

B. Helicobacter pylori, A Sex Transmitted Bacteria?

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Since oral sex is a very common sexual activity and recent evidence reported *H. pylori* exist in oral cavity as colonized site. Both facts indicated *H. pylori* may results sex transmitted disease such as vagina, breast and urethritis, however, further clinical studies and lab confirmation should be follow.

Helicobacter Pylori (*H. pylori*) passes through the mouth on its way to colonizing the stomach, where chronic infection is associated with ulcers, gastritis, and gastric adenocarcinoma. *H. pylori* are the only proven oncogenic bacterial species and detecting, preventing, or curing infection in the early stages is essential if gastric disease is to be prevented. However, beside of stomach *H. pylori* infection, there are several reports indicated non-gut organs have been harbored of *H. pylori*, such as vagina [1], nasopharyngeal sinus cavities [2], coronary plaque [3], otitis media [4], breast [5]. Now it is time we should answer a question; is *H. pylori* a sex transmitted bacteria?

Seroprevalence studies have shown that in sex partners with a man/woman who is infected with *H pylori* the non-infected individual has an increased risk of transmitting the infection. Studies have shown that prevalence rates were statistically significant between couples with and without *H pylori* infection (83.3% v 28.5%) respectively [6,7].

Ethnicity may also be an important predictor of sexual transmission of *H pylori* infection. A number of studies have shown that the highest rates of sexually transmitted infections occur in ethnic minorities. The high prevalence of sexually transmitted infections correlates well with the high *H pylori* prevalence rates that exist among these ethnic groups [8,9].

Molecular studies have produced evidence of *H pylori* transmission between spouses. Schutze et al [7] found that re-infection had been caused by the same *H pylori* strain and identified the spouses of the patients as carriers of the identical strain. This was supported by other studies. Moreover, it has been shown that multiple strains of *H pylori* may infect the same individual.

There was very limited research published in association of sex transmitting disease with *H. pylori*. For example, Eslick GD [10] report no study conducted in the prevalence of *H pylori* infection is increased in female sex workers when compared with the general population by met analysis.

H. PYLORI INFECTION TRANSMITTED SEXUAL VIA ORAL-GENITAL CONTACT

There have been many reviews that have been published looking at possible transmission; however, few published papers have examined the possibility of sexual transmission via the vagina. Several theoretical links exist such *H. pylori* has been shown to colonize yeast within the vagina and has also been associated with biofilm formation, making it possible that *H. pylori* is one of many bacterial species seen in biofilms present in bacterial vaginosis leading to treatment failure. *H. pylori* are commonly found in the stomach and upper gastrointestinal tract, one of the few bacterial able to colonize this acidic environment. Eslick hypothesized that *H. pylori* may be able to also colonize the acidic vaginal environment, acting as a reservoir and allowing sexual transmission of the bacteria. *H. pylori* isolates have also been found to have an end symbiotic relationship with *Candida albicans*, with bacterial isolated from within this yeast. This relationship may allow *H. pylori* to colonize the vagina. *Candida* isolated from the vagina has been found to contain *H. pylori* specific genes, one mechanism for the vertical transmission of *H. pylori*. The colonization of the high rates of treatment failure and infection recurrence in some women with bacterial vaginosis [1].

A few studies have been conducted in an attempt to isolate *H. pylori* from the vagina. Early attempts produced negative results. Recently, de-Argila et al conducted a study which attempted to find *H. pylori* in vaginal secretions by taking vaginal brushings and using Polymerase Chain Reaction (PCR), culture, and Gram stain. However, the diagnostic methods used failed to detect *H. pylori*. This may be because the concentration of *H. pylori* in vagina is low. The technology of culture based on stomach where the concentration of *H. pylori* is very high.

An early case report which was published a year before Warren and Marshall's original paper on "spiral bacteria in the stomach" may have found strains of *H. pylori* in a woman's vagina

associated with vaginitis. The article reports finding comma-shaped rods (1-4 μm in length), with a characteristic corkscrew motility having between four and eight flagellae. Some of the organisms were cultured under microaerophilic conditions and cultured after 72 hours' incubation at 37°C. The biochemical profile of these unknown organisms matched very closely with that of *H. pylori*, although definitive tests such as urease activity were not undertaken. Could this unknown organism have been a *H. pylori* species? Vertical transmission may also occur during birth if *H. pylori* are present in the vagina. Studies have shown that the prevalence of *H. pylori* in pregnant women is about 20% [11,12]. The question remains, why hasn't a *H. pylori* species been recovered from the female vagina when so many vaginal swabs have been done to culture organisms like *Neisseria gonorrhoea*? Several reasons may be that *H. pylori* species are difficult to grow in culture that key difficulties in cultivating oral *H. pylori* result from oral specimen collection, preservation, small colonies of *H. pylori* culture, and competition with other oral bacteria and *H. pylori* colonies. Because the concentration of *H. pylori* in stomach is three magnitudes higher than that of the oral cavity (10⁵ CFU/mL versus 10² CFU/mL [13,14]), it would be insufficient to use conventional stomach culturing techniques for detecting oral *H. pylori*. The method must be adapted to obtain a high positive rate of oral *H. pylori* culture.

H. PYLORI INFECTION TRANSMITTED SEXUAL VIA ORAL-BREAST CONTACT

Kast RE report a case that oral contact with the nipple may result in retrograde propulsion of *H. pylori* into breast ducts that leading fibrocystic breast changes which is a heterogenous group of benign. The woman had *H. pylori* serology was negative. After antibiotic eradication, her breasts normalized and pain and tenderness stopped which leading to this hypothesis [5].

H. pylori in fecal were seen in half of all breastfed 3-day old neonates whose mothers have documented *H. pylori* antigenuria [15], *H. pylori* was found in 4 out of 66 milk samples from parturient [16].

H. PYLORI INFECTION TRANSMITTED SEXUAL VIA ORAL-ORAL CONTACT

Overall, inadequate sanitation practices, low social class, and crowded or high-density living conditions seem to be related to a higher prevalence of *H. pylori* infection due to oral-oral contact. The poor hygiene and crowded conditions may facilitate transmission of infection among family members and is consistent with data on intrafamilial and institutional clustering of *H. pylori* infection. Understanding the route of *H. pylori* transmission is important if public health measures to prevent its spread are to be implemented. For the general population, the most likely mode of transmission is from person to person, by either the oral-oral route (through vomitus or possibly saliva). The person-to-person mode of transmission is supported by the higher incidence of infection among institutionalized children and adults and the clustering of *H. pylori* infection within families. Also lending support to this concept is the detection of *H. pylori* DNA in vomitus,

saliva, dental plaque, gastric juice, and feces. Waterborne transmission, probably due to fecal contamination, may be an important source of infection, especially in parts of the world in which untreated water is common.

Dye BA et al. report that a total of 4504 participants who completed a periodontal examination and tested positive for *H. pylori* antibodies that show periodontal pockets with a depth of 5 mm or more were associated with increased odds of *H. pylori* seropositivity (odds ratio [OR]=1.47; 95% confidence interval [CI]=1.12, 1.94). The conclusion was that poor periodontal health, which is characterized by advanced periodontal pockets, could be associated with *H. pylori* infection in adults [17]. Fernández-Tilapa G et al. found that the prevalence of *H. pylori* in the oral cavity was higher among seropositive subjects than seronegative ones [18]. Furthermore, Nisha KJ et al. reported that there is a highly significant association between periodontal disease and the colonization of *H. pylori* in dental plaque [19]. Tsami A et al. detected *H. pylori* in subgingival dental plaque of children and their family [20].

Several reports have indicated that *H. pylori* colonies could be grown only from root canals but not from plaque. The root canals of endodontic-infected teeth could be a reservoir for live *H. pylori* that could serve as a potential source of transmission [21,22].

If the discover of oral cavity is second colonized site beside stomach has been established [23], then saliva may contains *H. pylori* that can be a risk of *H. pylori* infection transmitted sexual via oral-oral contact such as wet kiss.

H. PYLORI INFECTION TRANSMITTED SEXUAL VIA INTERCOURSE

H. pylori infection transmitted sexual may via intercourse that depends both of sex parties may have oral sex action or not. If saliva contains *H. pylori* that can transmitted to vagina. The intercourse can be a risk that *H. pylori* can cost urethritis.

H. PYLORI INFECTION TRANSMITTED SEXUAL VIA ORAL-ANAL

The possibility of sexual transmission via the vagina in adults would contribute, in part, to the low rates. Previous data have suggested that sexual behaviour may be important in the transmission of *H. pylori*. The majority of these studies have concentrated on the possibility of oro-anal transmission between male homosexuals.

H. PYLORI INFECTION AND URETHRITIS

The idea that *H. pylori* or another species of Helicobacter could cause urethritis has never before been proposed. There have been three conflicting studies conducted to determine if sexual contact plays any role in the transmission of *H. pylori* oral sex is one of the most common sexual practices in the world and it is possible that *H. pylori* could be transmitted via the act of fellatio to the urethra leading to infection. This organism may be the 'missing link' in explaining the large proportion of males with non-gonococcal urethritis where no other responsible organisms can be isolated. This is the first article to suggest a link between *H. pylori* infection and urethritis [24].

Oral yeasts were isolated more frequently from normally-delivered neonates. The frequency of *H. pylori* genes in mothers' vaginal yeasts was significantly higher than in mothers' oral yeasts. A significant correlation was found between the occurrence of *H. pylori* genes in vaginal yeasts and that in neonates' oral yeasts, occurrence of *H. pylori* genes in mothers' vaginal yeasts or neonates' oral yeasts, and UBT+ results in mothers. Calbicans which colonizes the oral cavity of neonates through vaginal delivery or contact with environment or healthcare workers could be an important reservoir of *H. pylori*. Vaginal yeasts are more potent in accommodating *H. pylori* than oral yeasts. Accordingly, vaginal yeast is proposed as the primary reservoir of *H. pylori* which facilitates *H. pylori* transmission to neonates [22].

Oral sex (fellatio) is a very common sexual activity. *H. pylori* is mainly a gastric organism, but studies have reported that infected individuals may permanently or transiently carry *H. pylori* in their mouth and saliva [22-24]. The existing studies support the hypothesis that *H. pylori* could be a causative agent of non-gonococcal urethritis. It is possible that *H. pylori* may be transmitted via the act of fellatio in the urethra. Further research is required to explore the role of *H. pylori* in sexually transmitted urethritis [25].

CONCLUSION

Since the evidence of oral *H. pylori* had been established, oral sex is a very common sexual activity. The risk of *H. pylori* infection transmitted sexual via oral contact, breast, and prostate vagina infection may exist.

References

1. Minakami H, Hayashi M, Sato I. Does Hp colonize the vagina of pregnant women? J infect. 2000; 41: 112-113.
2. 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.
3. 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 human coronary atherosclerotic plaque. J Physiol Pharmacol. 2001; 52: 3-31.
4. 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.
5. 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.
6. Singh V, Trikha B, Vaipheh K, Nain CK, Thennarasu K, et al. *Helicobacter pylori*: evidence for spouse-to-spouse transmission. J Gastroenterol Hepatol. 1999; 14: 519-522.
7. Schutze K, Hentschel E, Dragosics B, et al. *Helicobacter pylori* reinfection with identical organisms: transmission by the parents' spouses. Gut. 1995; 36: 831-833.
8. Zenilman JM. Ethnicity and sexually transmitted infections. Curr Opin Infect Dis. 1998; 11: 47-52.
9. Mollison LC, Lecons RJ, Thein-Htut, Rajabalendran N, Perera C. Upper gastrointestinal endoscopy in central Australian aborigines. Med J Aust. 1994; 160: 182-184.
10. Eslick GD. Hp infection transmitted sexual via oralgenital contact: a hypothetical mode. Sex Tran. 2000; 76: 489-492.
11. Blecker U, Lanciers S, Keppens E, Vandenplas Y. Evolution of *Helicobacter pylori* positivity in infants born from positive mothers. J Pediatr Gastroenterol Nutr. 1994; 19: 87-90.
12. Yan P, Eslick GD, Xia HH-X, H. Murray, B. Spurrett, N.J. Talley. Association between *Helicobacter pylori* infection and fetal intrauterine growth retardation (IUGR). Gastroenterology. 2000; 118: A734.

13. Me'graud. F and Lehours.P. *Helicobacter pylori* Detection and Antimicrobial Susceptibility Testing. Clin Microbiol Rev. 2007; 20: 280-322.
14. 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: 1967-1968.
15. Fujimura S, Kato S, Nagai K, et al. Detection of Hp in the stools of newborn infants. Pediatr Infect Dis J. 2004; 23: 1055-1056.
16. Kitagawa M, Natori M, Kato M, Sugimoto K, Omi H, Akiyama, et al. Maternal transmission of Hp in the period. J Obstet Gynaecol Res. 2001; 27: 225-230.
17. Dye BA, Kruszon-Moran D, McQuillan G. The relationship between periodontal disease attributes and *Helicobacter pylori* infection among adults in the United States. Am J Public Health. 2002; 92: 1809-1815.
18. Fernández-Tilapa G, Axinecuilteco-Hilera J, Giono-Cerezo S, Martínez-Carrillo DN, Illades-Aguiar B, Román-Román A. vacA genotypes in oral cavity and *Helicobacter pylori* seropositivity among adults without dyspepsia. Med Oral Patol Oral Cir Bucal. 2011; 16: e175-180.
19. Nisha KJ, Nandakumar K, Shenoy KT, Janam P. Periodontal disease and *Helicobacter pylori* infection: a community-based study using serology and rapid urease test. J Investig Clin Dent. 2014; 7.
20. Tsami A, Petropoulou P, Kafritsa Y, Mentis YA, Roma-Giannikou E. The presence of *Helicobacter pylori* in dental plaque of children and their parents: is it related to their periodontal status and oral hygiene? Eur J Paediatr Dent. 2011; 12: 225-230.
21. Hirsch C, Tegtmeyer N, Rohde M, Rowland M, Oyarzabal OA, Backert S. Live *Helicobacter pylori* in the root canal of endodontic-infected deciduous teeth. J Gastroenterol. 2012; 47: 936-940.
22. Ogaya Y, Nomura R, Watanabe Y, Nakano K. Detection of *Helicobacter pylori* DNA in inflamed dental pulp specimens from Japanese children and adolescents. J Med Microbiol. 2015; 64: 117-123.
23. Yee J.K.C. Oral Cavity is Second Colonized Site beside Stomach- a milestone discovery. World J Gastroenterol. 2016; 22: 641-648.
24. Guy D. Eslick. Non-gonococcal urethritis,*Helicobacter pylori* infection and fellatio: a new me´ nage a` trois? Microbiology Comment. 2001; 150: 520-522.
25. Siavoshi F, Taghikhani A, Malekzadeh R, Sarrafnejad A, Kashanian M, et al. The role of mother's oral and vaginal yeasts in transmission of *Helicobacter pylori* to neonates. Arch Iran Med. 2013; 16: 288-294.
26. Yee KC, Wei MH, Yee HC, Everett KD, Yee HP, et al. A screening trial of *Helicobacter pylori*-specific antigen tests in saliva to identify an oral infection. Digestion. 2013; 87: 163-169.
27. Wang XM, Yee KC, Hazeki-Taylor N, Li J, Fu HY, 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.
28. Dimitriadi D. *Helicobacter pylori*: a sexually transmitted bacterium? Cent European J Urol. 2014; 67: 407-409.