Effects of Periodontitis on Systemic Health

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Abstract- Periodontitis is a chronic inflammatory disease affecting the supporting structures of the teeth, including the gingiva, periodontal ligament, and alveolar bone. Beyond its local effects in the oral cavity, increasing evidence indicates that periodontitis has significant systemic implications. The dissemination of periodontal pathogens and inflammatory mediators into the bloodstream can contribute to the development or progression of several systemic conditions. This article reviews the relationship between periodontitis and various systemic diseases such as diabetes mellitus, hypertension, cardiovascular disease, metabolic syndrome, rheumatoid arthritis, chronic obstructive pulmonary disease, chronic kidney disease, malignancy and adverse pregnancy outcomes. The mechanisms underlying these associations include systemic inflammation, immune dysregulation, oxidative stress, and endothelial dysfunction triggered by periodontal infection. A bidirectional relationship has been particularly observed between periodontitis and certain systemic diseases, notably diabetes mellitus and metabolic syndrome. Understanding the link between oral health and systemic health highlights the importance of early diagnosis and effective management of periodontal disease. Maintaining periodontal health may therefore contribute not only to preserving oral structures but also to improving overall systemic health and quality of life.

Keywords - Periodontitis, Systemic health, Diabetes Mellitus, Cardiovascular disease, Hypertension, Adverse pregnancy outcomes, COPD, Rheumatoid arthritis, Malignancy.

I.INTRODUCTION

Periodontal disease, one of the most common oral health problems in the world, It is an inflammatory disorder that affects the supporting structures of the teeth, such as the gingiva and alveolar bone. This disease not only affects the Periodontium but also affect systemically. Several studies have found a link between periodontal disease and various systemic conditions. There is a substantial evidence showing that periodontal diseases are associated with systemic conditions such as Diabetes mellitus, Metabolic syndrome, Atherosclerosis, Coronary artery disease, Hypertension, Chronic Obstructive Pulmonary Disease, Rheumatoid arthritis, Chronic kidney disease, Adverse pregnancy outcomes, Alzheimer's disease, Malignancy. In this article we are going to discuss about how the Severity and progression of periodontal disease affects the Systemic Health.[1]

II.FOCAL INFECTION THEORY

In 1900, British physician William Hunter proposed that oral microorganisms were responsible for various systemic conditions that were not immediately recognized as infectious in nature. He claimed that restoring carious teeth rather than extracting them resulted in infectious agents being trapped under the restoration. In addition to caries, pulpal necrosis, and periapical abscess, Hunter also identified gingivitis and periodontitis as foci of infection. He advocated for tooth extraction to remove the source of sepsis. He stated that the severity of systemic effects caused by oral sepsis was determined by the virulence of the oral infection and the individual's resistance.

Finally, Hunter believed that the tooth extraction shows improvement of systemic health. Hunter's theory became widely accepted in Britain and in

United states. However, the focal infection theory fell into dispute during the1940's and 1950's, when extraction failed to reduce or eliminate the systemic conditions. Hunter and other advocates of the theory were unable to explain how focal oral sepsis produced these systemic maladies. Today's era of evidence-based medicine and dentistry provides an excellent environment in which to examine the possible relationships between oral infection and systemic disorders.[2]

III.EFFECT OF PERIODONTITIS ON SYSTEIC HEALTH

Increasing evidence shows that periodontal infection may influence systemic health through inflammatory mediators and bacteria entering the bloodstream. It has been associated with cardiovascular, respiratory, endocrine, kidney, autoimmune, and neurodegenerative diseases, highlighting the oral–systemic health connection.[23]

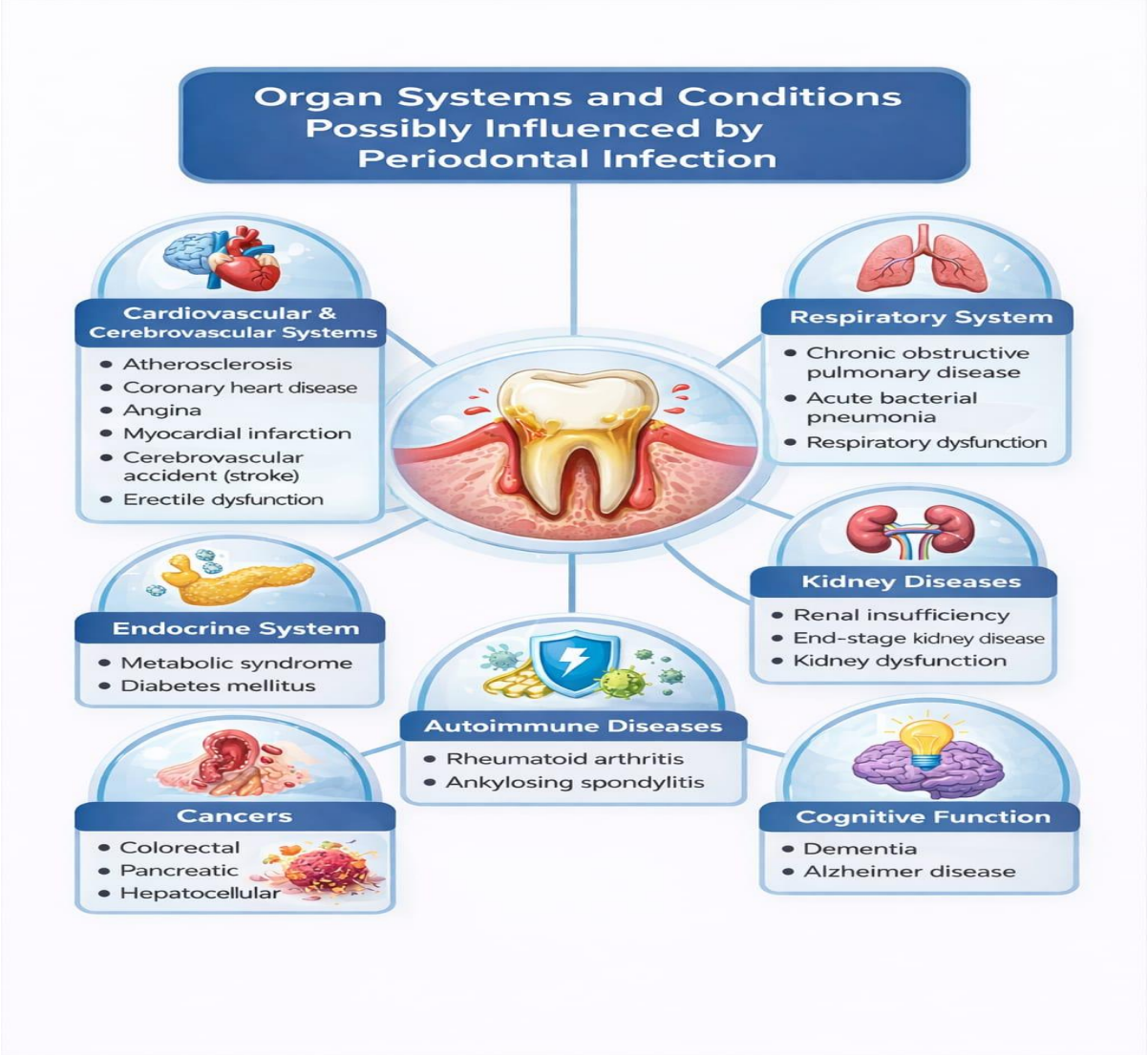

Fig1: Organ system and conditions possibly influenced by periodontal infection

IV.DIABETES MELLITUS

Diabetes mellitus is a complex metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both, in which the fasting blood glucose concentration is more than 6.1 mmol/L There are two main types of diabetes, type 1 and type 2 diabetes. Type 1 diabetes is an autoimmune disorder in which the body's immune

system targets and destroys insulin-producing beta cells in the pancreas, causing insulin deficiency. On the other hand, type 2 diabetes is a progressive condition where the body becomes resistant to the effects of insulin or doesn't produce enough insulin to maintain normal glucose levels.[3] There are five complications of diabetes mellitus namely, cardiac, vascular, renal, ophthalmic and neurological. In 1999 Löe described periodontal disease (PD) as the sixth complication of diabetes mellitus (DM) because this latter group of patients has a 3 – 4 times greater risk of developing PD when compared with non-diabetics. This rises to 10 times for smokers.[4]

The bidirectional relationship between diabetes mellitus (DM) and periodontal disease (PD) is well established, with each condition influencing the onset and progression of the other. Poorly controlled diabetes increases susceptibility to periodontal disease, while periodontitis negatively affects glycemic control. Type 2 diabetes is closely associated with systemic inflammation, which can impair pancreatic β-cell function, promote apoptosis, and induce insulin resistance. This inflammatory burden may be exacerbated by periodontal pathogens entering the bloodstream, providing a pathophysiological link between the two diseases. Central mechanisms underlying this association include dysregulation of advanced glycation end-products (AGEs), oxidative stress pathways, and activation of their receptors within periodontal tissues.[1]

Oxidative stress induced by chronic hyperglycemia results in an overabundance of advanced glycation end products, which attach to receptors on various cells. This interaction triggers the release of pro-inflammatory cytokines, including TNF-α and IL-6, thereby exacerbating both local and systemic inflammation. Furthermore, reduced neutrophil function in patients with diabetes impairs pathogen removal, while elevated CRP levels contribute to delayed wound healing. Pathogens such as Porphyromonas gingivalis worsen systemic insulin resistance by triggering inflammatory cytokine cascades.[5] Both diabetes and periodontitis exhibit elevated inflammatory markers such as IL-1β, TNF-α, IL-6, and an increased RANKL/osteoprotegerin ratio, alongside heightened oxidative stress and upregulation of TLR-2 and TLR-4, further reinforcing their interrelationship. Diabetes is also associated with periodontal ligament destruction and tooth loss, with higher levels of inflammatory mediators detected in gingival crevicular fluid and saliva of diabetic patients with periodontitis.[1]

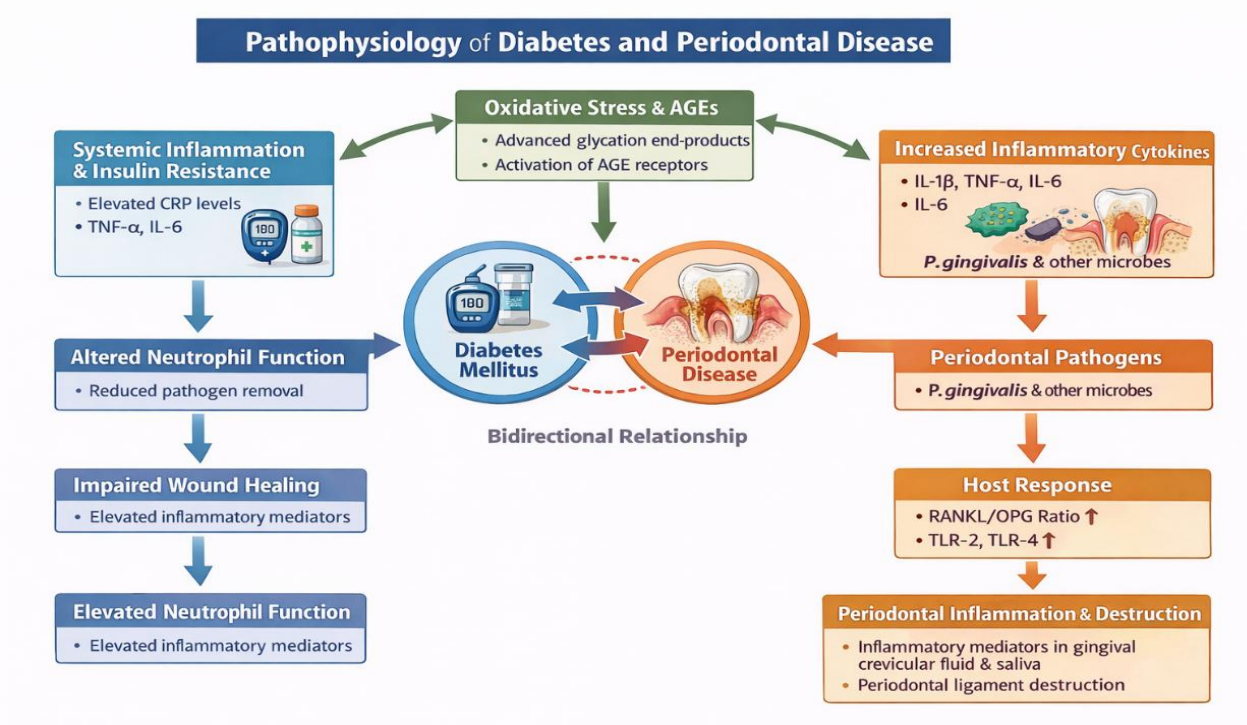

Fig 2: Pathophysiology of Diabetic mellitus and Periodontitis

HYPERTENSION

Arterial hypertension is one of the most common cardiovascular threat factors. In the world, about 45 people suffer from arterial hypertension, and its prevalence increases with age. According to a 2014 World Health Organization (WHO) report, arterial hypertension was the cause of 51 of deaths from stroke. The reason for the high frequence of arterial hypertension and its poor control is due to low knowledge of hypertensinogenic factors. One similar factor is periodontitis, which is a complaint of social significance.[6] Periodontitis can contribute to the overall inflammatory burden in individuals, leading to elevated CRP levels and, subsequently, an increased risk of CVD and hypertension. Gingival bleeding, indicative of periodontal inflammation, is consistently associated with increased systolic blood pressure and an elevated risk of developing hypertension.[1] Dental biofilms produce a range of biologically active substances, such as bacterial lipopolysaccharides, chemotactic peptides, protein toxins, and organic acids. The production and secretion of proinflammatory prostaglandins and cytokines, including interleukin-1 beta (IL-1β), interleukin-6 (IL-6), interleukin-8 (IL-8), and tumour necrosis factor-alpha (TNF-α), are activated in reaction to dental biofilm stimuli. The destruction of periodontal tissue is caused by the products produced by biofilms and the responses of the host.

Increased leukocytes infiltration and production of cytokines exaggerate oxidative stress and inflammation, eventually causing a disturbance to the normal endothelial function in regulating BP. Endothelial cells play a crucial part in BP homeostasis through the synthesis of vasodilators such as nitric oxide (NO), prostacyclin (PGI_2), and endothelium-derived hyperpolarising factor (EDHF) and vasoconstrictors such as endothelin-1, thromboxane (TXA_2), and angiotensin II. During endothelial dysfunction, the balance between these vasodilators and vasoconstrictors is disturbed.[7]

V.CARDIOVASCULAR DISEASE AND ARTHEROSCLEROSIS

Cardiovascular disease (CVD) is the primary cause of mortality globally, responsible for around 17.9 million deaths annually. CVD is a comprehensive term for disorders of the heart and blood vessels. Atherosclerosis serves as a fundamental cause of CVD. Atherosclerosis is a long-lasting inflammatory condition of the blood vessels characterized by the build-up of lipids (plaque) within the arterial walls. The development and progress of atherosclerosis can reduce blood flow in the arteries, leading to ischemia in various tissues or organs, as well as promoting clot formation. There is robust association between CVD and PD. There's a strong link between cardiovascular disease (CVD) and periodontal disease (PD).

Bacteria from the periodontium can enter the bloodstream and adhere to the endothelial cells that line blood arteries through the direct route. Oral bacteria, including the most prevalent Streptococcus mutans, Aggregatibacter actinomycetemcomitans, Tannerella forsythia, Prevotella intermedia, and Porphyromonas gingivalis, have been linked by PCR studies on atherosclerotic plaque. Some pathogens, especially P. gingivalis, may cause foam cell formation, inflammation, and endothelial cell dysfunction, all of which contribute to the development of atherosclerosis. According to the indirect mechanism, periodontal disease increases inflammatory mediators including TNF-α, IL-1, IL-6, and IL-8. These mediators raise fibrinogen and C-reactive protein (CRP), cause oxidative stress, interfere with lipid metabolism, and encourage endothelial dysfunction and atherosclerosis. [8]

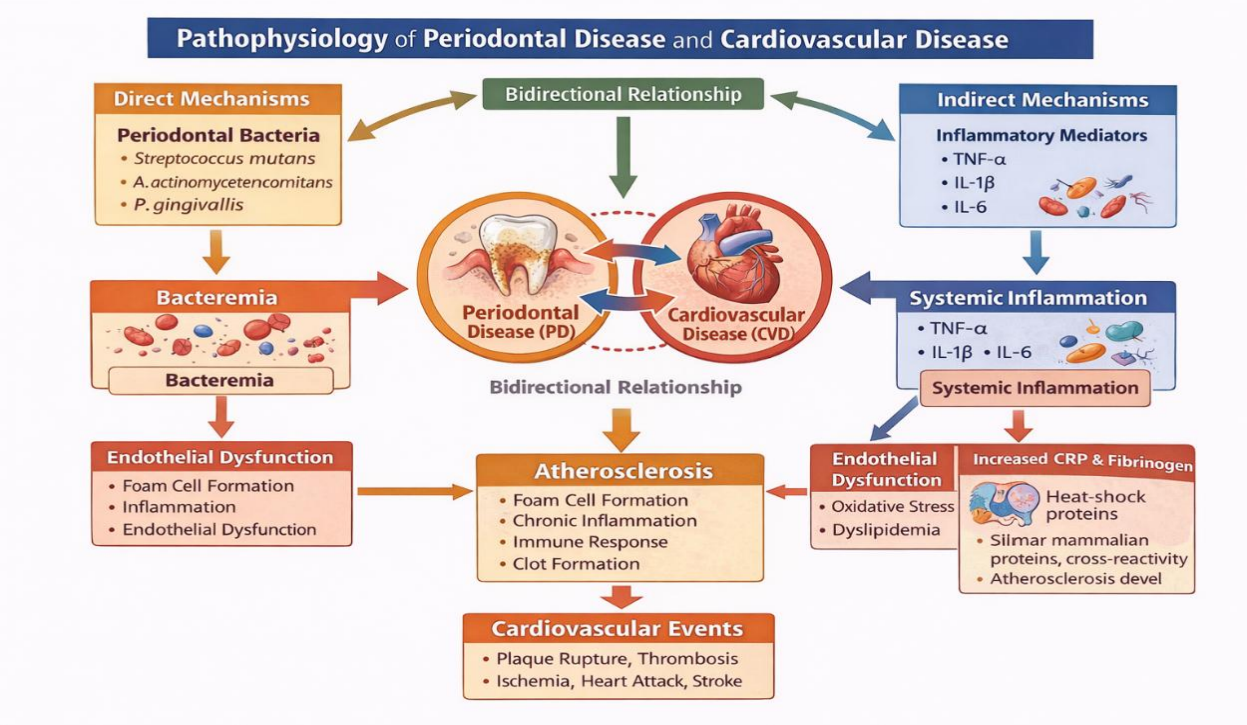

Fig3: Pathophysiology of cardiovascular disease and periodontitis

According to immunological studies, heat-shock proteins (HSPs) are the focus of an autoimmune reaction. Similar mammalian proteins in gingival connective tissue may cause the immune system to cross react after being primed by HSPs from periodontal pathogens like P. gingivalis.14 Atherosclerosis development has also been linked to this pathway.[1]

VI.PREGNENCY OUTCOMES

Delivery that takes place between 23 and 37 weeks of gestation is considered preterm. In addition to higher rates of chronic health issues like obesity and type 2 diabetes mellitus, preterm birth babies are more likely to experience neurological impairments like cerebral palsy, blindness, and deafness, respiratory issues like asthma, lower respiratory tract infections, and chronic lung disease, and behavioural disorders like attention deficit hyperactivity disorder. Preterm births account for 12.9 million births worldwide, or around 9.6% of all births. Due to their tiny size and immaturity, preterm babies are thought to have a higher risk of newborn mortality and morbidity. In underdeveloped countries, low birth weight (LBW), which is defined as an infant weighing less than five pounds eight ounces (2.5 kg), may be considered preterm birth. The average weight of newborns in india is about 2,900 g [6 lb 6 oz] [9].Preterm low birth weight may be caused by periodontitis through a number of interconnected processes. Gram-negative anaerobes like Fusobacterium nucleatum and Porphyromonas gingivalis are found in periodontal pockets. These organisms and their virulence factors can enter the bloodstream through normal actions like chewing or brushing one's teeth, enabling transfer to the fetoplacental unit. Prostaglandin E_2, tumour necrosis factor-α, interleukin-1β, and interleukin-6 are examples of pro-inflammatory mediators and circulating bacterial lipopolysaccharides that can cause uterine contractions, weaken foetal membranes, and impair placental perfusion, which can lead to premature labour or limit foetal growth. [10]

VII.METABOLIC SYNDROME

Concomitant disorders that double the risk of cardiovascular disease and double type 2 diabetes are together referred to as metabolic syndrome. According to the definition, a person must have at least three of the following risk factors: (a) increased abdominal circumference; (b) low plasma levels of high-density

lipoprotein cholesterol; (c) elevated plasma triglyceride values; (d) elevated blood pressure; and € elevated glucose levels. Prediabetes is considered part of metabolic syndrome because it is linked to insulin resistance and strongly predicts the future development of type 2 diabetes.[11]

The mechanism shows Proinflammatory cytokines from the gingiva enter the bloodstream and raise oxidative stress, which may promote insulin resistance and atherosclerotic alterations, both of which may contribute to the development of MetS. This is one of the underlying mechanisms that connect periodontitis to MetS.

The relationship is reciprocal since oxidative stress in the gingiva may be exacerbated by inflammatory cytokines produced by MetS components. Increased blood sugar causes a number of proinflammatory reactions that affect many body systems, especially the periodontal tissues. TNF-α, IL-6, and leptin are examples of adipokines from adipose tissue that cause inflammation. Advanced glycation end products (AGEs) are deposited in periodontal tissues as a result of hyperglycemia, which causes local cytokine production and modified inflammatory responses through the receptor for AGE (RAGE). Additionally, diabetes alters neutrophil activity, which increases the death of periodontal tissue by delaying apoptosis and magnifying the respiratory burst. Through systemic exposure, local cytokine production in periodontal tissues may reciprocally affect insulin signaling and glycemic control.[11]

VIII.RHUMATOID ARTHRITIS

Rhumatoid arthritis is accumulation of leukocyte inflammatory infiltrate in the synovial membrane and mediators like PGE2, TNF-α, IL-1β, IL-6, IL-12, IL-17, IL-18, IL-33, granulocyte macrophage colony-stimulating factor (GM-CSF), monocyte colony-stimulating factor (M-CSF), RANKL, MMPs, and NO in the synovial fluid. [12]

Chronic inflammation is a characteristic feature of both PD and RA, and TNFα is a key proinflammatory mediator. In RA, joint macrophages are the primary generator of TNFα . According to a study by Nilsson et al., the degree of systemic inflammation is correlated with plasma levels of TNFα, which may impact the development of Parkinson's disease in RA patients. [13].Globul et al., explained the link between RA and PD by two hit theory, the periodontal microenvironment's increased concentration of anaerobic microbes and their antigens is the initial "hit." Increased production of bone-resorptive cytokines (IL-6, IL-1, TNF-α) and tissue-destructive proteinases (MMPs) are among the destructive events of periodontitis that are triggered by this initial hit. The second "hit" is a systemic illness like RA that raises serological indicators of systemic inflammation (e.g., CRP, IL-6, IL-1β, PGE2, MMPs, and TNF-α). In a process that is similar to cytokine-driven osteoclast activation and bone destruction during the pathogenesis of RA, elevated serum levels of inflammatory mediators may further stimulate immune cells in the periodontium and increase the production of MMPs and RANKL, aggravating the destruction of non-mineralized and mineralized connective tissues in the periodontium.[14]

IX.CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The set of long-term lung conditions known as chronic obstructive pulmonary disease (COPD), which includes emphysema and chronic bronchitis, is defined by a restriction in airflow. Chronic inflammatory diseases including periodontal disease and chronic obstructive pulmonary disease (COPD) have a substantial impact on a person's general health and wellbeing.

Patients with COPD typically have a higher prevalence of periodontitis than people without the disease, which may be partially explained by common COPD risk factors such inflammation, smoking, and respiratory infections. Specifically, by encouraging inflammatory reactions and bacterial infections, periodontitis may worsen the course of COPD and further impair the respiratory system.[15]

Periodontal bacteria Aggregatibacter actinomycetemcomitans, Capnocytophaga sputigena, Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola were found in the tracheal aspirate of patients with severe acute exacerbation COPD, suggesting that dental bacteria may play a role in the pathophysiology of severe acute exacerbation COPD. These two conditions have certain pathogenic traits, including neutrophils, oxidative stress,

protease/anti-protease imbalance, T helper 17 (Th17) cell lineage, and inflammatory cytokines. Th17 and the cytokines it produces and secretes have a major impact on the degenerative processes of both COPD and periodontitis. The proinflammatory cytokine Interleukin-17 (IL-17), which is released by Th17 cells, promotes persistent inflammation, one of the main pathologic features of both periodontitis and COPD.

One of the primary clinical characteristics of both COPD and periodontitis is chronic inflammation, which is promoted by Th17 cells' secretion of the proinflammatory cytokine Interleukin-17 (IL-17). Chemokine (C-X-C motif) ligand 1 (CXCL1), chemokine (C-X-C motif) ligand 5 (CXCL5), interleukin-1β (IL-1β), interleukin-6 (IL-6), interleukin-10 (IL-10), interleukin-12 (IL-12), tumour necrosis factor-alpha (TNF-α), stromelysin, prostaglandin E2 (PGE2), and other proinflammatory cytokines. In a similar vein, IL-17 causes airway epithelial cells to release CXCL1, CXCL5, Granulocyto-colony-Stimulating-Factor (G-CSF), Granulocyte-macrophage colony stimulating factor (GM–CSF), and Chemokine (C-X-C motif) ligand 8 (CXCL8) in order to exacerbate lung tissue inflammation and damage.

Tissue destruction in COPD occurs due to an imbalance between proteases and antiproteases, especially neutrophil elastase and α1-antitrypsin (AAT). Increased elastase activity and AAT deficiency are linked to emphysema and other chronic respiratory diseases. MMPs contribute to lung tissue destruction in COPD and are normally regulated by tissue inhibitors in healthy conditions. Higher levels of neutrophil elastase and imbalance between AAT and elastase are also associated with periodontal diseases and tissue breakdown. Increased oxidative stress markers and reactive oxygen species (ROS) are found in both COPD and periodontitis, contributing to inflammation and tissue damage.[16]

X.CHRONIC KIDNEY DISEASE

An global public health concern is chronic kidney disease (CKD). According to the Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease (KDIGO), 9.1% of people worldwide are predicted to have chronic kidney disease at some point in their lives. According to the World Health Organization (WHO), chronic kidney disease (CKD) is the fourteenth most common cause of mortality globally. Furthermore, according to WHO projections, CKD could rank as the fifth most common cause of mortality by 2040. This is because serious infections, diabetes mellitus, cardiovascular illnesses, and other conditions have significant morbidity and mortality rates that result in expensive medical care.[17] Women are more likely than men to have chronic kidney disease (CKD), It is a prevalent illness of the urinary system with an average population prevalence of roughly 10% (Suriyong et al., 2022; Li et al., 2024).

Periodontal pathogens such as *Porphyromonas gingivalis* and their lipopolysaccharides enter the bloodstream through ulcerated gingival tissues, activating systemic immune responses. Local periodontal inflammation causes immune cell infiltration and release of cytokines like IL-1 and TNF-α, which spread through circulation and trigger systemic inflammation. Systemic inflammation increases oxidative stress, which activates the NLRP3 inflammasome and promotes the release of pro-inflammatory cytokines such as IL-1β, contributing to renal inflammation and fibrosis.

Circulating inflammatory mediators activate glomerular mesangial cells and tubular epithelial cells, inducing expression of inflammatory genes and causing localized kidney inflammation. Periodontal infection increases reactive oxygen species (ROS) production beyond antioxidant capacity, leading to mitochondrial dysfunction, lipid peroxidation, DNA damage, and impaired renal cell metabolism. Persistent inflammation and oxidative stress lead to glomerulosclerosis, tubulointerstitial fibrosis, endothelial dysfunction, reduced renal blood flow, and RAAS activation, ultimately impairing kidney structure and function[18]

XI.ALZEIMERS DISEASE

Alzheimer's disease (AD), a progressive neurological illness, rises exponentially with age. It is typified by an irreversible loss of neurons and neural connections that starts in the hippocampus and spreads throughout the brain. Alzheimer's patients gradually lose their autonomy and cognitive capacities. As a result, these

symptoms cause progressive dementia, which ultimately results in death.[19]

Periodontal diseases linked to Socransky's Red Complex is Porphyromonas gingivalis. Gingipains are virulence factors produced by this late colonising, Gram-negative, anaerobic bacterium. It has been shown that P. gingivalis can encapsulate virulence factors in its outer membrane vesicles, which can then be shed to enter host cells. These outer membrane vesicles have the ability to pass through biological barriers and ultimately arrive in the brain. These outer membrane vesicles of the common gut infection Helicobacter pylori have been shown to be absorbed by astrocytes, resulting in amyloid-β pathology and dementia.

Gingipains, lipopolysaccharide, and P. gingivalis DNA have all been found in the brain tissue of Alzheimer's patients. Red complex organisms like T. denticola and T. forsythia are among the other periodontal infections that have been shown to exist in different brain tissues . Furthermore, the brains of individuals with and without Alzheimer's disease have been found to contain antibodies against the periodontal infections P. gingivalis, T. denticola, A. actinomycetemcomitans, F. nucleatum, and P. intermedia. When patient had both periodontitis and Alzheimer's disease, their antibody levels rise. Additionally, Riviere et al. showed that Alzheimer's disease-affected brains were more likely than control brains to harbour Treponema species. Treponema may infect the brain by branches of the trigeminal nerve, as evidenced by the detection of Treponema in the trigeminal ganglia of both Alzheimer's disease and control specimens.

Herpes simplex virus type 1 (HSV-1) has been linked to Alzheimer's disease-affected brains and periodontitis. In the trigeminal ganglia, HSV-1 is known to exist in a latent condition that is occasionally awakened. The molecular pathways of HSV-1 pathogenesis in Alzheimer's disease, such as the production of neuroinflammation, oxidative stress, and mitochondrial dysfunction, are well supported by evidence.[20]

XII.MALIGANCY

Periodontal disease has been linked to several factors, including dental calculus, overhang restorations, tooth location, smoking, nutrition, diabetes mellitus, blood dyscrasia, age, and genetic changes. There is now plenty of information showing a connection between malignant alteration of the afflicted oral epithelium and persistent inflammation. For example, there have been reports of oral lichen planus, a persistent inflammatory condition, turning cancerous. The primary cause of cancer-related mortality, oral squamous cell carcinoma (OSCC) accounts for up to 90% of all mouth cancers. Other types include Colorectal, Pancreatic, Hepatocellular. A number of variables have been identified as risk factors, including alcohol, tobacco, betel quid consumption, malnutrition, viral infections, and oral microbiota. There are two types of cytokines: pro-inflammatory and anti-inflammatory. Tissue damage might occur from any disparities in their relative concentrations. Leukocytes and other immune mediators are drawn to periodontal inflammatory foci by chemokines. Chemokines, and their receptors also have a role in angiogenesis, cancer growth, cell motility, proliferation, and metastasis. Neutrophils, lymphocytes, monocytes/macrophages, fibroblasts, osteoblasts, endothelial cells, epithelial cells, and mast cells are among the cell types that produce chemokines in the oral cavity.[21]

Oral pathobionts may contribute to oral squamous cell carcinoma (OSCC) through multiple biological mechanisms. First, they can induce epigenetic modifications that alter gene expression, as seen in inflammatory genes such as GP1BB, MIR193, and ZAP70. Bacteria like Fusobacterium nucleatum and Porphyromonas gingivalis can downregulate tumor-suppressor pathways, including p53 and Ku70, through histone H2A phosphorylation, thereby enhancing cancer cell proliferation. Second, certain microbes directly promote carcinogenesis by producing toxins and metabolites—such as cytolethal distending toxin, reactive oxygen species, nitrosamines, and acetaldehyde—that damage DNA and impair repair mechanisms. Third, the periodontitis-associated microbiome can stimulate chronic inflammation through Toll-like receptor (TLR) signaling and activation of NF-κB and MAPK pathways, increasing cell proliferation and inhibiting apoptosis. Additionally, bacteria like P. gingivalis and Treponema denticola disrupt epithelial barrier integrity by degrading junctional proteins such as occludin and zonula occludens-1, increasing tissue

permeability. Finally, these pathogens may enable immune evasion by upregulating immune checkpoint ligands B7-H1 and B7-DC (PD-L1 related), which suppress T-cell responses and facilitate tumor progression. [22]

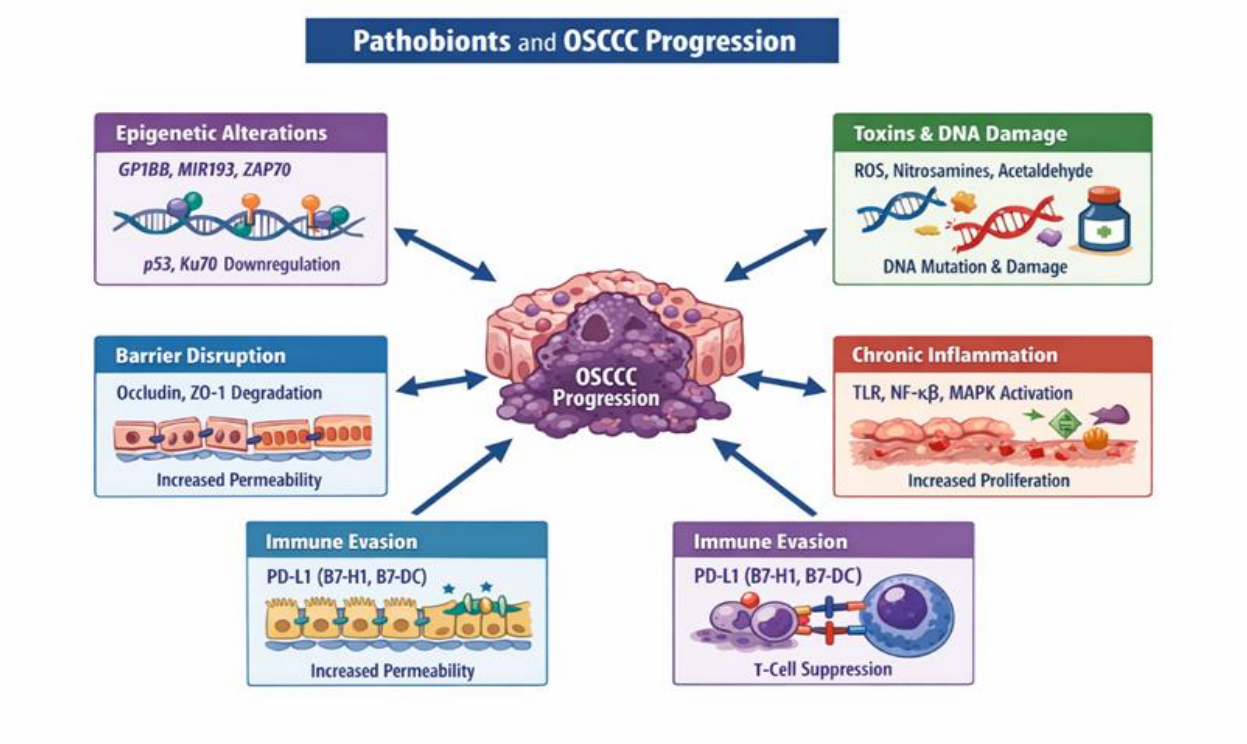

Fig4: Pathobionts and OSCCC Progression

Periodontitis is a common oral health problem worldwide, primarily caused by changes in the oral microbiome. The resulting inflammatory responses can increase the risk of genetic alterations and malignant transformation. Oral cancer develops through a complex, multistep process involving multiple cellular signaling pathways. Therefore, understanding these signaling pathways and identifying reliable biomarkers are important for the early detection and prevention of oral cancer.[21]

XIII.CONCLUSION

Periodontitis is not merely a localized oral disease but a chronic inflammatory condition with potential systemic consequences. Scientific evidence suggests that periodontal infection can influence the development and progression of multiple systemic diseases through inflammatory mediators, bacterial dissemination, and immune responses. Conditions such as diabetes mellitus, cardiovascular diseases, hypertension, metabolic syndrome, rheumatoid arthritis, malignancy, chronic kidney disease, chronic obstructive pulmonary disease, and adverse pregnancy outcomes have shown significant associations with periodontal disease.

These relationships are often bidirectional, where systemic conditions may also worsen periodontal status. Therefore, maintaining good oral hygiene and providing timely periodontal treatment are essential for overall health. Greater collaboration between dental and medical professionals is necessary to promote comprehensive patient care. Further research is still needed to better understand the biological mechanisms linking periodontitis with systemic diseases and to develop effective preventive and therapeutic strategies.

CONFLICT OF INTEREST: Nil

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