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Review Article

An Overview on Periodontitis

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Abstract

Periodontitis is one of the most common oral diseases affecting worldwide. It is characterized by periodontal tissue support breakdown, Smoking, improper oral hygiene, dietary habits, bruxism, tongue thrusting, mouth breathing are known major risk factors of periodontitis. Habits and behaviour to life circumstances affect the immune system and thereby by vital part of periodontal therapy. This review delves into the relationships between chronic periodontitis and other chronic diseases, including cardiovascular diseases, diabetes, cancer, chronic respiratory diseases, and pregnancy complications. We will investigate how shared inflammatory risk factors, such as environmental stressors, contribute to the development of both periodontal diseases and systemic disorders.

Additionally, we will explore how chronic diseases can have impact on oral health. Our analysis will focus on inflammatory cytokines like TNF-alpha, IL-1, and IL-6, which are increased in patients with chronic periodontitis, chronic systemic diseases, and those with co-occurring conditions. By examining these connections, we can gain a deeper understanding of the intricate relationships between oral health and systemic well-being [1].

Keywords: Periodontitis, Clinical features, Staging, Grading, Disease severity, Diagnosis

Introduction

Periodontal disease is a major contributor to the global oral health burden and has common risk factors with various chronic diseases. Recognizing its significance, the World Health Organization (WHO) has emphasized the need for enhanced global control and management of periodontal disease. This acknowledgment underscores the importance of addressing periodontal disease as a public health priority, particularly in the context of its interconnectedness with other chronic conditions [1]." Periodontal disease can be broadly categorized into two main groups:

1. Gingivitis: Diseases that primarily affect the gingiva (gums)
2. Periodontitis: Diseases that impact the underlying periodontal tissues, which support the teeth.

Inflammation is the primary pathological process driving periodontal disease, except in rare cases where other conditions may be present [2]. The progression of periodontal disease is typically measured by:

1. Clinical assessment: Loss of probing attachment
2. Radiographic evaluation: Loss of alveolar bone

Previously, periodontal disease was thought to progress continuously and slowly. However, recent clinical research suggests that the disease course is more complex way, with periods of

1. Exacerbation (worsening)
2. Remission (improvement)

Advanced automated probes have revealed diverse patterns of disease activity, which may be influenced by the disease threshold.

While subtraction radiography remains a valuable diagnostic tool, its widespread adoption is limited by cost and methodological challenges [3].

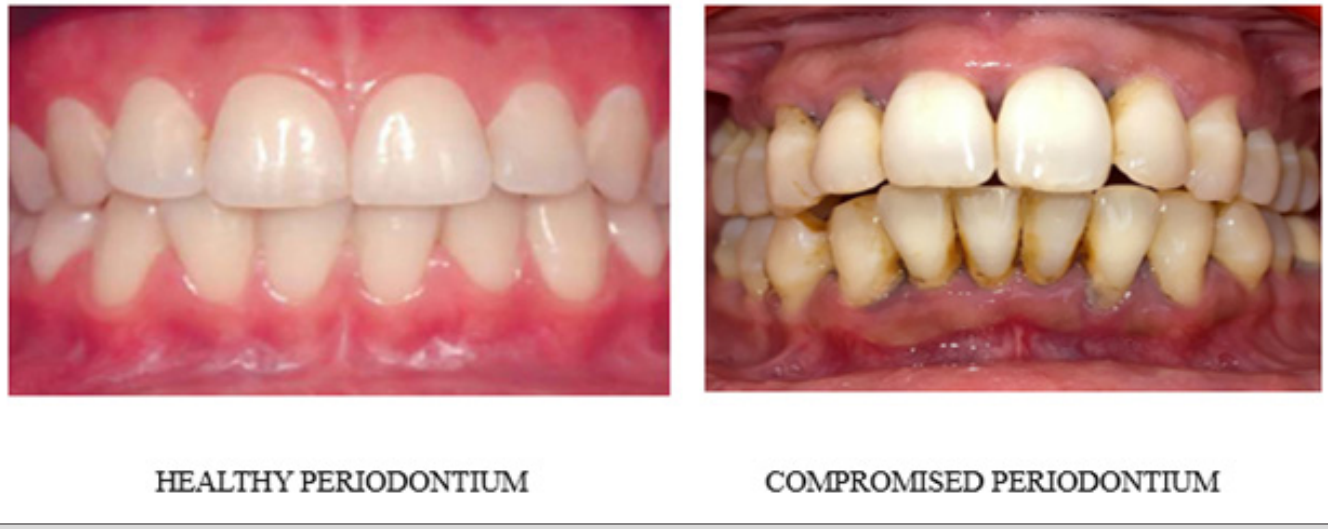

Figure 1:

Classification

In 1989, the American Academy of Periodontology (AAP) held a workshop at Princeton, leading to further revisions in the classification of periodontal diseases. This revised classification remained widely accepted for the next decade and included the following categories:

Table 1:

FORMS OF PERIODONTITIS	DISEASE CHARECTERISTICS
Adult periodontitis	• Age of onset>35years
	• Slow rate of disease progression
	• No defect in host defences
Early -onset periodontitis [may be pre pubertal, juvenile, rapidly progressive	• Age of onset<35 years
	• Rapid rate of disease progression
	• Defect in host defences
	• Associated with specific microflora
Periodontitis associated with systemic diseases	• Systemic diseases that pre dispose to rapid rate of periodontitis
	• Disease: diabetes, down syndrome, HIV infection, papillon-lefevre syndrome
Necrotising ulcerative periodontitis	Similar to acute necrotising ulcerative gingivitis but with associated clinical attachment loss
Refractory periodontitis	Recurrent periodontitis that does not respond to treatment

European workshop classification in periodontology-1993:

Table 2:

FORMS OF PERIODONTITIS	DISEASE CHARECTERESTICS
Adult periodontitis	Age of onset-fourth decades of life
	Slow rate of disease progression
	No defects in host responses
Early -onset periodontitis	• Age of onset- before fourth decades of life
	• Rapid rate of disease progression
	• Defect in host responses
Necrotising periodontitis	Tissue necrosis with attachment and bone loss

AAP international workshop classification-1999:

Table 3:

FORMS OF PERIODONTITIS	DISEASE CHARECTERSTICS
Chronic periodontitis	• Prevalent in adult
	• Slow to moderate rate of progression
	• Environmental factors such as cigarette smoking and emotional stress
Aggressive periodontitis	• Rapid attachment loss and bone destruction
	• Amount of microbial deposits inconsistent with disease severity
Periodontitis as a manifestation of systemic diseases	1. Hemetological disorders
	• Acquired neutropenia
	• Leukemias
	2. Genetic disorders
	• Down syndrome
	• Glycogen storage disease
	• Cohen syndrome
	• Hypophosphatasia
	• others

2017 Classification of Periodontal Disease

The 2017 classification of periodontitis is a system used to diagnose and categorize periodontitis, a chronic inflammatory disease affecting the gums and bone supporting the teeth. This classification was introduced by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) in 2017.

The 2017 classification system includes the following categories:

Table 4:

Necrotizing periodontal disease	Periodontitis	Periodontitis as a manifestation of systemic disease

Staging of Periodontitis:

Table 5:

	PERIODONTITIS	STAGE 1	STAGE 2	STAGE 3	STAGE 4
SEVERITY	Interdental CAL [at the sight of greatest loss]	1-2mm	2-4mm	>5mm	>5mm
	RBL	Coronal third 15-33%	Coronal third 15-33%	Extending to middle third of the root and beyond	Extending to middle third of the root and beyond
	TOOTH LOSS [DUE TO PERIODONTITIS]	No tooth loss		<4 teeth	>5 teeth
COMPLEXITY	Local	Max. probing depth<4mm	Max. probing depth<5mm	Probing depth>6mm	Need for complex rehabilitation due to;
		Horizontal bone loss	Horizontal bone loss	Mostly vertical bone loss >3mm	Masticatory dysfunction
				Furcation involvement class 2 or 3	Occlusal trauma
				Moderate ridge defects	Severe ridge defects
					Bite collapse ,drifting,-flaring.
EXTENT AND DISTRIBUTION	Add to stage as descriptor	Localized			
		Generalized			
		Molar/incisor pattern			

Grading of Periodontitis:

Table 6:

	PROGRESSION		GRADE A -SLOW RATE	GRADE B-Moderate rate	GRADE C-rapid rate
PRIMARY CRITERIA [whenever available direct evidence should be used	Direct evidence of progression	Radiographic bone loss	No loss over 5 years	<2mm over 5 years	>2mm over 5 years
	Indirect evidence of progression	%of bone loss / age	<.24	<.25-1.0	>1.1
		Case phenotype	Heavy biofilm deposition with low levels of destruction	Destruction commensurate with biofilm deposition	Destruction exceeds expectation of the biofilm deposits
GRADE MODIFIERS	RISK FACTORS	Smoking	Non smokers	<9cigarettes per day	10+cigarettes per day
		Diabetes	No diagnosis of diabetes	HbA1c<6.99 in case of diabetes patients	HbA1c >7 in case of diabetes patients

Clinical Features

"To accurately identify abnormal findings, it is essential to be familiar with the characteristics of healthy gingival tissues. Normal healthy gingiva typically exhibits:

i. A coral pink colour (considering individual variations in pigmentation)

ii. A thin, knife-like edge

iii. A smooth and regular texture

Additionally, healthy gingiva should be free of:

i. Plaque

ii. Calculus (tartar) on the teeth

Recognizing these normal features enables clinicians to detect deviations from health and identify potential issues [5].

"Periodontitis is marked by microbially induced, host-mediated inflammation, resulting in periodontal attachment loss, measured as Clinical Attachment Loss (CAL). However, it's essential to note that:

a. CAL can occur in conditions other than periodontitis.

b. Relying solely on radiographic bone loss to define periodontitis has limitations, including:

*Lack of specificity

*Inability to detect mild to moderate periodontitis

In cases where CAL measurement is challenging, such as during tooth eruption or mixed dentition, radiographic bone loss assessments can be used, utilizing bitewing radiographs typically taken for caries detection [6].

Prevalence

Studies have revealed three distinct bacterial groups in the subgingival biofilm:

1. Health-associated bacteria
2. Periodontitis-associated bacteria
3. Core species, equally present in both healthy and diseased individuals

Fusobacterium nucleatum (F. nucleatum) is the most prominent core species, playing a key role in the subgingival biofilm. Its physical interactions with various microorganisms, including P. gingivalis, Aggregatibacter actinomycetemcomitans, and others, facilitate the formation of a complex biofilm. F. nucleatum acts as a connector, linking early and late colonizers, and is essential for the ecological succession that leads to periodontitis progression [7].

Disease Severity

Research has identified a potential link between chronic periodontitis and an increased risk of various cancers, including oral and squamous cell carcinoma. However, limitations in controlling for confounding variables, such as tobacco and alcohol use, impact the validity of these findings. There is growing interest in exploring the relationship between periodontal disease and overall cancer risk, with systemic inflammation being a key focus. A recent meta-analysis supports a positive association between periodontal disease and the risk of oral, lung, and pancreatic cancers, but highlights the need for more robust studies with adequate sample sizes, improved periodontal disease measurements, and thorough adjustment for risk factors.

Furthermore, a significant body of evidence demonstrates that inflammation plays a profound role in all stages of cancer development, from initiation to progression. Additionally, an emerging concept is that cancer should be viewed as a systemic disease, rather than a localized one, emphasizing the need for a more comprehensive understanding of its underlying mechanisms [5].

Diagnosis

Diagnosing periodontal diseases typically involves evaluating clinical signs and symptoms, supported by radiographic evidence. Gingival changes, such as altered colour, contour, texture, and bleeding on probing, indicate plaque-induced gingival diseases. Non-plaque induced gingival diseases may require additional investigations like histopathology, microbiology, or serology.

Periodontitis is diagnosed by the presence of gingival changes, plus reduced tissue resistance to probing, deeper gingival sulcus or "pocket," and loss of periodontal attachment. It's essential to consider both horizontal and vertical dimensions of "pockets" and evaluate furcation involvements, requiring knowledge of tooth and furcation anatomy.

Tooth mobility and migration should also be assessed, but it's important to note that mobility alone is not diagnostic of periodontitis and may be caused by occlusal trauma or other factors. Mobility and migration related to periodontitis are typically late symptoms and are more relevant for prognosis and treatment planning [9].

Family history and risk-modifying factors, such as smoking, stress, drugs, or sex hormones, should be considered to further describe the type of disease. Radiographs can confirm attachment loss by showing marginal bone loss, but their role in diagnosis will be discussed in another article.

It's important to remember that:

i. A healthy gingival crevice can range from 1-3mm

ii. The distance from the cementoenamel junction to the alveolar bone crest can vary from 1-3mm in health

iii. Attachment loss alone does not constitute periodontitis, which is an inflammatory lesion

iv. Health can exist with severe attachment loss and recession, and a healthy periodontium can exist at different levels along the root after successful treatment [4].

Conclusion

Diagnosing periodontal diseases involves classification, which is an evolving process. However, all existing classification systems have limitations and criticisms. Some experts, like Van der Velden, argue that the 1999 reclassification was unhelpful and propose alternative systems, such as age-based classification with additional considerations for extent, severity, and clinical characteristics. This approach enables accurate clinical diagnoses for periodontitis patients.

Others, like Milward and Chapple, criticize the 1999 classification for being overly complex and unsuitable for general dental practice. From an epidemiological perspective, Lopez and Baelum suggest that simple clinical attachment loss measurements (e.g., ≥ 3 mm) are sufficient, advocating for a minimalist approach that doesn't differentiate between periodontitis types.

The debate highlights the challenges in developing a universally accepted classification system for periodontal diseases, with varying opinions on the ideal approach [8].

Conflict of Interest

Nil.

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