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Review Article

Oral disease linked with cardiovascular disease

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ABSTRACT

As far as the pathogenesis of cardiovascular disease the spotlight has customarily been on dyslipidaemia. understanding of the pathogenesis of CVD has increased, and infections, including those caused by oral bacteria, are more likely involved in CVD progression than previously thought. While many examinations have now shown a relationship between periodontal infection and cardiovascular disease, the components supporting this relationship stay hazy. This review gives a brief overview of oral diseases with host-bacterial interactions and various studies which suggest the relationship of oral disease and cardiovascular disease.

1. Introduction

The mouth has essentially the teeth, gums, tongue. Teeth are being encircled by 2hard tissue and 2 soft tissues. Delicate tissues unite gingiva, periodontal tendon. Hard tissues are bone and cementum. Dental caries affects hard tooth surfaces and, thus, do not cause systemic exposure to the bacteria. However, if left untreated, tooth caries progress to the dental pulp (nerve and blood vessels teeth), which leads to a root canal infection that spreads to the supporting structures including bones, and significantly increases the level of systemic exposure.[1]

According to Queensland Health, CVD is the second highest cause of disease burden in Queensland in 2003, accounting for 17.8% of the total disease burden (the number of lives lost due to premature death and disability: disable adjusted life year (DALY) with 77% of cardiovascular (CV) associated load with premature death. With regard to certain conditions, coronary heart disease (CHD) is a major contributor to the burden of disease, accounting for 10.2 and 10% of disable adjusted life years (DALY) in Queensland and Australia, respectively.[2] The fundamental reason for Cardiovascular disease in most is atherosclerosis.[3] While they have made extraordinary advances in the treatment of Cardiovascular disease, the degree of the ailment keeps on rising. The underlying cause of most cases of CVD is atherosclerosis.[3] While great progress has been made in the treatment of CVD, the prevalence of this disease is increasing. Up to 50% of individuals with CVD do not have a CV-traditional risk factors such as hypercholesterolemia, hypertension, smoking, and obesity,[4] suggesting that other factors must contribute to the disease. Identification of other risk factors for CVD, along with their mechanisms of action, are essential if morbidity and mortality from this disease should be reduced.[4] The role of infection and inflammation in atherosclerosis has become increasingly evident. Chronic inflammatory periodontal disease is one of the most common human infections with 10-15% of the population has advanced forms of the disease.[5,6] Besides, a balanced examination indicated that the bacterial weight of Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, Treponema denticola, and Tannerella forsythia in subgingival plaque tests was related to carotid intima-

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media thickening.[7] While many studies have shown a relationship between periodontal disease and CVD, some studies have shown a weak or no association between the two diseases.[8–10] The fact that periodontitis and CVD share common risk also makes the interpretation of complex clinical studies. Meta-analysis has been done, however, and a small but significant association has been demonstrated.[11–13] The vulnerable atherosclerotic apolipoprotein E deficiency mouse models have provided further support for the role of oral bacteria in atherosclerosis indicate that inoculation with P. gingivalis results in advanced atherosclerotic lesions compared with control mice.[14–16]

2. Oral infections links to cardiovascular problem by

Inflammation plays a major role in both oral infections such as periodontitis and CVD.[17–20] oral inflammation caused by bacteria in the biofilm consists microbiome that forms on teeth.[21] Vascular inflammation is induced by hyperlipidemia, hypertension, smoking, and several other known and unknown factors. In the following sections we consider the recent evidence for the role of oral infections in CVD as well as the direct and indirect actions of oral bacteria through stimulation of local and systemic inflammation.

3. What does oral bacteria does on vasculature?

Extensive ulceration of the epithelial surface layer of periodontal pockets in periodontitis patients have generally been estimated to be between 8-20 cm2.[22] Bacteria in the ulcerated gingival sulcus approach provides a portal for entry of bacteria into the systemic circulation, thereby bacteremia. As a result of this interaction, the interface is very populated by phagocytic cells which can also carry the bacteria to the remote site[23] Periodontal bacteria contribute to atherosclerosis track was recently reviewed by Reyes and colleagues.[24] A series of studies initiated in 2000[25] reported the genomic DNA of some periodontal bacteria in human atheromas. This study replicated and extended by others who find some species of bacteria in atheromas. Finally, human atherosclerotic plaques shown to harbor viable Porphyromonas gingivalis and Actinobacillus actinomycetemcomitans invasive.[26] This study shows the localization of oral organisms in atheromatous plaques cause a large number of mechanistic studies and animal models geared to evaluate seven 'evidence' which has been proposed as necessary to show that periodontal bacteria that contribute to afactor in atheromatous disease.[24]

A recent study[27] of the microbiome of atherosclerosis plaques showed that 84 different bacterial taxa detected in atheromas and the walls of blood vessels of patients with atherosclerotic vascular disease and periodontal disease, while 18 different taxa identified in blood vessels tissue of patients with vascular disease and little or no periodontal disease. Some taxa of bacteria commonly found in the oral cavity were detected in diseased blood vessel tissue, including Streptococcus, Prevotella, Capnocytophaga, Veillonella, and Porphyromas species, show that the oral cavity can be a source for the spread of bacteria to the blood vessel network. Other sources are proposed to contribute to skin and gut.[27]

Evidence began to accumulate showing that in addition bacteremias therapy-related acute dental and other procedures in which the mucosal flora is introduced into the bloodstream, there is a chronic seeding of bacteria into the bloodstream from other sources. Recent studies state that dendritic cells from the periodontal pathogens menfagositosis sick bags and carry them through the bloodstream until they are stored at the site of vascular

The study followed healthy and periodontitis patients with or without ASVD, and shows that carry blood myeloid dendritic cells known periodontal pathogens such as P. gingivalis and other oral species, and that the carrier cell count was highest in the group suffered from both ASVD and periodontitis simultaneously. This study suggested that blood dendritic cells play an important role in spreading the oral organisms to atherosclerotic plaques, and that these organisms provide an important signal for the differentiation of dendritic cells and conversion atherogenic after they entered atheroma. Furthermore, parallel in vitro cultured P. gingivalis isolated from atherosclerotic plaques found them alive for up to 24 hours in dendritic cells, while they rapidly kill dendritic cells when outside by polymorphonuclear leukocytes (PMN).[23]

In summary, there is evidence for two modes of cardiovascular tissue invasion by periodontal bacterial species, in the words of phagocyte-mediated bacteremia and other modes of translocation of bacteria from the periodontal lesion to vascular tissue. related bacteremia bacteria attack the lining of endothelial production of pro-inflammatory cytokines such as monocyte chemoattractant protein 1 MCP-1.[28] In the latter mode, resettled phagocytes save the proposed oral bacteria to spread to atheromas, where they contribute to the growth of the atheroma. Further evaluation of these pathways and their specific contribution to the formation of atheroma is necessary to better understand the role of bacteria in atheromas. oral infections and systemic inflammation A clear distinction must be made between active bacteria.

4. Various studies for better understanding

4.1. Intervention studies

Interventional trials may offer strong evidence for a direct correlation between periodontitis and CVD because they try to cope with the effects of periodontal therapy on CVD indicator. There are no studies that evaluate the impact

of treating periodontal disease in a primary event (first myocardial infarction - MI), and only a feasibility study that examined the secondary event (prevent both MI) has been published.[29] interventional trials also offer a way to understand more about the magnitude of the proposed mechanism link periodontal and CVD, by intervening to control the suspected mechanism and observe the impact on the magnitude of the link. Trial use of antibiotic interventions specifically target microorganisms isolated from atherosclerosis plaque has helped us to understand more about the lack of direct infectious aetiology theory magnitude as the link between periodontal disease and ASVD.

4.2. Animal trials

Animal studies offer very good, and it is easier to control and replicate, alternative to human intervention trials, which are often difficult to manage because of logistics or finance obstacles. Furthermore, as seen above, animal models offers a great opportunity to study in depth and confirmed the pathophysiology and treatment of experimental which is often impossible to perform in patients on ethical issues. Studies in animals have provided support for the hypothesis that molecular mimicry is a potential mechanism that links periodontitis and CVD. molecular mimicry is thought to occur when the sequence similarities between foreign and self peptides produce cross-activation of T cells or B of auto-reactive can cause tissue pathology or autoimmunity.[30] Crossreactive autoantibodies to periodontal bacterial lipopolysaccharide

and a heat-shock protein (Hsp) have been identified and proposed as a potential explanation for the alleged relationship between periodontal disease and ASVD.[31,32] Recent in vitro studies showed that a tooth infection with P. gingivalis increased endothelial injury in obese mice.[33] aortic tissue studied 6 weeks post-infection were found to have an increased number of cell apoptosis (TUNEL-positive) compared with uninfected controls. Immunohistochemical analysis of P. gingivalis was detected deep in aortic smooth muscle, and the number of P. gingivalis cells in the aortic wall was higher in obese mice than in control mice. The results highlight the complexity and importance of studying modifications potential link between periodontal disease and CVD.[34] presence or absence of other known risk factors.

5. Conclusion

As we move forward, we get to know of how oral bacteria can have a significant impact on systemic inflammation and perhaps initiate local vascular lesions leading to CVD. Of particular interest is the dynamic relationship between inflammation and the oral micro-biome, and the potential for modification of bacterial viru-lence both locally and systemically. Recent studies provide strong support for the role of systemic inflammation and immunological cross-reactivity in atherosclerosis. Furthermore, intervention studies have shown that periodontal therapy may reduce systemic markers of inflammation and blood vessel health and the antibody response to heat shock protein. Future studies are now needed to determine the long-term effects of periodontal therapy on CVD outcomes. Nevertheless, reports a new consensus on periodontitis and CVD atherosclerosis, published in the American Journal of Cardiology and the Journal of Periodontology, has recommended that patients with moderate to severe periodontitis should be informed about the increased risk of the possibility of CVD and those with more than one cardiovascular risk factors must undergo a medical evaluation of cardiovascular risk.

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7. Conflict of Interest

None.

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