

BIOCHEMICAL AND CLINICAL EVALUATION OF CORRELATION OF PERIODONTITIS AND EARLY CAROTID ATHEROSCLEROSIS

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Abstract

Periodontal disease is a disease characterized by inflammation of the periodontal tissues caused by microorganisms in the oral cavity and affects approximately 90% of the world population. Losses in the alveolar bone and connective tissue are observed due to periodontal diseases, which may lead to tooth loss. In addition, periodontal disease has been associated with many systemic diseases such as coronary artery diseases, stroke, head and neck cancer, pregnancy complications and diabetes. Periodontal disease is affected by risk factors such as smoking and diabetes, similar to vascular diseases. Hence based on above findings the present study was planned for Biochemical and Clinical Evaluation of Correlation of Periodontitis and Early Carotid Atherosclerosis.

The present study was planned in Department of Dentistry, Anugrah Narayan Magadh Medical College and Hospital, Gaya, Bihar, India. In the present study 20 cases of the Generalized chronic Periodontitis were enrolled in the Group A as study group patients. And 20 cases of Periodontally healthy individuals were enrolled in Group B as control group patients for comparative evaluation. Under aseptic measures, venous blood samples were drawn by venipuncture in antecubital fossa using 5ml syringe and collected in a plain vacuum tubes and transported to clinical laboratory for lipid profile analysis. Both the groups were subjected for ultrasonography for the assessment of Carotid Intima-Media Thickness (IMT) and Lipid Profile.

The data generated from the present study concluded that periodontal disease has association with early carotid atherosclerosis which is a risk factor for cardiovascular diseases. Thus oral health and systemic health are closely related and overall systemic health of an individual can be improved by maintaining a proper oral health regimen. It is important in terms of health policies that there is no need for an additional cost for this early diagnosis.

Keywords: Periodontitis, Early Carotid Atherosclerosis, Heart disease, etc.

Introduction:

Periodontal disease, also known as gum disease, is a set of inflammatory conditions affecting the tissues surrounding the teeth. In its early stage, called gingivitis, the gums become swollen, red, and may bleed. In its more serious form, called periodontitis, the gums can pull away from the tooth, bone can be lost, and the teeth may loosen or fall out. Bad breath may also occur. Periodontal disease is generally due to bacteria in the mouth infecting the tissue around the teeth. Risk factors include smoking, diabetes, HIV/AIDS, family history, and certain medications. Diagnosis is by inspecting the gum tissue around the teeth both visually and with a probe and X-rays looking for bone loss around the teeth.

Treatment involves good oral hygiene and regular professional teeth cleaning. Recommended oral hygiene include daily brushing and flossing. In certain cases antibiotics or dental surgery may be recommended. Globally 538 million people were estimated to be affected in 2015. In the United States nearly half of those over the age of 30 are affected to some degree, and about 70% of those over 65 have the condition. Males are affected more often than females.

The most common type of gingivitis involves the marginal gingiva and is brought on by the accumulation of microbial plaques in persons with inadequate oral hygiene. Gingivitis proceeds through an initial stage to produce early lesions, which then progress to advanced disease.

The initial stage of an acute exudative inflammatory response begins within 4 or 5 days of plaque accumulation. Both gingival fluid and transmigration of neutrophils increase. Deposition of fibrin and destruction of collagen can be noted in the initial stage. At approximately 1 week, transition to early lesions is marked by the change to predominately lymphocytic infiltrates. Monocytes and plasma cells also may be present. With time, lesions become chronic and are characterized by the presence of plasma cells and B lymphocytes. As chronic local inflammation progresses, pockets develop where the gingiva separates from the tooth. These pockets deepen and may bleed during tooth brushing, flossing, and even normal chewing. As this persistent inflammation continues, periodontal ligaments break down and destruction of the local alveolar bone occurs. Teeth loosen and eventually fall out.

ANUG is a completely different syndrome caused by acute infection of the gingiva with organisms such as *Prevotella intermedia*, alpha-hemolytic streptococci, *Actinomyces* species, or any of a number of different oral spirochetes. ANUG may result in accelerated destruction of affected tissues, as well as local or systemic spread of infection.

Frequency is difficult to determine because of the lack of agreement on measurement criteria. Many people believe that gingivitis begins in early childhood and that 9-17% of children aged 3-11 years have gingivitis. At puberty, prevalence rises to 70-90%. In recent years, periodontal disease, the endpoint of chronic gingivitis, slowly has decreased among adult Americans. However, chronic periodontitis is still the most prevalent chronic inflammatory condition in the elderly. [1] ANUG may be a clinical problem in immunocompromised patients during chemotherapy. Gingivitis and resulting periodontal disease are seen more frequently in patients with either diabetes or HIV.

Studies in Australia, Sweden, England, and Switzerland report gingivitis in 48-85% of children aged 3-6 years, but whether this range reflects population differences or whether it is due to different criteria used to define the disease is difficult to know. In adolescence, incidence around the world is comparable to US data (70-90%). ANUG may be found in areas where those at risk, particularly children, face poor living conditions. Recent publications show several cases in areas such as

Nigeria, where ANUG and noma were observed in children younger than 14 years. [2]

Periodontal disease has been shown in some studies to be an associated factor in coronary artery disease (CAD) and cerebrovascular disease/ischemic stroke. [3] Elevated levels of chronic inflammation (eg, C-reactive protein) have been shown to fall after treatment of periodontal disease. These elevated markers have clear association with vascular disease, so treatment of periodontal disease may theoretically have an impact on CAD and ischemic CNS disease. However, a clear cause-and-effect relationship has not been demonstrated between treatment of periodontal disease and improvement of atherosclerotic diseases or outcomes. A recent study that induced gingivitis in healthy volunteers was associated with a clear increase in inflammatory markers. [4]

A study by Sen et al that included data from 10,362 stroke-free participants, and 584 participants that had incident ischemic strokes over a 15-year period reported that periodontal disease was associated with cardioembolic (hazard ratio, 2.6; 95% confidence interval, 1.2-5.6) and thrombotic (hazard ratio, 2.2; 95% confidence interval, 1.3-3.8) stroke subtypes. The study also added that a lower stroke risk was associated with regular dental care utilization (hazard ratio, 0.77; 95% confidence interval, 0.63-0.94). [5]

A large study that enrolled 805 patients with a first myocardial infarction (MI) and 805 control patients without MI concluded that the risk of a first myocardial infarction was significantly increased in patients with periodontitis. The study found that 43% of MI patients vs 33% of matched controls had mild to severe periodontitis. After researchers controlled for smoking, diabetes, education, and marital status, individuals with periodontitis had a 28% increased risk of MI. [6]

Periodontal disease in pregnancy has been associated with an increase in preterm birth and adverse pregnancy outcomes. However, treatment of periodontal disease in pregnancy has not been shown to improve pregnancy outcomes. Periodontitis coexisting with bacterial vaginosis is associated with higher vaginal bacterial counts. Periodontitis in a person with diabetes has been associated with exacerbation of both conditions. Treatment of periodontitis in persons with type 2 diabetes has improved glycemic control. [7]

The primary cause of gingivitis is poor or ineffective oral hygiene, which leads to the accumulation of a mycotic and bacterial matrix at the gum line, called dental plaque. Other contributors are poor nutrition and underlying medical issues such as diabetes. Diabetics must be meticulous with their homecare to control periodontal disease. New finger prick tests have been approved by the Food and Drug Administration in the US, and are being used in dental offices to identify and screen people for possible contributory causes of gum disease, such as diabetes.

In some people, gingivitis progresses to periodontitis – with the destruction of the gingival fibers, the gum tissues separate from the tooth and deepened sulcus, called a periodontal pocket. Subgingival microorganisms (those that exist under the gum line) colonize the periodontal pockets and cause further inflammation in the gum tissues and progressive bone loss. Examples of secondary causes are those things that, by definition, cause microbial plaque accumulation, such as restoration overhangs and root proximity.

Smoking is another factor that increases the occurrence of periodontitis, directly or indirectly, and may interfere with or adversely affect its treatment. [8] It is arguably the most important environmental risk factor for periodontitis. Research has shown that smokers have more bone loss, attachment loss and tooth loss compared to non-smokers. The reason for this is that smoking has several effects on the immune response including: Decreased wound healing, Suppresses antibody production, It reduces neutrophilic phagocytosis. Ehlers–Danlos syndrome is a periodontitis risk factor and so is the Papillon–Lefèvre syndrome also known as palmoplantar keratoderma.

If left undisturbed, microbial plaque calcifies to form calculus, which is commonly called tartar. Calculus above and below the gum line must be removed completely by the dental hygienist or dentist to treat gingivitis and periodontitis. Although the primary cause of both gingivitis and periodontitis is the microbial plaque that adheres to the tooth surfaces, there are many other modifying factors. A very strong risk factor is one's genetic susceptibility. Several conditions and diseases, including Down syndrome, diabetes, and other diseases that affect one's resistance to infection, also increase susceptibility to

periodontitis. Another factor that makes periodontitis a difficult disease to study is that human host response can also affect the alveolar bone resorption. Host response to the bacterial-mycotic insult is mainly determined by genetics; however, immune development may play some role in susceptibility.

According to some researchers periodontitis may be associated with higher stress. Periodontitis occurs more often in people from the lower end of the socioeconomic scale than people from the upper end of the socioeconomic scale. Genetics appear to play a role in determining the risk for periodontitis. It is believed genetics could explain why some people with good plaque control have advanced periodontitis, whilst some others with poor oral hygiene are free from the disease. [9]

As dental plaque or biofilm accumulates on the teeth near and below the gums that is some dysbiosis of the normal oral microbiome. As of 2017 it was not certain what species were most responsible for causing harm, but gram-negative anaerobic bacteria, spirochetes, and viruses have been suggested; in individual people it is sometimes clear that one or more species is driving disease. Research in 2004 indicated three species gram negative anaerobic species: *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, *Bacteroides forsythus* and *Eikenella corrodens*. [8]

Plaque may be soft and uncalcified, hard and calcified, or both; for plaques that are on teeth the calcium comes from saliva; for plaques below the gumline, it comes from blood via oozing of inflamed gums.

The damage to teeth and gums comes from the immune system as it attempts to destroy the microbes that are disrupting the normal symbiosis between the oral tissues and the oral microbe community. As in other tissues, Langerhans cells in the epithelium take up antigens from the microbes, and present them to the immune system, leading to movement of white blood cells into the affected tissues. This process in turn activates osteoclasts which begin to destroy bone, and it activates matrix metalloproteinases that destroy ligaments.[48] So, in summary, it is bacteria which initiates the disease, but key destructive events are brought about by the exaggerated response from the host's immune system.[8]

The cornerstone of successful periodontal treatment starts with establishing excellent oral hygiene. This includes twice-daily brushing with daily flossing. Also, the use of an interdental brush is helpful if space between the teeth allows. For smaller spaces, products such as narrow picks with soft rubber bristles provide excellent manual cleaning. Persons with dexterity problems, such as arthritis, may find oral hygiene to be difficult and may require more frequent professional care and/or the use of a powered toothbrush. Persons with periodontitis must realize it is a chronic inflammatory disease and a lifelong regimen of excellent hygiene and professional maintenance care with a dentist/hygienist or periodontist is required to maintain affected teeth.

Removal of microbial plaque and calculus is necessary to establish periodontal health. The first step in the treatment of periodontitis involves nonsurgical cleaning below the gumline with a procedure called "root surface instrumentation" or "RSI", this causes a mechanical disturbance to the bacterial biofilm below the gumline.[8] This procedure involves the use of specialized curettes to mechanically remove plaque and calculus from below the gumline, and may require multiple visits and local anesthesia to adequately complete. In addition to initial RSI, it may also be necessary to adjust the occlusion (bite) to prevent excessive force on teeth that have reduced bone support. Also, it may be necessary to complete any other dental needs, such as replacement of rough, plaque-retentive restorations, closure of open contacts between teeth, and any other requirements diagnosed at the initial evaluation. It is important to note that RSI is different to scaling and root planing: RSI only removes the calculus, while scaling and root planing removes the calculus as well as underlying softened dentine, which leaves behind a smooth and glassy surface, which is not a requisite for periodontal healing. Therefore, RSI is now advocated over root planing.[8]

Nonsurgical scaling and root planing are usually successful if the periodontal pockets are shallower than 4–5 mm (0.16–0.20 in). The dentist or hygienist must perform a re-evaluation four to six weeks after the initial scaling and root planing, to determine if the person's oral hygiene has improved and inflammation has regressed. Probing should be avoided then, and an analysis by gingival index should determine the presence or absence of inflammation. The monthly

reevaluation of periodontal therapy should involve periodontal charting as a better indication of the success of treatment, and to see if other courses of treatment can be identified. Pocket depths of greater than 5–6 mm (0.20–0.24 in) which remain after initial therapy, with bleeding upon probing, indicate continued active disease and will very likely lead to further bone loss over time. This is especially true in molar tooth sites where furcations (areas between the roots) have been exposed.

If nonsurgical therapy is found to have been unsuccessful in managing signs of disease activity, periodontal surgery may be needed to stop progressive bone loss and regenerate lost bone where possible. Many surgical approaches are used in the treatment of advanced periodontitis, including open flap debridement and osseous surgery, as well as guided tissue regeneration and bone grafting. The goal of periodontal surgery is access for definitive calculus removal and surgical management of bony irregularities which have resulted from the disease process to reduce pockets as much as possible. Long-term studies have shown, in moderate to advanced periodontitis, surgically treated cases often have less further breakdown over time and, when coupled with a regular post-treatment maintenance regimen, are successful in nearly halting tooth loss in nearly 85% of diagnosed people.[10]

Local drug deliveries in periodontology has gained acceptance and popularity compared to systemic drugs due to decreased risk in development of resistant flora and other side effects. A meta analysis of local tetracycline found improvement. Local application of statin may be useful. [11]

Once successful periodontal treatment has been completed, with or without surgery, an ongoing regimen of "periodontal maintenance" is required. This involves regular checkups and detailed cleanings every three months to prevent repopulation of periodontitis-causing microorganisms, and to closely monitor affected teeth so early treatment can be rendered if the disease recurs. Usually, periodontal disease exists due to poor plaque control, therefore if the brushing techniques are not modified, a periodontal recurrence is probable.

Most alternative "at-home" gum disease treatments involve injecting antimicrobial solutions, such as hydrogen peroxide, into periodontal pockets via slender applicators or oral irrigators. This process

disrupts anaerobic micro-organism colonies and is effective at reducing infections and inflammation when used daily. A number of other products, functionally equivalent to hydrogen peroxide, are commercially available, but at substantially higher cost. However, such treatments do not address calculus formations, and so are short-lived, as anaerobic microbial colonies quickly regenerate in and around calculus.

Doxycycline may be given alongside the primary therapy of scaling (see § initial therapy).[63] Doxycycline has been shown to improve indicators of disease progression (namely probing depth and attachment level). Its mechanism of action involves inhibition of matrix metalloproteinases (such as collagenase), which degrade the teeth's supporting tissues (periodontium) under inflammatory conditions. To avoid killing beneficial oral microbes, only small doses of doxycycline (20 mg) are used. [12]

Dentists and dental hygienists measure periodontal disease using a device called a periodontal probe. This thin "measuring stick" is gently placed into the space between the gums and the teeth, and slipped below the gumline. If the probe can slip more than 3 mm (0.12 in) below the gumline, the person is said to have a gingival pocket if no migration of the epithelial attachment has occurred or a periodontal pocket if apical migration has occurred. This is somewhat of a misnomer, as any depth is, in essence, a pocket, which in turn is defined by its depth, i.e., a 2-mm pocket or a 6-mm pocket. However, pockets are generally accepted as self-cleansable (at home, by the person, with a toothbrush) if they are 3 mm or less in depth. This is important because if a pocket is deeper than 3 mm around the tooth, at-home care will not be sufficient to cleanse the pocket, and professional care should be sought. When the pocket depths reach 6 to 7 mm (0.24 to 0.28 in) in depth, the hand instruments and ultrasonic scalers used by the dental professionals may not reach deeply enough into the pocket to clean out the microbial plaque that causes gingival inflammation. In such a situation, the bone or the gums around that tooth should be surgically altered or it will always have inflammation which will likely result in more bone loss around that tooth. An additional way to stop the inflammation would be for the person to receive subgingival antibiotics (such as minocycline) or undergo some form of gingival surgery to access the depths of the pockets and perhaps even change the pocket depths

so they become 3 mm or less in depth and can once again be properly cleaned by the person at home with his or her toothbrush.

If people have 7-mm or deeper pockets around their teeth, then they would likely risk eventual tooth loss over the years. If this periodontal condition is not identified and people remain unaware of the progressive nature of the disease, then years later, they may be surprised that some teeth will gradually become loose and may need to be extracted, sometimes due to a severe infection or even pain. According to the Sri Lankan tea laborer study, in the absence of any oral hygiene activity, approximately 10% will suffer from severe periodontal disease with rapid loss of attachment (>2 mm/year). About 80% will suffer from moderate loss (1–2 mm/year) and the remaining 10% will not suffer any loss.[13]

Periodontal disease is a disease characterized by inflammation of the periodontal tissues caused by microorganisms in the oral cavity and affects approximately 90% of the world population. Losses in the alveolar bone and connective tissue are observed due to periodontal diseases, which may lead to tooth loss. In addition, periodontal disease has been associated with many systemic diseases such as coronary artery diseases, stroke, head and neck cancer, pregnancy complications and diabetes. Periodontal disease is affected by risk factors such as smoking and diabetes, similar to vascular diseases. Hence based on above findings the present study was planned for Biochemical and Clinical Evaluation of Correlation of Periodontitis and Early Carotid Atherosclerosis.

Methodology:

The present study was planned in Department of Dentistry, Anugrah Narayan Magadh Medical College and Hospital, Gaya, Bihar, India. In the present study 20 cases of the Generalized chronic Periodontitis were enrolled in the Group A as study group patients. And 20 cases of Periodontally healthy individuals were enrolled in Group B as control group patients for comparative evaluation. Under aseptic measures, venous blood samples were drawn by venipuncture in antecubital fossa using 5ml syringe and collected in a plain vacuum tubes and transported to clinical laboratory for lipid profile analysis. Both the groups were subjected for ultrasonography for the assessment of Carotid Intima-Media Thickness (IMT) and Lipid Profile.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: Cases of Both male and female patients aged 20-50 years participated in the study. Patients having BMI ranging between 25-30 Kg/m².

Exclusion Criteria: Pregnant or Lactating women and smokers. Patients with history of any antibiotics therapy 3 months prior to study enrolled or any other regular medication. Patients who underwent periodontal therapy for last six months.

Results & Discussion:

Atherosclerotic cardiovascular diseases describe a group of diseases including fatal or non-fatal coronary artery diseases (myocardial infarction, angina), ischemic cerebrovascular diseases (stroke) and peripheral arterial diseases. Tonetti et al. reported strong epidemiological evidences describing the relationship between periodontal disease and cardiovascular diseases. It was stated that the relation of periodontitis with cardiovascular diseases may be caused by the microbiota, which plays a role in the pathogenesis of periodontitis, directly or indirectly increasing the systemic inflammation and affecting the pathogenesis of athero-thrombogenesis. [14]

One of the main causes of the cardiovascular diseases is atherosclerosis which is characterised by thickening and the loss of elasticity of the arterial walls. Chronic periodontitis is one of the risk factor of coronary artery disease. Immune response to chronic periodontitis may exert an endothelial cytotoxic effect known to be a risk for atherosclerosis and periodontal pathogens have been demonstrated in atherosclerotic plaques removed during carotid endarterectomy in previous studies.

Chronic Periodontitis (CP) is distinguished as loss of periodontal attachment and alveolar bone. [15]. Previous studies demonstrated microorganisms related to periodontal diseases in carotid endarterectomy which establishes, an association between chronic periodontitis and carotid atherosclerosis. [16]

Table 1: Demographic Details

Group	Group A	Group B
Cases of	Study Group	Control Group
Cases of	Generalized chronic Periodontitis	Periodontally healthy individuals
Age:		
20 – 30 years	1	2
31 – 40 years	8	17
41 – 50 years	10	11
Sex:		
Males	13	9
Females	7	11

Table 2: Clinical Parameters

Group	Group A	Group B
Cases of	Study Group	Control Group
Cases of	Generalized chronic Periodontitis	Periodontally healthy individuals
Body Mass Index kg/cm ²	26.1 – 28.3	25.9 – 29.2
Plaque Index (PI)	0.83 – 1.29	0.45 – 1.32
Gingival Sulcus Bleeding Index	0.32 – 1.01	0.19 – 0.53
Probing Pocket Depth (PPD)	1.89 – 3.57	0.86 – 1.63
Clinical Attachment Level (CAL)	0.39 – 2.2	0

Table 3: Serum Lipid Profile

Group	Group A	Group B
Cases of	Study Group	Control Group
Cases of	Generalized chronic Periodontitis	Periodontally healthy individuals
Total Cholesterol (mg/dl)	175.4 – 235.8	161.7 – 198.3
Triglycerides (mg/dl)	158.4 – 267.5	94.8 – 155.3
HDL(mg/dl)	37.8 – 44.2	34.2 – 40.5
LDL (mg/dl)	91.5 – 148.	101.4 – 147.3
VLDL(mg/dl)	27.4 – 57.6	15.8- 34.3

Table 4: Carotid Intima-Media Thickness

Group	Group A	Group B
Cases of	Study Group	Control Group
Cases of	Generalized chronic Periodontitis	Periodontally healthy individuals
CIMT: Carotid Intima-Media Thickness	0.59 – 1.02	0.53 – 0.89

Studies report that people with periodontal disease or with few or no teeth experience an elevated risk of cardiovascular disease. Chronic infection with inflammation and change in diet are the proposed pathways linking tooth loss and cardiovascular disease.[17] Using an index based on the severity of caries, periodontitis, periapical lesion and pericoronitis they found that patient admitted to hospital for acute myocardial infarction had high scores on the dental index than matched controls of the population. Adults in United States have an average of 10-17 decayed, missing or filled teeth and most of them have experienced periodontal disease.[18] Periodontal pathogens, for example *Bacteroides forsythus* (*Tannerella forsythensis*), *Porphyromonas gingivalis*, and *Prevotella intermedia*, have been identified in

atherosclerotic plaques as well as in human aortic and coronary endothelium.[19]

Infectious agents relevant in oral health such as *Streptococcus sanguis* and *actinobacillus*, *actinomycetemcomitans* have also been shown to have possible direct effects contributing to the pathogenesis of atherosclerosis and thrombosis. Oral hygiene index seemed to have a stronger association, indicating that oral health indices may be general indicators of personal health behaviour. Instead of being casually related to coronary heart disease.[20]

Research studies continue to investigate the possible relationship between periodontal disease and cardiovascular disease. Some studies have shown that bacteria in the mouth that are involved in the development of periodontal disease can move into the blood stream and cause an elevation in C-reactive protein, a marker for inflammation in the blood vessels. These changes can, in turn, increase the risk of heart disease and stroke. It has been suggested that periodontal disease-associated bacteria can penetrate gingival tissues and enter the blood stream. Periodontal disease associated bacteria could enter the blood stream and play a direct or indirect role in the Progression of atherosclerotic coronary artery plaque lesions. [21] Periodontal disease caused by the gram negative bacteria found in the oral flora is common among the adults. Over time the bacterial endotoxins in the mouth may enter the systemic circulation through gingival connective tissue causing vascular injury. [22]

The probable link between oral and systemic disease dates back to 1900 when the concept of 'Oral sepsis' was put forward by a British physician, William Hunter. Subsequently in 1912, it was superseded by 'focal infection' by Frank Billings. Two major mechanisms of focal infection were proposed - an actual metastasis of organisms from a focus and the spread of toxins or toxic products from a remote focus to other tissues by the blood stream. Though the theory was accepted initially, it was later disregarded due to various reasons. [24]

Over the last two decades, the whole concept of focal infection has resurfaced with the work done by Matilla et al., in 1989 who found a highly significant association between poor dental health and acute myocardial infarction. [24] Of the various oral health related conditions, researchers have investigated the

relationship between periodontitis and atherosclerotic cardiovascular disease and have thrown light on the underlying biologic plausibility that exists between them.

In 1996, Beck et al. [47] proposed an initial hypothesis to explain the relationship between PD and atherosclerosis. The authors developed their theory by taking into account individual differences in host response against bacterial assault. This host response may be abnormally large and cause the release of important proinflammatory mediators such as prostaglandin (PGE) 2, interleukin (IL) 1- β , or tumor necrosis factor (TNF)- α by monocytes. This particular phenotype is called the monocytic hyperinflammatory phenotype, and patients with this phenotype produce 3–10 times more proinflammatory mediators in response to a stimulus by bacterial lipopolysaccharide. [25] Patients with this phenotype are at risk of developing PD because the cytokines produced by cells in contact with bacteria recruit inflammatory cells, degrade connective tissue, and induce bone destruction; from the cardiovascular perspective, these patients are at risk of CVD due to the over-expression of pro-inflammatory cytokines and dissemination through blood flow.

As periodontitis continues to have a high prevalence within the population and the fact that CVD remains a major cause of human death in developed countries, in light of these associations we can legitimately based on evidence state that oral health has an influence on systemic health in general & in CVD in particular & therefore we should promote oral health in general periodontal health in particular as part of a healthy life style and hence as an important component in the prevention of CVD.

Conclusion:

The data generated from the present study concluded that periodontal disease has association with early carotid atherosclerosis which is a risk factor for cardiovascular diseases. Thus oral health and systemic health are closely related and overall systemic health of an individual can be improved by maintaining a proper oral health regimen. It is important in terms of health policies that there is no need for an additional cost for this early diagnosis.

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