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Association Between Periodontal Health and Systemic Health: A Review Article

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Abstract:- Most current studies recommends that periodontal diseases correlated with systemic diseases which includes cardiovascular disorders, respiratory diseases, pregnancy, endocrine diseases, musculoskeletal system, nervous system, renal system and malignancy. The periodontal pathogens and its metabolic by products promotes the development of systemic diseases. A better understanding of oral microorganisms can contribute to the oral cavity to diagnose and possibly help in treating the systemic disease.

Keywords:- *Microorganisms, Oral Cavity, Periodontal Health, Systemic Diseases.*

I. INTRODUCTION

Oral cavity is the gateway to general health and mirror for well-being. General health and Oral health have a synergistic and dynamic relationship. Dental caries, endodontic and periodontal diseases are bacterially induced diseases which are originated by dysbiotic microbiomes. The focal sepsis theory suggested that these infections play a major role in the etiology of several systemic diseases. Periodontal disease is one of the familiar inflammatory diseases and is a constant potential source of infection that damages the gums and the jawbone, which is caused due to the periodontal ligament loss leading to destruction of alveolar bone. It is been postulated that the complex interplay between bacteria and its host responses results in periodontal diseases. The goal of this article is to view into depth of the various systemic disorders in relation to periodontal disease.

II. CARDIOVASCULAR DISEASES

An individual with periodontal disease have significant chance of developing cardiovascular disease than those without periodontal disease. It can be the result of either indirect or direct periodontal infection; an alternative pathway may be related to genetic and other host characteristics that promote vulnerability to both atherosclerosis/thrombosis and chronic periodontitis. [1].

When a periodontal infection is present, inflammatory mediators such C-reactive protein, TNF-, PGE2, IL-1, and IL-6 are produced. These mediators speed up the advancement of pre-existing atherosclerotic plaques and are also linked to an increase in the incidence of harmful cardiovascular disorders [2]. Furthermore, numerous investigations have shown that periodontal infections can cause platelet aggregation and atheroma development [3]. These systemic markers of inflammation are thought to function as predictors of current and upcoming cardiovascular events and illnesses.

Patients with periodontal disease generally have elevated fibrinogen levels and white blood cell counts. Individuals with poor oral health may have large elevations in coagulation factor VIII / von Willebrand factor antigen, raising the likelihood of thrombus formation. Periodontal infection may also promote increased blood viscosity and thrombogenesis, increasing the risk of central and peripheral vascular illness.

Periodontium functions as an endotoxin reservoir when periodontitis is present. Endotoxins enter the systemic circulation easily during normal activity, causing damage to the vascular endothelium and generating a variety of deleterious cardiovascular consequences.

Furthermore, in a study of 1163 men, a link between edentulousness and serum antibodies against P.gingivalis and A.actinomycetemcomitans and coronary heart disease was discovered [4]. An further investigation verified the presence of bacterial DNA in 42 atheromatous plaques extracted after endarterectomy. P. gingivalis was the most often discovered bacteria in this investigation, followed by A. actinomycetemcomitans, T. forsythia, Eikenella corrodens, Fusobacterium nucleatum, and Campylobacter rectus [5].

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Human atherosclerotic plaques included DNA from periodontal infections such P. gingivalis, A. actinomycetemcomitans, Prevotella intermedia, and T. forsythia, indicating that oral pathogens may spread to distant parts of the body [6].

Alveolar bone loss and aortic atherosclerosis are linked to bacterial infection, as evidenced by P. gingivalis and Treponema denticola [6].

Patients with periodontitis have a much higher prevalence and incidence of cardiovascular disorders.

III. RESPIRATORY DISEASES

People with periodontitis have 4.8 times more chances to have respiratory disease than people without periodontitis

The respiratory pathogen can colonise the mouth cavity because it is portable and next to the trachea. Patients with periodontal disease appear to have higher levels of pathogens in their saliva and dental plaque, which can lead to aspiration of such pathogens into the upper airway from the oropharynx.

Lung infections have been linked to a number of oral pathogens, including Actinomyces israelii, Capnocytophaga species, Chlamydia pneumoniae, E. corrodens, F. nucleatum, Fusobacterium necrophorum, P. gingivalis, P. intermedia, and Streptococcus constellatus. C. pneumoniae is well-studied respiratory pathogen and is associated with asthma, bronchitis and chronic obstructive pulmonary disease.

Lemierre's syndrome, which begins with pharyngitis and progresses to respiratory tract infection, was discovered to be caused by the common oral infections F. nucleatum and F. necrophorum.

The genetic similarity of respiratory pathogens isolated from dental plaque and Broncho alveolar lavage fluid from the same individuals supports the idea that dental plaque may serve as a substantial reservoir for respiratory pathogens [8].

The analysis of samples from the tongue, supragingival dental plaque, and tracheal aspirate revealed that 70% of the bacteria recovered from the tracheal aspirate were also present in the dental biofilm. According to the authors, the tooth biofilm serves as a reservoir for respiratory germs.

Salivary inflammatory indicators and GCF levels are higher in covid and post-covid patients, who also have more periodontal destructions. To support this claim, more investigation is needed.

In periodontitis, salivary enzymes modify the mucus surface, which increases the adherence and colonisation of respiratory infections. The destruction of salivary pellicles on pathogenic bacteria by periodontal disease-associated enzymes, as well as the modification of respiratory epithelium by periodontal disease-associated cytokines, enhances infection of the epithelium by respiratory pathogens [9].

IV. PREGNANCY

Maternal infections are linked to unfavourable pregnancy outcomes include preterm labour, preterm premature rupture of the membranes, pre-eclampsia, miscarriage, intrauterine growth retardation, low birth weight, stillbirth, and neonatal sepsis. Pregnant women are more prone to periodontal disease than non-pregnant ones because of hormonal changes in their bodies.

In order to explain the unfavourable pregnancy outcomes, two distinct pathways have been postulated.

The first mechanism is that oral pathogens directly migrate from an unhealthy oral cavity across the placenta and into the intra-amniotic fluid and fetal circulation [10]. The second mechanism is that endotoxins or inflammatory mediators produced during periodontal disease may spread throughout the body and affect fetal growth [11].

Preterm labour is caused by uterine muscle contraction brought on by bacterial infection and its microbial byproducts, such as LipoPolySaccharides (LPS), which encourages the activation of cell-mediated immunity and its subsequent production of cytokines like interleukins (IL-1, IL-6), tumour necrosis factor (TNF-a), and prostaglandins (PDE2) [12].

Infection causes aberrant production of the physiological mediators of parturition, which can cause births and also contributes to low birth weight.

F. nucleatum is frequently seen in intrauterine infections along with other oral subspecies. Patients who had premature deliveries also have P. gingivalis (and its endotoxins) in their placentas [12].

Recent research has shown that pregnant women with periodontal disease have a higher risk of developing gestational diabetes mellitus than pregnant women with healthy gums. [13]. Until now, the findings have been helpful in developing preventative strategies for pregnant women with periodontal disease. Females may develop gingival inflammation prior to menstruation as well as during ovulation due to elevated levels of progesterone, which inhibits collagen fibre repair and causes blood vessel dilatation. Lack of oestrogen lowers bone density after menopause, which can lead to the loss of alveolar bone and eventual tooth loss.

V. ENDOCRINE DISEASES

Diabetes mellitus is a chronic metabolic illness defined by hyperglycemia caused by a deficiency in insulin synthesis by pancreatic cells (type 1 diabetes), insulin resistance (type 2 diabetes), or a combination of both.

Bacteria from periodontal disease enter the circulation and trigger the body's immune system. These stimulated immune cells release inflammatory biological signals that have a damaging impact on the entire body.

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Periodontal disease can aggravate the body's resistance to insulin by raising levels of cytokines, C-reactive protein, TNF-, interleukin-6, and fibrinogen. This can prolong a state of chronic systemic inflammation [14].

The cells responsible for insulin generation in the pancreas are destroyed by prolonged high levels of cytokines, which can cause Type 2 diabetes even in people who have no other risk factors for diabetes.

The non-surgical periodontal treatment enhances the metabolic regulation of diabetic patients, which influences a decrease in glycine and glycemic haemoglobin levels [15].

However, if diabetes is not controlled, the recurrence of periodontal disease becomes more common and difficult to control. The link between periodontal disease and diabetes is well recognised.

VI. MUSCULOSKELETAL SYSTEM

There is an increased chance of developing rheumatoid arthritis in people with moderate to severe periodontitis.

Periodontal disease may play a causative role in the development and maintenance of the autoimmune inflammatory response that characterises rheumatoid arthritis. The basic pathogenic processes of RA and periodontal disease are similar [16].

Both disorders are thought to be driven by a poorly controlled inflammatory response, which results in oxidative stress-induced tissue injury [17].

Alveolar bone loss and tooth loss are common in RA patients and are also consequences of periodontal disease. There is also a significant link between systemic bone mass and oral bone loss.

VII. NERVOUS SYSTEM:

Age-related cognitive decline affects geriatric individuals' behaviour, particularly oral hygiene practises.

The Third NHANES-III data analysis found that people with poor cognitive function had high levels of the periodontitis-related blood marker (increased P. gingivalis IgG) [18].

Furthermore, a recent study by Kamer and colleagues demonstrated that clinical AL can encourage amyloid deposition in the brain, which can result in cognitive impairment [19].

Alzheimer's disease is a neurological condition that impairs memory, thinking, language, and learning abilities in a progressive and irreversible manner, eventually leading to death.

Alzheimer's disease is distinguished by the presence of activated glial cells, which release high levels of inflammatory cytokines.

Aside from the direct harm produced by -amyloid plaques and tau aggregates, the innate immune response seeks to remove these aggregates from the brain but instead aggravates neurodegeneration. As a result, pro-inflammatory cytokines are shown to be increased in older patients with periodontitis and Alzheimer's disease [20].

However, immunosuppressive cytokines and the IL-1 receptor antagonist can shield the brain from additional harm and slow the progress. The discovery of lipopolysaccharide (LPS) from periodontal infections including P. gingivalis and T. denticola in short-term postmortem human brains with Alzheimer's disease raises the possibility that these pathogens' virulence factors may contribute to the development of brain inflammation and Alzheimer's disease. Bacteria such as T. denticola and C. pneumoniae were found in postmortem Alzheimer's disease brains, implying that, in addition to inflammatory mediators, some periodontal infections may infiltrate the brain via the blood-brain barrier [21].

The symptoms of Alzheimer's disease and these periodontal bacteria appear to be related, but further research is required to draw a firm conclusion of Alzheimer's disease.

VIII. RENAL SYSTEM

Periodontal disease and chronic renal disease have a mutually reinforcing association. It was discovered by (Ioannidou and Swede) that those with CKD had a 30–60% higher risk of experiencing mild periodontitis [22].

According to a different study by Ioannidou et al., Mexican Americans with impaired kidney function had a twice as high risk of developing periodontal disease as those with normal renal function [23].

Similar findings were made by Iwasaki et al. in their study of older Japanese people , which linked periodontitis to decreased kidney function [24].

Ricardo et al. discovered that CKD patients with periodontitis had a 35% higher risk of mortality than CKD patients without periodontitis [25].

In-depth research is still required to firmly establish this truth.

IX. MALIGNANCY

The chance of acquiring haematological, lung, renal, pancreatic, and periodontal malignancies was found to be significantly correlated with periodontitis.

Patients with Oral Squamous Cell Carcinoma (OSCC) and Esophageal Squamous Cell Carcinoma (ESCC) were shown to have considerably higher levels of the periodontal pathogen P. gingivalis [26, 27].

P. gingivalis has been shown to encourage the invasion and metastasis of oral squamous cells in OSCC by upregulating the production of matrix metalloproteinase 9 (pro-MMP9) [26]. Oral F.nucleatum has the potential to move and colonise the human gastrointestinal tract, causing harmful inflammatory illnesses. As the fourth most common cancer in the world, colorectal carcinoma (CRC) is linked to high levels of F. nucleatum and Clostridium difficile in the intestinal microbiota of affected individuals [28].

The potential of F. nucleatum to modify the lumen microbiota, mediate cytokine release, and activate tumorigenesis-related pathways provides a pro-inflammatory milieu that may hasten the course of neoplasia in colorectal adenoma-carcinoma [29].

Research is currently ongoing and calls for conclusive evidence of a link between periodontal diseases and cancer.

X. DISCUSSION

Poor oral hygiene is strongly correlated with a buildup of dental plaque, a high prevalence, and a worsening of periodontal illnesses; therefore, improving periodontal health may have some beneficial effects on overall health. There is a clear link between periodontal disease and the musculoskeletal, endocrine, cardiovascular, and respiratory systems as well as pregnancy. Further studies are required to substantiate the indirect relationship between the central nervous system, renal system, and cancer.

XI. CONCLUSION

This article comes to the conclusion that systemic and periodontal health are related, either directly or indirectly. Before we could demonstrate the conclusive relationship between periodontal disease and CNS, renal, and cancer, more long-term studies were required.

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