RESEARCH PAPER

Exploring the Past, Present, and Future of Periodontitis Management: A Holistic Review

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ABSTRACT

Periodontitis is a chronic multifactorial inflammatory disease that progressively destroys the supporting tissues of the teeth, including the gingiva, periodontal ligament, and alveolar bone. It is primarily initiated by microbial biofilms, particularly dental plaque, which trigger a complex host immune-inflammatory response leading to connective tissue degradation and bone resorption. If left untreated, periodontitis results in tooth mobility, loss, and a decline in oral function and aesthetics. In recent decades, research has established its strong association with systemic disorders such as diabetes mellitus, cardiovascular diseases, rheumatoid arthritis, and inflammatory bowel disease, highlighting its significance beyond the oral cavity. Diagnosis involves clinical evaluation supported by biochemical, microbiological, radiographic, and genetic methods. Conventional mechanical debridement through scaling and root planning remains the foundation of therapy; however, adjunctive strategies like host modulation therapy, locally delivered antimicrobials, and regenerative surgical approaches significantly improve outcomes. Recent advancements in nanotechnology and novel drug delivery systems have transformed periodontal management by enabling site-specific, controlled, and sustained drug release while minimizing systemic side effects. Furthermore, regenerative techniques employing growth factors and biomaterials show promise in restoring lost periodontal structures. Overall, the integration of advanced technologies and a better understanding of host–microbe interactions represent a major step toward personalized and regenerative periodontal therapy.

Keywords: Periodontitis, Dental plaque, Host modulation therapy, Novel drug delivery system.

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INTRODUCTION

Oral health encompasses the condition of the teeth, gingiva, and the entire orofacial complex that facilitates essential functions such as smiling, speaking, and mastication. Periodontitis, commonly known as gum disease, is a prevalent inflammatory condition that leads to the destruction of the soft tissues and alveolar bone supporting the teeth.[1,2] If left untreated, it results in the gradual and progressive loss of the surrounding bone structure. Although periodontitis is widespread, it is largely preventable. The primary contributing factor is inadequate oral hygiene. Maintaining proper oral care through regular tooth brushing, daily flossing, and routine dental examinations significantly enhances treatment outcomes and lowers the risk of disease onset.[3,4] If unmanaged, periodontitis can ultimately cause tooth loss and has been associated with an elevated risk of systemic conditions such as cardiovascular diseases, including stroke and myocardial infarction. The primary etiological factor is dental plaque- a soft, sticky, and colorless biofilm that accumulates on tooth

surfaces. When plaque is not adequately removed, it can mineralize to form calculus (tartar), further exacerbating periodontal tissue destruction.[5]

Gingivitis represents the earliest and mildest manifestation of periodontal disease, affecting nearly 90% of individuals. It is characterized by gingival inflammation resulting from the accumulation of microbial plaque and debris along the gingival margin. This stage is reversible through effective oral hygiene practices.[6] However, in the absence of proper management, gingivitis can advance to periodontitis. Periodontitis is a chronic, progressive inflammatory disorder that leads to the destruction of periodontal supporting tissues. The defining pathological feature is the apical migration of the junctional epithelium, resulting in attachment loss and the formation of periodontal pockets. As pathogenic bacteria invade deeper periodontal structures, the host immune response is triggered to counter the infection.[7-9] Nevertheless, this immune reaction inadvertently contributes to connective tissue breakdown and alveolar bone resorption. With continued disease progression, further attachment loss and bone destruction occur, potentially culminating in tooth loss in advanced stages. In certain instances, gingival inflammation does not advance to periodontitis; however, the onset of periodontitis is invariably preceded by gingival inflammation.[10] Periodontal disease can be effectively prevented through consistent oral hygiene practices, including regular tooth brushing and the use of dental floss. Additionally, mouth rinses containing agents such as hydrogen peroxide, saline, alcohol, or chlorhexidine may serve as beneficial adjuncts in maintaining periodontal health. The use of powered or vibrating toothbrushes has also been shown to lower the risk of periodontitis compared to conventional manual brushing.[11] The primary objective of treatment is to reduce the bacterial load within the oral cavity, which involves maintaining proper oral hygiene at home in conjunction with regular professional dental care. Consequently, several preventive measures used to control gingival inflammation are also applicable in the management of established gingival disease. These include procedures such as scaling, root planning. According to the World Health Organization (WHO), periodontal disease is a highly prevalent chronic condition affecting populations worldwide. Its onset is typically associated with the accumulation of dental plaque on tooth surfaces, leading to the formation of microbial biofilms that harbor pathogenic bacteria and initiate localized gingival inflammation. If this condition is neglected, it may progress into a chronic form of periodontal disease. At this advanced stage, the structural components of the periodontium are damaged by the harmful enzymes and toxic metabolites released by periodontal pathogens.[12] These include leukotoxins, collagenase, and fibrinolytic enzymes produced by various microorganisms such as Bacteroides species (B. intermedius, B. gingivalis), fusiform bacteria like Actinobacillus actinomycetemcomitans, Wolinella recta, Eikenella species, Porphyromonas gingivalis, Tannerella forsythia, as well as diverse bacilli, cocci, spirochetes, amoebae, and Trichomonas species. If dental plaque is not eliminated during its early stage, it mineralizes to form tartar or calculus, which cannot be removed by routine toothbrushing or flossing. The presence of calculus facilitates bacterial invasion into deeper periodontal tissues, leading to the degradation of periodontal ligaments and subsequent resorption of the alveolar bone. This process results in the formation of a space between the gingiva and the tooth, known as a “periodontal pocket,” a hallmark feature of periodontitis or periodontal disease. The severity of the condition is largely determined by the extent and persistence of microbial plaque accumulation.[13] Periodontitis is a chronic inflammatory disorder initiated by pathogenic bacteria residing in the oral cavity. The persistence of inflammation leads to progressive destruction of periodontal tissues, including the gingiva, periodontal ligament, cementum, and alveolar bone. Severe alveolar bone loss results in diminished tooth support, ultimately causing tooth exfoliation. Tooth loss, in turn, leads to occlusal instability and a consequent decline in overall quality of life. In recent years, regenerative therapies have been developed with the aim of restoring periodontal tissues damaged by periodontitis; however, complete regeneration to the original structural and functional state remains a challenge. Moreover, periodontitis has been implicated in the development of various systemic diseases. Therefore, a comprehensive understanding of its pathogenesis is essential for advancing effective therapeutic strategies and improving disease management.[14,15] The management of periodontitis primarily involves the removal or reduction of dental plaque through mechanical debridement. However, this approach has certain limitations, particularly in accessing anatomically complex regions such as root concavities, furcation areas, and other oral sites that may harbor bacterial reservoirs. Despite thorough debridement, recolonization of periodontal pathogens within the periodontal pocket can occur within approximately 60 days, leading to recurrent infection and necessitating the use of adjunctive therapeutic agents. Various adjunctive modalities have therefore been employed alongside meticulous scaling and root planing to enhance the control and treatment of periodontitis. According to the Global Burden of Disease Study (2016), severe periodontal disease ranked as the 11th most prevalent condition globally. The reported prevalence of periodontal disease ranges between 20% and 50% worldwide. It is a leading cause of tooth loss, which can negatively impact mastication, aesthetics, self-esteem, and overall quality of life. The worldwide prevalence of periodontal disease is projected to continue rising in the coming years, primarily due to the growing aging population and the increased retention of natural teeth resulting from a decline in tooth loss among older individuals.[16-20]

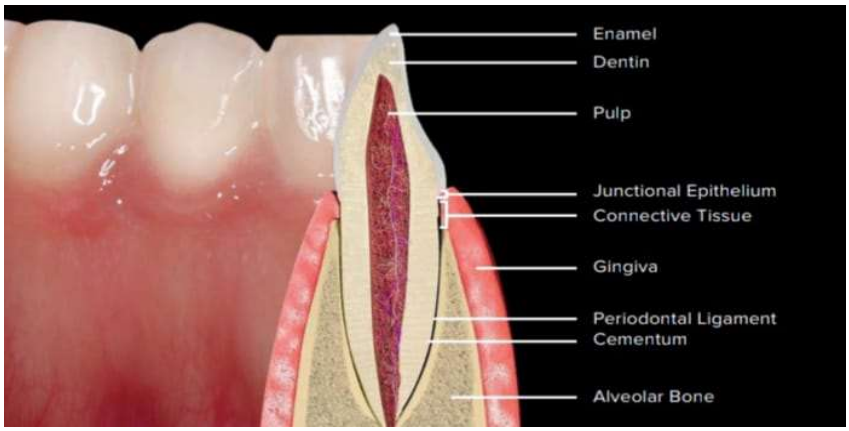

Fig. No. 1: The Periodontal Apparatus

ETIOPATHOGENESIS

Role of dental plaque and biofilm

Dental plaque is a complex microbial community that adheres to the tooth surface in the form of a biofilm, enclosed within a matrix composed of both host- and bacteria-derived polymers. Clinically, this is significant because biofilms exhibit reduced susceptibility to antimicrobial agents, and the microorganisms within them often demonstrate increased pathogenic potential through synergistic interactions. The structural organization of the plaque biofilm can impede the diffusion of antimicrobial substances, while bacteria attached to surfaces tend to grow more slowly and adopt distinct phenotypic traits, including decreased sensitivity to inhibitory agents. Despite its pathogenic potential, dental plaque is a natural component of the oral microbiota and plays a role—similar to other resident microbial communities in the body—in supporting the normal physiological development and immune defenses of the host.[21-23]

The complex composition of bacterial species and matrix components within dental biofilms makes understanding their exact role in periodontal disease pathogenesis challenging, requiring advanced multispecies models. In healthy conditions, symbiotic biofilms formed by resident microbiota help prevent colonization by pathogenic species. However, disruption of this balance leads to dysbiosis and the onset of periodontitis. Neutrophils (PMNs), while protective, contribute to inflammation-driven tissue destruction, aided by immunological factors such as antibodies, complement proteins, and cytokines. Specific T-cell subsets further sustain chronic inflammation and oxidative stress. Porphyromonas gingivalis, a key dysbiosis-associated pathogen, promotes immune evasion, inflammation, and overgrowth of pathogenic bacteria despite its low abundance. Understanding these host–microbe interactions is crucial for developing future therapies to prevent dysbiosis and manage periodontitis. A key feature of biofilms is their antimicrobial resistance (AMR), which refers to the capacity of microorganisms to survive or resist the effects of antimicrobial agents intended to eliminate or inhibit their growth. This phenomenon represents a major global health concern, as biofilm-associated infections, including those affecting the oral cavity, are particularly challenging to manage and are linked to increased morbidity rates. saliva and dental plaque serve as useful samples for studying the onset and progression of periodontitis. A balanced relationship between oral microorganisms and the host is essential for maintaining oral health, while disturbances in this balance lead to dysbiosis and periodontal disease. In healthy conditions, gram-positive bacteria such as Actinomyces and Streptococcus dominate, whereas poor oral hygiene promotes gram- negative and motile species. Dysbiosis reduces microbial diversity and triggers an exaggerated inflammatory response that damages periodontal tissues, which in turn alters the microbiome and bacterial virulence. Advanced techniques like metagenomics and metatranscriptomics have enhanced understanding of these microbial and host interactions in periodontitis and related systemic diseases.[24,25]

RISK FACTORS FOR PERIODONTITIS

1. Smoking: Cigarette smoking is a significant and modifiable risk factor for chronic periodontitis, as demonstrated by various epidemiological, longitudinal, and interventional studies, with attributable risk estimates ranging from 2.5 to 7.0. Smokers generally exhibit poorer periodontal health and greater tooth loss compared to non-smokers, even after controlling for confounding variables. Long-term studies indicate that smokers experience faster progression of chronic periodontitis and increased tooth loss, while both non-surgical and surgical periodontal treatments yield less favorable outcomes in this population. Interestingly, clinical signs of gingival inflammation may appear less evident in smokers due to nicotine-induced vasoconstriction and increased keratinization of gingival tissues.[26,27]

2. Stress: Individuals with poor stress management or defensive coping behaviors are at an increased risk of developing severe periodontal disease. Psychological stress has been linked to inadequate oral hygiene, elevated glucocorticoid levels leading to suppressed immune responses, heightened insulin resistance, and a greater susceptibility to periodontitis. Research indicates that

men experiencing daily anger have a 43% higher likelihood of developing periodontitis compared to those who rarely feel anger. Furthermore, indicators of periodontal disease, such as tooth loss and gingival bleeding, have been correlated with occupational stress and financial strain.[28,29]

3. Drug Induced Disorders: Certain medications can markedly reduce salivary flow, including antihypertensive agents, narcotic analgesics, tranquilizers, sedatives, antihistamines, and antimetabolites. Additionally, drugs in liquid or chewable formulations that contain added sugars can modify the pH and composition of dental plaque, enhancing its ability to adhere to tooth surfaces. Pharmacological agents may also contribute to the development of periodontal diseases; for example, anticonvulsants, calcium channel blockers, and cyclosporine are known to induce gingival overgrowth.[30,31]

4. Age: Multiple studies have demonstrated that both the prevalence and severity of periodontal disease increase with advancing age. Papapanou et al. reported that the mean annual rate of alveolar bone loss was 0.28 mm in individuals aged 70 years, compared to 0.07 mm in those aged 25 years. The heightened severity of periodontal destruction and bone loss in older adults is likely due to prolonged exposure of periodontal tissues to bacterial plaque, representing a cumulative effect of an individual's oral health history. Research from developed countries indicates evolving trends in the progression of periodontal disease, showing that severe periodontal destruction and bone loss are rarely observed in individuals younger than 40 years. Similar trends have been noted in elderly populations, where only a small proportion exhibit advanced disease. Nonetheless, in individuals already affected by severe periodontitis, further periodontal breakdown tends to occur with increasing age.[32,33]

5. Gender: Associations between gender and periodontal disease have shown inconsistent findings. One study, after controlling for factors such as oral hygiene, socioeconomic status, and age, reported that males exhibited significantly greater clinical attachment and bone loss. Research involving postmenopausal women suggests that estrogen may play a role in these gender-related differences in periodontal disease. In a comparative study, postmenopausal women undergoing estrogen supplementation demonstrated notably less gingival bleeding compared to those not receiving hormone replacement therapy. Another investigation indicated that estrogen supplementation could be linked to reduced gingival inflammation and a lower incidence of clinical attachment loss in osteopenic or osteoporotic women during early menopause.[34,35]

SYSTEMATIC DISEASES LINKED TO PERIODONTITIS:

1. Inflammatory bowel disease: There is strong two-way relationship between periodontitis and inflammatory bowel disease (IBD). Patients with IBD have a higher prevalence and severity of periodontitis compared to healthy individuals, indicating that intestinal health affects oral health. At the same time, poor periodontal health can worsen gut inflammation and contribute to IBD progression. Oral bacteria linked to periodontitis are often found in the intestinal tissues of IBD patients, suggesting that oral pathogens can migrate and disturb gut balance. The immune response activated in gum tissues can also influence inflammation in the gut, creating a shared inflammatory link. Overall, both diseases are connected through microbial imbalance and immune system interactions, where oral inflammation can aggravate intestinal disease and vice versa.[36]

2. Diabetes mellitus: Periodontitis and diabetes mellitus are closely linked through a two-way relationship. Poorly controlled diabetes increases the risk and severity of periodontitis due to impaired immune response and delayed healing. In turn, chronic inflammation from periodontitis can worsen blood sugar control and increase insulin resistance. This makes managing diabetes more difficult and raises insulin requirements. Diabetes also affects bone health by reducing the growth and function of cells that form bone, leading to more periodontal tissue damage. Additionally, inflammation in diabetes enhances bacterial growth and worsens gum destruction. Overall, both diseases influence and worsen each other through inflammation and metabolic imbalance.[37]

3. Cardiovascular diseases: There is a strong link between cardiovascular diseases and periodontitis. Research shows that periodontitis increases the risk of developing heart diseases such as coronary heart disease, stroke, and peripheral artery disease. This connection exists even after accounting for other risk factors like smoking, diabetes, and socioeconomic status. People with periodontitis are more likely to experience heart problems due to chronic inflammation and the spread of bacteria from the gums into the bloodstream. The overall risk of cardiovascular disease is found to be higher, especially in older adults. Studies also suggest that individuals with gum disease have greater chances of developing heart-related conditions than those with healthy gums. Overall, periodontitis contributes to worsening cardiovascular health through systemic inflammation and bacterial effects.[38]

4. Rheumatoid arthritis: Periodontal disease is common in people with rheumatoid arthritis (RA) and is believed to play a role in triggering the autoimmune response seen in RA. Both conditions share similar disease-causing mechanisms. People with RA often experience bone loss around the teeth and tooth loss, which are also typical outcomes of periodontal disease.[39]

5. Respiratory tract diseases: There is a clear connection between periodontitis and respiratory tract diseases. In people with periodontal disease, saliva and dental plaque can harbor harmful bacteria that may travel from the mouth to the lungs. These oral pathogens can cause or worsen respiratory infections such as pneumonia, bronchitis, and chronic obstructive pulmonary disease (COPD). The bacteria from dental plaque have been found in the lungs of infected patients, showing that the mouth can act as a source of infection. In addition, inflammatory substances and enzymes produced during periodontitis can damage the respiratory tract and increase infection risk. Some oral

bacteria, like Fusobacterium and Chlamydia pneumoniae, can also contribute to serious complications such as bloodstream infections and heart disease. Overall, poor oral health and periodontitis increase the likelihood of respiratory infections and related systemic diseases.[40]

6. Parkinson's disease: There is a noticeable link between Parkinson's disease and periodontitis. People with Parkinson's disease are more likely to have gum problems, including deeper periodontal pockets and higher rates of periodontitis, than those without the condition. This may be because motor difficulties and cognitive decline in Parkinson's patients make it harder to maintain proper oral hygiene. Additionally, both diseases share an inflammatory component, suggesting that chronic inflammation from periodontitis might worsen or contribute to nerve damage in Parkinson's disease. Although more research is needed, evidence indicates that inflammation caused by gum disease could play a role in the early development or progression of Parkinson's disease.[41]

TYPES AND STAGES

Types of Periodontal Diseases

1. Gingivitis: Gingivitis represents the initial and reversible stage of periodontal disease characterized by inflammation of the gingival tissues without any loss of connective tissue attachment or bone. It commonly results from the accumulation of dental plaque and can be effectively managed through proper oral hygiene practices, including regular brushing, flossing, and professional dental cleaning.[43]

2. Chronic Periodontitis: Chronic periodontitis is the most prevalent form of periodontal disease and is characterized by persistent inflammation of the gingiva, leading to progressive destruction of the periodontal ligament, alveolar bone, and connective tissues. Clinical signs include bleeding on brushing, halitosis (bad breath), gingival recession, and tooth mobility. Unlike gingivitis, tissue and bone loss in chronic periodontitis are irreversible, making early detection and management crucial to prevent tooth loss.[44]

3. Aggressive Periodontitis: Aggressive periodontitis can occur in localized or generalized forms and typically manifests at an early age, often between puberty and the third decade of life. It is marked by rapid attachment loss and bone destruction that may occur in otherwise healthy individuals. The condition may have a strong genetic component and involves a hyper-responsive immune reaction to specific periodontal pathogens.[45]

4. Necrotizing Ulcerative Gingivitis (NUG): Necrotizing ulcerative gingivitis is an acute infection characterized by painful ulceration, necrosis of the gingival tissue, and bleeding. It predominantly affects individuals with compromised immunity, malnutrition, or those suffering from systemic conditions such as HIV infection. The necrosis is primarily due to inadequate host defense and poor nutritional status, leading to tissue breakdown.[46]

5. Peri-Implant Mucositis: Peri-implant mucositis is an inflammatory condition affecting the soft tissues surrounding dental implants, without evidence of bone loss. Clinical features include redness, swelling, tenderness, and bleeding on brushing around the implant site. It is considered a reversible condition if managed early, but if left untreated, it may progress to peri-implantitis, involving bone resorption.[47]

6. Systemic Chronic Periodontitis: Systemic chronic periodontitis refers to periodontal inflammation associated with underlying systemic diseases such as diabetes mellitus, cardiovascular disease, and respiratory disorders. The systemic condition alters the host immune response, increasing susceptibility to periodontal breakdown. This bidirectional relationship highlights the impact of systemic health on periodontal status and the importance of an integrated approach to management. **[48]**

Stages of Periodontal Disease

Periodontal disease progresses through four major stages, each characterized by distinct clinical symptoms and radiographic findings, as described below:

1. Gingivitis: Gingivitis represents the initial and reversible stage of periodontal disease. It occurs due to plaque accumulation around the teeth and is usually painless. Common symptoms include bad breath, redness, swelling of gums, and bleeding during brushing or flossing. With proper oral hygiene practices and regular dental checkups, this stage can be fully reversed. Clinically, there is a 1–2 mm attachment loss, less than 15% bone loss around the root, and probing depth of 4 mm or less.

2. Early Stage (Initial Periodontitis): The early stage marks the beginning of irreversible tissue damage. Infection starts spreading to the supporting structures of the teeth, leading to gum inflammation, persistent bad breath, and bleeding during brushing. Small gaps or spacing between teeth may become visible and gradually increase. At this stage, 3–4 mm of clinical attachment loss and 15–33% bone loss are usually present, with probing depths of up to 5 mm. Although irreversible, the condition can still be managed through improved oral care and professional dental treatment.

3. Moderate Stage (Moderate Periodontitis): In this stage, the destruction of supporting periodontal tissues becomes more evident. The symptoms are similar to those of the earlier stage but more severe, including increased gum recession, tooth spacing, and sensitivity. Professional treatments such as deep cleaning, scaling, root planing, or flap surgery are often required. Typically, there is around 5 mm or more clinical attachment loss, tooth loss involving four or fewer teeth, probing depths exceeding 6 mm, and presence of Class II–III furcations or moderate ridge defects.

4. Advanced Stage (Severe Periodontitis): This is the final and most severe stage of periodontal disease, involving 50–90% loss of periodontal supporting tissues. Symptoms include swollen and pus-discharging gums, tooth mobility, pain during chewing, cold sensitivity, and severe halitosis. If untreated, it may lead to widening gaps between teeth and gums, gum recession, bite collapse, and eventual tooth loss, often requiring dentures. Radiographically, secondary occlusal trauma, severe ridge defects, and fewer than 20

remaining teeth (10 opposing pairs) may be observed. Regular dental care, professional cleaning, and good oral hygiene can help prevent further disease progression.[49,50]

Fig. No. 2: Stages of Periodontitis

CLINICAL FEATURES AND SYMPTOMS

Gingivitis

Gingivitis is a bacterially induced, chronic inflammatory condition of the gingival tissues. It represents the earliest and completely reversible stage of periodontal disease. The inflammation
results primarily from plaque accumulation along the gingival margin, leading to local irritation and vascular changes in the soft tissue. With proper oral hygiene and professional cleaning, gingivitis can be fully resolved without any permanent tissue damage.

The common clinical signs and symptoms of gingivitis, listed in descending order of frequency, include:

- Bleeding gums, especially during brushing or flossing.
- Swollen, soft, or puffy gums.
- Redness or change in gum color (erythematous appearance).
- Build-up of dental plaque or calculus (tartar) on tooth surfaces.
- Persistent bad breath (halitosis).
- Tenderness or mild discomfort in the gums.
- Shiny or smooth gum surface due to inflammation.

If left untreated, gingivitis can gradually progress to periodontitis, resulting in irreversible tissue destruction.

Periodontitis: Periodontitis is a chronic, irreversible inflammatory disease that affects the supporting structures of the teeth, including the periodontal ligament (PDL), cementum, and alveolar bone. It usually develops from untreated gingivitis and leads to progressive destruction of connective tissue attachment and bone, eventually causing tooth mobility and loss if not managed promptly.[51]

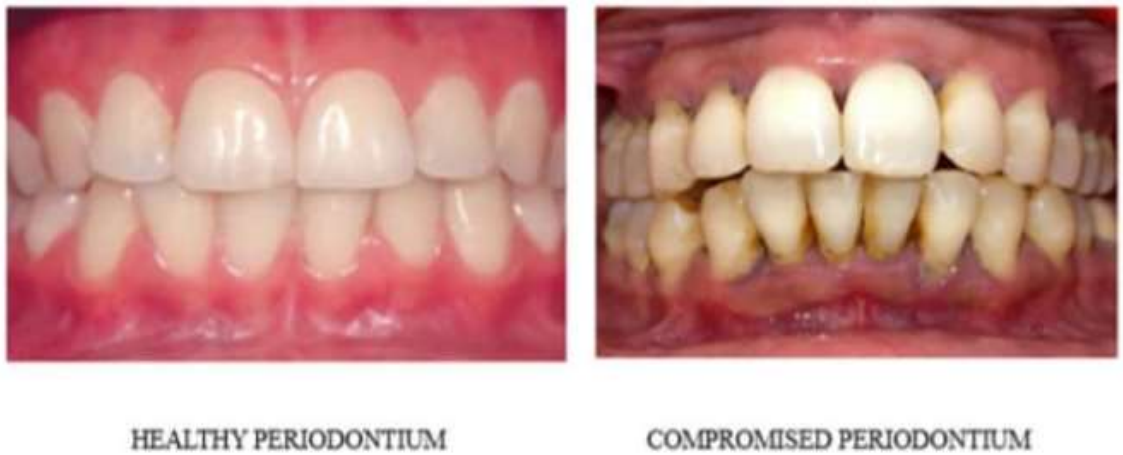

Fig No. 3: Healthy Vs Compromised Periodontium

The characteristic clinical features of periodontitis include:[52,53]

- Bleeding and pus discharge (suppuration) from the gums.
- Receding gums leading to visible tooth roots.
- Deep periodontal pockets on probing.
- Gaps or spaces developing between teeth (pathologic tooth migration).
- Loose or mobile teeth.
- Persistent foul taste or bad breath.
- Pain or discomfort while chewing.
- Gum recession and exposed root surfaces causing sensitivity.
- Changes in tooth alignment or bite due to bone loss.
- Partial or complete tooth loss in advanced cases.

DIAGNOSIS OF PERIODONTITIS

1. Bleeding on probing: Diagnosis of periodontitis using bleeding on probing (BOP) is based on the observation of bleeding from the gums when gentle probing is done around the teeth. The presence of bleeding indicates inflammation in the periodontal tissues, while its absence suggests periodontal stability and healthy gums. Sites that bleed on probing are more likely to show disease progression compared to those without bleeding. Persistent gingival bleeding over time is associated with future periodontal breakdown. The predictive value of BOP increases when it occurs along with deep periodontal pockets (≥6 mm). However, factors like smoking can reduce bleeding response, which may affect accuracy. Thus, BOP serves as a simple, non-invasive, and useful clinical marker for diagnosing and monitoring periodontal inflammation and disease activity.[54]

2. Periodontal probing: Periodontal probing is a key diagnostic method used to assess the depth of gum pockets around teeth, which helps determine the severity of periodontitis. A thin, calibrated instrument called a periodontal probe is gently inserted into the space between the tooth and gum to measure pocket depth and clinical attachment loss (CAL). These measurements indicate the extent of tissue destruction and bone loss. Over time, different generations of probes have been developed to improve accuracy and consistency. Manual, pressure-controlled, and automated probes provide precise readings of periodontal pockets. Despite their usefulness, factors like probe angulation, tissue condition, probing force, and patient discomfort can affect accuracy. Overall, probing remains an essential, simple, and reliable clinical method to detect and monitor periodontal disease. [55]

3. Radiographic assessment: Radiographic assessment is an important method for diagnosing periodontitis as it helps visualize bone loss and structural changes around the teeth. It allows evaluation of alveolar bone height and detection of bone defects that indicate disease progression. When combined with clinical probing, radiographs provide a clearer picture of periodontal health. However, their accuracy can be affected by factors like probing force, site anatomy, and projection errors. Intraoral radiographs may show overlapping anatomical structures and lack three-dimensional detail, making it difficult to distinguish certain bone areas such as furcations. Despite these limitations, intraoral and digital radiographs remain the most commonly used and reliable tools in dental practice for assessing the extent of periodontal damage and supporting diagnosis.[56]

4. Biochemical tests

a. Oral Fluid Nano Sensor Test (OFNASET): This test detects specific salivary proteins and genetic markers linked to oral diseases. It identifies biomarkers such as IL-8, IL-1β, and thioredoxin in saliva with high sensitivity and accuracy. Although mainly used for oral cancer screening, it demonstrates the potential of saliva- based diagnostics for periodontal disease detection.[57]

b. Electronic Taste Chip (ETC): This chip-based system measures C-reactive protein (CRP) levels in saliva, which increase during inflammation in periodontal disease. It can distinguish between healthy and diseased individuals by detecting very low concentrations of CRP. The method is faster and more sensitive than conventional ELISA tests.

c. OraQuick Test: OraQuick is a rapid, point-of-care diagnostic test that provides quick results using oral fluids. While originally designed for HIV detection, its principle demonstrates how saliva-based rapid tests can be adapted for detecting periodontal and other oral infections effectively within minutes.[58]

d. Integrated Microfluidic Platform for Oral Diagnostics (IMPOD): IMPOD is a handheld device that measures salivary biomarkers like MMP-8, which are elevated in periodontitis. It provides quick, accurate, and non-invasive detection of periodontal inflammation using small saliva samples, helping in early disease diagnosis and monitoring.[59]

5. Saliva analysis: Diagnosis of periodontitis through saliva analysis is based on detecting various biochemical and physical changes in saliva that reflect oral inflammation. Key parameters such as pH, buffer capacity, osmolarity, calcium, phosphate, and protein levels are evaluated, as they tend to alter in periodontal disease. Increased levels of inflammatory markers like alkaline phosphatase and lactoferrin indicate tissue destruction and active inflammation. The presence of leukocytes, nitrites, and blood components in saliva also suggests infection and periodontal breakdown. Changes in salivary viscosity and composition can further differentiate healthy from diseased individuals. Overall, saliva analysis offers a simple, non-invasive, and effective method for early detection and monitoring of periodontitis.[60]

6. Microbiological Tests

a. My PerioPath: This test identifies the specific bacteria responsible for periodontitis by analyzing saliva samples. It detects both the type and number of periodontal pathogens, helping determine the severity and cause of infection.[61]

b. Omnigene: This test uses DNA probes to identify major

periodontal pathogens such as P. gingivalis, A. actinomycetemcomitans, and T. denticola. It provides quick and accurate results, enabling clinicians to diagnose infection and plan targeted periodontal therapy effectively.[62]

7. Genetic testing: Genetic testing helps in diagnosing periodontitis by identifying specific genetic variations that increase a person's susceptibility to the disease. Certain gene polymorphisms, especially in inflammatory genes like IL-1 and IL-6, are linked to severe or chronic forms of periodontitis. These tests can reveal an individual's inherited risk, allowing early prevention and personalized treatment plans. They also help explain why some people develop advanced periodontal disease despite good oral hygiene. Genetic analysis thus serves as a valuable tool to predict disease risk, guide clinical decisions, and improve long-term periodontal health management.[63]

Conventional and non-surgical treatments

1. Scaling and root planing: Scaling and root planing (SRP) is a fundamental non-surgical approach used to treat periodontitis by thoroughly cleaning the tooth and root surfaces. Its main objective is to eliminate plaque, calculus, and endotoxins that cause gingival inflammation. The procedure promotes healing of periodontal tissues and reattachment of the gingiva to the tooth surface. Both manual and ultrasonic instruments effectively remove subgingival deposits and reduce bacterial load. The outcome depends on the operator's skill, access to deep pockets, and thoroughness of cleaning. Ultrasonic scalers are preferred as they are faster, efficient, and safe for surrounding tissues. SRP significantly helps in reducing bleeding, pocket depth, and inflammation, leading to overall improvement in gingival health and stability of periodontal support. Regular follow-up and maintenance therapy enhance the long-term success of this treatment. [64]

2. Locally delivered antibiotics: Locally delivered antibiotics are an important adjunctive non- surgical treatment for periodontitis. They are applied directly into the periodontal pocket to target the bacteria responsible for the infection. This method helps in achieving a high concentration of the drug at the diseased site while minimizing systemic side effects. There are several ways to deliver these agents locally, such as subgingival irrigation, mouth rinses, and controlled-release systems like gels, fibers, or microspheres. However, subgingival irrigation is less effective because the medication washes out quickly and doesn't stay long enough to act on the biofilm. Mouth rinses and toothpastes help control supragingival bacteria and reduce gum inflammation but cannot reach deep periodontal pockets effectively. In contrast, locally delivered antimicrobial systems are designed to stay in the pocket for an extended period, maintaining a therapeutic drug concentration. Common agents used include tetracycline, doxycycline, minocycline, metronidazole, azithromycin, and chlorhexidine, each with proven antibacterial activity against periodontal pathogens. These local delivery systems help in reducing bacterial load, inflammation, and pocket depth, and in promoting better healing of the periodontal tissues. Overall, local antibiotic therapy enhances the effectiveness of scaling and root planing and contributes to the long-term maintenance of periodontitis.[65]

Table No. 1: Types of locally delivered antibiotics

Tetracyclines	Broad- spectrum bacteriostatic antibiotics for Gram-positive and Gram-negative bacteria.
Doxycycines	Is a second-generation long- acting tetracycline antibiotic
Metronidazole	Chemotherapeutically active against that can kill Gram-positive and Gram-negative bacteria.
Minocycline	Is a semi-synthetic tetracycline antibiotic that is most lipid soluble and active. It targets bacteria with and without cell wall, as well as both Gram-positive and Gram-negative bacteria.

3. Host modulation therapy: Host Modulation Therapy (HMT) is an advanced adjunctive approach in the treatment of periodontitis that focuses on modifying the host's inflammatory response rather than just eliminating bacteria. It is used along with conventional treatments like scaling and root planing (SRP) and good oral hygiene. HMT aims to reduce excessive inflammation, tissue destruction, and bone loss caused by overactive host enzymes and cytokines. Drugs such as nonsteroidal anti-inflammatory agents (NSAIDs) and sub-antimicrobial doses of doxycycline (Periostat®) help inhibit enzymes like matrix metalloproteinases (MMPs) that degrade collagen and other periodontal tissues.[66]

This therapy helps in stabilizing the periodontium, reducing pocket depth, and improving tissue attachment. Chemically modified tetracyclines (CMTs) and curcumin-based compounds have also shown anti-inflammatory and MMP-inhibitory effects. Other potential HMT approaches include anti-cytokine therapy, probiotics, omega-3 fatty acids and resolvins, stem cell therapy, and complement inhibitors, all targeting different aspects of host response regulation. Overall, HMT helps achieve better control of inflammation, supports tissue healing, and enhances the long-term success of periodontal treatment when used alongside conventional mechanical therapy.[67]

SURGICAL/ADVANCED TREATMENTS

1. Bone grafting: Bone grafting is a surgical technique used in periodontitis to restore bone lost due to infection and inflammation. It involves placing graft material into the defective area to act as a filler and scaffold for new bone growth. These grafts are bioresorbable, biocompatible, and free from immune reactions. Bone grafts can be taken from the patient (autografts), another human donor (allografts), animals (xenografts), or made synthetically (alloplasts). As healing occurs, the natural bone gradually replaces the graft

material, resulting in complete regeneration. The main biological processes involved are osteoconduction, osteoinduction, and osteogenesis. This regeneration helps in restoring tooth support and function. Bone grafts, along with guided tissue regeneration and biomaterials, have significantly improved outcomes in periodontal therapy.[68]

2. Laser therapy: Laser therapy in periodontitis is used as an advanced surgical approach to remove infection and promote healing in the gums and surrounding tissues. It helps destroy or suppress disease-causing bacteria present on tooth surfaces and within periodontal pockets. Lasers provide precise and controlled removal of infected tissue while minimizing bleeding and discomfort. They also improve visibility and access to deep pockets or bony defects that are difficult to clean through nonsurgical methods. The laser energy stimulates tissue regeneration and enhances wound healing. Different types of lasers, such as diode, Nd:YAG, and Er:YAG, are used depending on the clinical need. Overall, laser therapy supports better infection control, reduced inflammation, and faster recovery compared to conventional methods.[69]

3. Photodynamic therapy: Photodynamic therapy (PDT) is a modern, non-invasive treatment used to eliminate bacteria in periodontitis. It involves applying a special dye called a photosensitizer (like methylene blue or toluidine blue) to the infected gums. This dye is then activated by light of a specific wavelength, producing reactive oxygen species (ROS) that destroy harmful bacteria and disrupt their cell walls and DNA. PDT effectively targets bacteria even in deep periodontal pockets and biofilms that are hard to remove by scaling alone. It also promotes tissue healing by stimulating new blood vessel and collagen formation. PDT can be used along with scaling and root planning to improve treatment results. It is safe, painless, and reduces the risk of antibiotic resistance. Overall, PDT helps in infection control, faster healing, and better periodontal health.[70]

4. Use of growth factors: Growth factors are natural proteins that help in the regeneration and healing of periodontal tissues damaged by periodontitis. In regenerative periodontal therapy, they stimulate the growth and differentiation of periodontal ligament (PDL) cells, promoting the formation of new bone, cementum, and connective tissue. To deliver these growth factors effectively, special systems like scaffolds, gels, or membranes are used to release them slowly and continuously at the target site. These delivery systems mimic the natural extracellular matrix (ECM), providing a suitable environment for cell growth. Materials such as collagen, hydroxyapatite, and biodegradable polymers are often used as carriers. Growth factors enhance tissue repair by encouraging cell migration, angiogenesis, and collagen synthesis. Different systems, like reservoir or biologically inspired systems, control how the growth factors are released. This controlled delivery ensures long-term effectiveness and minimizes side effects. Overall, growth factor therapy offers a promising approach for regenerating lost periodontal structures and improving treatment outcomes in periodontitis.[71]

5. Flap surgery: Flap surgery is a common surgical procedure used in periodontitis to gain direct access to the tooth roots and underlying bone for thorough cleaning. It involves carefully lifting a section of the gum tissue (flap) away from the tooth to remove plaque, tartar, and infected tissue from deep periodontal pockets. This allows for better visualization and debridement of the affected root surfaces and bone. After cleaning, the flap is repositioned and sutured back to promote healing and reduce pocket depth. Traditional flap surgery involves wider incisions for greater access, while modern Minimally Invasive Periodontal Surgery (MIPS) uses smaller openings and specialized instruments to reduce trauma. MIPS shortens surgical time, minimizes patient discomfort, and enhances healing. Both techniques aim to eliminate infection, reduce inflammation, and allow reattachment of healthy tissue. Overall, flap surgery helps in controlling disease progression and preserving natural teeth.[72]

ADVANCED THERAPIES AND CURRENT RESEARCH TRENDS

Nanotechnology and novel drug delivery systems have emerged as advanced therapeutic strategies for managing complex diseases like periodontitis. These approaches enable targeted, controlled, and sustained release of therapeutic agents directly to periodontal sites, improving efficacy and reducing systemic side effects. Current research focuses on nanoparticle-based formulations, liposomes, hydrogels, and microspheres to enhance drug stability and bioavailability. Such innovations hold great promise for regenerative therapy and effective management of periodontal inflammation and tissue destruction.[73-75]

Nanotechnology plays a big role in improving the prevention and treatment of periodontitis. Because nanomaterials are extremely small (less than 100 nm), they can easily reach deeper areas of the gums and tooth surfaces where normal treatments may not work well. Their large surface area allows better interaction with cells and bacteria, helping in faster healing. Nanoparticles can be used to deliver drugs directly to infected periodontal sites, maintaining effective drug levels for longer periods. They can also be used in coatings for dental implants to prevent bacterial growth and promote tissue attachment. Nanomaterials like nano-hydroxyapatite help in bone regeneration by mimicking natural bone structure. Nanofibers and nanocomposites are used in scaffolds for tissue engineering, encouraging gum and bone repair. Nanotechnology also improves diagnostic methods by enabling early detection of periodontal pathogens. Overall, it offers targeted, efficient, and faster healing options for managing periodontal disease.[76-78]

Novel drug delivery systems (NDDS) provide targeted and sustained delivery of therapeutic agents directly into periodontal pockets, which improves clinical outcomes compared to conventional therapy. Localized delivery allows higher drug concentrations at the site of infection while minimizing systemic exposure and adverse effects. Polymers such as polycaprolactone, chitosan, PLGA, and hydrogels have been utilized to create nanofibers,

nanoparticles, microspheres, liposomes, and gels that can efficiently load and gradually release antibiotics like metronidazole, doxycycline, minocycline, and azithromycin. These delivery platforms have shown significant improvements in pocket depth reduction, bleeding on probing, plaque index, and suppression of key pathogens including P. gingivalis and A. actinomycetemcomitans.NDDS enhance penetration through the dense biofilm and periodontal tissues, overcoming limitations of conventional antibiotics affected by biofilm-mediated resistance. Sustained-release formulations maintain therapeutic drug levels for longer durations, reducing the need for frequent administration. Biodegradable polymers further support tissue repair and regeneration by serving as scaffolds for healing. Some nanocarrier systems additionally exhibit anti- inflammatory actions by reducing cytokines such as TNF-α and MMP-8, which are involved in tissue destruction.[79-82]

Overall, NDDS support precision therapy by delivering agents site-specifically, enhancing antimicrobial efficacy, limiting systemic resistance, and promoting periodontal tissue regeneration. This approach aligns with modern periodontal therapy.[83-86]

CONCLUSION

Periodontitis remains one of the most prevalent chronic oral diseases worldwide, exerting a profound impact on both oral and systemic health. It originates from the persistent accumulation of bacterial plaque and the ensuing dysbiosis within the oral microbiome, which triggers an excessive immune-inflammatory reaction leading to the destruction of periodontal tissues. The progression of periodontitis is influenced by various risk factors, including poor oral hygiene, smoking, stress, systemic conditions such as diabetes, and the aging process. The interrelationship between periodontitis and systemic diseases underscores the importance of considering it not merely as a localized oral disorder but as a condition with significant systemic implications.

Traditional therapeutic approaches, including scaling, root planing, and surgical debridement, remain the mainstay of treatment and are aimed at reducing bacterial load and promoting periodontal stability. However, these methods often fall short in addressing deep-seated biofilms and recurrent inflammation. Adjunctive modalities such as locally delivered antibiotics, host modulation therapy, and laser or photodynamic therapy have enhanced clinical outcomes by improving infection control and tissue healing. Among recent developments, nanotechnology- based drug delivery systems and biomaterial-assisted regenerative therapies represent a paradigm shift in periodontal treatment. Nanocarriers such as nanoparticles, liposomes, and hydrogels allow targeted, sustained, and controlled release of drugs directly into periodontal pockets, improving therapeutic efficiency while minimizing systemic exposure. These systems also facilitate bone and tissue regeneration by acting as scaffolds for cellular growth.

In addition, the use of growth factors and stem cell-based regenerative approaches has opened new possibilities for restoring periodontal tissues to their original structure and function. The

incorporation of advanced diagnostics such as saliva-based biomarkers, genetic testing, and molecular imaging further strengthens early detection and personalized treatment planning.

Looking ahead, the integration of molecular biology, material science, and clinical dentistry is essential to develop innovative, patient-specific therapeutic strategies. Preventive measures, public health awareness, and regular professional maintenance remain vital for long-term disease control. Ultimately, a multidisciplinary and holistic approach combining prevention, advanced therapeutics, and patient education will redefine the future of periodontal care, contributing not only to oral health but also to overall systemic well-being.

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