Review Article

Helicobacter pylori and its role in oral diseases: A note on pathogenesis

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ABSTRACT

Helicobacter pylori belongs to a family of bacteria that colonize the mammalian stomach present in half of the world's population. It has been causally linked to chronic gastritis, peptic ulceration, and gastric adenocarcinoma. This review is about the pathogenesis and oral-related lesions associated with *H. pylori* infection.

Key words: Aphthous ulcer, gastrointestinal tract, *Helicobacter pylori*, oral lesions

INTRODUCTION

Helicobacter pylori (*H. pylori*) previously called '*Campylobacter pylori*' is a gram-negative, microaerophilic bacteria present in the gut. It was first isolated by Marshal and Warren, which ushered in a new era in gastric microbiology.[1] *H. pylori* belongs to a family of bacteria that colonize the mammalian stomach, and is causally linked to chronic gastritis, peptic ulceration, and gastric adenocarcinoma.[2] This bacterium colonizes over half of the world's population. Infection is usually acquired in childhood and in the absence of antibiotic therapy, persists for lifetime of the host. *H. pylori* is the only known bacteria that can persistently colonize the normal stomach, and is able to tolerate the harsh conditions of the stomach.[3]

Dental plaque is recognized as a reservoir of *H. pylori* by some researchers. It is considered to be a normal commensal by some, though being difficult to isolate due to the meager presence. There isevidently an increase in the number of oral *H. pylori* in chronic gastritis patients. Further, *H. pylori* can promote the development of oral mucosal lesions likerecurrent aphthous ulcer and may function as an intermediary in the route of oral to gastric infection.[4] *H. pylori* infection contributes to the pathogenesis of periodontal diseases, recurrent aphthous ulcers, glossitis, burning mouth syndrome, and some dermatological diseases.[5]

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Risk factors for *H. Pylori*

The risk factors for *H. pylori* include poor social economic status, poor hygiene practice, absence of hygienic drinking water, and unsanitary food preparation.[6,7]

Transmission of *H. Pylori*

The main route of organism entry has been charted as the following — oral to oral, gastro to oral, and fecal to oral. Transmission may occur in a vertical or horizontal mode. Oral carriage of *H. pylori* may play a role in the transmission of infection.[7]

Pathogenesis

H. pylori is well adapted to withstand low pH to gain entry to its preferred territory, the mucus layer of the mucous membrane. One of the survival capabilities is its ability to resist mucosa and migration towards epithelial cell. Once there, the bacterium resists the local and systemic immune responses. Colonization persists for life in the host if there is no exposure to antibiotics.[5] Once it escapes from the lumen, it modifies the bioenvironment in the area and starts releasing collagenases which degrade the collagen in the host enabling more space for the movement of the bacteria. The Fas Ag pathway of apoptosis is activated during *H. pylori*

infection. The combined action of the collagenases and stimulation of apoptosis leads to ulceration of the mucosa.[8]

Oral lesions associated with *H. Pylori*

Recurrent aphthous stomatitis

Recurrent aphthous stomatitisis one of the most common oral mucosal conditions presenting as recurrent, multiple, small, round ulcers, with circumscribed margins, having yellow or gray floors, and are surrounded by an erythematous halo. The predisposing factors implicated include genetics, trauma, tobacco, drugs, hematinic deficiency, celiac disease, inflammatory bowel disease, hormonal changes, stress, and microorganisms.[9]

Recently, *H. pylori* has been proposed to be one of the important etiological agents in the pathogenesis of recurrent aphthous stomatitis.[10] It has been showed that patients with recurrent oral ulcerations appear to suffer from active *H. pylori* infection in a high percentage of cases.[11] Surprisingly, eradication of *H. pylori* may have a reducing effect on the recurrence and healing period of recurrent aphthous stomatitis. Studies by Meleki *et al*., and Fritscher *et al*., have proved contradictory to the same.[12]

Periodontitis

Dental plaque typically forms on the supragingival and subgingival tooth surfaces lacking ingood oral hygiene measures. Plaque biofilms can enhance the survivability of some bacteria by providing access to urea reduced by urease-producing microorganisms and neutralizes the effects of acidification. Moderate and severe cases of periodontitis have shown a positive seropositivity to *H. pylori*.[3] Riggio and Lennon concluded from a study that subgingival plaque, particularly when the pocket depth is more than 5 mm in adult periodontitis cases, may function as a reservoir for *H. pylori*.[13,14] The inflammatory markers produced by *H. pylori* in the oral cavity further addsto the burden on the preexisting inflammation due to chronic periodontitis in the oral cavity. Conclusively, *H. pylori* is mainly associated with moderate to severe cases of periodontitis, and its role in the contributionto gastric reinfection needs to be substantiated by further research.[4]

Lichen planus

Lichen planusis a relatively common, chronic dermatomucosal disease that often affects the oral mucosa. Lichen planus is an autoimmune disease with a female predilection. Hamideh *et al*., found that levels of *H. pylori* using urease breath test was significantly higher in lichen planus patients than in controls.[15] These results support a definitive etiological role for *H. pylori* in lichen planus, which is in line with study done by Sikander *et al*. On the contrary, a couple of research findings were totally disagreeing with the concept of any relationship of lichen planus with *H. pylori*.[16]

Psoriasis

Psoriasis is an auto immune inflammatory disease that is often associated with comorbidities such as dyslipidemia, diabetes mellitus, obesity, and cardiovascular disorders. Studies done by Ana *et al*.,[17] Ali *et al*.,[18] Halasz,[19] and Ghada *et al*.,[20] have shown a close association between psoriasis and *H. pylori*. Further, there has been evidence of psoriatic lesions clearing following eradication of *H. pylori*. In a largest study, *H. pylori* showed positive results for moderate to severe cases of psoriasis. Hence, it can be concluded that although no clear association between the two, testing for presence of *H. pylori* can enhanceviable treatment option for psoriasis.

CONCLUSION

Thus, there is increasing evidence that the oral cavity acts as a reservoir and may also aid in the transmission of *H. pylori*. However, further studies have to be performed using controlled conditions, larger samples, and latest diagnostic tests to substantiate the role of this bacterium. Understanding the mechanisms of action of these bacteria offers the exciting hope of newer treatment options for the above diseases.

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