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Abstract:

Periodontal Disease is an inflammatory process affecting the Periodontium, the tissue that surrounds and supports the teeth. The process usually starts with an inflammatory process of the gum (gingivitis) but it may progress with an extensive involvement of the gum, as well as the periodontal ligament and the bone surrounding the teeth resulting in substantial bone loss. Current evidence suggested that there is a potential correlation between increased blood pressure and periodontitis. However there are only limited cross sectional studies are emerging now to associate the relation between the hypertension, periodontitis, gingivitis and healthy gingiva. This study is basically done to evaluate the relation between hypertension, periodontitis gingivitis and healthy ging

Introduction:

The periodontal diseases are a group of chronic inflammatory diseases, involving the soft tissue and bone surrounding the teeth in the jaws, or known as periodontium. Periodontal diseases including gingivitis and periodontitis are among the most common dental diseases after tooth decay in humans. Periodontal diseases are characterized by inflammation of tooth-supporting tissues caused by bacterial infection [1]. Gingivitis is a very common reversible condition, which manifests as redness, gum swelling, and bleeding during tooth-brushing and flossing. Gingivitis may progress into periodontitis with further destruction of periodontal tissues ligament and alveolar bone if left without appropriate treatment. Teeth may become mobile and eventually be exfoliated following the diminution of periodontal supporting tissues [2]. This process is attributed to the release of toxic products from the pathogenic bacteria plaque in addition to the inflammation of gingival tissues elicited by the host response [3–6].

Periodontitis is linked to an increased risk of cardiovascular diseases (CVD). The chronic inflammatory process of periodontitis and the host response provide the basis for the hypothetical association between periodontitis and CVD [7, 8]. Hypertension increases the risk of various adverse cardiovascular events such as atherosclerosis, stroke, and coronary heart disease. Oxidative stress and endothelial dysfunction have been hypothesized to be involved in the pathogenesis of hypertension. It is well known that hypertension and periodontitis share common risk factors, namely, smoking, stress, increased age, and socioeconomic factors. These risk factors may confound the association between hypertension and periodontitis. Nevertheless, according to the scientific statement issued by the American Heart Association (AHA) published in *Circulation*, observational studies support an association between periodontal disease and cardiovascular disease, independent of shared risk factors [9].

Although current epidemiological data are yet to provide sufficient evidence to prove a causal relationship between these two diseases, researchers have identified chronic inflammation as an independent link of periodontal disease in the development and progression of CVD in some patients [10]. Both AHA and the American Academy of Periodontology (AAP) were in agreement that more thorough and long-term interventional studies should be carried out in order to gain an in-depth knowledge of the diseases' mechanism. The present review is to examine the existing literature on the association between hypertension and periodontitis. In addition, we looked into the possible mechanisms to explain this link.

Discussion :**Role of Inflammation in Hypertension**

Inflammation is an essential component of immune response to pathogens, damaged cells, and other potent inflammatory stimuli including reactive oxygen radicals. While it provides a

pivotal defense mechanism against injurious agents, inflammation itself may cause injury to surrounding healthy bystander cells at the site. Inflammation is therefore a 'double-edged sword' as this adaptive response might eventually become maladaptive after a chronic time. In blood vessel, inflammation increases vascular permeability and alters cytoskeletal elements in endothelial cells, disrupting the endothelial functions in controlling vascular health. Hence, there is a potential association between vascular inflammation and hypertension.

Over the past three decades, the role of vascular inflammation as a mechanism that participates in the progression of hypertension has gained increasingly strong footing through a tremendous amount of supportive reports [3]. Khraibi et al. [4] have found that chronic immunosuppressive therapy with cyclophosphamide significantly attenuated blood pressure (BP) elevation in Okamoto spontaneously hypertensive rats (SHR). This finding supported the hypothesis regarding the involvement of inflammatory reaction in hypertension. In the following year, Norman et al. [5] demonstrated that the development of hypertension was delayed by correcting the immune imbalance state in SHR. Their works continued to show that immunological dysfunction is one of the key aetiologies of hypertension [4]. Dzielak [4] pointed out an inflammatory involvement in hypertension by observing an alteration in the serum immunoglobulin levels in both patients and laboratory animals. Furthermore, the interaction between inflammatory cells and endothelial cells was increased in hypertensive patients [5]. Kampus et al. also found an increase in C-reactive proteins (CRP) and vascular wall stiffness in untreated hypertensive patients. More recently, a link between hypertension and inflammatory responses to oxidized low-density lipoprotein was reported in patients, further suggesting that BP is directly correlated to immunological milieu [4].

The overall contribution of inflammation to vascular damage in hypertensive patients remains an interesting puzzle to be solved by scientists. Research during the last dozen years has shed light on some aspects of this puzzle. Endothelial cells, which line the intimal surface of blood vessel, are the primary target of immunological attack in inflammatory responses. Under normal conditions, the endothelium maintains a vasodilator, antithrombotic, and antiinflammatory state. However, a proinflammatory condition could contribute to endothelial dysfunction.

Certain inflammatory adhesion molecules are involved in the pathogenesis of hypertension and predictive of future cardiovascular events. Vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) are expressed by the endothelial cells. Their expressions are upregulated in response to inflammatory insult [5]. The increased expression of adhesion molecules on endothelial cells is a common process in response to inflammation [5]. VCAM-1 and ICAM-1 are recognized as important cardiovascular risk markers [3].

VCAM-1 and ICAM-1 mediate leukocytes binding to the endothelial lining.

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 (PGI₂), and endothelium-derived hyperpolarising factor (EDHF) and vasoconstrictors such as endothelin-1, thromboxane (TXA₂), and angiotensin II. During endothelial dysfunction, the balance between these vasodilators and vasoconstrictors is disturbed in favor of the latter. Ng et al. [5] have recently shown that the levels of VCAM-1 is directly associated with the imbalance between PGI₂ and TXA₂ in hypertensive rats. Therefore, inflammation may be involved in hypertension by directly damaging the endothelial BP regulation.

Evidences of Association between Hypertension and Periodontitis

Hypertension is a major global health disorder affecting about 972 million adult populations in year 2000. This number is expected to grow to 1.56 billion by the year 2025 [2]. Prevalence of hypertension in most developing countries is comparable to the developed countries [5]. Hypertension is defined when a patient has an elevated systolic BP greater than 140 mmHg and/or diastolic BP greater than 90 mmHg [29]. A patient with systolic BP ranging between 120 mmHg and 139 mmHg, and/or diastolic BP of 80 mmHg to 89 mmHg, is categorized as prehypertensive. Patients at this stage have the tendency to develop hypertension; hence medical approaches and life style need to be taken care of More than 700 [2]

Conclusion:

In summary, the current epidemiological data, mainly from cross-sectional studies, show an association between hypertension and periodontitis. However, there is no strong proof to indicate that a causal relationship exists. In order to connect the relationships between dentistry and medicine, additional issue needs to be addressed for the improvement in managing the overall health of patients. Future studies should be conducted to yield better understanding of the mechanisms and interactions between hypertension and periodontitis, which will further strengthen the involvement between dental and medical communities. Since previous studies demonstrated an elevation in BP which is associated with periodontitis, preventive approaches targeted at reducing BP should also be included in the management of periodontitis. Periodontal health is achievable in both individual level as well as the population level. These preventive measures should be emphasized in oral health promotion programme, in order to enhance overall health outcomes.

References:

- 1-F. Vidal, I. Cordovil, C. M. S. Figueredo, and R. G. Fischer, "Non-surgical treatment reduces cardiovascular risk in refractory hypertensive patients: a pilot study," *Journal of Clinical Periodontology*, vol. 40, no. 7, pp. 681–687, 2013.
- 2-O. Shaker, N. A. Ghallab, E. Hamdy, and S. Sayed, "Inducible nitric oxide synthase (iNOS) in gingival tissues of chronic periodontitis with and without diabetes: immunohistochemistry and RT-PCR study," *Archives of Oral Biology*, vol. 58, no. 10, pp. 1397–1406, 2013
- 3-C. Gray, M. Li, C. M. Reynolds, and M. H. Vickers, "Pre-weaning growth hormone treatment reverse hypertension and endothelial dysfunction in adult male offspring of mothers undernourished during pregnancy," *PLoS One*, vol. 8, no. 1, article e53505, 2013.
- 4-B. H. Al-Ghurabei, "Evaluation of serum anti-cardiolipin antibody, hs-CRP and IL-6 levels in chronic periodontitis as possible risk factors for cardiovascular diseases," *Journal of Baghdad College of Dentistry*, vol. 24, no. 2, pp. 161–165, 2012.
- 5-M. S. Gomes, T. C. Blattner, M. S. Filho et al., "Can apical periodontitis modify systemic levels of inflammatory markers? A systematic review and meta-analysis," *Journal of Endodontics*, vol. 39, no. 10, pp. 1205–1217, 2013.
- 6-A. R. Pradeep, M. Kumari, N. Kalra, and N. Priyanka, "Correlation of MCP- 4 and high-sensitivity C-reactive protein as a marker of inflammation in obesity and chronic periodontitis," *Cytokine*, vol. 61, no. 3, pp. 772–777, 2013.
- 7-H. Miyashita, T. Honda, T. Maekawa et al., "Relationship between serum antibody titres to *Porphyromonas gingivalis* and hs-CRP levels as inflammatory markers of periodontitis," *Archives of Oral Biology*, vol. 57, no. 6, pp. 820–829, 2012.