Helicobacter pylori in dental plaque and gastric mucosa: Correlation revisited

Saima Chaudhry[1], Hafiz Aamer Iqbal[2], Ayyaz Ali Khan[3], Mateen Izhar[4], Arshad Kamal Butt[5], M. Waheed Akhter[6], Faisal Izhar[7], Kamran Masood Mirza[8]

Department of Oral Health Sciences[1,7,8], Shaikh Zayed Federal Postgraduate Medical Institute, Department of Oral and Maxillofacial Surgery[2], SIMS/Services Hospital, Department of Oral Health Sciences[3], Department of Microbiology[4], Department of Gastroenterology[5], Shaikh Zayed Federal Postgraduate Medical Institute, School of Biological Sciences[6], University of the Punjab, Lahore.

Abstract

Helicobacter pylori (H. pylori) related gastric infection is highly prevalent in developing countries. Prevalence of bacterium in dental plaque from these regions is also reported to be high, but association between simultaneous colonization of H. pylori in both these sites has not been established yet. Aim of this paper is to review possible association between simultaneous oral and gastric H. pylori colonization in dyspeptic patients. Pertinent literature was reviewed and all available evidence collected from Medline and PakMedinet. Studies conducted in the developing world show conflicting results. Some report a positive relation between oral and gastric H. pylori colonization while others deny any association. This may be due to the population sampled or methodology applied. Further studies are recommended to confirm the association between concurrent presence of H. pylori in dental plaque and gastric mucosa of dyspeptic patients using sensitive and specific tests for detection of bacterium in oral samples.

Introduction

The presence of Helicobacter pylori (H. pylori) in humans is recognized as a chronic infection, which in most cases persists indefinitely.[1] H. pylori is a gram negative, motile, microaerophilic, rod-shaped bacterium that colonizes the human stomach. It lives beneath the gastric mucous layer adjacent to gastric epithelial cells.[2] It is a major human gastric pathogen responsible for gastritis and peptic ulcer disease.[3] It has also been designated a type I, or definite, carcinogen by World Health Organization, as it is associated with the development of gastric adenocarcinoma, the second most commonly diagnosed fatal cancer worldwide.[4] H. pylori is also associated with gastric MALTomas.[3]

Prevalence

Approximately 50% of the world's population is affected by gastric H. pylori infection.[5] Infection is significantly more prevalent in developing countries where reported prevalence in adult population is around 90%[6] as compared to less than 40% in developed nations.[7] Asians carry higher prevalence of H. pylori infection[8] with large inter-country variation. Infection is more frequent in less developed countries like Pakistan[6], India[9] and Bangladesh[10], as compared to Japan[11] and China.[12] H. pylori infection is very common in Pakistan with infection rates reported to be as high as 90% in adult population.[6] The exposure rate in children is around 33%[13] with infection rates of 67% in infants[14] and 30% in children under fifteen years of age.[15]

Route of transmission

Exact mode and route of transmission of H. pylori is still unknown. Faecal-oral and oral-oral routes are generally accepted as the most probable ones.[16] The bacterium is believed to be transferred through person to person contact occurring more in close contacts and in situations of poor social hygiene.. Overall, inadequate sanitation practices, low social class, and crowded living conditions are related to high prevalence of H. pylori infection.[17]

Extra gastric ecological niche

H. pylori is known to resides in human stomach. Other possible extra gastric ecological niche for H. pylori being evaluated recently is the oral cavity.[18] Human oral cavity has a complex flora of 350 different species. Presence of carbohydrate fermenting organisms and an optimum oral temperature of 35-37^{o}C provides an ideal environment and a vast static area of dental plaque (collection of microorganisms forming soft debris, covering the exposed tooth surface) for growth of H. pylori.[19]

The bacterium has been detected in saliva[20], in the microbiota from the dorsum of the tongue[21], on the surface of oral ulcerations[22], oral neoplasia[23] and in dental plaque.[24] High rates of H. pylori detection in dental plaque and saliva suggest that the oral cavity might be an important reservoir of the bacterium which can lead to infection in the stomach.[16] Kamada et al[25] reported three cases of H. pylori related acute gastric mucosal lesions following dental treatment in all patients. Furthermore, dental plaque has been implicated as a possible source of gastric reinfection after apparently successful gastric eradication as H. pylori eradication therapy successfully removes the bacterium from stomach but not from dental plaque.[19]

Detection rate in dental plaque

Rate of detection of H. pylori in dental plaque also varies among studies conducted on different populations with very low figures in Western countries[26] while extremely high rates have been reported from developing nations around the world[27], including Pakistan.[28]

Correlation of H. pylori in dental plaque and gastric mucosa

H. pylori related gastric infection is highly prevalent in developing countries.[6] Prevalence of bacterium in dental plaque from these regions is also reported to be high,[28,29] but correlation between simultaneous oral and gastric H. pylori colonization in dyspeptic patients has not been yet. established Studies done so far to find association between oral and gastric H. pylori infection have revealed conflicting results. Some report a positive relation[19,28,30-32] while others deny any association.[33,-35] Studies from Iran,[33] Brazil,[34] and Venezuelan Andes[35] have found no relation between the two, while those from Pakistan[19,36], India,[32] and Nigeria[31] have reported a positive association. A study from Venezuvela[30] also reported positive correlation between oral and gastric H. pylori. Variations in prevalence of infection in stomach among sampled populations and methods used for detection of the bacterium may account for conflicting results among studies.[16]

Two studies have been conducted so far in Pakistan addressing this issue. Butt et al reported it to be 100% in dental plaque of patients with symptomatic dyspepsia.[19] Another study reported majority of Pakistanis to have possible H. pylori colonization in dental plaque while about two-thirds had H. pylori associated chronic active gastritis. The researchers implicated that oral cavity may be the first place for colonization and then infection involves the gastric mucosa.[36]

Problems with Helicobacter pylori detection in Oral Samples

There are several popular methods for detection of Helicobacter pylori infection. Detection of H. pylori infection in gastric biopsy specimens by urease test, histology, culture and polymerase chain reaction (PCR) is well documented; however detection of this bacterium in the oral cavity is a topic of considerable debate.[16]

Detection of Oral H. pylori is quite complex. Oral cavity is residence to several urease-producing species, including Streptococcus, Haemophilus species, and Actinomyces species. It is inappropriate to conclude that high urease activity in dental plaque indicates presence of

H. pylori.[16] Histology, where H. pylori appear as Gram-negative, curved or spiral-shaped rods, provides low specificity in the case of oral samples, where spirochetes, including Treponema species, are present.[16] Culture of oral H. pylori is more sensitive than other methods but has not met with much success. While the bacterium has been isolated from dental plaque and saliva samples, there is a potential for false-positives and detection rates have been consistently low. A difficulty in culturing the bacterium from oral samples is presence of a viable coccoid form that is unculturable by conventional techniques. While this coccoid form is not associated with disease, it reverts to the infectious rod-shaped form under appropriate conditions, and so its presence can still be considered clinically significant.[16]

PCR based assays have proved to be promising tools for detecting H. pylori in dental plaque samples, but the sensitivity and specificity of the assay varies with the type of buffers and primers used and the detection method applied. Nested PCR using EHC-U/EHC-L primers, which amplifies a DNA fragment of 417 bp homologous to a DNA fragment of H. pylori of 860 bp, has been shown to carry highest sensitivity and specificity for detection of bacterium in oral samples.[37]

Conclusion

Studies conducted so far to find correlation between oral and gastric H. pylori infection in developing countries are not in full agreement and need to be treated with reservations as the methodology applied is not sensitive and specific enough for detection of bacterium in plaque samples. Some researchers have done rapid urease test for detection of bacterium in plaque samples whereas others have performed culture. Few studies conducted have done PCR on oral samples but the primers used in these studies are not sensitive and specific for H. pylori detection. Furthermore, studies done in the developing world have not performed nested PCR, using EHC-U/EHC-L primers, on plaque samples to find the correlation between oral and gastric colonization.

Future Research

The correlation between the presence of H. pylori in dental plaque and gastric mucosa of patients with symptomatic dyspepsia is still not clear. Future research should aim at confirming this association using highly sensitive and specific PCR assays for diagnosis of Helicobacter pylori in dental plaque samples.

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