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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 contradict 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 = 0028). 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 61.33% 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 reinfection.

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) C [13] is a gold standard for diagnosis of stomach *H. pylori*, but is not so for detection in the mouth. We found that UBT C [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 [5,9]. 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 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-polylysine (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 glycerol 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 oral *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 five magnitudes higher than that of the oral cavity (10^7 CFU/mL versus10^2 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

1. Marshall B. Speech at Helicobacter pylori infection of the Seventh National Forum of China. Beijing 2012.
2. Al-Ahmad A, Kürschner A, Weckesser S, Wittmer A, Rauberger H, Jakob T et al, Helicobacter pylori resident or transient in the human oral cavity?. J Med Microbiol. 2012; 61:1146-1152.
3. Al-Ahmad A, Hellwig E, Wittmer A, Kist M, Waidner B. Letter to the editor: re: The impact of the stone age diet on gingival conditions in the absence of oral hygiene. J Periodontol. 2010; 81: 338-339.
4. Lindén S. K, Wickström C, Lindell G, Gilshenan K, Carlstedt I. Four modes of adhesion are used during Helicobacter pylori binding to human mucins in the oral and gastric niches. Helicobacter. 2008; 13: 81-93.
5. Wang XM, Yee KC, Hazeki-Taylor N, Li J, Fu HY, Huang ML, et al. Oral Helicobacter pylori, its relationship to successful eradication of gastric H. pylori and saliva culture confirmation. J Physiol Pharmacol 2014; 65: 559-566.
6. Bharath TS, Reddy MS, Dhanapal R, Raj Kumar NG, NeeladriRaju P, Saraswathi T, et al. Molecular detection and corelation of Helicobacter pylori in dental plaque and gastric biopsies ofdyspeptic patients. J Oral Maxillofac Pathol 2014; 18: 19-24.
7. Song HY, Li Y. Can eradication rate of gastric Helicobacterpylori be improved by killing oral Helicobacter pylori?. World J Gastroenterol. 2013; 19: 6645-6650.
8. Momtaz H, Souod N, Dabiri H, Sarshar M. Study of Helicobacter pylori genotype status in saliva, dental plaques, stool and gastricbiopsy samples. World J Gastroenterol. 2010; 2105-2111.
9. Miyabayashi H, Furhata K, Shimizu T, Ueno I, Akamatsu T. Influence of oral Helicobacter pylori on the success of eradication therapy against gastric Helicobacter pylori. Helicobacter. 2000; 5: 30-37.
10. Zou QH, Li RQ. Helicobacter pylori in the oral cavity and gastricmucosa: a meta-analysis. J Oral Pathol Med. 2011; 40: 317-324.
11. Minakami H, Hayashi M, Sato I. Does Hp colonize the vagina of pregnant women?. J infect. 2000; 41:112-123.
12. Morinaka S, Ichimiya M, Nakamura H. Detection of Hp in nasal and maxillary sinus specimens from patients with chronic sinusitis. Laryngoscope. 2003; 113:1557-1563.
13. Kowalski M. Hp infection in coronary artery disease: influence of Hp eradication on coronary artery lumen after percutaneous transluminal coronary angioplasty. The detection of Hp specific DNA in juman coronary atherosclerotic plaque. J Physiol Pharmacol. 2001; 52:3-31.
14. Yilmaz T, Ceylan M, Akyon Y, Ozcakyr O, Gursel B. Hp: A possible

association with otitis media with effusion. Otolaryngol Head NeckSurg. 2006, 134:772-777.

15. Kast R.E. Some fibrocystic breast change may be caused by sexually transmitted H. pylori during oral nipple contact: Supporting literature and case report of resolution after gut H. pylori eradication treatment. Medical Hypotheses. 2007, 68:1041-1046.

16. Yee KC, Wei MH, Yee HC, Everett KD, Yee HP, Hazeki-Talor N, A screening trial of Helicobacter pylori-specific antigen tests in saliva to identify an oral infection. Digestion. 2013; 87: 163-169.

17. Yee JKC. Oral Cavity is Second Colonized Site Beside Stomach- a milestone discovery. World J Gastroenterolÿ. 2016; 22: 641-648.

18. Huang R, Ding P, Huang D, Yang F. Antibiotic pollution threatens public health in China. The Lancet. 2015; 385: 773-774.

19. Me´graud F, Lehours P. Helicobacter pylori Detection and Antimicrobial Susceptibility Testing. Clin Microbiol Rev, 2007; 22: 280322.

20. Song Q. Zirnstein GW, Swaminathan B, Gold BD. Pretreatment with Urea-Hydrochloric Acid Enhances the Isolation of Helicobacter pylori from Contaminated Specimens. J Clin Microbiol. 2001; 39: 19678.

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