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Comprehensive Review of Gingival and Dental Anatomy with Pathophysiological Perspectives of Periodontitis.

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Abstract

An oral cavity including gum, teeth and associating structures are essential for overall health and wellbeing of an individual. This article is an in-depth exploration of the anatomy and pathophysiology of oral cavity, with a particular focus on gingiva, dentition and supporting tissues. Because of the structural organization and intricate design, the gums and teeth act as a barrier against microbial invasion along with providing additional support for mastication and speaking. However, disturbances and inconvenience in these processes result to pathological conditions or illness involving gingivitis, periodontitis, dental caries, and various other systemic implications along with effects of oral diseases. This study focuses on the structural morphology and underlying disease pathologies of gingiva, teeth by emphasizing the gap between normal function and disease states.

Keywords:

Gingiva, teeth, mouth, pathophysiology, pathogenesis, periodontitis.

Introduction:

The health of our gums, teeth and mouth is significantly more intricate, complex and vital than just a beautiful radiant smile. It provides a view into the overall general health of the body, regulating everything from digestion to cardiovascular wellbeing. Beyond their essential roles in eating speaking and smiling, these components comprise a complex system that reflects our general health. The mouth, often referred to as the “gateway to the body,” plays an important role in our daily lives, from the first bite of food to the words we say. However, the complicated and complex relationship between our gums, teeth, oral tissues frequently goes unrecognized until an issue or a problem arises. Gums can be said as unsung heroes of oral health, serving and providing a protective foundation for our teeth. These soft tissues function as mediators against harmful bacteria and microorganisms while maintaining the structural integrity. However, neglecting gum care can develop to conditions like gingivitis and periodontitis, which not only threaten oral health but also risks to our health and its management.

Teeth on other hand, are amazing examples of natural engineering. They are made up of enamel, the hardest and strongest substance in human body, they withstand the forces of chewing and grinding every day. But teeth are not invincible: - poor hygiene, diet, food and lifestyle choices can result to cavities, erosion, and even tooth loss. The mouth cavity is a dynamic environment filled with bacteria, enzymes, and tissues that constantly interact to maintain and preserve balance. An imbalance in this ecosystem can cause in common issues such as bad breath, oral-mouth infections, and even systemic disorders. This article gives insight into the complexities of oral health, revealing the crucial roles of gums, teeth, and the mouth.

Anatomy:

Mouth

The mouth also known as the oral or buccal cavity. The mouth is a formed by cheeks, hard and soft palates, and tongue. The cheeks form the lateral walls of oral cavity. They are covered externally by skin and internally by mucous membrane, which consists of non-keratinized stratified squamous epithelium. Buccinator muscles and connective tissue are located in the middle of the skin and mucous membranes of the cheeks. The anterior portion of cheeks ends at the lips. (1)

The lips are fleshly folds (borders) surrounding the opening of mouth. They include the orbicularis oris muscle and are covered externally by skin and internally by a mucous membrane. The inner surface of each lip is attached to its corresponding the mouth into maltose, maltotriose and α-dextrin. Bicarbonate and phosphate ions that buffer acidic (pH 6.35-6.85). Salivary glands help remove waste molecules from the body, which includes for the presence of urea and uric acid in saliva. Mucus tends to lubricate food so it can be easily moved around the mouth, and form into a ball, then swallowed. Immunoglobulin A (IgA) helps to prevents attachment of microbes so they cannot penetrate the epithelium, and the enzyme lysozyme destroys the bacteria, however, these chemical substances are not present in large enough quantities to completely eliminate all oral bacteria. (1)

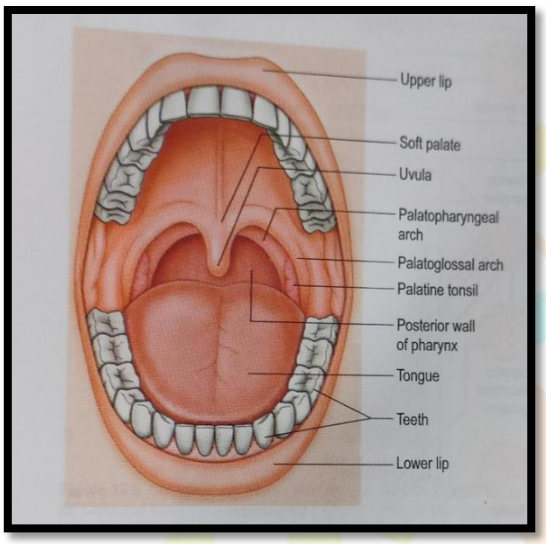


Fig 1. Structure in wide open Mouth (2) Fig 2. Inferior surface of Tongue (2)

The mount is made up of muscles and bones: –

Anteriorly – by the lips.

Posteriorly – it is continuous with the oropharynx.

Laterally – by the cheek muscles.

Superiorly – by the bone hard palate and muscular soft palate.

Inferiorly – by the muscular tongue and the soft tissues of the floor of the mouth. (3)

Tongue: Tongue is said to be the accessory digestive organ composing of the skeletal muscle covering them with mucous membrane. Together they are with associated muscles, it forms the floor of the oral cavity in the mouth. The tongue is separated into symmetrical lateral halves by a median septum that extends its entire length, and it is attached inferiorly to the hyoid bone, styloid process of temporal bone and mandibles. Each half of the tongue consists of an identical combination of extrinsic and intrinsic muscle. The extrinsic muscles of the tongue, which originate outside the tongue and insert into connective tissues in the tongue, consists the hyoglossus genioglossus and styloglossus muscle. The extrinsic muscles move the tongue from side to side and in and out to help food for chewing, shape the food into a round spherical mass and force the food, the back of the mouth for swallowing. They also help in forming the floor of the mouth and help in holding the tongue in position. The intrinsic muscles of the tongue originate in and insert into the connective tissue within the tongue. They alter and modify the shape and size of the tongue of for speech and swallowing. (1)

Functions: The tongue plays an important part in:

- Chewing (mastication)
- Swallowing (deglutition)
- Speech
- Taste

Nerve endings of taste are present in the papillae and widely distributed in the epithelium of the tongue. (3)

Gingiva:

The gingiva (gums) is pink- colored keratinized mucosa that surrounds and protects the teeth. It is perfused by multiple networks of small arteries that originate from branches coming off, of the carotid artery. It receives innervation by the nerves which are derived from the mandibular and maxillary divisions of the trigeminal nerve. The gingiva is the part of the periodontium, which includes the investing and supporting structures off the teeth. (4, 5)

Structure and Function: The gingiva is also known as the gums. They are specialized regions of the oral mucosa that surround the teeth and cover adjacent regions of the alveolar bone. (2) These are a specialized cells called as junctional epithelial (JE) cells. This junctional epithelial is strategically located at the bottom of gingival sulcus, where it acts as a barrier to both mechanical damage and microbiological insult. The oral gingiva is responsible for both sensation in the mouth and absorption of micronutrients along with an addition of its protective function. Epithelium plays a crucial role in an innate immune response to infectious inflammation in periodontal tissue. (4, 6) The gingival connective tissues composed of collagen fibers, cells and ground substance. Cells make up to 5% of gingival connective tissue while collagen fibers and ground substance make up to 60% and 35% respectively. The different types of cells present in gingival connective tissue includes fibroblasts, mast cells, macrophages and inflammatory cells. (4, 7)

Clinical Features: Normal gingiva in an adult covers the alveolar bone and tooth root to a level just coronal to the cementoenamel junction. The gingiva is anatomically separated into three marginal, attached, and interdental areas. Although each type of gingiva varies significantly in differentiation, histology, and thickness according to its functional demands and their requirements. (8, 9)

- ***Marginal Gingiva:*** The marginal, or detached, gingiva is the terminal edge or border of the gingiva surrounding the teeth in collar like form. The gingival sulcus has a soft tissue wall formed by the marginal gingiva. It can be separated from the tooth surface with a periodontal probe.
  Gingival sulcus: The gingival sulcus is the shallow crevice or space around the tooth bounded by the surface of the tooth on one side and epithelium lining the free margin of the gingiva on the side. It is V shaped, and barely permits the entrance of the periodontal probe.
- ***Attached Gingiva:*** The attached gingiva is continuous with marginal gingiva. The attached gingiva is firm, resilient, and is tightly bound to the underlying periosteum of alveolar bone loactaed on the jaw bones which hold the teeth. The facial aspect of the attached gingiva extends to relatively loose and movable alveolar mucosa and is demarcated by mucogingival junction.
- ***Interdental Gingiva:*** The interdental gingiva occupies the gingival embrasure, which is actually the interproximal space beneath the area of the tooth in contact. They (Interdental Gingiva) can be pyramidal or can have “col” shape. In the former the tip of one papilla is located immediately beneath the contact point; the latter presents a valleylike depression that connects facial and lingual papilla and conforms to the shape of interproximal contact. (8, 9)

Teeth:

The teeth also known as dentes are the accessory digestive organs that are located in the sockets of alveolar processes of the mandible and maxillae. The alveolar processes are covered are protected by gingiva or gums, which extend slightly into each socket. The sockets are lined with periodontal ligaments or periodontal membranes, which consist of dense connective tissue that binds the teeth to the socket walls and acts as a shock absorber during chewing and eating. A typical tooth has three major external regions: the crown, the root and the neck. The crown is the visible portion above the gum level. Embedded in the socket are one to three roots. The neck is constricted junction of the crown and roots near the gum line. Internally the dentin forms the majority of the tooth. Dentin consists of calcified connective tissue that gives the tooth the basic shape and rigidity. It is harder than a bone because it's higher content of hydroxyapatite. The crown is covered by enamel, it is the hardest substance in the body, and it serves to protect the tooth from wear and tear of chewing. It also protects against acids that can easily dissolve dentin. (1)

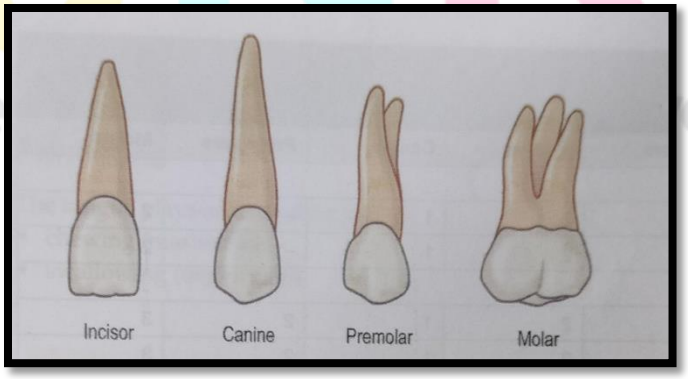


Fig 3. Shapes of the Permanent Teeth (3)

Humans have two dentitions (set of teeth): deciduous and permanent. The first of these - the deciduous teeth are also called as primary teeth, milk teeth or baby teeth- begin to erupt at about 6 months of the age and approximately two teeth appearing each month there after all the 20 are present. All the deciduous teeth are lost generally between is 6 to 12 years and are replaced by permanent i.e. secondary teeth. The permanent dentition has 32 teeth that erupt in between age the age group of 6 to adulthood. (1) 16 in the upper (top) jaw and 16 in lower jaw. On each side in both maxillary and mandibular arches are two incisor, one canine, two premolars, and three molar teeth. (2)

The **incisor teeth** are the "front teeth" and have one root and chisel-shaped crown, which "cuts."

The **canine teeth** are posterior to the incisors, are the longest teeth, have a crown with a single pointed cusp, and "grasp."

The **premolar teeth** (bicuspids) have a crown like shape with two pointed cusps, one on the buccal (cheek) side of the tooth and the other one on the lingual (tongue) or palatal (palate) side, they generally have one root (but the upper first premolar next to the canine may have two), and "grind"

The **molar teeth** are behind the premolar teeth, have three roots and crowns with three to five cusps, and "grind" (2)

Structure and their shape: Although the shapes of different teeth vary, the structure is the same and consists of:

The crown – protrudes from the gum

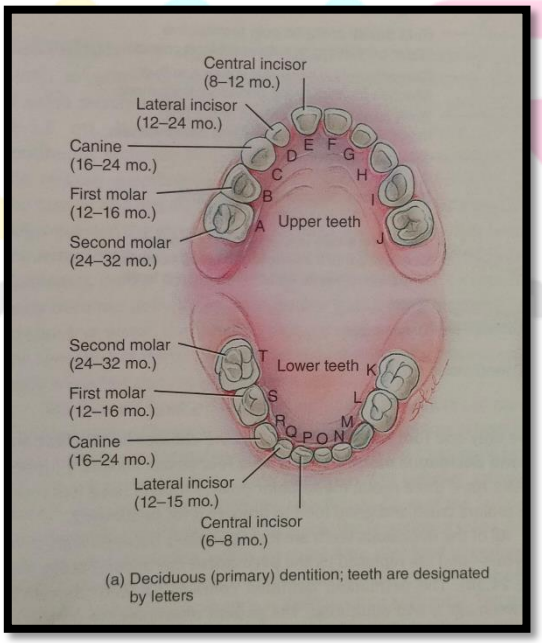
The root – the part embedded in the bone

The neck – the slightly narrowed region where the crown merges with the root. (3)

Jaw	Molars	Premolars	Canine	Incisors	Incisors	Canine	Premolars	Molars
Deciduous teeth								
Upper	2	–	1	2	2	1	–	2
Lower	2	–	1	2	2	1	–	2
Permanent teeth								
Upper	3	2	1	2	2	1	2	3
Lower	3	2	1	2	2	1	2	3

Fig 4. Number and Positions of Deciduous and Permanent Dentitions (3)

Given this can be seen in the below figures along with the time (age) of eruption in the child and adult. (2)



(a) Deciduous (primary) dentition; teeth are designated by letters

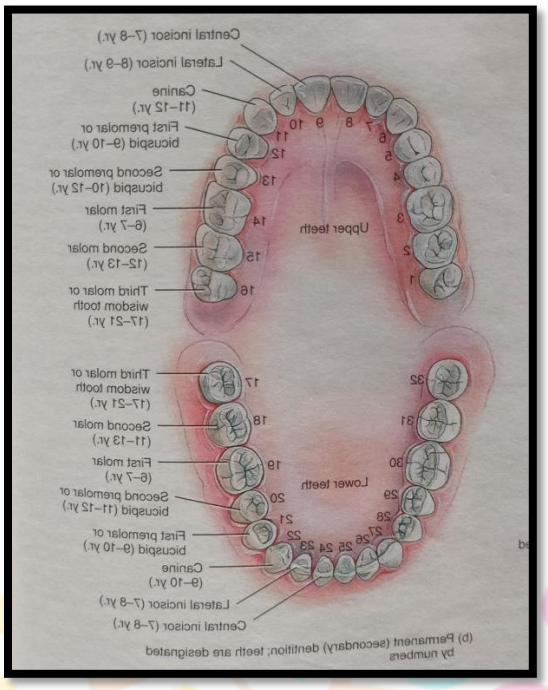


Fig 5 & 6. Dentitions and times of Eruption, There are 20 teeth in complete deciduous set and 32 teeth in a complete permanent set. (1)

Pathophysiology of Periodontal:

Dental plaque is the primary cause of gingivitis and periodontitis. A single human contains about 150 distinct kinds of bacteria, and the calculus of the tooth has been discovered to have 800 different species of microbes. These species include viruses, spirochetes, and Gram-negative anaerobic bacteria. The imbalance between two enzymes that causes periodontal tissue to deteriorate. Bacteria biofilms are a type of matrix that adheres to the surface of teeth and is fixed with colonies of various bacteria species. Plaque biofilm has seven phases. (10)

Periodontitis:

Inflammatory mediators are essential to the pathophysiology of periodontal tissue destruction. Current studies show that patients with AgP have significantly lower levels of IL-17 and IL-23 in their GCF compared to healthy subjects, as well as significantly higher levels of IL-1β and MMP-8 in both shallow and deep pockets. AgP patients had gingival cervicular fluid calprotectin levels that were considerably higher and positively correlated with bleeding index (BI), probing depth (PD), and clinical attachment loss (CAL). By encouraging p65 nuclear translocation in periodontal ligament cells (PDLCs), its subunit rhS100A8/A9 has been demonstrated to enhance nuclear factor-kB (NF-kB) activation and promote cell death. As a result, pro-inflammatory cytokines as COX2, IL-6, IL-8, and TNFa were expressed. (11, 12)

Proteolytic periodontal tissue degradation is another pathogenic phenomenon in AgP, where fibronectin degradation by HtrA1 (high temperature requirement A 1) can result in the expression and secretion of several MMPs. This is corroborated by the fact that AgP patients had higher levels of plasma cell HtrA1, which may have caused an excess of MMPs and, by blocking TGF-b, raised the inflammatory mediators IL-1b and TNF-a.Also, a notable rise in activated cytotoxic T cells, or CD8+/CD28+ cells, in both defect sites and systemic circulation blood samples distinguishes the phenotypic profile of blood mononuclear cells in generalized AgP patients. Severe tissue damage may result from these results, which are associated with inflammation. (11, 12)

Since nicotinamide phosphoribosyltransferase (NAMPT), commonly referred to as visfatin, can trigger the generation of inflammatory mediators like IL-6, IL-1β, and TNF-α, it has been identified as a proinflammatory marker for AgP. Common periodontal infections like P. gingivalis and F. Nucleatum may promote its synthesis in the gingival fibroblast cells and periodontal ligament. Visfatin expression in gingival tissues was elevated in AgP patients, indicating that it may play a part in etiopathogenesis. (11, 12)

The condition known as periodontitis affects the tissue that surrounds the tooth. Both systemic and local etiological variables influence the disease. One of the most prevalent conditions affecting the oral cavity is periodontitis. Prompt treatment of the disease is crucial since it not only causes tooth loss but also has an impact on the patient's overall health. The activity goes over the pathophysiology, diagnosis, and therapy of periodontitis. This exercise emphasizes how important the interprofessional team is to the treatment of patients with periodontitis. Along with a small number of harmful bacteria, the majority of the bacteria that live in the human mouth cavity are commensals. One of the most prevalent conditions affecting teeth is periodontitis, which causes the surrounding and supporting tooth

structure to deteriorate. The word "periodont-"(means "structure around the teeth") and "it is" (means "inflammation") and this combines to form the phrase as "periodontitis." Initially it affects the gingival tissue, periodontitis leads inflammation to spread to deeper tissues, changing the homeostasis of bones and ultimately leading to tooth loss if treatment is not received. The etiology of periodontal disease is complex. The bacterial biofilm that forms on the surfaces of teeth has been found to be the primary cause of periodontitis. In addition to local variables like calculus and plaque, the host reaction also influences the disease's course. Other factors include genetics, environmental factors, the patient's systemic health, lifestyle choices, and other social determinants. In addition to their detrimental effects on the periodontium, periodontopathogens also have a negative impact on patients' overall health. (13)

Signs of periodontal disease:

1. Gums that bleed when you brush or floss your teeth.

2. Gums those are red, swollen or tender.

3. Gums that have pulled away from teeth.

4. Infection including purulence (pus) between the teeth and gums when the gums are pressed.

5. Permanent teeth are loose or separating.

6. Any changes in the way teeth fit together when bit.

7. Any changes in the fit of partial denture.

8. Bad breath, itchy sensation.

Stages of periodontics: -

Early Periodontitis: When gum inflammation spreads to the deeper underlying structures and bone, it results in periodontitis. Gums that have the general form of periodontitis, plaque behind the gum line, may bleed readily, feel itchy, and appear bright red. A periodontal pocket, or space between the tooth and gum, deepens and becomes more bacterially dense as a result of the ligaments holding the tooth in its socket breaking down and the gums pulling away from the teeth. Bone and supporting ligaments begin to deteriorate.

Moderate periodontitis: Gums are painful, swollen, and red. For periodontal disease to exist, calculus and plaque do not need to be visible to the unaided eye. The gums are now healthy again following nonsurgical periodontal therapy, or scaling. Observe how the gums have retreated, revealing the tooth's root. This is because prior periodontal disease caused irreparable bone loss. Tooth sensitivity may result from this. In this deeper pocket are form and more bone and tissue are lost. In this PDD is 5mm and tissue damage by bacteria or another pathogen.

Advanced periodontitis: When periodontitis worsens and there is a significant loss of bone support, it is called advanced periodontal disease. The teeth could become loose as a result of the loss of support. This is a major type of periodontitis PDD is up to 6mm. Further destruction of teeth could pilot the damage or destroyed them.

Refractory Periodontitis: In this kind of periodontitis causes permanent damage of teeth can occur and permanently lead to loss of tooth.

When periodontitis reaches the advanced stage, pockets deepen, the gums pull away from the tooth, and they may become pus-filled.

- There might be pain when cleaning teeth, sensitivity to heat or cold, and swelling around the root. This is because the root surface is exposed by the drastically receding gums.
- As bone loss worsens, the teeth may become less stable and must be extracted to maintain oral health. (14)

Types of periodontitis: -

Chronic Periodontitis: This includes symptoms like gum inflammation, extreme poor breath, bleeding while brushing or flossing. Loss of epithelial tissue, bone and ligaments are irreversible.

Aggressive Periodontitis: this early-onset chronic periodontal inflammatory disease, which can be localized or generalized, usually appears between puberty and in the early third decade of life. Symptoms are similar to chronic periodontitis.

Necrotizing Ulcerative Gingivitis: It is commonly seen in individuals suffering from malnutrition, immunological suppression or HIV. Necrosis refers to death of cells or living tissues. It is often caused by lack of food, which is essential for wellness.

Systemic Chronic Periodontitis: this kind of chronic periodontal disease is seen in patients with systemic illness. Gum inflammation can be caused by systemic diseases like diabetes, heart issue or respiratory disease. (15)

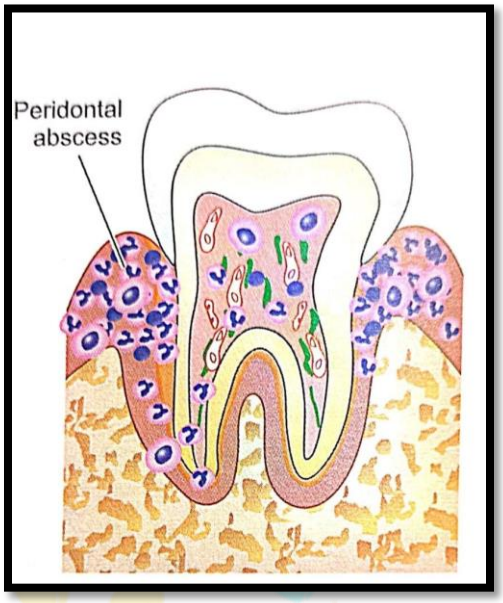


Fig 7. Periodontal Abscess (12)

Dental plaque:

The microbial film of dental plaque has been well examined and has been found to contain 800 distinct species in human dental plaque, as well as 150 species in a single individual. This bacterium is responsible for both causing and maintaining chronic gingivitis and periodontitis. The species is especially virulent and can cause sickness that hasn't been cured for ten years. Spirochetes, bacteria, and possibly viruses are among the suspected pathogens; nonetheless, it is likely that no single pathogen is responsible for the dysbiosis. When a few particular pathogens cause periodontal disease, the treatment approach focuses on changing the microbiota of plaque instead of removing biofilms. (16)

***Microbialbiofilm*:**

In cohort studies, colonization by particular clones of Aggregatibater actinomycetemocomitans has been linked to periodontal disease. Although the temporality of the microbial biofilm associated with progressive periodontitis is less well established, other species, such as Porphyromonas gingivalis, have been linked to it. According to a review, certain periodontal pathogens cause aggressive and chronic periodontitis, which suggests that microbial biofilms are the cause of comparable diseases. (16)

Calcification:

Both calcified and uncalcified kinds of dental plaque can be seen; supragingival plaque is usually uncalcified and more challenging to remove than subgingival plaque, which is usually dark in color and calcified. Salivary calcium and phosphate ions that aggregate within the plaque generate supragingival calculus, while ions from serum transudate caused inflammation in periodontal tissue calcify subgingival plaque (16)

Periodontal Disease Pathogenesis:

The nature of host-microbe interactions in periodontitis has been better understood thanks to recent microbiome and mechanistic research. In particular, research in people and animal models has shown that the microbiota linked to periodontitis is far more varied and complicated than previously believed, and that the bacteria implicated cause illness through dysbiosis and polymicrobial synergy. Put another way, periodontitis does not seem to meet the criteria for an infection in the traditional meaning of the word and is not brought on by a single or small number of bacterial species (also known as "periodontopathogens"). Instead, dysbiosis—a change in the number or impact of specific species within the polymicrobial community—is linked to periodontitis in proportion to their prevalence or impact on health. Destructive inflammation can result from dysbiosis, but the opposite is also true. In this way, the gingival crevicular fluid is exposed to the breakdown products of inflammatory tissue, such as broken collagen and substances that include heme, which are sources of iron and amino acids, respectively. These inflammatory waste products might be utilized as nutrients in the gingival pocket or crevice to support the selective growth of certain bacterial species (such as proteolytic and asaccharolytic pathobionts), aggravating the microbiota imbalance (dysbiosis).

Accordingly, adding serum, hemoglobin, or hemin to an oral multispecies community that has been created in vitro selectively causes pathobionts to develop. Interestingly, these pathobionts upregulate genes that encode hemolysins, proteases, and enzymes implicated in hemin acquisition.The original "homeostatic" community may become more proinflammatory as a result of this transformation into a "dysbiotic" one. Therefore, an early inflammatory reaction to the formation of subgingival biofilms (for example, as a result of poor oral hygiene) may favor "inflammophilic" pathobiotic bacteria (incipient dysbiosis), which can worsen inflammation when they continue to

grow in an inflammatory environment that is nutritionally favorable. For those who are vulnerable, this feed-forward cycle of inflammation and dysbiosis may eventually result in overt periodontitis (17)

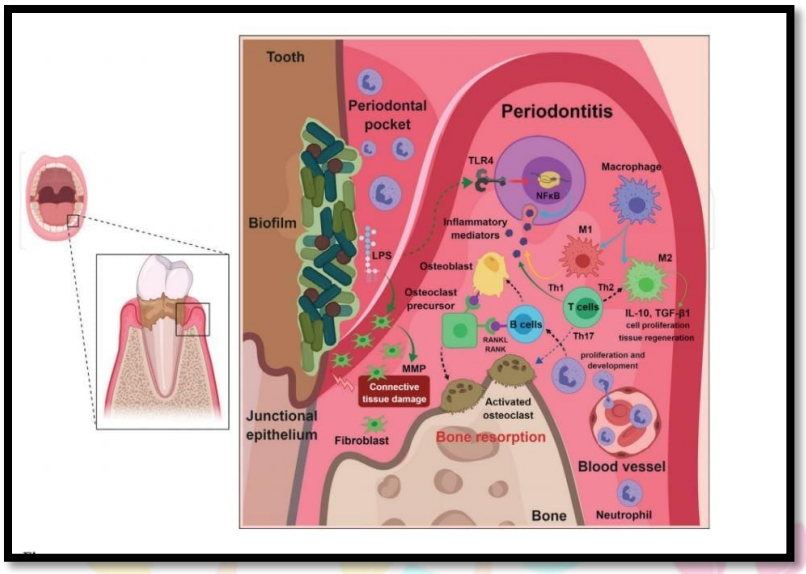


Fig 8. Innate and Adaptive Response during Periodontal Disease (18)

Histopathology:

The histology of periodontal disorders was initially characterized by Page and Shroeder. The disease progresses in four separate stages, each of which considers the tissues' clinical and histological features. Plaque marks the initial lesion, which causes vascular alterations and the production of intercellular gaps, which raises the amount of gingival crevicular fluid (GCF). Polymorphonuclear neutrophils are drawn to the lesion site by adhesion molecules. Particularly, T lymphocytes change the fibroblasts in the afflicted region. Clinically, the lesion is benign at this stage. The early lesion is identified by the development of redness at the location. After entering the area, polymorphonuclear neutrophils (PMN) eliminate the fibroblasts that are going through apoptosis.Collagen fibers break down as a result of the infiltration, creating more room for infiltrates.

The matrix of marginal connective tissue is degrading. Leukocyte aggregation and B cells, either plasma cells or lymphocytes, are primarily responsible for the formed lesion. They start the site's transformation by converting the sulcular and junctional epithelium into the pocket epithelium. The permeability and vulnerability of the pocket epithelium are very high. Clinically, this shows up as bleeding when the gingival tissues are gently probed.The progression to periodontitis is the last stage, referred to as advanced lesion. The migration of biofilm to the pocket creates the advanced len, providing an optimal niche for the growth of anaerobic bacteria. Histologically and clinically, there is an irreversible loss of connection and bone loss. This stage is characterized by the loss of alveolar bone and gingival fibers. Depending on the host and organism, this lesion can result in a number of alterations and is heavily controlled by the microbial components themselves (17)

Disease Immune response in periodontal:

When the balance between the infection control mechanisms and the subgingival biofilm—which includes Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Tannerella forsythia, and Treponema denticola—is upset, innate, inflammatory, and adaptive reactions are set off, which lead to the destruction of the tissues surrounding and supporting the teeth, and ultimately to the loss of tissues, bones, and ultimately the teeth. (13) The pathogenesis of periodontal disease is not solely caused by microbial films; the host cells immune system is also responsible for the degradation of periodontal ligaments.The balance between microbial biofilm and host cell is lost due to which remarkable variance in both dental plaque and Host immunity system occurs, which results in increment of Inflammatory cells leads to degradation of periodontal tissue and bone. Therefore decrease of anti-inflammatory cells Such as neutrophils, lymphocytes, granulocytes, etc. due To chronic persistence of microbial biofilms which results in severity of alveolar bone resorption by osteoclast and Leads to degradation of ligament fibers followed by chronic Periodontitis (10)

Gingivitis:

Poor oral hygiene causes bacterial plaque to build up, which, if left unchecked, can cause an acute inflammatory reaction in less than a week. This is gingivitis's early stage. Characterized by a rise in neutrophil counts and gingival crevicular fluid. Together with the fibrin deposition, the collagen fibers begin to break down. The change from neutrophilic lymphocytic infiltration to an early stage of gingivitis occurs at one week.An established lesion with primarily plasma cells and B lymphocytes results from further advancement into the chronic stage. Pockets create as it goes along, which causes the gingiva to separate from the tooth. Persistent inflammation causes the neighboring alveolar bone to resorb and the periodontal ligament to break down, which can eventually lead to tooth loss. (19)

Pathophysiology of Teeth:

The Two most common dental abnormalities are dental caries and malocclusion.

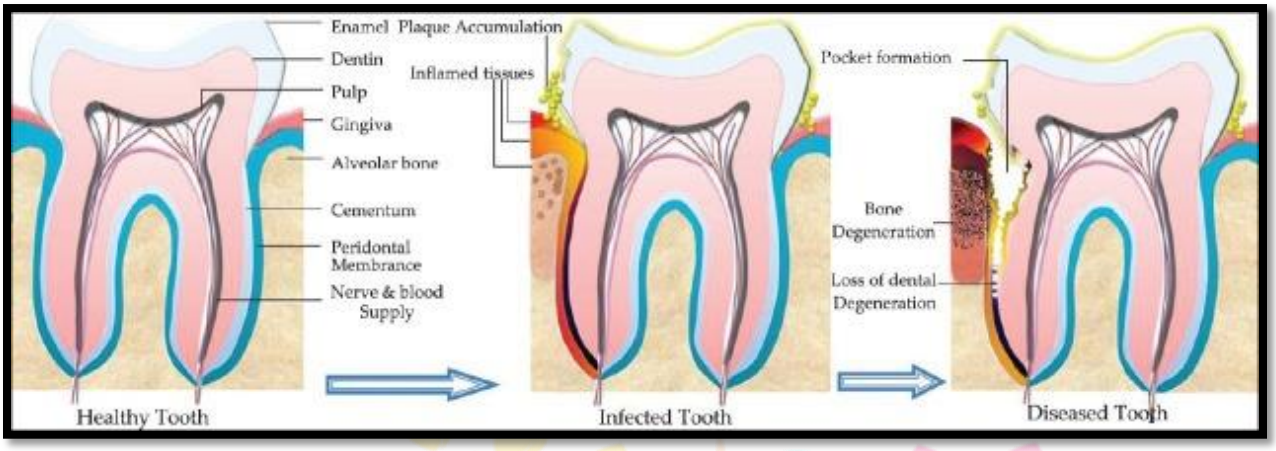


Fig 9. Healthy, Infected and Diseased Teeth. (16)

Dental Caries:

Dental caries, also referred to as cavities or tooth decay, is a complex condition that causes the teeth to become less mineralized and develop decayed areas, or "holes." Pain, infection, and tooth loss are signs of dental decay. Five times more common than asthma, dental caries is the most prevalent chronic pediatric illness in the US. (17) The most frequent bacteria that cause dental caries on teeth are Streptococcus mutans. The accumulation of plaque, a layer of food and salivary secretions, on the tooth is the catalyst for the development of dental caries. Carbohydrates are essential for the activation and growth of the bacteria that live in plaque. Additionally, these bacteria produce proteolytic enzymes and acids (lactic acid), which dissolve tooth calcium salts in the now extremely acidic environment and lead to caries. (20)

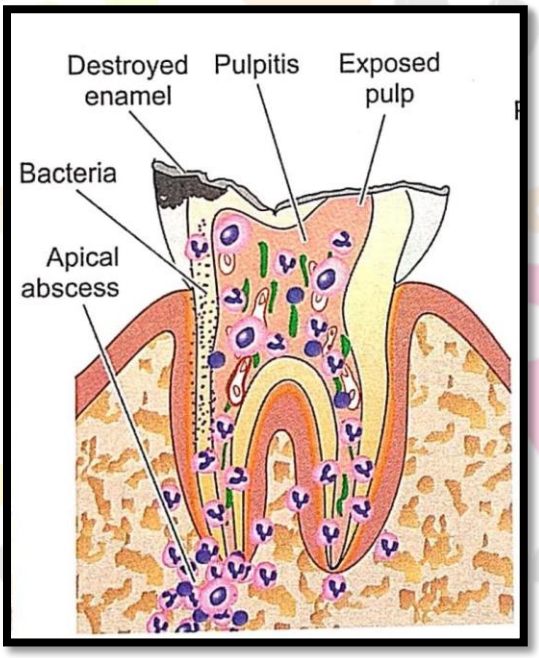


Fig 10. Dental Caries (12)

Etiopathogenesis:

Dental disease is primarily linked to a diet heavy in refined carbohydrates in contemporary society.For nearly a century, it has been known that when saliva and sugar or bread are combined with acidogenic oral bacteria, particularly streptococci, organic acids are produced that can decalcify enamel and The majority of enamel is made up of inorganic material, which essentially dissolves.After decalcification, biological material is still present in dentin. The remaining organic dentin material is proteolyzed by oral bacteria, finishing the degradation process. Carbohydrate-rich diets don't require much chewing, thus soft, sticky food sticks to the teeth instead

of being removed, especially around occlusal pits and fissures. Bacterial plaques develop in these areas of stagnation.The process of tooth decay starts if these plaques are not eliminated by brushing or vigorously chewing fibrous food.There is proof that consuming water with one part million (ppm) fluoride is enough to lower children's tooth decay rates. (12)

Malocclusion:

Misaligned teeth are referred to as malocclusion. Usually a genetic defect, malocclusion results in improper tooth interdigitation, which prevents the teeth from performing normal cutting and grinding functions. Sometimes, misaligned teeth can lead to abnormal displacement of the lower jaw relative to the upper jaw, which can cause tooth decay and mandibular joint pain.[8] An orthodontist is a graduate of dentistry school who specializes in dental development, occlusion, facial growth, and jaw alignment. They hold an advanced orthodontic degree. Braces are used by orthodontists to treat malocclusions by applying mild, continuous pressure on the teeth. The tooth is gradually moved to a new, desired position by this pressure, which results in the absorption of alveolar jaw bone on the compressed side of the teeth and the deposition of new bone on the tension side. (20)

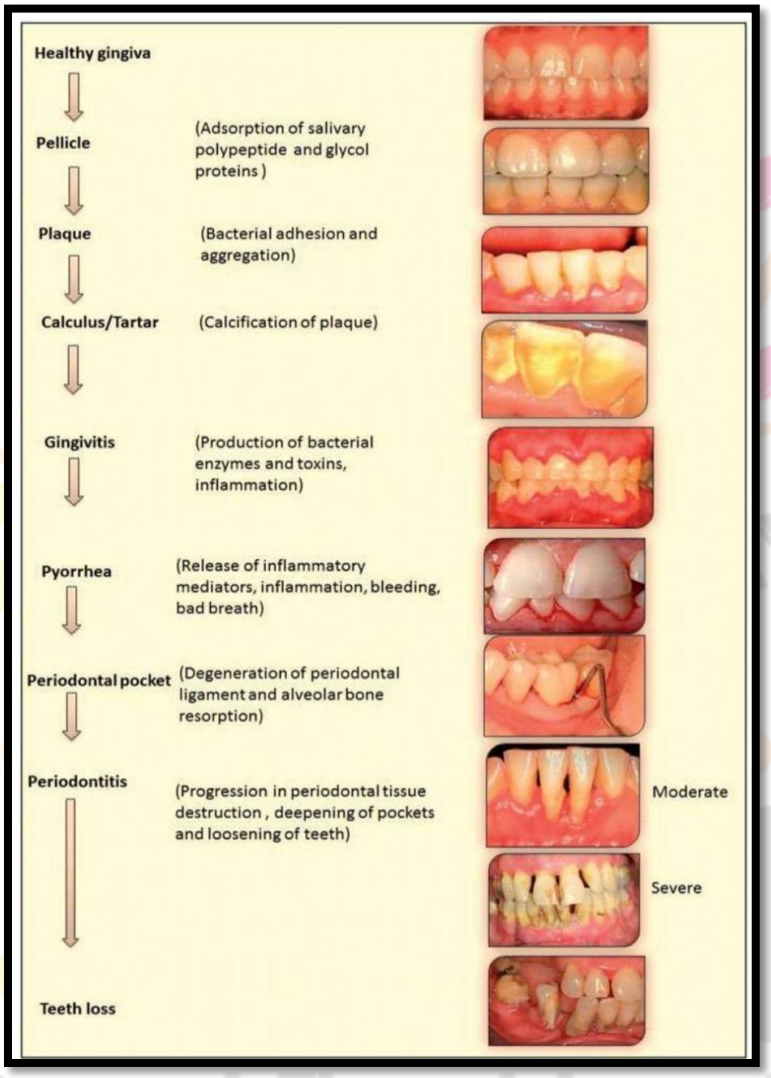


Fig 11. Various phases of periodontal diseases (15)

Conclusion:

The anatomy, pathophysiology of gums, teeth and mouth play a crucial role in maintaining overall oral heal health and systemic well-being. The gums provide structural support and protection for teeth, while teeth enable essential functions such a chewing and speech. The oral cavity, including the mucosa, salivary glands, and supporting structures, ensures proper digestion and defense against pathogens.

Understanding pathophysiological conditions, including gingivitis, periodontitis, and dental caries arising from microbial imbalances, poor oral hygiene, genetic factors and systemic diseases. These conditions can have far- reaching effects on overall health, linking oral health to conditions like diabetes and cardiovascular diseases. Maintaining good oral hygiene, regular dental check-ups and adopting healthy lifestyle choices are essential in preventing and managing oral diseases. Further research and advancements in oral and dental

care enhance health conditions, improving both individual well-being and public health outcomes. A deep understanding of all these aspects helps in early detection and prevention of oral diseases, acknowledging the importance of oral health care.

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