Periodontal pathology

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Periodontal pathology is a science or a study of periodontal diseases. Periodontal diseases can affect one or more of the periodontal tissues/structures (e.g. alveolar bone, periodontal ligament, cementum and gingiva). While there are many different periodontal diseases that can affect these tooth-supporting tissues/structures, by far the most common ones are plaque-induced inflammatory conditions,[1] such as gingivitis and periodontitis.[2] Often the term *periodontal disease* or *gum disease* is used as a synonym for periodontitis, specifically chronic periodontitis. Periodontal disease ranges from the mildest stage, known as gingivitis, to severe stage, known, as periodontitis.[3]

While in some sites or individuals, gingivitis never progresses to periodontitis,[4] data indicate that periodontitis is always preceded by gingivitis.[1]

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Diagnosis

In 1976, Page & Schroeder [5] introduced an innovative new analysis of periodontal disease based on histopathologic and ultrastructural features of the diseased gingival tissue. Although this new classification does not correlate with clinical signs and symptoms and is admittedly "somewhat arbitrary," it permits a focus of attention pathologic aspects of the disease that were, until recently, not well understood.[1] This new classification divided plaque-induced periodontal lesions into four stages:

1. initial lesion
2. early lesion
3. established lesion
4. advanced lesion

Initial lesion

Unlike most regions of the body, the oral cavity is perpetually populated by pathogenic microorganisms; because there is a constant challenge to the mucosa in the form of these microorganisms and their harmful products, it is difficult to truly characterize the boundary between health and disease activity in the periodontal tissues. The oral cavity contains over 500 different microorganisms. It is very hard to distinguish exactly which periodontal pathogen is causing the breakdown of tissues and bone. As such, the initial lesion is said to merely reflect "enhanced levels of activity" of host response mechanisms "normally operative within the gingival tissues."[1][6][7]

Healthy gingiva are characterized by small numbers of leukocytes migrating towards the gingival sulcus and residing in the junctional epithelium.[1][7] Sparse lymphocytes, and plasma cells in particular, may exist just after exiting small blood vessels deep within the underlying connective tissue of the soft tissue between teeth.[1][7] There is, however, no tissue damage, and the presence of such cells is not considered to be an indication of a pathologic change. When looking at the gums they look knife like and a very light pink or coral pink.

On the contrary, the initial lesion shows increased capillary permeability with "very large numbers" of neutrophils migrating from the dilated gingival plexus into the junctional epithlieum and underlying connective tissue (yet remaining within the confines of the region of the sulcus) and macrophages and lymphocytes may also appear. Loss of *perivascular collagen* occurs; it is thought that this is due to the degradative enzymes released by extravasating leukocytes, such that the collagen and other connective tissue fibers surrounding blood vessels in the area dissolve.[1][7] When this occurs the gums will appear bright red and either bulbous or rounded, from all the excess fluid building up in the infected area.

The initial lesion appears within two to four days of gingival tissue being subjected to plaque accumulation. When not generated through clinical experimentation, the initial lesion may not appear at all, and instead, a detectable infiltrate similar to that of the early lesion, explained below, appears.[8]

Features of the Initial Lesion:[1]

- Vasculitis of vessels subjacent to junctional epithelium
- Increased migration of leukocytes into junctional epithelium
- Extravascular presence of serum proteins, especially fibrin
- Alteration of the most coronal portion of junctional epithelium
- Loss of perivascular collagen

Early lesion

While the early lesion is not entirely distinct from the initial lesion, it is said to encompass the inflammatory changes that occur from days four to seven after plaque accumulation has commenced.[7] It is characterized by a matured leukocytic infiltrate that features mainly lymphocytes. Immunoblasts are

quite common in the area of infiltration, while plasma cells, if present, are only at the edges of the area.[1] The early lesion can occupy up to 15% of the connective tissue of the marginal gingiva and up to 60-70% of collagen may be dissolved.[9]

Fibroblasts appear altered, exhibiting electron-lucent nuclei, swollen mitochondria, vacuolization of the rough endoplasmic reticulum and rupture of their cell membranes, appearing up to three times the size of normal fibroblasts and found in association with moderately-sized lymphocytes.[9]

The early lesion displays acute exudative inflammation; exudative components and crevicular lymphocytes reach their maximum levels between days 6-12 after plaque accumulates and gingival inflammation commences[10] with the quantity of crevicular fluid being proportional to the size of the reaction site within the underlying connective tissue. The junctional epithelium may even become infiltrated with enough leukocytes so that it resembles a microabscess.[11]

Features of the Early Lesion:[1]

- Accentuation of features of the initial lesion, such as the considerably greater loss of collagen
- Accumulation of lymphocytes subjacent to junctional epithelium
- Cytopathic alterations in resident fibroblasts
- Preliminary proliferation of basal cells of junctional epithelium

Established lesion

The hallmark of the established lesion is the overwhelming presence of plasma cells in relation to the prior stages of inflammation. Beginning two to three weeks after first plaque formation, the established lesion is widespread in both human and animals populations[12] and can be seen commonly associated with the placement of orthodontic bands on molars.[13]

Similar to the initial and early lesions, the established lesion features an inflammatory reaction confined to the area near the base of the gingival sulcus, but unlike prior stages, displays plasma cells clustered around blood vessels and between collagen fibers outside the immediate area of the reaction site.[1] While most of the plasma cells produce IgG, a significant number do produce IgA (and rarely, some produce IgM).[14] The presence of complement and antigen-antibody complexes is evident throughout the connective and epithelial tissue.[14]

It is in the established lesion that epithelial proliferation and apical migration begin. In health, the junctional epithelium creates the most coronal attachment of the gum tissue to the tooth at or near the cementoenamel junction. In the established lesion of periodontal disease, the connective tissue lying subjacent to the junctional epithelium is nearly destroyed, failing to properly support the epithelium and buttress it against the tooth surface. In response to this, the junctional epithelium proliferates and grows

into the vacant underlying spaces, effectively causing the level of its attachment to migrate towards apically, revealing more tooth structure than is normally evident *supragingivally* (above the level of the gumline) in health.

While many established lesions continue to the advanced lesion (below), most either remain as established lesions for decades or indefinitely; the mechanisms behind this phenomenon are not well understood.

Features of the Established Lesion:[1]

- Predominance of plasma cells without bone loss
- Presence of extravascular immunoglobulins in the connective tissue and junctional epithelium
- Continuing loss of collagen
- Proliferation, apical migration and lateral extension of the junctional epithelium, with or without pocket formation

Advanced lesion

Many of the features of the advanced lesion are described clinically rather than histologically:[15]

- Periodontal pocket formation
- Gingival ulceration and suppuration
- Destruction of the alveolar bone and periodontal ligament
- Tooth mobility, drifting and eventual loss

Because bone loss makes its first appearance in the advanced lesion, it is equated with periodontitis, while the first three lesions are classified as gingivitis in levels of increasing severity.[1]

The advanced lesion is no longer localized to the area around the gingival sulcus but spreads apically as well as laterally around a tooth and perhaps even deep into the gum tissue papilla. There is a dense infiltrate of plasma cells, other lymphocytes and macrophages. The clusters of perivascular plasma cells still appears from the established lesion. Bone is resorbed, producing scarring and fibrous change.[1]

Features of the Advanced Lesion:[1]

- Extension of the lesion into alveolar bone, periodontal ligament with significant bone loss
- Continued loss of collagen
- Cytopathic alterations in plasma cells in the absence of altered fibroblasts
- Formation of periodontal pocketing
- Conversion of bone marrow into fibrous connective tissue

Treatment

The treatment of periodontal disease begins with the removal of sub-gingival calculus (tartar) and biofilm deposits. A dental hygienist procedure called scaling and root planing is the common first step in addressing periodontal problems, which seeks to remove calculus by mechanically scraping it from tooth surfaces.

Dental calculus, commonly known as tartar, consists almost entirely of calcium phosphate salt, the ionic derivative of calcium phosphate (the primary composition of teeth and bone). Dental calculus deposits harbor harmful bacteria. Clinically, calculus stuck to teeth appears to be hardened to the point requiring mechanical scraping for removal.

The bacteria responsible for most periodontal disease are anaerobic, and oxygenation reduces populations. Thorough brushing with dilute hydrogen peroxide, with emphasis on the gum line, and flossing, help prevent the formation of harmful biofilm, gingivitis, and tartar. Therapeutic mechanical delivery of hydrogen peroxide to subgingival pockets can be provided by a water pick. Wound "healing following gingival surgery was enhanced due to the antimicrobial effects of topically administered hydrogen peroxide". For most subjects, beneficial effects were seen with hydrogen peroxide levels above 1% though concentrations between 1% and 3% have been suggested, and commercial preparations contain 1.5% hydrogen peroxide.[16]

Enzymatic agents found in commercial preparations can loosen, dissolve, and prevent biofilm formation. Beneficial agents include lysozyme, lactoperozidase, glucose oxidase, mutanase, and dextranase.[17]

Another method for treatment of periodontal disease involve the use of an orally administered antibiotic, Periostat (Doxycycline). Periostat has been clinically proven to decrease alveolar bone loss and improve the conditions of periodontal disease with minimal side-effects. However, Periostat does not kill the bacteria, as it only inhibits the body's host response to destroy the tissue.

Prognosis

Plaque, also known as a biofilm, when examined under a microscope, is made of millions of bacteria, leucocytes, protozoa (*Entamoeba gingivalis* and *Trichomonas tenax*) and occasional fungi.[18][19] There are many different types of microbes contained in the biofilm of those with periodontal disease. Two major bacteria implicated are "Porphiromonas gingivalis"and "Aggregatibacter actinomycetemcomitans" A. actinomycetemcomitans is associated with acquired resistance to normal treatments against periodontal disease. P. gingivalis can produce harmful enzymes which disrupt the host immune system and lead to massive tissue destruction.[20] Since a microbe is a living organism, it maintains some of the same properties that humans do to survive. *Porphyromonas* have a life cycle, they have a digestive system, and they reproduce. Bacteria have to eat to survive; they also have to eliminate wastes and are constantly reproducing. Naturally, bacteria are always present in the oral cavity. However, when plaque is not removed on a daily basis, trouble begins.

Bacteria around teeth cause the destruction and foul odors in a person with gum disease, specifically sulfur-containing compounds. Bone is considered to be the foundation and supporting structure of teeth. Bacteria will initially colonise the spaces between teeth and release or exhibit compounds that the body's

immune response responds to through inflammation, resulting in bone loss. As bacteria proliferate, the immune response increases and teeth will eventually become loose and either fall out on their own, or are extracted by a dentist. This process is lengthy and does not happen overnight.

It is recommended that a dental prophylaxis and thorough examination of the mouth be done every six months, preventing plaque buildup on teeth. Plaque or bacteria, if left for a long period of time, eventually die off. Dead plaque hardens and calcifies and is then referred to as tartar, or calculus. Once the calculus builds up around the teeth, in between them, and the gums, it causes the gums to pull away from the teeth. When the gums pull away from the teeth, a pocket is created which allows food and debris to accumulate, harboring even more bacteria. This also allows bacteria to enter the bloodstream.

Studies have shown that heart disease is almost twice as likely to occur in people with gum disease, although a causal relationship between gum disease and heart disease has not yet been sufficiently established in such studies.[21] In 2012 an American Heart Association scientific statement was released that was based on reviews of the latest periodontal pathology research regarding cardiovascular disease, which concluded that while there is an association between periodontal disease and cardiovascular disease independent of some confounding variables (e.g., cigarette smoking), more research needs to be conducted to sufficiently establish a causal relationship between periodontal disease and cardiovascular disease while better accounting for other confounding variables (e.g., socioeconomic status).[22][23][24] Studies have also shown that the most common strain of bacteria found in dental plaque may cause blood clots.[25] When blood clots escape into the bloodstream, there is a relation to increased risk of heart attacks, and other illnesses.

History

Investigation into the causes and characteristics of periodontal diseases began in the 18th century with pure clinical observation, and this remained the primary form of investigation well into the 19th century.[15] During this time, the signs and symptoms of periodontal diseases were firmly established:[1]

1. Rather than a single disease entity, periodontal disease is a combination of multiple disease processes that share a common clinical manifestation.
2. The etiology (cause) includes both local and systemic factors.
3. The disease consists of a chronic inflammation associated with loss of alveolar bone.
4. Advanced disease features include pus and exudates.
5. Essential aspects of successful treatment of periodontal disease include initial debridement and maintenance of proper oral hygiene.

The advent of microscopy allowed later studies performed at the turn of the 19th century to report the histological structures and features of periodontal lesions, but most were limited to advanced stages of the disease. High correlation with protozoa *Entamoeba gingivalis* and *Trichomonas tenax* was then established.[26] Progress in microscopy in the 1960s, such as advances in histopathology and stereology, allowed researchers to focus on earlier stages of inflammatory processes while the innovation of experimentally-induced periodontal disease in both human and animal models allowed for more detailed research into the temporal progression of the pathogenesis of plaque-induced periodontal disease.[6]

Historically, chronic plaque-induced periodontal diseases were divided into three categories:[10]

1. subclinical gingivitis
2. clinical gingivitis
3. periodontal breakdown

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External links

- American Academy of Periodontology Home Page (http://www.perio.org)
- Periodontal Disease as a Specific, albeit Chronic, Infection: Diagnosis and Treatment (http://www.ncbi.nlm.nih.gov/pmc/articles/PMC89001/)