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AND REFRESHER COURSE IN DENTISTRY

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A SYSTEMATIC REVIEW OF PERIODONTAL DISEASE AND CARDIOVASCULAR DISEASE

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ABSTRACT

Periodontal disease known as an oral inflammation that has a relation with systemic disease, including cardiovascular disease. The relationship about periodontal disease and cardiovascular disease has many researchers finding which are the biomarkers linking between them. The purposes of this systematic review are finding data about correlation between periodontal disease and cardiovascular disease. Literature Studies: The search process of this systematic review is based on the publication of the Journal of MEDLINE, EMBASE based on EBSCOhost or google search, based on the keywords: “periodontal disease”, “cardiovascular disease”, “periodontitis”, “periodontal disease” and “cardiovascular disease”; “periodontitis” and “cardiovascular disease”. We review for last 5 years publication with full text publication. We found about hundreds publication about periodontal disease and cardiovascular disease. But we found 10 full-text publications supported our data variable. Discussion: We found that periodontal disease affecting cardiovascular disease with many biomarkers between them, including periodontal pathogen, lipopolysaccharide, carotid artery intima–media thickness (CCA IMT), C-reactive protein (CRP), anti-human heat shock protein 60 (hHSP60) antibodies, levels of sCD40 L and MCP-1 in serum and gingival crevicular fluid (GCF), plasma levels of E-selectin and myeloperoxidase (MPO), plasma levels of systemic inflammation (CRP, fibrinogen and interleukin-6) and established cardiovascular risk factors (systolic and diastolic blood pressure (SBP and DBP), left ventricular mass (LVM) and arterial stiffness). Conclusions: Researches showed many indicator and factors linking the correlation between periodontal disease and cardiovascular disease. The emergence about this correlation has impacted importance maintain periodontal health keeping cardiovascular health. This systematic review could informed and strengthen evidence about this to the society including cardiologist, general practitioner, stakeholders and patients.

Key words: periodontal disease, cardiovascular disease, oral infection, literature review.

INTRODUCTION

Based on Indonesian National Health Survey in 2005, population deaths caused by blood circulating system are in the first rank with percentage 26.4. Based on WHO (2012) each year people died caused by cardiovascular disease over the other disease, and in 2030 expected 23.6 million people died caused by cardiovascular disease. Cardiovascular diseases (CVD) or ‘total cardiovascular diseases’ include rheumatic fever/rheumatic heart disease, hypertensive diseases, ischemic (coronary) heart disease, pulmonary heart disease and diseases of pulmonary circulation, other forms of heart disease, cerebrovascular disease (stroke), atherosclerosis, other diseases of arteries, arterioles, and capillaries, diseases of veins, lymphatic’s, and lymph nodes not classified elsewhere as well as other and unspecified disorders of the circulatory system. When data is available, congenital cardiovascular defects are also included (AHA, 2013). Factors of cardiovascular disease, and as infection disease periodontitis are one of risk factors of cardiovascular disease. Periodontal disease are an inflammation disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganism, resulting in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth formation, recession, or both. Periodontal disease known as an oral inflammation that has relation with systemic disease, including cardiovascular disease.

According to Periodontitis and atherosclerotic cardiovascular disease; consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases (2013) concluded the association between periodontitis and cardiovascular disease examined plausibility, epidemiology, and early results from intervention trials. In intervention trials mentioned periodontal treatment reduces systemic inflammation as evidenced by reduction in C-reactive protein (CRP) and improvement of both clinical and
surrogate measures of endothelial function, but there is no effect of lipid profiles. The relationship about periodontal disease and cardiovascular disease has many researchers finding which are the biomarkers linking between them. In 2012, the American Heart Association (AHA) issued a scientific statement regarding the association between cardiovascular disease and periodontal disease. The AHA statement suggests that there are significant gaps in the scientific understanding of the interaction of oral health and cardiovascular disease. Therefore, it is stated that while there is an association between cardiovascular disease and periodontal disease, there is not a causal relationship.

Many of research done to investigate the findings including periodontal pathogen, lipopolysaccharide, carotid artery intima–media thickness (CCA IMT), C-reactive protein (CRP), anti-human heat shock protein 60 (hHSP60) antibodies, levels of sCD40 L and MCP-1 in serum and gingival crevicular fluid (GCF), plasma levels of E-selectin and myeloperoxidase (MPO), plasma levels of systemic inflammation (CRP; fibrinogen and interleukin-6) and established cardiovascular risk factors (systolic and diastolic blood pressure (SBP and DBP), left ventricular mass (LVM) and arterial stiffness). The purposes of this systematic review are finding data about correlation between periodontal disease and cardiovascular disease.

OBJECTIVES

In this paper will be discussed through evidence-based systematic review on the relationship of periodontal disease and cardiovascular disease.

LITERATURE STUDIES

Methods in a systematic review of evidence-based

Study selection used as the protocol of this systematic review is the journal that has been published. Inclusion studies including randomized clinical trial, control clinical trials, and conducted follow-up approximately 6 months thereafter, cross-sectional, and cohort research. In this paper it is limited for research that written in the last five years, from year 2010 until year 2015.

The search process of this systematic review is based on the publication of the journal of MEDLINE, EMBASE based on EBSCOhost or google search, based on the keywords: “periodontal disease”; “cardiovascular disease”; “periodontitis”; “periodontal disease” and “cardiovascular disease”; “periodontitis” and “cardiovascular disease”.

The expected outcome of a systematic review is evaluation of non-surgical periodontal treatment in cardiovascular patients in terms of level of biomarkers, periodontal pathogen, and also from carotid artery intima–media thickness (CCA IMT).

Control the quality of systematic reviews

To ensure the quality and focus of this paper, the question of this review is: “Is any relationship and causation between periodontitis and cardiovascular disease?”. After that, of each journal that will be reviewed and question which can be seen emerging, namely: (1) title and abstract; intervention, random or random allocation, etc ?; (2) Method of and intervention: how to do sample selection, whether it is in accordance with the specific categories which will be discussed in this paper ?; (3) Outcome: whether the expected outcome is in conformity with the criteria for inclusion?; (4) Results: The analysis of the obtained data, follow-up period of each technique.

Literature Studies in journal findings

Exposure findings journal

Journal publication select from period from 2010 to 2015. From the searches conducted, obtained 685,000 journal from the search results back which has a number of full text journals, and obtained 29 journals of the keyword “periodontal disease” and “cardiovascular disease”. Examination and selection of journals, obtained 4 journal of evaluation of non-surgical periodontal treatment in cardiovascular disease, 4 journal of biomarker analysis in cardiovascular patient in healthy and with periodontitis (chronic and aggressive), 2 journal of evaluation of carotid intima-media thickness. The journal search by multilevel stratified by using keywords. In the final results, obtained several journals that match the criteria for the systematic review and comparison of data processing is done. The processing described in Figure 1. Selection process of studies included.

Figure 1. Selection Process

Relationship of Periodontal Disease and Cardiovascular Disease

Many systemic diseases have been implicated as risk factors in periodontal disease. Clinical and basic research over the past several decades has led to an improved understanding and appreciation for the complexity and pathogenesis of periodontal disease. Periodontal infection may affect the onset or progression of the atherosclerosis and coronary heart disease through both direct and indirect pathway mechanism. Periodontal infection increased fibrinogen level, while blood cell count and von Willebrand factor that increased blood viscosity and cause ischemic heart disease. Periodontal infection that released toxic production of gram-negative bacteria associated lipopolysaccharide (LPS) that cause endothelial damage, platelet adhesion (aggregation), monocyte infiltration (proliferation) that leading to cytokine and growth factors production and thrombus formation, leading to atheroma formation, vessel wall thickening and thromboembolic events.
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<th>Journal</th>
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<th>Indicator</th>
<th>Treatment</th>
<th>Result</th>
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<td>Camila 2019</td>
<td>Randomized Clinical Trial</td>
<td>62 patients; ages between 30 - 60 years old Group I (Experimental group): with periodontitis Group II (Control group): without periodontitis</td>
<td>Periodontal examination: Periodontal examination (Gingival Index (GI), Plaque Index (PI), probing depth (PD), gingival recession, Loss of attachment (CAL)), and CRP level collection</td>
<td>To: first examination: questionnaire, periodontal examination (Gingival Index (GI), Plaque Index (PI), probing depth (PD), gingival recession, Loss of attachment (CAL)), and CRP levels collection</td>
<td>Periodontal examination: Group control: absence of periodontitis 100%. Gingival index 0.2 Group experimental: T0: absence of periodontitis 0%  T2: absence of periodontitis 70% T0: gingival index 0.6 T2: gingival index 0.2 juice level CRP at different times of collection in the experimental group (initial exam = 2.99 ± 3.2 mg/dL, 8 days after treatment = 4.26 ± 4.5 mg/dL and 45 days after treatment = 2.23 ± 2.51 mg/dL; and in the control group (2.30 ± 2.77 mg/dL)</td>
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<td>Cauza, 2014</td>
<td>Randomized clinical trial</td>
<td>64 patients; severe chronic periodontitis group I: Test group: immediately periodontal treatment group II: Control group: delayed periodontal treatment</td>
<td>Clinical periodontal: PPD, CAL, BO, Plaque index (PI) Laboratory examination: Erythrocyte sedimentation rate (ESR), Triglycerides, C-reactive protein (CRP)</td>
<td>To: baseline T2: 2 months of periodontal treatment T6: 6 months after periodontal treatment</td>
<td>Periodontal examination: To: similar between group I and II T12: significant difference in all periodontal parameters between the test and control groups 2 and 6 months after treatment (T2 and T6). Laboratory examination: To: no differences; T2: significant difference after treatment between the test and control groups for the median values of CRP and ESR. T6: significant differences in test group. Reduction of CRP and ESR. Conclusion: periodontal treatment was effective in improving the periodontal parameters and reducing CRP level, ESR, total cholesterol, and triglycerides after 6 months.</td>
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<td>Pejcin, 2011</td>
<td>Randomized clinical trial</td>
<td>Test group: 50 patients with periodontitis, age 30 – 74 years Control group: 25 volunteers</td>
<td>Periodontal examination: BOP, PPD, CAL Laboratory examination: C-Reactive Protein (CRP) and presence of Erythrocyte and A. actinomycescens</td>
<td>To: before treatment Treatment: Scalling and root planning, Oral hygiene instructions, Periodontitis treatment T6: 6 months after treatment T12: 12 months after treatment</td>
<td>After the therapy, there was a statistically significant decrease of mean values of all examined periodontal clinical parameters (p &lt; 0.01) in the study group. There were statistically significant decreases of periodontal pathogens – Porphyromonas gingivalis (p &lt; 0.001) and Aggregatibacter actinomycetemcomitans (p &lt; 0.01). Levels of inflammatory markers at baseline and after treatment were different. After treatment, median serum of CRP decreased from the baseline value of 6.69 mg/L to 4.25 mg/L.</td>
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<td>Vidal, 2013</td>
<td>Interventional prospective cohort pilot study</td>
<td>26 patients (53.6 ± 8.0 years old) diagnosed with refractory hypertension and generalized chronic periodontitis</td>
<td>Plasma levels of systemic inflammation (CRP, fibrinogen and interleukin-6) and established cardiovascular risk factors (systolic and diastolic blood pressure (SBP and DBP), left ventricular mass (LVM) and arterial stiffness) were assessed at three time points</td>
<td>To: baseline T3: 3 months after treatment T6: 6 months after treatment</td>
<td>No significant difference was detected between the clinical periodontal data between baseline and the 3 months re-evaluation previous to periodontal therapy, except for BOP and percentage of sites with PPD ≥ 6 mm. Mean percentage of sites with VPI, BOP, PPD ≥ 4 mm, PPD4-5 mm, PPD5-6 mm, CAL 4-5 mm and CAL ≥ 6 mm was significantly reduced 6 months after periodontal treatment. Six months after periodontal treatment, a significant reduction in the median values of SBP and DBP and of the mean values of LVM and PWV was observed. The median values of the inflammatory markers, CRP, IL-6 and fibrinogen were significantly reduced 6 months after periodontal treatment</td>
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Table 2. Review of evaluation of relation of periodontal disease that shown by biomarker.

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<td>Lata, 2014</td>
<td>75 systematically healthy subjects to analyze CRP using ELISA</td>
<td>Group I: nonperiodontitis Group II: chronic generalized periodontitis Group III: aggressive periodontitis</td>
<td>CRP</td>
<td>No treatment</td>
<td>Mean CRP levels were significantly greater in both group II (2.31±0.56) and III (4.61±0.58) as compared to group I (0.96±0.13) and group III having greater level than group II.</td>
</tr>
<tr>
<td>Mill, 2015</td>
<td>Cross sectional</td>
<td>15 healthy patient 30 severe chronic periodontitis</td>
<td>Periodontal examination: PI, GI, PPD, CAL Biomarkers: sCD40L MCP-1</td>
<td>Scaling and root planing examination after 6 weeks</td>
<td>The sCD40L levels correlated strongly with MCP-1 levels in both GCF (r=0.886) and serum (r=0.861) in patients of chronic periodontitis. The relationship between the levels of the two markers was maintained in GCF (r=0.866) and serum (r=0.750) after Phase I periodontal therapy.</td>
</tr>
<tr>
<td>Ramirez, 2014</td>
<td>evaluate the endothelial function, systemic inflammatory biomarkers and subgingival microbial profile association in patients with and without periodontal disease</td>
<td>44 patients with moderate to severe periodontitis gingivitis and incipient periodontitis</td>
<td>Periodontal examination Biomarkers: E-selectin MPO ICAM-1</td>
<td>No treatment</td>
<td>Significantly higher plasma levels of E-selectin (64.5±30.9 vs 43.8±22.2; P=0.026) and myeloperoxidase (MPO) (103±114.5 vs 49.1±35.6; P=0.032) in cases than controls.</td>
</tr>
<tr>
<td>Letchman, 2012</td>
<td>To determine the relationship between periodontal pathogen load and anti-human heat shock protein 60 (hHSP60) antibodies in patients with established cardiovascular disease (CVD)</td>
<td>Participants were cardiovascular patients (n=74) with a previous hospital admission for myocardial infarction.</td>
<td>Concurrent periodontal pathogen load of P. gingivalis, F nucleatum, T forsythia and A actinomyces comites PCR Serum antibodies: GroEL and hHSP60 ELISA</td>
<td>No treatment</td>
<td>The strongest positive correlations were found between anti-hHSP60 levels and numbers of T. forsythia (r=0.43; p&lt;0.001) and between anti-hHSP60 and anti-GroEL levels (r=0.39; p=0.001).</td>
</tr>
</tbody>
</table>

DISCUSSION

Review journal of evaluation of non-surgical periodontal treatment in cardiovascular disease

We review for last five years publication with full text publication. We found 4 journal of evaluation of impact of non-surgical periodontal treatment of cardiovascular disease that shown by C-reactive protein (CRP) level. Our findings concluded in Table 1:

We review four journals about how the non-surgical periodontal treatment affecting cardiovascular patient. In the first review, research from Camila et al (2010) resulting in higher C-reactive protein (CRP) level in the first examination higher in experimental group and increase 8 days after treatment, and decrease similar to control group. In research from Cauca et al (2014) periodontal treatment was effective in improving the periodontal parameters and reducing CRP level, ESR, total cholesterol, and triglycerides after 6 months. In Pejicic (2011), the present study demonstrates that basic nonsurgical periodontal treatment in patients with moderate to severe periodontitis results in clinically and statistically significant improvements in periodontal status, reduction of subgingival bacterial levels leading to a significant suppression of serum C-reactive protein. Serum CRP levels were significantly decreased in patients with periodontitis (p<0.001) in 6 months and 12 months evaluation. After treatment, serum CRP levels decreased (4.94mg/l and 4.25mg/l) and the differences were statistically significant (p<0.001). In Vidal et al (2013), resulting in the median values of the inflammatory markers, CRP, IL-6 and fibrinogen were significantly reduced 6 months after periodontal treatment. From these journals, they discuss how non-surgical periodontal treatment affecting cardiovascular disease by CRP level. Periodontal disease is oral inflammations that produce lipopolysaccharides that may then enter the systemic circulation. These products can trigger the host inflammatory response and elevate serum concentration of acute- phase reactants and inflammatory mediators such as CRP. Increased levels of circulating inflammatory mediators are thought to contribute to the inflammatory processes leading to atherosclerosis. C-reactive protein has shown to play a role in the pathogenesis of atherosclerosis and recent evidence linking periodontal disease with a high
Table 3. Review of evaluation of relation of periodontal disease and cardiovascular disease by intima-media thickness

<table>
<thead>
<tr>
<th>Journal</th>
<th>Methods</th>
<th>Patients and Groups</th>
<th>Indicator</th>
<th>Treatment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jung, 2014²⁰</td>
<td>Cross sectional</td>
<td>5404 patients age ≥ 50 years</td>
<td>Periodontal examination; PPD, CAL, BOP. Determine common carotid artery intima-media thickness (CCA IMT) and the presence of carotid plaques</td>
<td>No treatment</td>
<td>Number of missing teeth was associated with increased CCA IMT, and BOP% was associated with increased CCA IMT in females only</td>
</tr>
<tr>
<td>Yu, 2014²¹</td>
<td>Cross sectional</td>
<td>847 patients</td>
<td>Periodontal examination</td>
<td>No treatment</td>
<td>Each 1-mm increase in mean clinical attachment loss corresponded to a 0.018-mm increase in maximal cIMT. The risk of atherosclerotic plaque increased by 18.3% with each 1-mm increase in mean clinical attachment loss.</td>
</tr>
</tbody>
</table>

The risk of atherosclerotic plaque formation is the main cause of all cardiovascular diseases. CRP level in CVD patient reduce 6 months after treatment.

Review journal of evaluation of biomarkers in cardiovascular disease without treatment

We review for last five years publication with full text publication. We found 4 journal of evaluation of relation of periodontal disease that shown by biomarker. Our findings concluded in Table 2:

We review four journals related to biomarkers of systemic and periodontal disease. In Lata, 2014 found that CRP levels are significantly different from healthy, chronic periodontitis and aggressive periodontitis patient. CRP levels increasing as well as increasing of periodontal examination included pocket depth and loss of attachment in chronic periodontitis patient. In Mili, 2015 they choose sCD40 L and MCP-1 as the biomarker. A significant association was seen that is, an increase in the quantity of sCD40 L corresponded with an increase in levels of MCP-1 in both GCF and serum. The findings of these studies substantiated the possible pathogenic role played by the two markers in causing severe periodontal disease and increasing the systemic inflammatory load. In Ramirez, 2014 case control study showed that patients with periodontitis compared with periodontally healthy controls have increased levels of E-selectin, MPO and ICAM-1, which these inflammatory proteins have been associated in several observational studies with endothelial dysfunction, atheroma plaque develop-ment and cardiovascular events. In Leishman, 2012 demonstrated that in CVD patients, elevated levels of anti-HSP60 antibodies occurred in those with more extensive periodontal pocketing and with periodontal pathogens (P. gingivalis, P. nucleatum, T. forsythia and A. actinomycetemcomitans).

From these two journals, we concluded the number of missing teeth and BOP% are associated with carotid atherosclerosis. Carotid intima–media wall thickness (cIMT) is an indicator of the risk of vascular disease. Increased cIMT and presence of atherosclerotic plaque are considered subclinical atherosclerosis.

CONCLUSION

Preservation of periodontal health is a key component of oral and overall health and as such is a fundamental human right (Consensus of the European Workshop on Periodontal Education, Baehni & Tonetti 2010).

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