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The Role of *Helicobacter Pylori* in Chronic Tonsillitis

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Abstract

Chronic tonsillitis is a common inflammatory disease of the tonsillar tissue that causes various ENT pathologies. Microbiological studies have shown a possible correlation between tonsillitis and Helicobacter pylori. Patients with chronic tonsillitis and H. pylori infections have a higher risk of complications. PCR testing revealed H. pylori DNA in 30.8% of patients with chronic tonsillitis, compared to a lower rate in healthy volunteers. Complications of chronic tonsillitis include infectious endocarditis, scarlet fever, and psoriasis. Carcinoma of the tonsil may be a possible complication in patients with tonsillitis and H. pylori. Detecting markers of H. pylori in oral cavity and eradicating the pathogen is recommended for patients with chronic tonsillitis.

Introduction

Helicobacter pylori is a non-spore-forming microaerophilic gram-negative spiral organism. It has a unique characteristic of producing urease enzyme that is not produced by other bacteria of the human digestive tract. As a result of this enzyme, it has the ability to survive in the acid environment of the stomach. This is the main factor that allows researchers to detect this organism in the stomach. The stomach has long been believed to be the only habitat of the Helicobacter pylori. Recently, some articles have demonstrated the presence of Helicobacter pylori in dental plaque and suggested that oral occurrence of these bacteria may underlie a state of chronic colonization in extra-gastric sites or gastro-oral transmission.[1,18,19]

Tonsils have been under intense research and discussion for many years. Some authors suggest that the tonsils may be a significant extra-gastric source of Helicobacter pylori, which is responsible for stomach re-colonization after eradication. It is believed that re-colonization happens through the natural and/or surgical connections between the stomach and the mouth. This paper aims to analyze published data on the topic to present the available research findings.[2]

Helicobacter pylori: Characteristics and Pathogenesi

Structure and Morphology

H. pylori also utilizes type IV secretion systems for the exportation of certain cytoplasmic proteins across its inner membrane. VirB7 and VirB9 are standard inner-membrane proteins that interact within the type IV secretion system. When addressing the periplasmic protein VirB10, these three proteins successfully cross the outer membrane. QssB is a VirB7-like protein that aids in the interaction between the inner membrane protein VirB9 and the outer membrane protein VirB10 to facilitate secretion. The flagellum is the largest structure of the Helicobacter pylori cell, with the hook and its junction with the basal body contributing to the generation of torque during Helicobacter pylori swimming. Primary topic editors do not receive any external funding for their contributions to Scholarpedia. [3]

Helicobacter pylori utilizes a double layer of peptidoglycan to maintain its spiral shape. The periplasmic space features peptidoglycan-binding proteins and a thin outer shell combined with an inner layer beneath the surface. This structure requires all materials to pass through porins in the bilayer, with the size of the porins being dictated by the overall thickness of the H. pylori envelope. As a result, porins are the unique proteins expressed from the bacterial chromosome, contributing to diverse cell size classes. [4]

Virulence Factors

The initial step in the process of bacterial infection is the adhesion of bacteria to host cells. H. pylori is linked to a variety of adhesion factors related to its disease-causing capabilities. Adhesion molecules are crucial for enabling host cells to attach to tissues and for facilitating important cell-to-cell interactions that aid in communication within and between cells. [5]

The BabA adhesion factor is a crucial molecule on H. pylori's surface. BabA is a 74.1 kDa protein with 598 amino acids and is present in seven-to-nine specific H. pylori strains. These factors are related to ABO and Lewis histo-blood group antigens, specifically, the Lewis b antigen. Additionally, adhesion factors are categorized based on their binding abilities, with BabA being highly volatile and recognizing the Lewis b antigen, while BabB has weaker binding. [6]

Another significant attachment factor of H. pylori is the adhesion protein associated with the bacterium's adherence. The surface structure of the bacterium contains Hop sequences that bind to B or O blood groups or act as a protein with a higher level of bicapavity, potentially serving as parietal cell adhesion. HopQ is shielded from the body's mucous membrane surface by adhesion reliant on host cells, causing increased damage to the host cells. HopQ is functionally dependent on BabA for adhesion, host cell cycles, and plays a role in causing damage to gastric tissue. [7]

Chronic Tonsillitis: Definition and Etiology

Chronic tonsillitis (CT) is an infectious-inflammatory disease of the tonsils with a prolonged course. It can either be acute or gradually arise from chronic inflammation of the tonsils. Currently, the etiology of CT is being studied again, which has sparked a large interest among researchers from different fields such as pathologists, pathophysiologists, allergists, endocrinologists, pediatricians, and otolaryngologists. Recent publications indicate a variety of different organisms that contribute to chronic tonsil diseases. It is evident from recent studies that the colonization of patients by specialists has had a positive impact on the understanding of this issue. [8]

Clinical Presentation

Symptoms include subjective feelings of redness in the oral cavity, difficulty swallowing, occasional dry cough, and slight fever. On examination, there may be no visible changes or, in some cases, the affected area may show inflammation of the tonsils. In some cases, there may also be a small localized abscess or fistula. Pain is usually reduced or absent due to decreased sensitivity in the affected area. The size of the tonsils may be as large as half the normal size during chronic streptococcal tonsillitis, and may later increase or decrease. [9]

In certain instances, patients may present with a pre-existing anatomical abnormality such as hernia, hypoplasia, or palatalism. If this is the case, corresponding sized cones will appear during the same period of the illness. Their previous absence, potentially due to activated lymphoid tissue or stagnant secret focus, will be observed following a tonsillectomy. Since the condition is typically non-specific, it can be challenging to distinguish from emerging external or internal tumor-like growths. Only a biopsy can accurately determine the nature of the issue and define the risk or clinical significance of its progression. [10]

Risk Factors

The search for risk factors associated with Helicobacter pylori infection is crucial, given its prevalence and concerns about transmission. A literature review found that risk factors include large households, male gender, marital status, income sources, factory/vendor-prepared food consumption, dining out, shift work, prior treatment for related conditions, drug use, smoking, alcohol consumption, dyspeptic symptoms, and fast eating under stress.

People who live in clean homes and those who work different shifts faced a noticeably higher risk, even when other factors were taken into account. The research involved people of various ages, indicating that there may be other risk factors to consider. In summary, the type of diet a person has and the social environment they are in are linked to a higher risk of H. pylori infection. Unhealthy eating habits such as drinking coffee with meals and regular alcohol intake are especially obvious. More research is necessary to explore these and other transmission-related factors. From the current information, it seems that social environment plays a more significant role than individual lifestyle in the transmission of H. pylori.[11]

Evidence for the Presence of H. pylori in Tonsillar Tissues

The study findings revealed that 18.6% of the tonsillar tissues examined tested positive for Helicobacter pylori (H. pylori). Significantly higher rates of H. pylori presence were observed in individuals with hypertrophic tonsils compared to the control group, while the recurrent tonsillitis group had the lowest positive rate. There was no correlation between the presence of H. pylori and factors such as age, sex, Helicobacter species, or urea breath test results. [12]

Multiple logistic regression analysis showed that the presence of intratonsillar food debris or foreign bodies significantly impacted the incidence of H. pylori. These results suggest a potential link between intrapinguating food debris or foreign bodies and the colonization of H. pylori within the tonsillar niche. The study also indicates variations in histological changes and bacterial function between recurrent tonsillitis and other tonsillar diseases. [2] Although H. pylori is primarily found in the stomach, it is also commonly present in extragastric sites. The study aimed to thoroughly investigate the presence of H. pylori in tonsillar tissues and its association with clinicopathological characteristics of patients who underwent tonsillectomy at the Otolaryngology Department. Patients were divided into pathology-confirmed hypertrophic tonsils, chronic tonsillitis, and recurrent tonsillitis groups, with the control group consisting of individuals with normal tonsils during septum surgery. [13**]**

Association Between H. pylori and Chronic Tonsillitis

Various studies have consistently discovered a link between chronic tonsillitis and the existence of Helicobacter pylori, although few have delved into the microorganism's role in the development of the condition. This association is often overlooked by healthcare workers and organizations, possibly due to the infrequency of infection in certain countries. However, carrying the bacteria since childhood may have detrimental effects, leading to a heightened immune response and significant immunopathology. Clinical observations also indicate a potential increase in allergic symptoms in affected organs after tonsil removal, particularly in individuals harboring Helicobacter pylori. The full extent of Helicobacter pylori's role in chronic tonsillitis remains unrecognized, and further research is essential to reveal the mechanisms of immune protection and the long-term relationship between tonsils and the bacteria in their tissue environment. [14]

The upper respiratory tract and the digestive tract contain high amounts of lymphoid tissue. Submucosas of the intestinal tracts have the highest proportion of tissue areas occupied by lymphocytes, similar to the tonsils. Animals with larger lymphoid structures are at a disadvantage, particularly in the immune response to antigens, hence various strategies have been developed by microorganisms that invade mammals. These microorganisms have a cutaneous and mucosal front, and the interest in understanding this aspect of infection arises from the ability of many mucosal barrier defects to favor infections and alterations in the presence of abnormal microbial densities. This is why corticosteroids applied to the oral mucosa to treat aphthous ulcers alter Helicobacter pylori's microorganism content, which can be responsible for diseases of an important known character. [15]

Lymphoid tissue constitutes the organ providing a starting response to most antigens. The histological structure of lymphoid tissue acts as such, not only structurally but also by providing

antigenic selection processes in which the so-called primary humoral response arises, which is rapidly replaced by a secondary immune response. The latter response is characterized by establishing a depot of long-lived memory B cells and long-lived plasma cells that maintain specific antibody levels for prolonged periods. However, by promoting a high diversity of immunological memory cells with moderate survival, the diversity of secondary responses remains high, and it is mammals' organ that has the highest metabolic requirement. [16]

Immunological Responses in H. pylori-Associated Tonsillitis

Tonsillar inflammation caused by Helicobacter pylori is usually chronic, diffuse, and often catarrhal-purulent. Mononuclear leukocytes, lymphocytes, and epithelial cells can penetrate the tonsillar parenchyma and exfoliate into the tonsillar crypts and surface. H. pylori often localizes in the crypts among the exfoliating epithelial cells and the phagocytes that have invaded them, which in turn is characteristic of H. pylori tonsillitis. This pathogen has evolved special virulent factors. Chronic or recurring tonsillitis caused by H. pylori is observed in 12.9% of cases of chronic tonsillitis and 27.4% in intractable chronic tonsillitis. Extensive tonsillitis can accelerate the progression of other infections, as a constant focus of infection influences the development, maturation, and function of immune elements, which leads to tonsillar infections and not only exacerbates the infection but also can cause other foci of infection. [17]

Inflammations of other organs related to H. pylori have the potential to be the systemic source of inflammation and impact immune reactions. For example, in patients with both H. pylori and tuberculosis infections, this stimulation can hinder the development of immune reactions. The development of immunity in chronic tonsillitis caused by H. pylori is also inadequate; T-lymphocytes, particularly suppressive T-lymphocytes involved in immunoregulation, are suppressed in patients with tonsillitis positive for H. pylori. Additionally, T-cells in inflamed tonsils are unable to function properly. Phagocytosis and respiratory burst in the infected tonsils are adversely affected, leading to immune tolerance for H. pylori metabolites and creating conditions for proliferation. The decreased quantity of H. pylori in tonsillitis rates may be the mechanism for its removal from tissues even after radical tonsillectomy. Early diagnosis and elimination of H. pylori along with traditional treatment are necessary for intractable chronic tonsillitis, especially in cases of H. pylori tonsillitis. Removing H. pylori from inflamed tonsils reduces the number of exacerbations of the inflammatory process in cases of chronic hyperplastic adenotonsillitis. [14]

Conclusion

In conclusion, the evidence suggesting a potential association between H. pylori infection and chronic tonsillitis is intriguing and highlights the need for further investigations to clarify the role of this bacterium in the pathogenesis of tonsillitis. Elucidating the mechanisms underlying this association may lead to a better understanding of chronic tonsillitis and the development of more effective treatment strategies.

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