

RESEARCH COMMUNICATION

Lack of Association Between *Helicobacter pylori* Infection and Oral Lichen Planus

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Abstract

Oral lichen planus is a premalignant chronic inflammatory mucosal disorder with unknown etiology. It is a multifactorial disease and in addition to genetic background, infections, stress, drug reactions are suggested as risk factors. *Helicobacter pylori* which is involved in development of many gastrointestinal lesions may also be implicated in oral lichen planus induction. This is of clear importance for cancer prevention and the present study was performed to determine any association between *H. pylori* infection and oral lichen planus in southwestern Iran. Anti *H. pylori* IgG levels were determined in 41 patients and 82 sex-age matched controls. The results showed no association between *H. pylori* infection and oral lichen planus (51% in patients vs. 66% in control), or any of its clinical presentations.

Keywords: Oral lichen planus - *Helicobacter pylori* - etiological factor - Iran

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Introduction

Oral lichen planus (OLP) is a chronic inflammatory disease with a prevalence rate of 0.1 to 4% in different populations (Seoane et al., 2004; Thongprasom et al., 2011). OLP often occurs in middle-aged adults and affects men 2 to 3 times more than women (Laeijendecker et al., 2005). OLP usually affects stratified squamous epithelium (Eisen et al., 2005) and causes lesions or plaques on buccal mucosa, tongue, and gingiva (Mollaoglu, 2000). Reticular, popular, plaque like, erosive, atrophic and bullous are different clinical presentations of OLP. Reticular and erosive types are common clinical forms (Mollaoglu, 2000). In contrast to cutaneous lesions which are usually self limiting, symptomatic patients with OLP often need intensive treatment and topical corticosteroids are the main medication in such cases. Although there is little evidence for helpfulness of usual treatments, long-lasting oral lesions may lead to malignant transformation (Chan et al., 2000; Dissemond., 2004; Eisen et al., 2005).

OLP is a multifactorial disease and in addition to genetic background, infections, stress and drug reactions are suggested as risk factor (Seoane et al., 2004; Konidena and Pavani, 2011). *Helicobacter pylori* (*H. pylori*) is involved in gastrointestinal lesions but its role in OLP is still suspicious (Attia et al., 2010), this study was designed to find any probable association between *H. pylori* infection and OLP in southwestern Iran.

Materials and Methods

This case/control study involved patients with OLP referred to the department of oral medicine, Shiraz School of Dentistry. After obtaining informed consent, a total of 41 new OLP patients were enrolled in this study. OLP was diagnosed based on clinical examination (Jontell and Holmstrup, 2008). Eighty two sex-age matched individuals without any kind of mucocutaneous inflammatory diseases were selected from the same geographic region and the same ethnicity as control group. Individuals who had taken NSAIDs, corticosteroids or antibiotics during last two months were excluded. Individuals who had a history of gastrointestinal or cardiovascular problems or reported gastrointestinal disorders in their first degree relatives were also excluded.

Whole blood (2 mL) was collected from the patients and controls and serum was separated and stored at -20 °C before use. The level of anti *H. pylori* IgG was measured in each serum sample by ELISA method in duplicate. Chi square test was used for finding the relation between sex, age and anti *H. pylori* IgG levels in patient and control groups. For evaluation of association between *H. pylori* infection and the disease condition after controlling demographic factors binary logistic regression analysis was used. SPSS 16 statistical software was used for data analyses and P-value less than 0.05 was considered statistically significant.

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Table 1. Group Division of *H. Pylori*

	Patients n=41 (%)	Controls n=82 (%)	P value
Triple group division			0.28
Negative	15 (36.6)	20 (24.4)	
Equivocal	5 (12.2)	8 (9.8)	
Positive	21 (51.2)	54 (65.9)	
Double group division			0.117
Negative	20 (48.8)	28 (34.1)	
Positive	21 (51.2)	54 (65.9)	

Table 2. Independent Parameters

	β	SE	OR (95% CI)
<i>H. pylori</i>	0.731	0.404	2.1 (0.94-4.59)
Age	0.29	0.016	0.97 (0.94-2.002)
Sex	0.192	0.404	1.21 (0.55-2.67)

Results

In this study, 41 OLP patients (25 women and 16 men) ranged in age from 24 to 78 years (mean age 45.17±14.61 years) and 82 controls (48 women and 34 men) ranged in age from 20 to 65 years (mean age 41.18±11.52) were assessed for anti *H. pylori* IgG serum levels.

There was no significant difference between patients and controls in demographic factors. However the percent of seropositive individuals were higher in control group than patients, this difference was not significant (Table 1). The most common clinical presentation of our OLP patients was keratotic plaques (34.1%), 31.7% of the patients showed both keratotic and atrophic plaques, 24.4% had both keratotic and ulcerative lesions and 9.8% were involved with more than two forms of the plaques. No correlation was observed between any type of OLP clinical presentations and the serum levels of anti *H. pylori* IgG.

The results of binary logistic analysis showed no association between anti *H. pylori* IgG levels and the development of OLP after controlling sex and age factors in patients and controls (Table 2).

Discussion

OLP is an inflammatory mucosal disorder with uncertain etiology. Both genetic background and environmental factors (certain dental materials, some drugs, infectious agents and stress) are considered in the development of this disease. Because of inflammation caused by allergy to dental materials or reaction to infectious agents in some patients with OLP, an immune-mediated pathogenesis is suspected in OLP (Seoane et al., 2004; Konidena and Pavani, 2011).

H. pylori infection which serves as a main cause of peptic ulcer and may lead to gastritis and cancer has also been considered in different oral lesions such as aphthous stomatitis and periodontitis. However no strong association was observed between the presence of *H. pylori* in oral cavity and oral lesions in patients with recurrent

aphthous stomatitis (Victoria et al., 2003; Fritscher et al., 2004), it was detected in the saliva and subgingival samples in a high percentage of patients with periodontitis (Gebara et al., 2004; Souto and Colombo, 2008).

However based on some evidence oral cavity can be considered a reservoir for *H. pylori* in non-dyspeptic individuals, some researchers believe in temporary presence of *H. pylori* in such situation (Martinez-Gomis et al., 2006; Burgers et al., 2008). Other researchers believe that non-infectious *H. pylori* might exist in the oral cavity which is just able to form colonies and invade tissues under special conditions such as concomitant infections (Martinez-Gomis et al., 2006). *H. pylori* colonization might be necessary for the induction of OLP but not crucially detectable in the generated OLP lesions by PCR or bacterial culture (Riggio et al., 2000; Shimoyama et al., 2000).

Although OLP can be considered a result of bacterial pathogenesis or direct immune responses against bacterium which also affect mucosal tissue, as Attia et al., detected *H. pylori* DNA in oral lesions in all their patients with erosive OLP (Attia et al., 2010), an autoimmune-based mucosal destruction due to epitope spreading following the release of autoantigens from the injured cells after bacterial infection or antigenic mimicry between *H. pylori* and one of the antigens in mucosa membrane is more likely (Tchernev and Nenoff, 2009). In such conditions, neither the detection of *H. pylori* in the OLP lesions nor simultaneous gastric infection with this bacterium is essential in OLP formation as Taghavi Zenouz et al. did not find any relationship between OLP and functional *H. pylori* infection by urease breath test (Taghavi Zenouz et al., 2010).

In this study we looked for anti *H. pylori* IgG to determine if there is an association between OLP and a serologic record of previous exposure to this bacterium.

Conflicting evidence exists regarding the link between *H. pylori* and skin disorders. There is some evidence to support a negative relation between *H. pylori* infection and SLE, while positive association between *H. pylori* and chronic urticaria or immune thrombocytopenic purpura has also been reported (Sawalha et al., 2004; Hernandez-Harder et al., 2009). To reduce confounding factors, we selected the individuals without any mucocutaneous inflammatory diseases as control group.

Although we detected anti *H. pylori* antibody in 51% of our patients, no significant association was observed between OLP and anti *H. pylori* IgG. It might be explained by the high percentage of *H. pylori* seropositive individuals among the control group (66%). According to the previous report more than 50% of the human population has a history of *H. pylori* infection (Wedi and Kapp, 2002).

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