

Helicobacter Pylori in the dental plaque

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Received: 03-01-2014

Revised: 25-02-2014

Accepted: 27-03-2014

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ABSTRACT

Background: The oral cavity has been suggested as a reservoir of *Helicobacter pylori*, but the assumption that the oral microflora may be a permanent reservoir is still controversial.

Objective: The aim of this work is to see if the plaque can be a reservoir of *H. Pylori* based on data from clinical study and literature.

Materials and Methods: 35 patients were recruited; oral bacterial samples were taken from patients before performing endoscopy and gastric samples. These samples underwent microbiological and histological examination with bacterial identification.

Results: In periodontal samples, the urease test was positive in 33 of 35 patients, whereas no culture was positive. In gastric biopsies, the Urease test was positive for 20 samples from 35, the culture showed that the bacterium was present in 16 of the 35 patients, the Urease test and culture was positive for 15 samples from 35. On histological examination, 22 samples were positive among 35.

Conclusion: Whatever the role of the oral cavity (transient or permanent reservoir of *H. Pylori*), a support dental and periodontal case is essential in all patients with *H. Pylori* in the stomach. Prescribing antibiotics for the eradication of *H. Pylori* in the stomach does not allow disposal at the dental biofilm; Mechanical disruption of the latter by scaling and root planing is essential.

Key Words: *Helicobacter pylori*, oral cavity, reservoir, dental plaque, samples

Introduction

Helicobacter Pylori (*H. Pylori*) is a Gram negative, curved or spiral, microaerophilic and urease-producing. This germ is involved in several gastrointestinal diseases such as chronic gastritis, peptic ulcer, gastric cancer or lymphoma of the gastric mucosa. Infection of the stomach

caused by *Helicobacter pylori* are prevalent worldwide and can cause serious medical problems, ranging from gastritis to gastric cancer. Thus, the search for *H. Pylori* in the oral cavity has been the subject of several recent studies: the oral cavity has been suggested as a reservoir for *H. Pylori*, but the hypothesis that oral

microflora may be a permanent reservoir is still controversial. [1, 2]

Current studies show that *H. Pylori* is present in dental plaque, although the number of organisms in the individual samples is very small and it seems to vary from one location to the other of the mouth. The presence of this organism in dental plaque may occur intermittently, may be as a result of gastro - oesophageal reflux. It is unclear whether the small number of *H. pylori* detected in the mouth of most patients would be sufficient to become a source of infection or reinfection causing gastric disorders. [3, 4]

The aim of this work is to see if the plaque can be a reservoir of *H. Pylori* based on data from clinical study in gastroenterology service of Ibn Sina Hospital in Rabat and the supporting as data from the literature.

Material and Methods

Patients were recruited from the gastroenterology service (Medicine C) CHU Rabat during the academic year 2002-2003, between January and May. This work was conducted with 35 patients who were selected based on the following criteria:

- Be older than 18 years
- Did not have prior to endoscopy
- We're not taking anti-ulcer during the last three months
- Not suffering from systemic diseases requiring antibiotic prophylaxis
- Not be treated with antibiotics systemically or in *H. pylori* receptor antagonists

- And with no vomiting to eliminate possible contamination gastro-oral by the bacteria

Review of patients and samples:

Oral clinical examination was performed to assess the state of oral mucosa and periodontal status by raising the gingival index and performing periodontal probing.

Once completed the oral examination, oral samples are taken at the periodontium, saliva and dental plaque using sterile swabs. For each patient, three mouth swabs are made: first, the sample is spread on a slide in order to perform cytology-second sample is placed in a sterile tube containing saline to achieve culture. The rapid urease test is performed by placing the sample in the third urea-indole medium.

Gastric and Retrieval Endoscopy

Upper gastrointestinal endoscopy and duodenal was performed in all patients to describe aspects of the lesions and to perform biopsies in the antrum:

- The first biopsy was placed in formalin for histological examination,
- And the other two kept in saline at 4 ° C, and were sent to the microbiology laboratory where Gram stain and culture were performed after crushing the samples.

Review microbiology:

The rapid urease test: The samples were placed in the middle of urea-indole: when *H. Pylori* is present, the large amount of urease present results the hydrolysis of urea to ammonia and bicarbonate. The liberated ammonia increases the pH of the medium from which the color change the pH indicator.

Culture: Two culture media were used:

- A non-selective medium enriched with 5% sheep's blood more growth supplement (Polyvitatex*) for homogenates of gastric biopsies,
- And an enriched and selective (*H. pylori* Selective Supplement: Selective Supplement tooth) SR 147 E® containing antibiotics (vancomycin, cefsulodin, trimethoprim, and amphotericin B) for buccal swabs.

The buccal swabs and gastric biopsy homogenates were placed on the curb for a perfect homogenization, respectively and then cultured in selective medium or not and incubated at 37° C in microaerophilic atmosphere for 12 days.

Histology

Histological examination was performed on gastric biopsies by Giemsa staining.

BACTERIAL IDENTIFICATION

Produced by:

- the study of morphological characters after Gram staining of colonies (Gram negative curved);
- the study of biochemical characteristics (bacteria oxidase +, catalase +)

Results

The gram negative curved rods were observed at gastric level. The rapid urease test at oral level was positive in 33 (94.2%) patients of the 35 patients examined. Culture, bacteriological examination of reference, showed that gastric level, the bacterium is present in 16 patients among the 35 is 45.7%, whereas in the mouth prevalence is 0%. (Fig. 1, 2) We noted the

presence of aphthous lesions in 10 patients among the 35 patients, or 28.5% (Fig. 3)

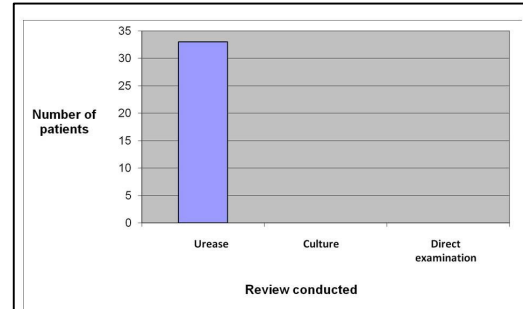


Fig. 1 Presence of *H. pylori* in the oral cavity depending on the type of examination performed

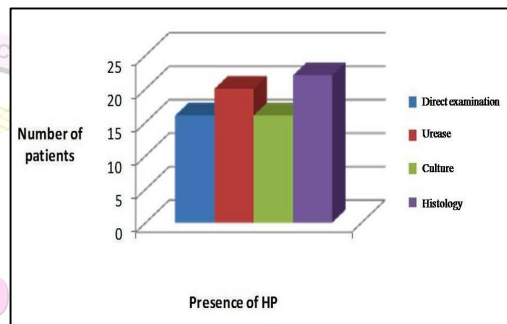


Fig. 2 The presence of *H. Pylori* in the stomach

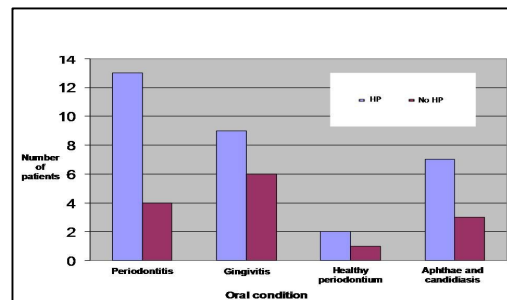


Fig. 3 Presence of *H. Pylori* in oral conditions

We also found a defective oral status of patients examined with 17 cases of chronic periodontitis in 35 patients, or 48% (Fig. 4)

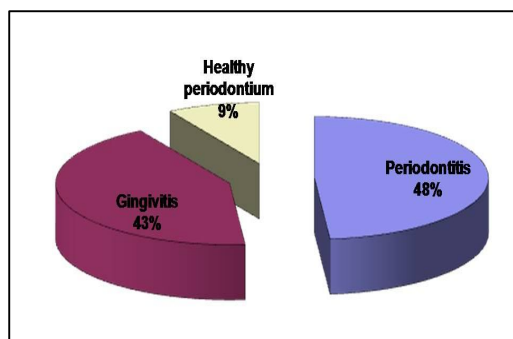


Fig. 4 Periodontal status of patients examined

Discussion

The rapid urease test in the mouth was positive in 94.2% of patients examined (33/35).

This positivity was detected in the half hour following the deposit of samples in the middle indole urea. This is in favor of the presence of H. pylori in the oral cavity. The urease test is quick, simple and inexpensive. It also presents a sensitivity and specificity depend on the type of test used urease and also the time of reading. According to Williams⁵, H. Pylori bacteria would present the most important urease activity among all known bacteria. And several studies have reported the presence of H. Pylori in the mouth on the basis of different types of tests urease (Table I).

However, some studies have shown that other bacteria were producing urease and their presence in the plaque could give false-positive urease, such as pseudomonas and enterbacteriaceae sp⁶. Other types of microbiological tests such as culture or PCR have also detect H. Pylori in dental plaque. In our study, the culture

showed that H. Pylori in the stomach was present in 16 patients (45.7%), while in the mouth cultures were negative. These data show that the stomach is the main reservoir of H. Pylori.

This result is supported by histological examination, which showed the presence of H. pylori in 22 patients. According to our results, the oral cavity is not a reservoir of H. Pylori. Previous studies have failed to isolate the organism in the mouth on the basis of culture, while other works have isolated it at varying rates (Table I).

This divergence of results can be related to the intermittent passage of the bacteria in the oral cavity, or in the presence of oral bacteria inhibit the growth of H. pylori, namely Streptococcus mutans and Prevotella intermedia. Some authors also explain this negativity by converting bacillary forms in Coccicoides forms that are very difficult to identify by conventional culture buccal swabs.^[6, 7, 8]

Negativity of culture in our sample can be attributed to the small sample size, since on larger samples, the prevalence of H. pylori in the mouth is small, but the bacterium was isolated.

This demonstration is currently facilitated by the use of PCR, which allows the identification of viable and nonviable bacteria. According to the PCR tests, oral cavity may be a reservoir of H. Pylori particularly at the plaque.

Table 1: Literature data on the concomitant presence of *Helicobacter pylori*

Authors	sample	Urease test	Culture	PCR
Desai et al (1991; India)	43 dyspeptic patients	98 %	-	-
Bernander et al (1993; Suede)	94 dyspeptic patients	-	0%	-
Pytko-Poloczk et al (1996; Poland) ⁹	100 dyspeptic patients with endoscopically normal gastro-duodenal 55 patients with peptic ulcer	100 % 100%	88% 100%	- -
Berroteran et al (2002; Venezuela) ¹⁰	32 patients	-	-	37,5%
Umeda et al (2000; Japan) ¹¹	45 dyspeptic patients	-	-	40 %
Kim et al (2000; Korea) ¹²	46 patients	-	-	6,9%
Commarota (1996)	31 patients	-	-	3,2%
Kamat et al (1998; India)	156 dyspeptic patients 92 healthy patients	23,7% 22,8%	0% 0%	5,1% 4,3%
Oshowo et al (1998) ¹³	208 patients	-	0,009	7 %
Ozdemir et al (2001; Turkish) ¹⁴	81 dyspeptic patients	79%	-	-
Hu et al. (2002; China) ¹⁵	32 patients with chronic gastritis	84,4%	-	-

Whatever the role of the oral cavity (transient or permanent reservoir of *H. Pylori*), a support dental and periodontal case is essential in all patients with *H. Pylori* in the stomach. Prescribing antibiotics for the eradication of *H. Pylori* in the stomach, does not eliminate the bacteria in dental biofilm (plaque). Mechanical disruption of the latter by scaling and root planing is essential.

It is also worth noting that the practitioner dentist is at high risk of contamination by *H. Pylori*. This finding is in favor of the presence of *H. Pylori* in the oral cavity of patients with this germ in the stomach.

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Cite this article as: Regragui A, Benaouda A, EL Feydi EA, Abdedine A, Ennibi OK. *Helicobacter Pylori* in the dental plaque. *Int J Med and Dent Sci* 2014; 3(2):396-402.

Source of Support: Nil
Conflict of Interest: No

