HELICOBACTER PYLORI & ORAL CAVITY INFLAMMATION

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ABSTRACT

Nowadays there are multiple studies suggesting that inflammation of the oral cavity caused by bacteria, Microbial and fungi is accompanied by gastric inflammation. Helicobacter pylori infection is considered as one of the most common infectious diseases throughout the world, the carriage rate of Helicobacter pylori is reported to be 20-80% for adults in the developed world, and more than 90% in the developing world. It is also associated with peptic ulcer and gastric carcinoma and specially a cofactor in the instance of happening again of aphthous ulceration, gastric colonization and mucosal attachment. Current studies indicate that H. pylori is present in dental plaque, although the number of organisms in individual samples is very low, and these numbers appear to vary from one site to another within the mouth. The presence of this organism in plaque may be fitful, perhaps occurring as the result of gastroesophageal reflux. In our study the most important finding was that patients with recognized inflammation in the oral cavity in the form of stomatitis prothetica hyperplasica.

Keywords: Oral cavity, Helicobacter pylori, Cofactor.

1. INTRODUCTION

An initial portion or the gate of the gastro-intestinal tract (GIT) is Oral cavity, many diseases of oral cavity may affect the integrity of mucous cover of oral activity and the remaining portions of the GIT. It also associated with the changes in oral biology, including its microbial and bacterial and fungal colonization and then infection in the oral cavity. Oral cavity can have serve as a reservoir of those microorganisms and the source of the infection of the stomach and the gut, or alternatively, it may serve as the transmission gate of external germs for further colonization of GIT. The aim of the present study was the critical analysis of the literature the relationship between the oral cavity and Helicobacter Pylori. Approximately 50% of the world’s population is believed to be infected [1]. Developed countries typically have a lower prevalence of Helicobacter pylori infection at all ages, but this difference is especially noticeable among younger people [2]. Most infections are probably acquired in childhood, although the exact route of transmission is unknown. These bacteria are also found in plaque and feces, so the route of infection could be oral–oral or fecal–oral. Dental plaque is a soft gelatinous mass adhering to the tooth surface and hard surfaces and comprising of bacteria embedded in an organic matrix. After isolation of helicobacter pylori form dental plaque, controversies regarding the existence of this bacterium in the dental plaque as a probable secondary reservoir for it have been never ceased [3,4]. While the presence of helicobacter pylori in dental plaque was a common finding in some studies [5], it was not detected in others as frequently[6].

2. DISCUSSION

Helicobacter pylori is a microaerophilic, Gram negative, spiral and mobile bacterium which is believed to be one of the major factors responsible for gastritis, gastroduodenal ulcers as well as gastric cancer [7]. Fourty Patients (16 men and 24 women; mean age, 24 years; range,8 to 45 years) were included in this study. There are different opinions concerning the presence of Helicobacter pylori in the oral cavity. Some authors[8] have suggested that Helicobacter pylori may belong to the normal oral flora of the human oral cavity, maintaining a commensal relation with the host, but present in very low numbers such that reliable identification is difficult. Others[3] have suggested that Helicobacter pylori is not consistently present in dental plaque and, when present, may be the result of
occasional gastroesophageal reflux. Young and others[9] found no morphological differences in Helicobacter Pylori cells obtained from gastric biopsy and dental plaque and examined by scanning electron microscopy, both rod and coccoid forms were seen. The findings of the present study show the higher prevalence of Helicobacter pylori infection in the dental plaque in male subjects. Similar findings, showing higher and more consistent prevalence of helicobacter pylori infection in male sex, have been reported previously[10]. We have to admit, however, that the detection of Helicobacter pylori in the oral cavity does not necessarily provides an evidence that the oral cavity is the reservoir of bacteria for the further parts of GIT. Certain number of viable bacteria is required for successful infection of gastric mucosa and the bacteria may be present in the oral cavity in the number too low to infect gastric mucosa after passing into the stomach with saliva or swallowed food. It was shown for the first time by B. Marshall, who drank the pure culture of Helicobacter pylori, that caused an immediate acute hemorrhagic and erosive gastritis confirmed by gastroscopy but no ulcer developed. That was the proof that the oral cavity may serve as the gate for the transmission of the Helicobacter pylori to the stomach.

Helicobacter in Oral Cavity:
Birek and others[11] determined the frequency of detection of Helicobacter Pylori DNA in oral samples from recurrent aphthous ulcers (RAUs), tongue, saliva and plaque. A total of 71.9% of the RAU samples exhibited Helicobacter Pylori DNA. The results for plaque and saliva indicated that these were not likely sources of Helicobacter pylori in healthy individuals. Birek and others[12] raised the possibility that adherence of Helicobacter pylori to the oral cavity and subsequent production of autoantibodies to epitopes shared by oral epithelium cells and Helicobacter Pylori might result in the tissue destruction associated with RAU. Because of similarities in the inflammatory process that produces gastritis associated with Helicobacter Pylori and that causes RAU, they postulated that Helicobacter Pylori may be a cofactor in the pathogenesis of RAU, especially in people sensitized through gastric colonization and mucosal attachment. Riggio and others[11] who also used PCR analysis, detected Helicobacter Pylori DNA at a much lower frequency (11%) from RAU biopsy samples. They suggested that this finding could result from variation within different patient groups. They concluded that their results did not support a definitive causative role for Helicobacter Pylori in RAU, although the possibility that this organism may be involved in a small proportion of RAU cases could not be excluded.

3. CONCLUSION
Helicobacter Pylori is a micro-organism that is associated with a Microbial and Bacterial infection. This infection responds to treatment by antibiotic, although in some patients the infection can be difficult to eradicate and there is a significant rate of recurrence. Whether the rate of recurrence can be reduced by concomitant emphasis on improving oral hygiene and treating periodontal disease remains to be clarified. We found that there is no gastric Helicobacter pylori infection without Helicobacter pylori presence in the oral cavity. Previous studies have suggested that the oral cavity might be a reservoir for H. pylori and the source of infection/reinfection after eradication of gastric infection and transmission. Having looked for the presence of H. pylori in dental samples, saliva, these studies again showed extreme variations in findings. For example, the detection rate of H. pylori by PCR assay from dental plaque and saliva samples varied from 0% to 87%. In the present study we were able to detect H. pylori in samples from the dorsum of the tongue (10.8%) using PCR techniques. Collectively, these data suggest that the oral cavity may be a reservoir for H. pylori in some individuals and transmission of the disease may be via an oral-to-oral route.

4. REFERENCES


