

Medical Implication of Oral Amoebiasis

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Summary

Periodontal disease affects a majority of adult population. *Entamoeba gingivalis* is always present as confirmed by phase contrast microscopy. Tissue destruction may be caused by proteolytic enzymes in part released from host PMN leucocytes lysed by amoebas. Local consequence is destruction of the maxillary bone surrounding teeth. Elimination of *E. gingivalis* ensures rapid healing of chronic and aggressive form of the disease. Periodontitis is known to adversely affect glycemic control in diabetes, is associated with an increased risk of cardiovascular complication and increase risk for pre-term low birth weight delivery. Although bacterial infection is primary, oral amoebiasis has important medical implications.

Introduction

Periodontitis is an infectious disease, with primary bacterial etiology followed by inflammatory process leading to destruction of the supporting structures of the teeth. Main bacterial species implicated in the disease are Gram-negative, anaerobic, and facultative microorganisms. Many authors¹⁻⁵ reported 100% incidence of secondary infection with *E. gingivalis*. Phase contrast observation in our dental practice during 20 years confirm the presence of the amoeba in all area of bone destruction. Normal health biofilm present typical morphotypes: coccoidal bacteria, non motile filaments and association of both (Fig. 1). Motility is absent. Periodontal deterioration present numerous polymorphonuclear neutrophils, very motile bacteria, spirillae, spinnings rods and protozoa (Fig. 2). Amoebae are visible on fresh biofilm smear mounted on patient saliva and taken from deepest lesion area. Mouth and lower gastrointestinal tract share similar protozoan activities. Pathology of *E. histolytica* in amebic dysentery is linked to lytic activity, invasion of intestine mucosa and formation of acute liver abscess. Mechanisms include motility of the trophozoite,

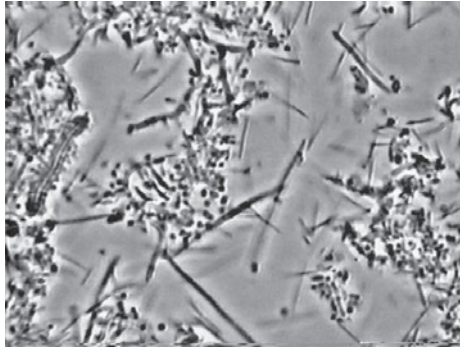


Figure 1.

phagocytic abilities, and discharge of a peptide promoting lysis and degradation of cell.⁶ Denucleated cell formation around the invading ameba leads to rapid lysis, tissue necrosis and abundant microhaemorrhaging.⁷ Lyons investigations on oral protozoa supported the pathogenicity of *E. gingivalis*, and offered protocols for microscopic examination and specific local and systemic treatments targeting the ameba. Elimination of the protozoa abrogated the disease and promoted its resolution.

Materials and methods

Subjects with severe periodontal disease have higher risk of cardiorenal mortality.⁸ Inflammation and the systemic immune response are believed to play a central role in the initiation and propagation of atherosclerosis.⁹ Low birth weight delivery incidence is equivalent to 12.5%.¹⁰ The prevalence in non-treated subjects with periodontal disease is high.¹¹ Production of cytokines and consequent release of prostaglandins are responsible for uterine contraction. Recent hypothesis suggests Type 2 diabetes be a disorder of the innate

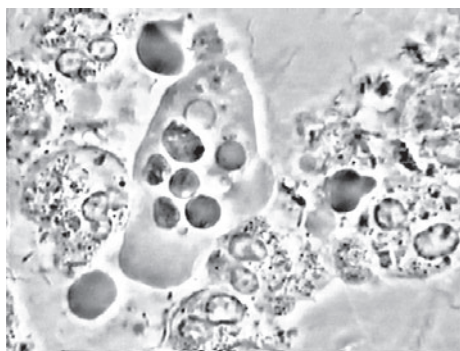


Figure 2.

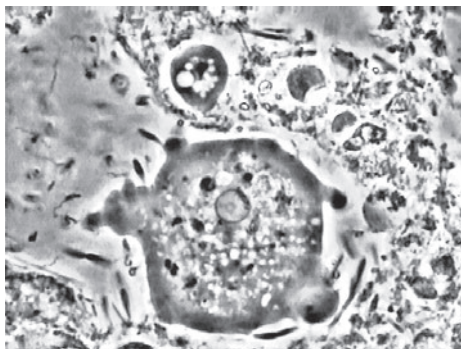


Figure 3.

immune system and result of a chronic, low-grade inflammatory process.¹² Our microscopic investigation for 20 years period supports the findings of Kofoid, Lyons and Keyes as *E. gingivalis* is present in all cases of periodontal disease and absent in healthy or gingivitis patients.

Results

Strict topical and systemic pharmaceutical elimination of parasites accompanied by good hygiene habits brings about cessation of bleeding, elimination of pus and gingival healing. Microscopy reveals complete disappearance of inflammatory cells and reappearance of normal flora. The concept basis for pathogenicity of Entamoeba genus comes from adherence to mammalian cell, chemotaxis, penetration, invasion, cytolysis and interaction with symbiotic flora. *E. gingivalis* structurally present all similar characters: motility (Fig. 3), phagocytosis resulting in uncontrolled denucleated neutrophils (Fig. 4), adhe-

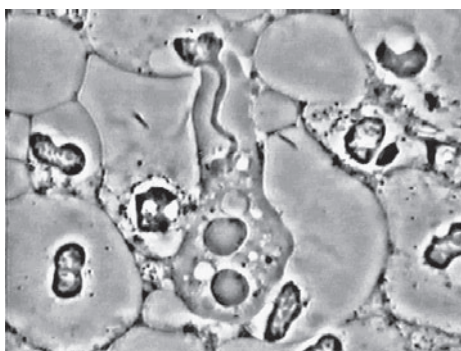


Figure 4.

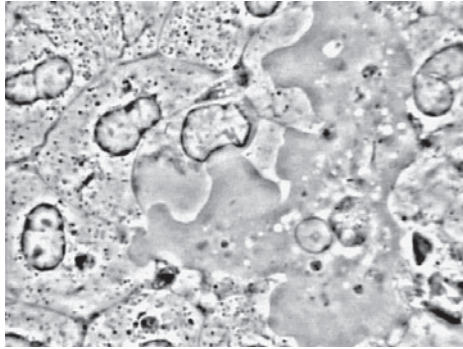


Figure 5.

sion and lysis of cell (Fig. 5) and tissues invasion (Fig. 6) as in amebic liver ulceration. The continued presence of amoebiasis in bone deterioration area seem to act as a secondary infection after primary bacterial gingivitis. All evidence show for the pathogenicity of *E. gingivalis*. The periodontium may serve as a reservoir for ameba, bacteria, inflammatory and immune mediators which can interact with other organ systems remote from the oral cavity. Professional opinions on the pathogenicity of certain microorganisms evolved with time, as illustrated by *Giardia intestinalis* and *Helicobacter pylori*, in which case a radical change has taken place from surgical to pharmacological therapy.

Conclusions

E. gingivalis is highly motile, has phagocytic abilities and exhibits typical pathogen characteristics. It is associated with destruction of the maxillary bone. Its presence is a constant in periodontal diseases. Its elimination ensures healing of chronic and aggressive forms of periodontitis as demonstrated by

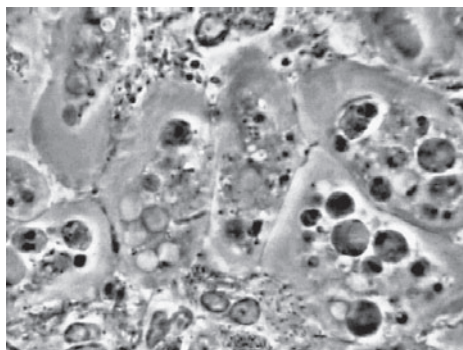


Figure 6.

microscopic and clinical examination. Presence of *E. gingivalis* has therefore important medical and epidemiological consequences. Our dense urban lifestyles make the transmission of this organism easily accomplished through direct social contact, air born particles and through contaminated cooking utensils. It may also play a key role in serious, debilitating and incapacitating disturbances of the general health. Its adverse impact on the human race will remain unappreciated unless it is fully investigated, not just as a specific agent of oral disease, but as a general factor of systemic disease.

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