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ARTICLE

The Relationship of Helicobacter pylori and Dental Plaques with Gastric Dyspepsia

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Abstract

Introduction: *Helicobacter pylori* (*H. pylori*) is a pathogen which is capable of colonizing the gastric mucosa and is associated with some pathological changes that may lead to dyspeptic manifestations. Recently, *H. pylori* was found to be present in the oral cavity and there was a growing body of evidence that the organism is linked to some dental diseases such as periodontitis and gingivitis. **Materials and Method:** In this study, 123 patients were investigated to explore the possible relationship between the presence of oral *H. pylori* and gastric dyspepsia. Patients were classified into oral disease group (viz. gingivitis and periodontitis) or gastric disease group (viz. gastritis, gastric ulcer, and duodenal ulcer). A third group had neither had oral nor gastric disease. Based on the presence or absence of *H. pylori*, patients were also classified as having either oral, gastric, or both oral and gastric organism. Some of the patients were neither having oral nor gastric infection. **Results:** 59 patients showed the presence of *H. pylori* in the gastro-duodenal area, 7 showed presence of oral *H. pylori*, whereas 18 patients showed *H. pylori* in both gastro-duodenal and oral specimens. Differences between groups were not statistically significant. **Conclusions**: Although there was a suggestion of a clinical and laboratory evidence of an association between the presence of oral *H. pylori* and the studied gastric diseases, this association was not statistically significant.

Key words: Helicobacter pylori (H. *pylori*), Gingivitis, Periodontitis, Gastritis, Gastric ulcer, Duodenal ulcer

Introduction

For years, studies on the relationship between *Helicobacter pylori* (*H. pylori*) in the mouth and the gastric mucosa have not resulted in a final conclusion justifying the presence of *H. pylori* in the oral cavity as pathogenic or just as a transitory organism. An association between oral infections and systemic diseases has been suspected

for centuries (1). *Helicobacter* pylori is a Gram-negative microorganism which is able to colonize the gastric mucosa and is associated with peptic ulcer, gastric carcinoma, and gastric mucosa-associated lymphoid tissue lymphoma (2). In addition, some oral diseases have actually been confirmed to be related to *H. pylori* infection. A typical one is periodontal disease, which has been demonstrated to be positively linked with *H. pylori* infection (3). However, the potential relationship between many other oral diseases and *H. pylori* infection has not been demonstrated (3). The oral cavity may be the first place for colonization by *H. pylori*, and then the infection involves the gastric mucosa (4). Transmission of *H. pylori* infection is fecal-oral, oral-oral, and gastro-oral through vomiting (5). The bacterium can live, among others, on the saliva (6,7), in dental plaque (7), in the mucus membrane of cheeks, tongue (8) and stool (6). The aim of this study was to evaluate the presence of *H. pylori* n the oral cavity of patients with functional dyspepsia (epigastric pain syndrome).

Material and Methods

Patients presented to periodontology department, Oran Algeria, with periodontitis and epigastric pain were evaluated. Patients with periodontitis who had never suffered from gastritis/peptic ulcers were selected as patient's control. The criteria for selection of study subjects were as follows: patients with no previous treatment for ulcer or gastritis three months before the start of the study. Patients were classified into orally presented disease (i.e. gingivitis and periodontitis) or gastric presented disease (i.e. gastritis, gastric ulcer, and duodenal ulcer) (Table1). Dental plaques from 123 patients were obtained before gastroscopy. Gastric *H. pylori* infection was diagnosed using rapid urease test, Giemsa staining and culture. Subgingival plaque samples were collected with standard dental scaling hand instruments and sent to microbiological laboratory for detection of *H. pylori* by direct Gram stain as well as culture. We followed the method used by Cheng LH et al. (9): Equal amounts of dental plaque and gastric mucus were dispersed in 5 ml of normal saline solution for culture and 5 ml of modified urea broth with phenyl red indicator to detect urease activity. Samples of dental plaque were divided with sterile equipment into two equal halves; separate gastric brushings from the same region of the stomach were necessary to produce two similar specimens. Matched samples of soft dental plaque and gastric mucus from each patient were centrifuged and inoculated onto a nonselective 10% horse blood agar and selective Skirrow's agar 18 that consisted of lysed blood with the addition of antibiotic supplements (vancomycin, trimethoprim, and polymyxin B). The agar plates were incubated in a microaerophilic atmosphere (produced by an oxoid gas generating kit specifically for Campylobacter) at 37° C for 5 days. A Gram stain was then performed. Those colonies that demonstrated the characteristic curved gram-negative rod appearance were further tested by oxidase, catalase, and urease tests. Data were collected, described and compared using Chi square-test of significance and the probability were measured at 0.05 level

Results

The oral *H. pylori* strain was urease, catalase, and oxidase positive, curved Gram-negative bacteria that did not grow on the aerobic control, which is typical for *H. pylori* (5). The oral strain was resistant to metronidazole (MIC, 32 mg/L) and clarithromycin (MIC, 1 mg/L) (6). Of the total 123 patients enrolled in this study, 39 patients (31.7%) suffered from duodenal ulcer, 25 patients (20.3%) suffered from gastric ulcer, and 22 patients (17.9%) suffered from gastritis. Regarding oral disease, 52 patients (42.3%) presented with periodontitis, and 21 patients (17.1%) were suffering from gingivitis. 59 patients (48.0%) showed presence of *H. pylori* in the gastro-duodenal area, 7(5.7%) showed presence of oral *H. pylori*, whereas 18 patients (14.6%) showed *H. pylori* in both gastro-duodenal and oral specimens. Comparing the relation of the presence of oral and gastric *H. pylori*, Chi^2 was 1.184 (NS). Comparing the presence of gastric disease with oral infection with *H. pylori*, and the presence of oral disease with the gastric infection with *H. pylori*, Chi^2 were 0.055, and 1.97, respectively, (both NS). Comparing the relationship between both oral and gastric diseases in patients suffering from *H. pylori* infection, Chi^2 was 2.378 (NS).

Discussion

Conflicting reports by searchers was perceived in the published articles about *H. pylori* in the oral cavity. In fact, several authors have identified *Helicobacter pylori* in the oral cavity and more exactly in the dental plaque. Some of them agree with the hypothesis that the oral cavity, dental plaque or saliva presents a potential reservoir for the *H.* pylori (10,11). *H. pylori* was detected in the supragingival plaque of individuals with periodontal disease and upper gastric diseases (2). Its detection in gastric antrum, dental plaques, feces and water was confirmed (12). Interestingly, others few authors deny any relationship between *H. pylori* in oral cavity and stomach and approve that the oral cavity may not be a reservoir for *H. pylori* (13-18). However, the

Table 1. The relationship between gastric disease and periodontal diseases

Patients symptoms	Confirmed diagnosis	Periodontal disease	
		Yes	No
Gastric problem	Duodenal/Gastric ulcer	39	25
	Gastritis	10	12
No Gastric problem	No gastric disease	24	13

present study suggests that the primary and vital reservoir for *H. pylori* is the stomach and duodenum. Its small rate in dental plaque in our experimentations allows us to support the hypothesis cited by many researchers that the oral cavity or dental plaque is a transient reservoir for this microorganism. Many authors have reported the transient role played by *H.pylori* in the oral cavity (14,19-21).

Diagnosis of *H. pylori* infection in the oral cavity is more difficult than in the stomach. We assume that its presence in low percentage in the dental plaque may also be due to microbiological methods used for its isolation. This is due to a number of factors, among them a small oral population of *H. pylori* and a lack of a simple method to detect *H. pylori* in the oral cavity. Such as media of culture, the methods of incubation (amount of oxygen present was higher) or the kit for Campylobacter used and who gives best results in the specimens from the stomach was not appropriate for the culture of *H. pylori* in the mouth. The immunodiagnosis test was successfully applied to detect *H. pylori* antigens in stool, now it appears to be also effective in the diagnosis of *H. pylori* infection in the oral cavity. Its absence may be due to the population sampled or methodology applied (22). The researchers substantiate the inability to identify *H. pylori* in the oral cavity may be due to a complex oral microflora and the sensitive nature of *H. pylori*, which requires special conditions such microaerophilic environment, supplemented media, and up to seven days incubation for its growth (23). Furthermore, the possibility of genetic diversity of *H. pylori* made our culture with non-important rate because we apply the same methods of culture as well as oral and gastric so it's possible that it might have undergone a genetic variation and changed its nutritional requirements. Loster BW et al., found that the oral bacteria and those originating from the stomach are completely different (21). Cai H et al, reported genetic variation of *H.*pylori in the oral cavity and stomach (15). The overabundance of the oral cavity by millions of microorganism species can also be another factor. Indeed, it could be that certain types of microorganisms have the ability to inhibit the growth of *H. pylori* in the mouth by their virulence factors like releasing some toxins or enzymes. Overgrowth by oral species others then *H.pylori* is likely, and direct growth inhibition of *H. pylori* by oral species in vitro was reported (23).

We have detected *H. pylori* in 18 patients (20.63%) suffering from gastric and periodontal disease. Namiot DB et al. (10) detected it only in three cases among 157 patients. *H. pylori* was detected in dental plaque with a rate of 34.1% (16) and in 82.3% by Gao J et al. (24). Boyanova et al. (6) isolated oral strain from a child with both gingivitis and chronic gastritis. In the study of Anand et al., the occurrence of *H. pylori* in the dental plaque of patients with gastric *H. pylori* infection was higher than in controls (25). Sudhakar U et al. (1), identified five patients (10%) positive for *H. pylori* by culture. *H. pylori* was identified by culture in 2/208, and by PCR in 15/208 (7%) (26). This result demonstrates the importance of using several methods for detecting *H pylori* infection (26). YanSong Z et al.,(27) found *H. pylori* in 1.69% of patients with periodontitis.

We isolated this organism in 7 cases (5.7%) with only periodontal diseases. For Bürgers R et al. *H. pylori* may be present in the oral cavity independently of the colonization of the stomach (28). According to Zou QH and Li RQ, the prevalence of *H. pylori* infection in the oral cavity (45.0%) of the patients with gastric *H. pylori*-positive patients was considerably higher than that in cases with gastric *H. pylori*-negative patients (23.9%) (29). Additionally, 30% only of the patients with non-periodontitis harbor *H. pylori* in their mouth and stomach versus 78% of patients with periodontitis had a positive test result for the coexistence of *H. pylori* in both dental plaque and the stomach (11). The

work of Silva Rossi-Aguiar, et al., 30 patients' harbored *H. pylori* in the stomach, but it was not possible to detect *H. pylori* in any oral samples (18). Therefore, another study confirms the absence of *H. pylori* from any of the 290 oral samples (14). It is very important to detect and eradicate early *H. pylori* in the oral cavity, especially in high-risk patients, such as tobacco users, alcohol consumers (30). Our preliminary data focused on the use of non-invasive method. The ability to detect *H. pylori* in dental plaque samples offers the potential for a non-invasive test for infection by this bacterium (1).

In conclusion, determination of the presence of *H. pylori* in the oral cavity cannot substitute the diagnosis of this infection in the stomach, because the presence of *H. pylori* in the mouth cavity may or may not be associated with the presence of gastric dyspepsia. However, we plan to investigate the exact role played by *H. pylori* in the mouth. Thus, non-invasive methods who can help the gastroenterologists to diagnose a very early infection in patients at high-risk of infection by this organism. We will use more than one method for its detection and we will increase the number of samples to ensure that all our hypothesis are correct or not. The detection *H. pylori* in the mouth will make diagnosis of infection easier and avoid the use of invasive methods which are expensive and traumatic for patients. Finally, to eradicate early the bacterium with appropriate treatments.

Disclosures

Authors' contributions

All authors contributed substantially by data collection, analysis and review and approval of the final version of the manuscript.

Conflicts of interests

None of the authors declared any multiplicities of interests that may potentially jeopardize the integrity of the study.

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Compliance with ethical principles:

The human subjects were conducted in accordance to the principles of medical ethics. All data are collected anonymously.

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