the Student's T test was used and the Chi Square and Mann-Whitney tests were used for nominal or ordinal values, respectively. The effect of different variables on the results of the UBT was determined by logistic or linear multivariates models of regression analysis, as indicated. These variables included various types of aphthous lesions (minor, major, or hepetiform RAS; as dummy variables), severity of lesions, gender, age, and gastrointestinal diseases. Type I error (α) was set at 0.05.

Results

In each group there were 15 males. There were no differences in gender in the two groups (X^2=0.006; p=0.938). The mean age (±SD) was 25.0±9.4 years (range 8 to 53, median: 24 yrs) in RAS patients and 23.7±5.2 years (range 7 to 35, median: 24 yrs) in controls (t=0.826; p=0.411). Documented gastrointestinal diseases are shown in Table 1.

In RAS patients four (9.3%) had major RAS and 39 patients (90.7%) had minor RAS. There was no case of herpetiform type. Mild symptoms were recorded in 23 patients (53.5%). Nineteen cases (44.2%) had moderate symptoms and one patient (2.3%) had severe symptoms.

UBT was positive in 16 individuals of RAS group (37.2%) and 14 controls (31.8%). This difference was not statistically significant (X^2=0.280; p=0597).

According to regression models, only age was related to a positive breath test (linear multivariate model; adjusted R^2=0.053; p=0.032). The mean age (±SD) of individuals with a positive breath test was 26.7±6.7 years vs. 23.1±7.7 years in individuals with a negative breath test. The frequency of major RAS was 12.5% (two patients) in patients with a positive test and 7.4% (two patients) in patients who had a negative test (X^2=0.309; p=0.578).

There was a significant relationship between the severity of symptoms and results of the UBT in RAS patients (Table 2).

Discussion

H. pylori plays a critical role in peptic ulcer disease and gastritis. Investigations on the relationship between this bacterium and oral disease originated from biologic and epidemiologic issues including bacterial oral-fecal transmission, acidic environment of the oral cavity, and ability of bacteria to induce epithelial ulceration, besides histological similarities between peptic ulcers and aphthous lesions. The cellular immune system is involved in both of these diseases. *H. pylori* can produce heat shock proteins and several lymphocyte chemotactic factors and may stimulate the release of some cytokines, such as IL-8. Also, neutrophilic

Table 1. Frequency of documented gastrointestinal disease among RAS patients and control group.

Disease	Frequency (%) in RAS Patients	Frequency (%) in Controls
Gastric ulcer	1 (2.3)	1 (2.3)
Duodenal ulcer	4 (9.3)	2 (4.5)
Gastritis	1 (2.3)	2 (4.5)
Irritable bowel syndrome	1 (2.3)	1 (2.3)
Gastro-esophageal reflux	1 (2.3)	-
Chronic constipation	1 (2.3)	-
No documented disease	34 (79.1%)	39 (88.6%)

Table 2. Severity of symptoms in patients with RAS, according to the result of the UBT.

Severity	Frequency (%) in positive test	Frequency (%) in negative test
Mild	5 (31.3)	18 (66.7)
Moderate	11 (68.8)	8 (29.6)
Severe	-	1 (3.7)
Mann-Whitney test:		
Mean rank	26.4	19.4
p value	0.041	

infiltration and production of free radicals with cytotoxic effects and inducing mucosal injury and apoptosis are seen in both conditions.[6] However, these findings are not conclusive.

H. pylori was first isolated from dental plaque of patients with *H. pylori* associated gastritis.[23] Consequently, it was shown isolates from the oral cavity were identical to gastric ones.[24] Some investigators suggested oral colonization might be the result of gastro-esophageal reflux.[6] Many researchers have suggested the oral cavity is a constant reservoir of bacteria,[14,25-29] but this is not a firm conclusion with respect to this phenomenon. In other studies, the frequency of *H. pylori* in dental plaque was less than 5%.30,31 It seems some patients have *H. pylori* only in their dental plaque. Mechanical cleaning of the oral cavity can affect bacterial growth and some products in saliva. For example, lactoferrin and specific IgA and IgG anti-*H. pylori* antibodies can eliminate or reduce bacterial colonization in the oral cavity and saliva.[32,33] In addition, oral bacteria, such as *Streptococcus mutans* and *Streptococcus sobrinus*, may limit bacterial growth.[34]

In the present study, no association between *H. pylori* and RAS was found. Findings of several studies, with different methods (mainly, anti-*H. pylori* antibodies) PCR, reported similar observations.[2,5,7,13] It seems *H. pylori* may be present and colonized in the oral cavity but it is not necessarily associated with aphthous lesions. There is some evidence for finding a probable association. *H. pylori* can induce peptic ulcers leading to iron deficiency due to apparent or occult bleeding or vitamin B12 deficiency due to inflammation and specially atrophic gastritis.[25,35,36]

Conflicting data from several studies may be a result of patient characteristics and different inclusion criteria. A relationship between severity of pathologic injury and bacterial species[37] or focal density has been reported.[38,39] Frequency of an *H. pylori* infection has also been reported to be related to ethnic, racial, and socioeconomic factors as well as age.[40] The association between a positive UBT and age was seen in the present study. These factors may have a confounding role in uncontrolled studies. Birek et al.[6] showed 71.8% of specimens from aphthous lesions have *H. pylori* DNA sequences based on the PCR technique. However, results of the PCR may be a false positive.[41] PCR results are not always in concordance with bacterial culture methods.[7] In one study there were 16 HIV positive patients, and the diagnosis of RAS is not definite in these patients.[42] Additionally, urease tests may not be valid in the oral cavity due to presence of several urease-positive bacteria.[16,26] There are more than 350 bacterial species in the oral cavity that may contribute to false positive results in the detection of *H. pylori*.[43] The UBT used in the present study has sensitivity and specificity of more than 90% but it is not the gold standard used to detect *H. pylori*. Multiple approaches are required to reach a definite diagnosis. Bacterial culture of specimens from RAS lesions and PCR on specific genes obtained from direct biopsies may be suitable.

Conclusion

In the present study no statistically significant difference was found between the frequency of a positive UBT in RAS patients and the control group.

Clinical Significance

Since the probability of a positive UBT test was higher in the more severe RAS cases this needs to be considered in the diagnosis and treatment of RAS.

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