Risk factors for periodontal diseases

Vaishali Sachan [1], Janardhana Amaranath B.J. [2], Shruti Gupta [3], Neelam Das [4]

[3] *PG student, Department of Periodontology, Rama Dental College Hospital and Research Centre Kanpur*

[2]*Professor and head, Department of Periodontology, Rama Dental College Hospital and Research Centre Kanpur,*

[3-4] *Assistant Professor, Department of Periodontology, Rama Dental College Hospital and Research Centre Kanpur,.*

Abstract

The role of risk factors in the prediction of clinical periodontal outcomes, and thus in patient management, continues to be a subject of high professional interest and clinical relevance globally. Identifying risk factors as well as undertaking measures that can reduce the risk, can help in maintaining oral health and prevent the onset of any form of periodontal disease. Risk management involves dental care providers to identify patients and populations at increased risk of developing periodontal disease. The awareness of risk factors also helps for identification and treatment of co morbidities in the general population as many periodontal disease risk factors are common to other chronic diseases such as diabetes, cardiovascular diseases and stroke.

Keywords: Periodontal disease, risk factors, smoking, diabetes.

Introduction

Periodontal disease is a highly prevalent, multifactorial, chronic inflammatory disease of periodontium eventually leading to destruction of supportive tissues of teeth and tooth loss.[1] Risk can be identified in terms of risk factors, risk indicators or risk predictors. Risk factors play an important role in the etiopathogenesis of periodontal diseases, as well as they affect the overall treatment plan designed for the patient. Risk factors are biologically related to the occurrence of the disease, but they do not necessarily imply cause and effect, i.e., just because a patient possesses a risk factor does not mean that they will definitely develop the disease. Equally, absence of a risk factor does not mean that the disease wills not develop. [2] Therefore, aim of this review is to assess risk factors which are contributing in the progression of periodontal disease.

A. Tobacco smoking

Tobacco smoking is considered one of the true risk factors and is known to be independently related to periodontal destruction.[3] The common forms of tobacco smoking are cigarette, beedi, chutta and hookah, with cigarettes being the main product smoked.[4] Tobacco use is directly related to a variety of medical problems including cancer, low birth weight, pulmonary disease and cardiovascular diseases.[5] The increased prevalence and severity of periodontal destruction associated with smoking suggests that the host-bacterial interactions normally seen with chronic periodontitis are altered, resulting in more extensive periodontal breakdown. This imbalance between bacterial challenge and host response may be caused by changes in the composition of the subgingival biofilm (e.g., increases in the numbers and virulence of pathogenic organisms), changes in the host response to the bacterial challenge, or a combination of both. [4]

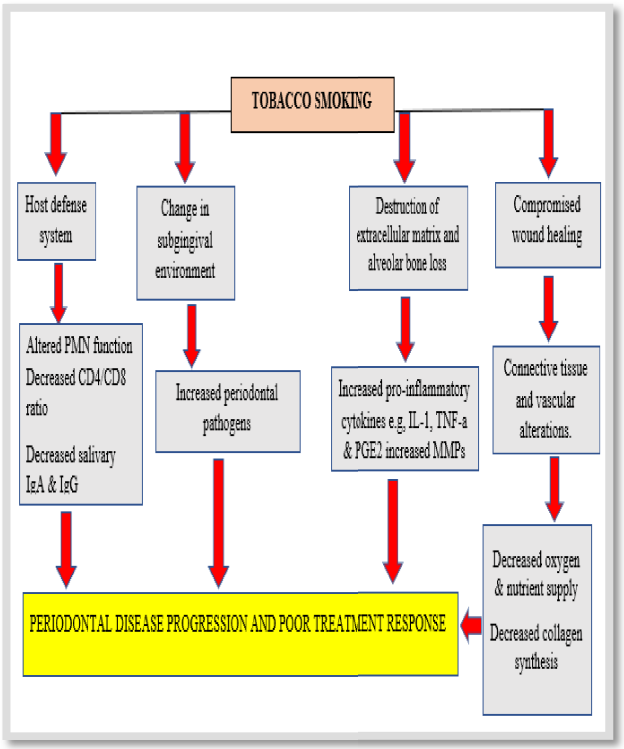

Figure 1: Smoking effects on cellular level [5]

B. Diabetes

Diabetes mellitus is the most common endocrine disorder, characterized by an inability of the body's cells to utilize glucose. It is of two types: Type 1 diabetes results from cellular mediated autoimmune destruction of pancreatic ß-cells, usually leading to total loss of insulin secretion. Type 2 diabetes, which is also known as non-insulin dependent diabetes, results from insulin resistance, which alters the use of endogenously produced insulin at the target cells. [6, 7]

Periodontitis is the most common chronic oral infection, caused by Gram-negative microorganisms and major cause of tooth loss in adults and has been considered the sixth complication of diabetes mellitus.[8] Conversely, periodontitis was shown to be a risk factor for poor glycemic control in patients with diabetes[6] due to bacteria and their byproducts in the inflamed periodontal tissue constituting a chronic source of systemic challenge to the host.[9] The bacteria involved in periodontitis are usually anaerobic gram-negative bacteria. They cause damage to periodontal tissues directly by releasing their virulence factors and indirectly by initiating the release of various inflammatory mediators by host immune cells. Gram-negative bacteria-derived lipopolysaccharide (LPS) is a potent inducer of TNF-α from monocytes and macrophages. Some locally produced proinflammatory immune mediators like tumor necrosis factor-alpha (TNF-α), which is known for inducing insulin resistance, may be found from the ulcerated periodontal pockets into systemic circulation and subsequently increase insulin resistance and exacerbate glycemic metabolic control in patients with diabetes. [10] For example, a dose–response relationship was observed between the severity of periodontitis and the serum TNF-α level in patients with type II diabetes. [11]

Signs of gingivitis and periodontitis in diabetic patients [12]

1. Inflamed gingival tissues that bleed easily.
2. Bluish/purple gingiva due to vascular changes.
3. Multiple periodontal abscesses results in bone loss & leads to mobility of teeth.
4. Proliferation of tissue at the gingival margin.
5. Lack of resolution of gingival signs after non-surgical periodontal therapy.
6. Delayed wound healing following oral surgical or non-surgical procedures.

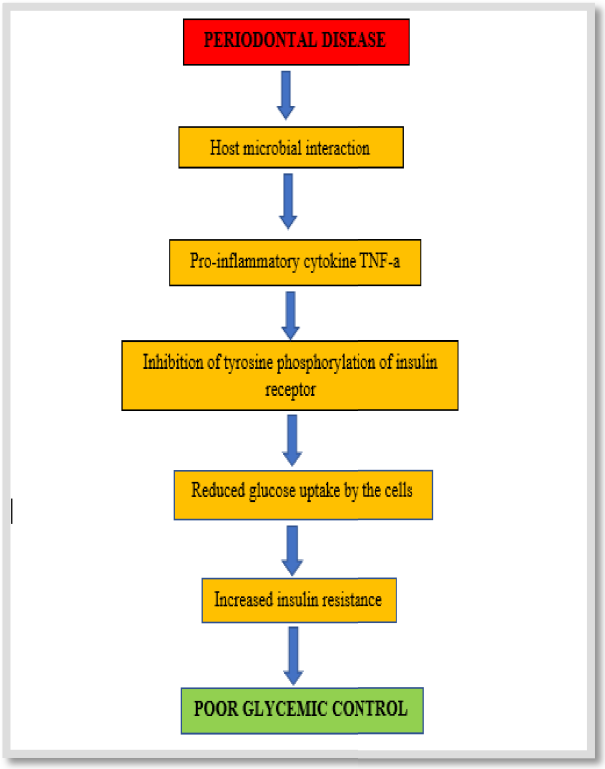

Figure 2: The effect of periodontal disease on diabetes mellitus [11]

C. Pathogenic bacteria & microbial tooth deposits

About 300 to 400 bacterial species are found in sub-gingival plaque but only about 10 to 20 species may play a role in the pathogenesis of destructive periodontal disease. It is only those species that are able to colonize that result in damage to periodontal tissues. Colonization requires the ability to attach to periodontal tissues, to multiply, to compete with other microbes within the oral environment and the resilience to survive the host defense mechanisms. The microbes involved with periodontal disease are largely gram-negative anaerobic bacilli, as mentioned, with some anaerobic cocci and a large quantity of anaerobic spirochetes. The main organisms linked with deep destructive periodontal lesions are Porphyromonas gingivalis, Prevotella inter-media, Bacteroides forysthus, Actinobacillus actinomycetemcomitans, and Treponema denticola. The accumulation of bacterial plaque at the gingival margin results in the development of gingivitis and that the gingivitis can be reversed with the implementation of oral hygiene measures.[13] Often, patients with severe loss of attachment have minimal levels of bacterial plaque on the affected teeth, indicating that the quantity of plaque is not of major

importance in the disease process. However, although quantity may not indicate risk, there is evidence that the composition, or quality, of the complex plaque biofilm is of importance. [13]

D. Cardiovascular disease

Periodontal diseases might affect heart disease through direct involvement of oral bacteria, indirectly by bacterial toxins, and by inducing inflammatory mediators that enter the bloodstream and contributing to chronic and systemic vascular challenge. Bacterial DNAs of periodontal pathogens such as Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, Aggregatibacter actinomycetemcomitans and Campylobacter rectus have been detected in stenotic coronary artery plaque samples.[14] The DNA of these microorganisms has also been detected in aneurysm walls and aneurysmal thrombus tissues.[15] Another possibility is that the inflammation caused by periodontal disease induces inflammatory cell infiltration into major vessels,[16] vascular smooth muscle proliferation, vascular fatty degeneration, and increasing plaque build-up, which contributes to swelling and thickening of the arteries.[17]

Association between periodontitis and atherosclerosis

The bacteria entering the systemic circulation from periodontal sites may contribute to the development of atherosclerosis. [18] The non-surgical periodontal therapy has been shown to cause transient bacteremia which causes initial, transient elevation in inflammatory and pro thrombotic mediators and an overall decrease of endothelial function [19]. Furthermore, following completion of the treatment, a reduction in the inflammatory markers, both at levels of local and systemic levels. [20] The periodontal infection may indirectly facilitate atherosclerosis by the end toxins [21] and cytokines that enter the circulation overload and atherosclerosis. [22]

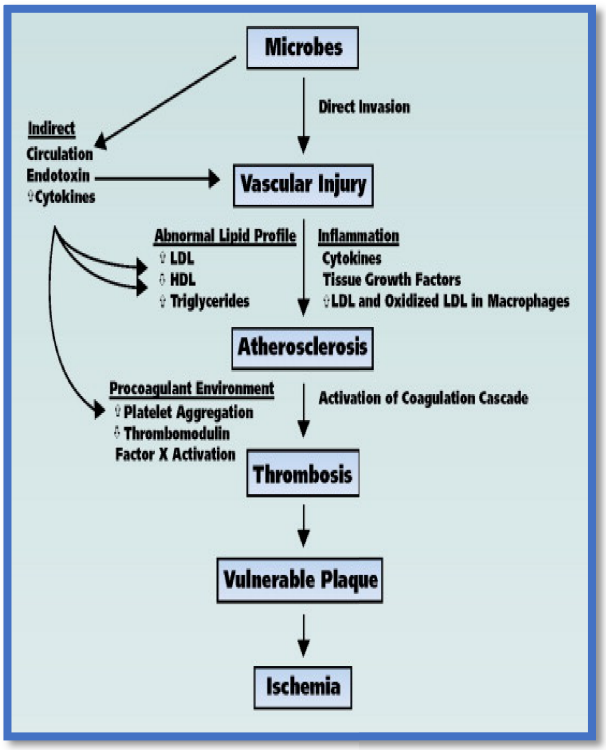

Figure 3: Periodontitis as a risk factor for coronary heart disease [22]

Infective endocarditis

Bacterial endocarditis is an infection of the inner surface of the heart or the heart valves, caused by bacteria, usually found in the mouth, intestinal tract or urinary tract. It can seriously damage the heart valves and cause other serious complications if it is not treated quickly with antibiotics. Certain oral bacteria are known to be the causative agents associated with infective endocarditis. Oral bacteria Streptococcus viridans is responsible for about 50% of all bacterial endocarditis cases. [23]

Myocardial Infarction

The periodontal infections have direct and indirect effects on systemic conditions, directly by microbes and their products and indirectly by chemical mediators or pro-inflammatory cytokines. The periodontal pathogens or their lipopolysaccharides are systemically disseminated via the blood flow and directly infect the vascular endothelium, producing an atherosclerotic lesion and subsequent myocardial ischemia. The endotoxins produced by these microorganisms, along with inducing the production of pro-inflammatory cytokines also induce the production of acute-phase proteins in the liver, such as C-reactive protein (CRP). These proteins can form deposits in damaged blood vessels, with the

consequent activation of phagocytes and release of nitrous oxide, contributing to the formation of atheromas". CRP has been shown to be not only a prognostic indicator of acute coronary syndromes, but also a predictor of future coronary events. [22]

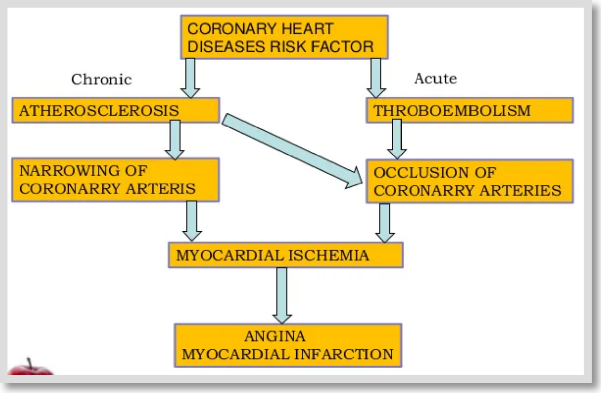

Figure 4: Relationship between Periodontal Disease and CVD [22]

E. Familial & Genetic Factors

Periodontitis is a multifactorial disease in which both environmental and genetic factors play a role. Genetic susceptibility to multifactorial diseases is usually due to several gene polymorphisms instead of a single, or few, gene mutations. [18] Common variation in the genetic code may results in altered expression or in functional changes of the encoded molecules, therefore resulting in an increase of disease severity or making individuals with genotypes more susceptible to a given disease. [19]

F. Psychological Factors

Psychosocial stress is thought to play an important role in the etiology and progression of periodontal diseases, and the outcome of treatment.[20] There is a strong relationship between periodontal diseases and psychological factors such as stress, distress, anxiety, depression and loneliness.[21] The factors responsible for such an association could be due to psychologically induced modulation of the immune system, leading to increased blood levels of adrenocorticotropic hormones, alteration of crevicular cytokine levels, depressed polymorphonuclear leukocyte chemotaxis and phagocytosis, reduced proliferation of lymphocytes, changes in blood circulation and healing, modifications of the salivary flow and its components and endocrine changes leading to immunosuppressant.[22]

G. Dietary habits

A significant association between poor overall diet quality and higher periodontitis prevalence has also been reported. [23] The mechanism underlying the association between diet quality and periodontitis could be related to the local and/or systemic effect of diet on periodontal health. The type and amount of food consumed has been linked to the development and survival of plaque biofilm, which is the primary etiologic factor for periodontal disease, by providing a direct nutrient source or by altering its surrounding environment. [24] Moreover, a natural texture diet (i.e., fruits and vegetables) has been suggested to reduce plaque accumulation, while a softer diet may promote plaque accumulation and subsequently contribute to the development of periodontal disease. The consumption of excessive quantities of refined carbohydrates and softer diets which clings to teeth and therefore predisposing to plaque accumulation at the approximal risk site. [25]

H. Occlusal Discrepancies

Periodontal disease has multifactorial etiology, among them is trauma from occlusion & malalignment of teeth. Thus, pathologic occlusion has been proposed as a risk factor for periodontitis. It is hypothesized that in certain cases, traumatogenic occlusion can exacerbate periodontal destruction, and therefore, occlusal adjustment occasionally is indicated as part of periodontal therapy. [26] Every individual tooth should be investigated, diagnosed, and subsequently treated for TFO. This philosophy is best summed up by Ramfjord and Ash, who stated that “the need for adjustment should be based on a definite diagnosis of a traumatic lesion rather than the location of some occlusal interference which may be of no significance. [27]

I. Pregnancy

Since the 1960s, it has been hypothesized that periodontal health is affected during pregnancy, and many studies have explored the mechanism of how pregnancy exacerbates gingival inflammation. Role of hormones during pregnancy Progesterone reaches 100 ng/mL (10 times the peak luteal phase of menses) during pregnancy. [28] Estradiol, the principal estrogen in plasma, may reach levels that are 30 times higher than during the reproductive cycle. In early pregnancy (trimester 1), the corpus luteum overproduces estrogen and progesterone. As the pregnancy advances, the placenta starts producing estrogen and progesterone. During pregnancy, both progesterone and estrogen are considered to have a drastic effect on the periodontium. [29] It has been suggested that estrogen and progesterone can

modulate the microbial community structure in the periodontium and increase the ratio of Anaerobic: Aerobic bacteria during pregnancy. Previous studies suggest that estradiol or progesterone increases in saliva from the first month and reaches a peak in the ninth month of gestation. [30]

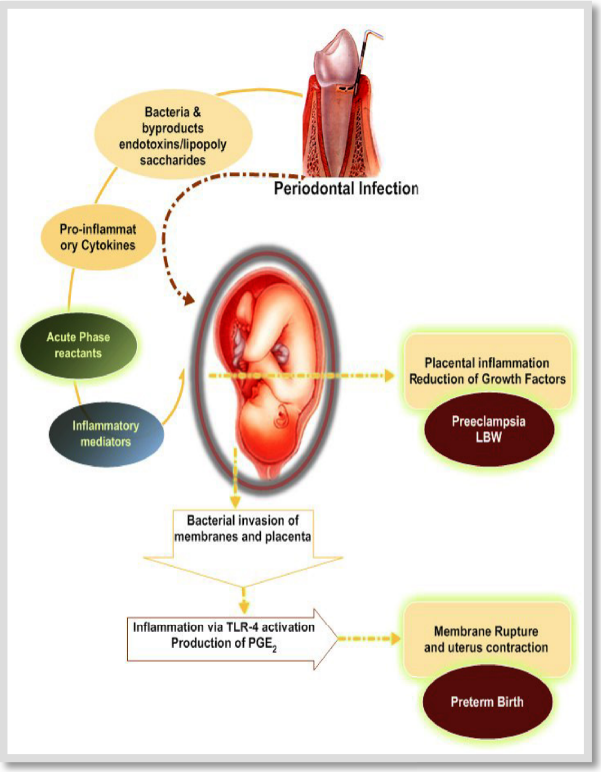

Figure 5: Schematic diagram of periodontal infection associated with preterm labor [30]

J. Nutritional deficiencies

Nutrition has a significant role to play in the development of periodontal diseases. Dietary inadequacies have been found to impact prognosis with malnutrition attributed to a more rapid progression. [31-33] the inflammatory response, characteristic of the disorders, can be attenuated by increasing consumption of vitamins C and D. [34]

Conclusion

As periodontal disease is a highly prevalent, inflammatory and multifactorial, an effective disease management requires a clear understanding of all the associated risk factors. Risk factors is an important part of modern-day periodontal practice and it involves identifying elements that either may predispose a patient to developing periodontal disease or may influence the progression of disease that already exists. These patients may require modification of their prognosis and treatment plan. In addition to an evaluation of the factors contributing to their risk, these patients should be educated concerning their risk, and when appropriate, suitable intervention strategies should be implemented.

References

1. Laddha R. Sikka L Periodontal Infectogenomics: a review. Int J Dent Med Res. 2015; 1(6):189–92.
2. Divaris K. Genome wide association study of periodontal pathogen colonization. J Dent Res. 2012; 91(7):21–28.
3. Bergstrom J, Eliasson S. Cigarette smoking and alveolar bone height in subjects with a high standard of oral hygiene. J Clin Periodontal. 1987; 14(8):466–9.
4. Rosa GM, Lucas GQ, Lucas ON. Cigarette smoking and alveolar bone in young adults: a study using digitized radiographs. J Periodontal. 2008; 79(2):232–44.
5. Wald NJ, Hackshaw AK. Cigarette smoking: An epidemiological overview. Br Med Bull 1996; 52:3-11.
6. Mealey BL, Rose LF. Diabetes mellitus and periodontal disease. Periodontal 2000. 2007; 44(1):127-53.
7. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Position statement. Diab Care 2005; 29 (1):37-42.
8. Loe H. Periodontal disease: The sixth complication of diabetes mellitus. Diab Care 1993; 16:329-334.
9. Taylor GW, Borgnakkev WS. Periodontal disease: Associations with diabetes, glycemic control and complications. Oral Dis. 2008; 14:191-203.
10. Pontes ACC, Flyvbjerg A, Buschard K, Holmstrup P. Relationship between periodontitis and diabetes: Lessons from rodent studies. J Periodontol. 2007; 78:1264-1275.
11. Engebretson S, Chertog R, Nichols A, Hey-Hadavi J, Celenti R, Grbic J. et al. Plasma levels of tumor necrosis factor-a in patients with chronic periodontitis and type 2 diabetes. J Clin Periodontol. 2007; 34:18-24.
12. Manouchehr-Pour M, Spagnuolo PJ, Rodman HM, Bissada NF. Comparison of neutrophil chemotactic response in diabetic patients with mild and severe periodontal disease. J Periodontol. 1981; 52 (8):410-5.
13. Loe H. Experimental gingivitis in man. J Periodontol. 1965; 6(36):177.
14. Nakano K, Nemoto H, Nomura R, Inaba H, Yoshioka H, Taniguchi K et al. Detection of oral bacteria in cardiovascular specimens. Oral Microbiol Immunol. 2012; 23(5); 78-84.
15. Ishihara K, Nabuchi A, Ito R, Miyachi K, Kuramitsu HK, Okuda K. DNA in carotid coronary stenotic artery plaque and in dental plaque Correlation between detection rates of periodontopathic bacterial samples. J Clin Microbial 2004; 42(3):1313-5.
16. Jin LJ, Chiu GK, Corbet EF. Are periodontal diseases risk factors for certain systemic disorders-What

matters to medical practitioners. Hong Kong Med J. 2003; 9:31-7.
17. Dorn BR, Dunn WA, Progulske-Fox A. Invasion of human coronary artery cells by periodontal pathogens. Infect immun. 1999; 67(11); 5792-8.
18. Heitz M L J. Disease progression: identification of high-risk groups and individuals for periodontitis J Clinical Periodontol. 2005; 32(6):196-209.
19. Craandijk J, Krugten VMV. Verweij CL, Velden VU, Loos BG et al. Tumor necrosis factor-alpha gene polymorphisms in relation to periodontitis. J Clin Periodontol. 2002; 29: 28-34.
20. 20. Vandyke, Hart TC, Shapira L, and Thomas E et al. Neutrophil defects as risk factors for periodontal diseases. J Periodontol. 1994; 65(5): 521-529.
21. Korman KS, Page RC, Tometti MS. The host response to the microbial challenge in periodontitis; assembling the players. Periodontology 2000. 1997; 14: 33-52.
22. Saroch N. Periobasics. 2nd edition. 2019.18
23. Kaplan, H. B. Social psychology of the immune system: A conceptual framework and review of the literature. Social Science & Medicine, 1991; 33(8): 909-923.
24. Tezal M., Grossi SG. Ho A. W, Genco R. J. et al. Alcohol consumption and periodontal disease. J clin Periodont. 2004; 31(7): 484-488.
25. 25. Newman H. N. Diet, attrition, plaque and dental disease. British dental journal. 1974; 136(12): 491.
26. 26. Brochut P, Marin I., Baehni P., Mombelli A. Behaviour basis oral health. J. Clin. Periodontol. 2007; 32(7), 695-701.
27. Stillman PR. The management of pyorrhea. Dent Cosm. 1917; 59:405.
28. Otomo-Corgel J. Periodontal therapy in the female patient. Newman and Carranza's Clinical Periodontology. 13th ed. Elsevier; 2019:944
29. Lindhe J, Brånemark PI. Changes in vascular permeability after local application of sex hormones. J Periodontal Res. 1967; 2(4):259-265.
30. Lieff S, Boggess KA, Murtha AP et al. The oral conditions and pregnancy study: periodontal status of a cohort of pregnant women. J Periodontol. 2004;75(1):116-126Tezal
31. T. França. Impact of Malnutrition on Immunity and Infection 2009; 15.
32. Enwonwu CO. Cellular and molecular effects of malnutrition and their relevance to periodontal disease. J. Clin. Periodontol. 1994; 21(10): 643–657.
33. Shilpa VP, khadtare Y, Patil P. Nutrition and its influence on periodontal disease. Int. J. Periodontol. Implantol. 2018; 3 (1):1–3.
34. P.J. Moynihan. The role of diet and nutrition in the etiology and prevention of oral diseases. Bull. World Health Organ. 2005; 83 (9): 694–699.

To cite this article: Risk factors for periodontal diseases: Vaishali Sachan, Janardhana Amaranath B.J., Shruti Gupta, Neelam Das, Rama Univ. J. Dent. Sci. 2022 December; 9 (4): 12-17