Short Note

Oral H. pylori Infection

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During the past 25 years, there has been disagreement regarding the existence of *H. pylori* in the oral infection. It was proposed that no living *H. pylori* exists in the oral cavity and that the positive results detected by a polymerase chain reaction (PCR) in the oral cavity may be a fragment of *H. pylori*, instead of living bacteria, as part of a reflux from the stomach: *H. pylori* could not be cultivated from PCR-positive samples. The *H. pylori* comes from the stomach reflux only to survive in the oral cavity for a few hours because of the high oxygen concentration in the oral cavity. If the above proposed idea is correct, then the fragment or dead *H. pylori* should not have any negative effect on the drug eradication of *H. pylori* infections of the stomach [1-4].

However, the proposed idea contradicts with PCR studies, because there are a number of studies using PCR as the indicated research tool; they indicated PCR is a sensitive and reliable test for detecting oral *H. pylori* [5]. The proposed idea contradicts with the fact of oral hypoxia environment, because the subgingival plaque of the oral cavity has microaerophilic environments favorable for the growth of this bacterium, and *H. pylori* was detected in the supragingival plaque of individuals with *H. pylori* gastric diseases by a rapid urease test and real-time PCR analysis [4]. The proposed idea contradicts with eradication can’t eliminate oral *H. pylori* infection, because there are a number of studies that show when patients received drug treatment for stomach *H. pylori*, the drug did not eliminate oral *H. pylori*. Also, a study shows that mouth-rinse treatment alone or combined with periodontal treatment can, to some extent, reduce the prevalence of oral *H. pylori* and improve the eradication rate of gastric *H. pylori* [6,7]. The proposed idea contradicts with *H. pylori* can be cultured in the oral cavity. In the PCR-positive saliva sample, can *H. pylori* be confirmed by culture? The answer is yes! One study showed that *H. pylori* from a saliva sample can be cultured in individuals with all positive test results (HPS and HPF) of the oral cavity [5]. The proposed idea contradicts with the same original source of oral and stomach *H. pylori*. The remarkable genotype diversity among stomach, saliva, and stool samples showed that more than one *H. pylori* genotype may exist in the same patient [8]. The proposed idea contradicts with the fact of lower rate of eradication on stomach *H. pylori* when oral *H. pylori* positive. Miyabayashi et al. [9] found the eradication success rate was significantly lower in the oral *H. pylori*-positive cases (12/23, 52.1%) than in the negative cases (22/24, 91.6%) at 4 weeks after the therapy (p = 0.028). Two years later, only 16 of the 23 (69.5%) oral *H. pylori*-positive cases were disease-free, as compared to 23 of the 24 (95.8%) oral *H. pylori*-negative cases (p =018). The eradication efficiency in the stomach was 85.8% (187/218), while in the oral cavity it was only 5.7% (9/158) (OR = 55.59, P < 0.00001). *H. pylori* was more difficult to eradicate in the oral cavity than in the stomach, and may be a source of re-infection.

They concluded *H. pylori* in the oral cavity affected the outcome of eradication therapy and was associated with a recurrence of gastric infection and recommend that oral *H. pylori* should be examined by nested PCR and, if positive, should be considered a causal factor in refractory or recurrent cases. Our study show the efficacy rate of treatment on stomach *H. pylori* infection at 82.26% for patients received treatment of mouthwash combined with drug eradication; but only at 61.3% efficacy when patients received drug eradication on stomach. So treatment of oral cavity *H. pylori* raise about 20% efficacy when combined treatments of both mouth and stomach [5]. The proposed idea contradicts with Meta analysis. A meta-analysis published in 2011 indicated that the prevalence of *H. pylori* infection in the oral cavities of gastric *H. pylori*-positive patients was significantly higher than in gastric *H. pylori*-negative patients (45.0% vs 23.9%). The pooled OR was 3.61 and the 95% CI was 1.91-6.82 (P < 0.0001). Different diagnostic methods produced different pooled ORs with PCR the highest (OR = 5.11, 95% CI: 2.08-12.54, P = 0.0004) and the rapid urease test the lowest (OR = 2.00, 95% CI: 0.80-5.00, P = 0.14). The 44.8% (91/203) prevalence of *H. pylori* infection in the oral cavity in patients with clinical and/or histologic gastroesophageal diseases was significantly higher than the 13.2% (21/159) in patients with non-ulcerous dyspepsia or healthy controls (OR = 5.15, 95% CI: 2.97-8.92, P < 0.00001) [10]. The eradication efficiency in the stomach was 85.8% (187/218), while in the oral cavity it was only 5.7% (9/158) (OR = 55.59, P < 0.00001). *H. pylori* was more difficult to eradicate in the oral cavity than in the stomach, and may be a source of re-infection.

There are several reports indicated non-gut organs have been harbored of *H. pylori*, such as vagina [11], nasopharyngeal sinus cavities [12], coronary plaque [13], otitis media [14], breast [15]. However, further confirmation study should be follow up.

After review of studies in past 5 years, there are four important facts here we want emphasized in *H. pylori* colonized in oral cavity:

1. Urea Breath Test (UBT) is an agold standard for diagnosis of stomach *H. pylori*, but is not so for detection in the mouth. We found that UBT [13] has color blind that see *H. pylori* in the stomach, but can’t detecting *H. pylori* in oral cavity. In medical
practice, patients with negative results in UBT C [13] suggest that their stomach infection of *H. pylori* is cured. In fact, patients can present negative UBT results and yet exhibit *H. pylori* infection due to oral infection. The clinical study provides evidence that *H. pylori* oral infection is nonetheless present. In Asia, more than 90% of the population suffered from oral *H. pylori* infection but had negative UBT results [16]. This study also showed that oral antigen screening test could identify individuals who have no risk for *H. pylori* gastric infection. It further identified persons with no symptoms but with antigenic evidence of possible oral *H. pylori* infection who are thus at risk for developing gastric disease. This information was not provided by UBT methods [17].

2. Drug treatment on stomach *H. pylori* infection has no effective in *H. pylori* infection of oral cavity. *H. pylori* exists in between the teeth and gums called “bio- film membrane” (Bio film), also known as plaque barrier. It is resistance when the drug into this area. This is why conventional treatment for *H. pylori* eradication *H. pylori* infection, but is not efficacy of oral *H. pylori* in dental plaque [5]. Our study show the efficacy rate of treatment on stomach *H. pylori* infection at 82.26% for patients received treatment of mouthwash combined with drug eradication; but only at 61.33% efficiency when patients is received drug eradication on stomach. So treatment of oral cavity *H. pylori* raise about 20% efficacy when combined treatments of both mouth and stomach [5].

3. There is non-antibiotic treatment for oral *H. pylori* infection available. Our studies indicated e-polysylline (L) and the Glycerol Monolaurate (GM) used in mouth washing solution. The L is typically produced as a homo-polypeptide of approximately 25-30 L-lysine residues. The epsilon (e) refers to the linkage of the lysine molecules. In contrast to a normal peptide bond that is linked by an alpha carbon group, the lysine amino acids are molecularly linked by the epsilon amino group and the carboxyl group. L belongs to the group of cationic polymers. In water, L contains a positively charged hydrophilic amino group. It is adsorbed electro statically to the cell surface of the bacteria, followed by a stripping of the outer membrane. This eventually leads to the abnormal distribution of the cytoplasm, causing damage to the *H. pylori* cell. GM is the mono-ester formed from glycercol and lauric acid. *H. pylori* is extremely sensitive to GM, however there are no reports of L or GM killing *H. pylori* in vivo. Since both have had a safe record in the food industry, we use L-GM successfully eliminate *H. pylori* of oral cavity within 2 to 3 months. In China alone, more than 280 million people carry *H. pylori*, which results in 28 million recurrences of stomach *H. pylori* infection and the abuse of antibiotics by over use [18]. The massive antibiotic pollution that appears in food, water, and children’s urine has become a serious concern worldwide. Antibiotic abuse kills 80,000 Chinese people every year and leads to extra medical spending of 11.7 billion dollars across the country, which could become a global problem. This is why we recommended use non-antibiotic formula to take care on *H. pylori* in oral cavity.

4. There are three important technologies developing to make a strong foundation of a colonized site of the oral cavity. PCR is a high sensitivity test for oral *H. pylori*, but it is not convenient in clinical settings. So, first a high sensitivity and specificity test in saliva such as HPS test should be established. Then we will have a much easier time of running clinical trial on a large number of patients to obtain a greater number of data, to find the positive correlation between oral and stomach *H. pylori* infection [16]. Second most important technology is developing a cell culture suitable for low concentration *H. pylori* in the oral cavity. Because the concentration of *H. pylori* in stomach is 5 magnitudes higher than that of the oral cavity (10⁷ CFU/mL versus 10⁵ CFU/ML [19, 20]), it would be insufficient to use conventional stomach culturing techniques for detecting oral *H. pylori*. After the method of a new cell culture is established, we would confirm if HPS technology can be confirmed by cell culture data [9]. As a final step, we need to develop a technology to eliminate *H. pylori* from the oral cavity instead of an antibiotic drug.

REFERENCES


