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Does Helicobacter Pylori Play a Role in Pathogenesis of Primary Acquired Nasolacrimal Duct Obstruction?

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Abstract

Nasolacrimal duct obstruction is a common ophthalmic problem, comprising 3% of clinic visits in some series. Primary acquired dacryostenosis results from inflammation of unknown cause that eventually leads to occlusive fibrosis. Helicobacter pylori is a microaerophilic, Gram-negative spiral organism that has been shown to be the causative factor in a large proportion of patients with gastric ulcer and gastritis. Until now, no attempt has been made to investigate the prevalence of H. pylori in idiopathic acquired nasolacrimal duct obstruction and its relationship with the pathophysiology of this entity.

We suggest two hypotheseses to explain possible role of H. pylori infection in pathogenesis of nasolacrimal duct obstruction: Direct transmission of H. Pylori infection from the nasal cavity to nasolacrimal duct and release of proinflammatory and vasoactive substances.

Keywords
Nasolacrimal duct obstruction, Helicobacter pylori, Infection, Dacryocystitis

Introduction

Primary Acquired Nasolacrimal duct obstruction

Lacrimal obstruction is a common ophthalmic problem, comprising 3% of clinic visits in some series (1).The symptoms of epiphora, dacryocystitis, or both may not develop until the end stages of the initial inciting event. In the absence of any apparent etiology, this entity has been termed primary acquired nasolacrimal duct obstruction (2). Primary acquired dacryostenosis results from inflammation of unknown cause that eventually leads to occlusive fibrosis (3).

Trauma, tumors, systemic inflammatory diseases, and infections are occasionally responsible for secondary acquired lacrimal excretory system obstruction (4,5).

Diagnosis and treatment of this condition requires a thorough understanding of the lacrimal apparatus and its ocular and nasal relationships. We mentioned here some opinions about pathophysiology of idiopathic dacryostenosis.

The lacrimal sac and nasolacrimal duct as parts of tear duct system are surrounded by a network of large capacitance vessels connected caudally with the cavernous body of inferior turbinate. Histologicaly, the whole vascular plexus is embedded in helical system of collagen bundles as well as elastic and reticular fibers. It has been suggested that these specialized blood vessels, while regulating blood flow, also permit opening and closure of the lumen of lacrimal passage effected by the engorgement and subsidence of the cavernous body, thus at the same time regulating tear flow. An important role in absorption and drainage of lacrimal fluid has also been hypothesized (6).

Descending inflammation from the eye or ascending inflammation from the region of the nose may initiate malfunction of the cavernous body with reactive hyperemia, swelling of the mucus membrane, and temporary occlusion of the passage. Then repeated isolated episodes of dacryocystitis may lead to structural epithelial and subepithelial changes. Loss of typical goblet and epithelial cells, as well as fibrosis of the helical system of connective tissue fibers in the area of lacrimal sac and nasolacrimal duct, and destruction of specialized blood vessels of the cavernous body, may exacerbate malfunctions of the tear outflow mechanism and start up a vicious cycle(7).

Helicobacter Pylori and the eye

Helicobacter pylori is a microaerophilic, Gram-negative spiral organism that has been shown to be the causative factor in a large proportion of patients with gastric ulcer and gastritis (8,9). It has also been associated with gastric cancer (10).

Although H pylori infection is widespread throughout the world, the exact mode of transmission has not been yet fully understood. Possible modes of transmission are oral-oral, fecal-oral, and gastrointestinal-oral routes (11).

Human stomach was considered to be the only reservoir of H pylori until bacteria were discovered in human dental plaque, oral lesion, and in saliva. Recently this organism was also detected in tonsil and adenoid tissue (10).

This organism has been implicated in a variety of extradigestive vascular conditions including ischemic heart disease, ischemic cerebrovascular disorders (12), and functional vascular disorders caused by vascular dysregulation (eg, Raynaud phenomenon and migraine) and with some autoimmune conditions such as Sjögren syndrome(13).

Moreover the role of H. pylori infection in ophthalmic diseases such as chronic ocular inflammation, glaucoma (14), rosacea, and chronic blepharitis has been postulated (15). The complex interactions between H.pylori and ocular diseases is a field of ongoing research ,though the association remains unclear. H.pylori infection may influence the pathophysiology of glaucoma by releasing proinflammatory and vasoactive substances , as well as by influencing the apoptotic process, These parameters may also exert their own effects in the induction and progression of glaucomatous neuropathy(16). Production of oxidative stress and circulating lipid peroxides by H.pylori infection could affect pathophysiology of glaucoma too (17). The precise pathophysiology of central serous chorioretinopathy is also uncertain. Choroidal microcirculation abnormalities, vascular wall antigens and antibody reactivity could be involved. H.pylori may contribute to CSR by increasing endothelin 1, nitric oxide, inducible nitric oxide synthase, proinflammatory and vasoactive substances. The prevalence of H. pylori infection has been found significantly higher in central serous chorioretinopathy-affected subjects (18).

Acne rosacea is a characteristic combination of skin manifestations with ocular involvement. Acne rosacea has long been associated with gastritis. Gastrin is a potent vasodilator that its serum levels are elevated in patient with H.pylori associated gastric disease. Gastrin could produce vasodilation of the skin and the other manifestations of acne rosacea (19,20).

Until now, no attempt has been made to investigate the prevalence of H. pylori in idiopathic acquired nasolacrimal duct obstruction and its relationship with the pathophysiology of this entity.

Hypothesis

We suggest two hypotheseses to explain possible role of H. pylori infection in pathogenesis of nasolacrimal duct obstruction.

1. Direct infection:

H pylori have been found in the mouth (11), but it is not known whether the oral cavity acts as a permanent reservoir for this bacterium. Furthermore the presence of H. pylori in nasal polyp, nasal mucosa, and maxillary sinus mucosa was detected in some patients with chronic sinusitis and gastric H. pylori infection(21). So direct transmission of H. Pylori infection from the nasal cavity to nasolacrimal duct and possibly lacrimal sac may trigger inflammatory process and

repeated episodes of dacryocystitis. These events may eventually lead to chronic inflammation and structural changes such as loss of goblet cells and epithelial cells and fibrosis in the area of nasolacrimal duct and lacrimal sac.

2. Release of proinflammatory and vasoactive substances:

Release of large amounts of variable proinflammatory and vasoactive substances such as cytokines (interleukin [IL] 1, IL-6, IL-8, IL-10, IL-12, tumor necrosis factor α, and interferon γ), eicosanoids, and acute phase proteins following gastric colonization by H pylori may be involved in a number of vascular disorders thought to be associated with the bacterium(22,23). Increasing Gastrin (15), nitric oxide; both are potent vasodilators, inducible nitric oxide synthase (24) and releasing proinflammatory and vasoactive substances may be involved in pathogenesis of nasolacrimal duct obstruction. These substances may initiate malfunction of cavernous body and accordingly affect passage of tear through the nasolacrimal duct.

Evaluation of the Hypothesis

Our Hypothesis could be evaluated by cohort studies on H.pylori positive populations and following them for Nasolacrimal duct obstruction. Furthermore we can asses whether eradication of H.pylori infection decrease incidence of nasolacrimal duct obstruction in these patients. We can also design studies for detection of H.pylori in tissue specimens from nasolacrimal duct or lacrimal sac mucosa. It is difficult to isolate such a fastidious strain in a highly colonized environment. Furthermore, unsuitable conditions for H.pylori induce morphological change to coccoid form. This dormant morphology is not culturable in vitro (25). So we should use more sensitive techniques such as RT-PCR (Reverse transcription polymerase chain reaction), CLO test (urease test), and immunohistochemical analysis to investigate the H.pylori status of tissue specimens (21).We have designed a study using these techniques to support our hypothesis.

However, in order to prove that the link is a casual relationship it is better to design more studies to prove that samples from obstructed nasolacrimal ducts have more infection in comparison to a matched control group .

Conclusion & Discussion

The etiology of primary acquired nasolacrimal duct obstruction is unknown. Although the first event in nasolacrimal obstruction remains uncertain, many authors have suggested that stasis and secondary infection lead to complete obstruction. Descending pathogens from the conjunctival sac, as well as diverticula of the lacrimal passage, have been suggested to be causal. Other specialists claim that the origin of idiopathic dacryostenosis is in the nose. Simple infections of the nasal mucous membrane or diseases of sinuses have been suggested (7). Infectious causes of secondary acquired lacrimal obstruction include trachoma, Aspergillus, Actinomyces, Diphtheria and Streptococcus organisms. More commonly, infections occur secondary to nasolacrimal duct obstruction with the trapped lacrimal sac contents serving as culture media for such microorganisms as Staphylococcus and Streptococcus, and gram negative rods(2). H.pylori is widespread through out the world. Recently this organism is detected in saliva, oral lesion, dental plaque, adenoid tissue, nasal mucosa, nasal polyp and maxillary sinus(21,26). According to our hypothesis H.pylori can get access to nasolacrimal duct and possibly lacrimal sac from nasal mucosa and start inflammation. Furthermore release of inflammatory and vasoactive substances after colonization of H.pylori may leads to malfunction of tear passage system. So there may be relationship between Helicobacter pylori infection and pathogenesis of nasolacrimal duct obstruction but investigations are needed to prove this theory. Probably eradication of H.pylori can prevent development of nasolacrimal duct obstruction in H.pylori positive dyspeptic patients. We can also treat H.pylori infection in patients with unilateral nasolacrimal duct obstruction whose specimens were positive for H.pylori in order to prevent the other side involvement.

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