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The most common oral disease Worldwide-Chronic Periodontitis - A Review

[1]Pooja Bharadwaj, Senior Resident, Department of Periodontology, Rishiraj College of Dental Science and Research Centre, Bhopal, Madhya Pradesh, India.

[2]Roopali Patel, Civil Hospital, Bairagarh Bhopal, Madhya Pradesh, India.

Corresponding Author: Pooja Bharadwaj, Senior Resident, Department of Periodontology, Rishiraj College of Dental Science and Research Centre, Bhopal, Madhya Pradesh, India.

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Introduction

Periodontology is a speciality of dentistry that deals with structures surrounding and supporting the tooth. The surrounding structure is the gingiva whose main function is the protection of the underlying supporting tissues. The supporting tissues of the teeth are the periodontal ligament, cementum, and alveolar bone whose main function is to firmly hold the tooth in its socket.[1] Any kind of defect in the function of these surrounding and supporting tissue leads to a change from physiologic one to pathologic one. This pathology is termed as gingivitis and Periodontitis. Gingivitis refers to the inflammation of the surrounding tissue that is gingiva but without the loss of attachment. When this inflammation extends to the supporting tissues of the teeth, loss of attachment occurs, and this type of pathology is known as Periodontitis.[2]

Keywords: Gingivitis, Periodontitis, Biofilm.

Etiological and amplifying factors

The oral cavity of a human being is full of microbes. As soon as the child is born, its oral cavity gets occupied with both the aerobic and anaerobic microbes. In a healthy oral cavity, a balance exists among the aerobic and anaerobic microbes. Once this balance get disturb in the sense, that there is a dominance of particular microbes over the other one, disease results. The microbes which causes a disease lives in film called as biofilm which is usually resistant to antimicrobials and host defense mechanism. This biofilm later get modified to the structured, resilient, yellowish grayish substance called plaque which is the main etiological factor for the causation of gingivitis and Periodontitis.[3] However though the microbes are the prior source of in fection, but there are certain factors which determine the pathogenicity of the disease and these factors are known as amplifying factors. Over these past few years, a new concept had come into existence that is the host response. host response refers to how the body of the host respond to these microbes and it is because of this

host response factor only that some individuals are at a higher risk of more prone disease while some are at the low or null risk to get the disease.[1] Now this host response is modifiable or amplified by certain factors which increases or decreases the individual risk of getting the disease. These are the environmental factors such as smoking which has an additive effect on making the individual more prone to disease. Another factor is the genetic one which also determine the individual risk of getting the disease. The third factor are the systemic factors like the diabeties which play an important role in increasing the susceptibility of an individual to disease.[2]

Etiopathogenesis of chronic periodontitis

The main etiological factor for the causation of gingivitis and periodontitis is plaque. Plaque in a basic term means a microbial colony that consists of mostly anaerobes and a few aerobes. Microbes initiate the inflammation through their virulence factors like lipopolysaccharide, gingipains, and direct invasion into the host through their fimbriae. As a result of this initiation of inflammatory and infectious response by the microbes, host immune response comes into play. As a result of the microbial virulence factors, many host derived inflammatory mediators such as cytokines are released from a large number of cell types like neutrophil, macrophages, lymphocytes. These cytokines signal, broadcast, and amplify immune responses which help in combating infections.

However, these immunoinflammatory responses that develop in periodontal tissues in response to the long-term presence of subgingival biofilm are protective by intent but result in considerable tissue damage. This has sometimes been referred to as bystander damager who denotes that the host response is mainly responsible for the tissue damage that occurs, thereby leading to the clinical signs and symptoms of periodontal disease. The prolonged and excessive production of cytokines and other inflammatory mediators in the periodontium leads to the connective tissue and alveolar bone destruction through the induction of fibroblast and osteoclast to produce proteolytic enzymes such as matrix metalloproteinases that break down the structural components of this connective tissue. Thus it is clear that the microbes are important for initiating and perpetuate the inflammation, but the great majority of tissue breakdown in periodontal disease results from the host inflammatory processes.[4]

Diagnosis

Chronic periodontitis is diagnosed both clinically and radiographically. however clinically we rely more on the diagnosis of chronic periodontitis as compared to radiographically due to the fact that radiograph always gives the past experience of how much bone is lost while clinical condition always is a present stage condition of the patient. And this is also a fact that bone loss is not necessarily related to the pocket depth always. Radiographically chronic periodontitis is diagnosed by orthopantomogram, and clinically by means of following characteristics features:[5]

1. Supragingival and subgingival plaque and calculus- this is due to increase in the depth of gingival sulcus because of which there is a food impaction, which cannot be removed by the patient himself and this food debris later on get converted to plaque, which later on further get calcified into a deposit called as calculus which is present both supragingival Ly and sub gingivally.
2. Gingival swelling and redness- this is because of the destruction of gingival collagen fibres as a result of which gingiva loses its firmness and gets swelled up and there is an increase in vascularity so the gingiva becomes red in color.

3. Pocket formation -this is mainly because of the apical migration of junctional epithelium and the destruction of collagen fibres resulting in pathological deepening of gingival sulcus, leading to loss of resistance to probe penetration, resulting in pocket formation and increase in pocket depth.
4. Bleeding on probing -due to enlargement and dilation of capillaries resulting in increased vascularity and due to presence of microbial species Porphyromonas gingival is, Tannerella forsythia, and Treponema denticola resulting in increase bleeding on probing.
5. Bone loss and clinical attachment loss- due to increased in the osteoclastic activity by the immune inflammatory mediators.
6. Furcation involvement- due to loss of interradicular bone in the advanced stages.
7. Increased tooth mobility- due to the destruction of alveolar bone
8. Pathologic tooth migration
9. Recession

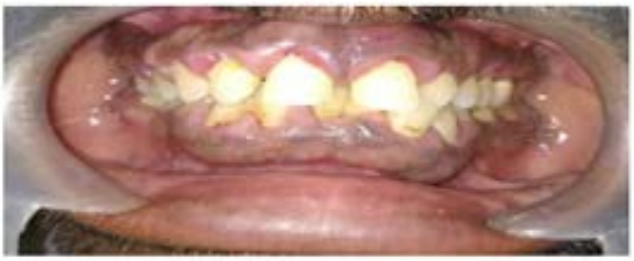

FIG 1. TYPICAL CHARACTERISTIC FEATURES OF CHRONIC PERIODONTITIS

Classification

A recent classification had been given by World Workshop of Periodontology (2017) which is divided into stages and grading on the basis of interdental clinical attachment loss, radiographic bone loss, number of tooth loss due to periodontitis, probing pocket depth, and amount of biofilm deposits[6]

Periodontitis - staging

Staging intends to classify the severity and extent of a patient's disease based on the measurable amount of destroyed and/or damaged tissue as a result of periodontitis and to assess the specific factors that may attribute to the complexity of long term case management. Initial stage should be determined using clinical attachment loss (CAL). If clinical attachment loss is not available, radiographic bone loss should be used. Tooth loss due to periodontitis may modify stage dentition. One or more complexity factors may shift the stage to a higher level.[6]

Table 1:

	Periodontitis	Stage 1	Stage 2	Stage 3	Stage 4
Severity	Interdental CAL (for the sight of greatest loss	1-2 mm	3-4 mm	≥5mm	≥5mm
	Radiographic bone loss (RBL)	Coronal third (<15%)	Coronal third (15%-33%)	Extending to middle third of root and beyond	Extending to middle third of the root and beyond.
	Tooth loss (due to periodontitis)	No tooth loss	No tooth loss	≤4teeth	≥5 teeth

Complexity	local	Maximum probing depth ≤4mm. mostly horizontal bone loss	Maximum probing depth ≤5mm. mostly horizontal bone loss	In addition to stage 2 completely: Probing depths ≥6mm Vertical bone loss ≥3 mm Furcation involvement class 2 or class 3 Moderate ridge defects	In addition to stage 3 completely: Need for complex rehabilitation due to: Masticatory dysfunction Secondary occlusal trauma (tooth mobility degree ≥2) Severe ridge defects Bite collapse, drifting, flaring <20 remaining teeth

Periodontitis grading

Grading aims to indicate the rate of periodontitis progression, responsiveness to standard therapy, and periodontal impact on systemic health.

Table 2:

	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 Indirect evidence of progression	Radiographic bone loss or CAL % Bone loss/age Case phenotype	No loss over 5 years <0.25 Heavy biofilm deposits with low levels of destruction	< 2 mm over 5 years 0.25to 1.0 Destruction commensurate with biofilm deposits	≥2mm over 5 years >1.0 Destruction exceeds expectations given biofilm deposits, specific clinical patterns suggestive of periods of rapid progression and/or early onset disease.
Grade modifiers	Risk factors	Smoking Diabeties	Non-smoker Normoglycemic/no diagnosis of diabeties	≥10 ciggrates/day HbA1c>7% in patients with diabeties	

Indicators of periodontitis

- **Pocket probing depth**

The normal gingival sulcus depth is around 2-3 mm. this normal sulcus depth is because of the presence of collagen fibres which provide firmness to the gingiva and resistance to periodontal probe penetration. Once this sulcus depth is increased it will result in a pathology. This pathological deepening of gingival sulcus is called as periodontal pocket and the resistance lost to periodontal probe penetration is called as periodontal pocket depth or pocket probing depth. This loss of resistance is mainly because of the destruction of collagen fibres mainly as a result of which there is an apical migration of junctional epithelium. Periodontal pocket depth is measured by means of a probe which is a pointed, tapered, calibrated instrument in millimetres. Periodontal pocket depth can be measured mainly by either two types of probe i.e., the Williams probe (in which 4 & 6 divisions are missing), and the second one is the UNC 15 Probe, which has black bends markings at 5,10, and 15 mm respectively.[2]

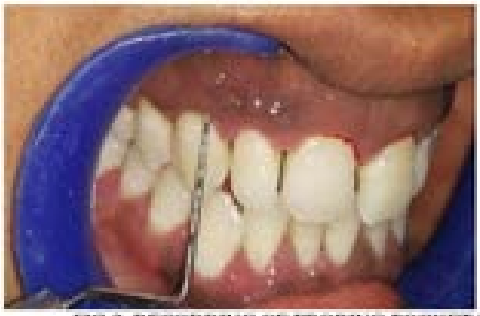

FIG 2. RECORDING OF PROBING POCKET DEPTH BY UNC-15 PROBE

Orthopantomogram (opg)

Refers to a full mouth x-ray which gives an idea about the condition of entire teeth in the dentition. From periodontal point of view, the main thing to identify is the amount of bone loss. Bone remaining in the middle third has a good prognosis, while bone remaining in the apical third has a fair to poor prognosis. However, bone loss is not always related to periodontal pocket depth. The best example for this is in the cases of recession, where we get a low pocket depth, but a greater amount of bone loss and the clinical attachment loss.[1]

FIG 3. OPG OF CHRONIC PERIODONTITIS PATIENT

Management of chronic periodontitis

Treatment of chronic periodontitis is divided based upon the severity of the disease:[7]

1. **Mild chronic periodontitis**

Convincing evidence of the central role of microorganisms in the etiology of gingivitis and periodontitis has been presented. Since periodontal diseases are primarily infectious, in general, early diagnosis and cause related therapy are considered the main principles in treatment and may well be the key to successful results in the overall clinical management of periodontal diseases. Mild chronic periodontitis refers to the presence of probing depth of 3-5 mm. Treatment at this depth mainly aim to be treated non-surgically with a thorough supragingival and subgingival scaling, root planning along with the use of antimicrobials or host modulating agents to modify the host response.

2. **Moderate chronic periodontitis**

By definition, moderate chronic periodontitis involves the probing depth of 5-6 mm with destruction of periodontal structures and noticeable loss of bone support, possibly accompanied by an increase in tooth mobility. There may be furcation involvement in

multirooted teeth. Probing depth varies with the position of the gingival margin. In most instances, probing depth closely correlates with the attachment loss. However there are also instances, where inflammatory enlargement of the tissue causes a deeper probing depth and instances where recession of the gingival margin causes a lesser probing depth. Radiographs generally show a corresponding loss of alveolar bone. The pattern of bone loss in most of the cases will be horizontal. The purpose of treatment is to interrupt a sequence of destructive events called periodontitis. Certainly, more than one treatment mode can be used in treating a patient. The treatment consists of following approaches:

A. Scaling and root planning

The clinician must weigh all the factors of the case and the response of the patient in determining whether closed scaling and root planning will be effective. For example, a patient with 4-6 mm probing depths and corresponding attachment loss, horizontal bone loss, no furcation problem, and soft and oedematose tissue certainly would be a better candidate for this approach, than a patient with vertical bone loss, shallow two wall interproximal bony craters, and furcation involvement and /or fibrous thick tissue. The crux of the decision must be related to the ability of the clinician to cleanse and detoxify the diseased root surface. Once the area is cleaned, it must be maintained. Therefore, if adequate access can be obtained non-surgically, then scaling and root planning is the treatment of choice, but if the root debridement cannot be accomplished with a closed approach, then surgical intervention is the treatment of choice.

B. Surgical approach

The most common surgical approach to periodontal disease is the flap approach because it allows the therapist access to the underlying structures for visualization and thorough debridement and correction of any anatomic defects and deformities of the bony tissues and teeth. The flap approach also permits repositioning of the tissue. The flap approach gained its popularity because of dissatisfaction with the results of other treatment techniques such as clo9sed scaling and root planning and gingivectomy techniques. Also, the flap approach allows for preservation of the gingival tissue which then permits more rapid healing. Once the flap is raised, efforts can be concentrated on removal of granulation tissue, debridement and detoxification of the root surfaces, correction of anatomic defects created by the periodontal disease process, either by bony contouring or by regenerative procedures, and correction of anatomic problems related to tooth structures. Flap approaches can be a conventional flap approach, modified Widman flap, apically repositioning flaps, osseous respective surgery.

3. **Severe chronic periodontitis**

The patient who presents with advanced chronic periodontitis will have clinical and radiographic evidence of severe periodontal destruction. The bone loss may be horizontal and /or vertical and localized or generalized in nature. Commonly the probing depth exceeds 6 mm and clinical attachment loss is equal to or greater than the probing depth. In addition, there is grade 2 or grade 3 furcation invasion. In order to correct these problems, a whole array of treatment modalities can be used. These usually include extraction, scaling and root planning alone, osseous surgery, root amputation/hemi section, orthodontic repositioning, interdental denudation, coronally repositioning flap, open flap debridement with or without chemotherapeutic root conditioning, bone grafts, alloplastic grafts, and guided tissue regeneration with or without bone grafts. The clinician must weigh the advantages and disadvantages

of each of these procedures and select the proper one based on therapeutic objectives for a particular patient.

Supportive periodontal treatment for chronic periodontitis patient

Supportive periodontal therapy is an important but often overlooked phase of periodontal therapy. A series of studies on choosing the appropriate therapy for patients with inflammatory periodontal diseases was started at the University of Michigan in 1960 and continued into the 1980. Patients were placed on 3 months SPT recall interval following active therapy. It was concluded that the majority of the patients remained stable regardless of which therapy was used as long as the patients stayed on a regular SPT interval. As a result of these and other studies, it can be said that while supportive periodontal treatment has limitations, regular interval of Supportive periodontal therapy can maintain the dentition of patients with periodontitis when scheduled following active therapy. The question now arises, do most patients receive regular supportive periodontal treatment. This is true because most periodontal problems are chronic and are closely associated with the bacterial flora often found in the oral cavity. This scenario is complicated by the fact that many dental professionals have been trained to deal with an active problem that can be removed and the damage readily repaired. To successfully deal with periodontal lesion, one must realize that these problems are chronic. The concept of cure is a transient one, since the best treated case will usually breakdown without adequate supportive periodontal treatment or personal oral hygiene. Although, it may be possible to truly cure some patients, at present no technology is available to confirm the cessation of disease. This means that frequent visits to the professional for disease monitoring and appropriate therapy are needed. The following protocol is followed for supportive periodontal treatment.[7]

1. **Health history update**

Patient can undergo significant medical and dental health changes in few weeks between supportive periodontal treatment visits. Any changes should be recorded in the patients chart. The following things should be asked to the patient such as has there been any change in your health or medications, or have you been hospitalized since your last visit, is there anything about your mouth or jaws, that is of concern to you.

2. **Periodontal probing pocket depth**

This distance has been defined as the distance from the gingival margin to the apical depth of periodontal probe tip penetration. These measurements should be taken and recorded at six points around each tooth. This parameter should be checked at each supportive periodontal treatment visit and any changes from baseline recorded

3. **Gingival recession**

Measurement of gingival recession is important at each SPT visits so as not only to document any continued recession, but also to provide a perspective for probing pocket depth measurements.

4. **Disease activity**

This is measured in terms of bleeding upon probing and suppuration upon each SPT visit.

5. **Fremitus**

Movement of teeth during function (fremitus) is simple to measure and has been associated with advancing disease. Therefore, it should be checked, noted and eliminated if it is found on a tooth that has clinical signs of active periodontal breakdown.

6. **Assessment of prostheses**

Any prostheses, fixed or removable, should be examined at each SPT visit and repairs or replacements made when indicated.

7. **Caries examination**

Each tooth should be checked clinically for caries at each SPT visit. Radiographs, if available, should be reviewed.

8. **Soft tissue examination**

The intraoral and extraoral soft tissues should be examined for any abnormalities and the findings recorded.

9. **Removal of supragingival and subgingival deposits**

the clinician providing the care at SPT visit should complete needed subgingival scaling and root planning before beginning to remove materials that have accumulated supragingival Ly. This may require additional visits by the patient.

The average SPT visit for a patient with inflammatory periodontal disease requires about an hour. However this is only the average since many patients may require a longer time for completion of required SPT. the time interval between SPT intervals is based upon Merin's classification

Merin classification for recall intervals for various classes of recall patients[2]

Merin classification	Characteristics	Recall interval
First year	Routine therapy & uneventful healing. Or First year patient-difficult case with complicated prosthesis, furcation involvement, poor crown to root ratio, or questionable patient cooperation	3 months Or 1 to 2 months.
Class A	Excellent results well maintained for 1 year or more. Patient displays good oral hygiene, minimal calculus, no occlusal problems, no complicated prosthesis, no remaining pockets, and no teeth with less than 50 percent of alveolar bone remaining	6 months to 1 year.
Class B	Generally good results maintained reasonably for one year or more, but patient displays some of the following factors like inconsistent or poor oral hygiene Heavy calculus formation, systemic diseases leading to periodontal breakdown, some remaining pockets, ongoing orthodontic therapy, positive genetic test.	3 to 4 months
Class C	Generally poor results following periodontal therapy and/or several negative factors such as heavy calculus, poor oral hygiene, remaining pockets, occlusal problems, periodontal surgery indicated, but not performed for medical, psychologic, or financial reasons. teeth with less than 50 percent of alveolar bone remaining.	1 to 3 months

Summary and conclusion

Chronic Periodontitis is by far the most common dental disease affecting worldwide. Chronic periodontitis related to the infectious inflammatory disease leading to destruction of bone as well as connective tissue and is modified by host response. Periodontitis occurs in mild, moderate, and severe stage, leading to variability of the treatment outcome. Chronic periodontitis is identified both clinically and radiographically, however to some extent more clinically because clinical condition of the patient is a present condition of his or her periodontium as compared to radiographically which is the past condition of how much bone is lost. Its very necessary for an individual to know about their gingival health as the negligence can lead to from initiation of bleeding upon probing to the tooth loss and since the tooth is firmly held in position by the supporting tissues of the teeth and these supporting tissues of teeth are in turn protected by the gingiva. So ultimately tooth health as well as the systemic health is dependent upon the condition of gingiva. So healthy the gingiva is, healthy the individual is.

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