ASSOCIATION BETWEEN DEPRESSION AND CHRONIC PERIODONTITIS - A REVIEW

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Abstract

Periodontal diseases are infections associated with specific pathogenic microorganisms and host response alterations. The variations in development and progression of periodontitis between individuals is attributed to non-oral risk factors apart from differences in individual host responses and adaptation. One of the important non-oral risk factors which contributes to the development and progression of periodontal disease are the psychosocial factors. Depression is an important psychosocial domain which has been associated with depression. Recent research findings show that depression contributes to periodontal disease through two mechanistic links namely biologic and behavioral. These findings have important clinical implications because they suggest that addressing psychological factors like depression forms an integral part in overall preventive periodontal maintenance. This article emphasizes the role of depression in the etiopathogenesis of periodontal disease and the need to use depression scales in periodontal practice.

Key words: Periodontal disease, Depression, Psychosocial factors.

1. Introduction

Destructive periodontal diseases are infections associated with specific pathogenic microorganisms and host response alterations (Socransky, S.S. & Haffajee, A.D 1992). However consistent data in the literature show that plaque accumulation alone is not enough to trigger the onset of periodontal disease (Löe, H et al 1986). The development of periodontitis and its progression varies from individual to individual because of differences in individual host responses and adaptation. The contribution of non-oral risk factors that strongly associate with the development of periodontitis have been demonstrated by numerous studies. Such non-oral risk factors include smoking, HIV, genetics, diabetes mellitus, chronic renal disease, obesity, alcohol dependence, osteoporosis and psychosocial factors (Breivik, T et al 2006). One of the important psychosocial domain associated with periodontitis is depression (Elter, J.R et al 1999). Depression is the most common psychiatric illness which poses substantial health risks. Evidences from literature indicates that emotional factors like depression are linked and a wide array of medical conditions. The relationship between Periodontal diseases and psychosocial factors is well established in ANUG. Pre-existing gingivitis, smoking and acute psychological disturbances such as stress and depression, have been correlated with ANUG (Melnick, S.L. et al 1988). This association has been proved through clinical studies, which has shown an increased incidence of ANUG among people subjected to stressful situations for a period of time. In addition, ANUG has been significantly associated with high levels of trait anxiety, depression and other emotional disturbances (Cohen-Cole et al 1983). Several case-control studies indicate associations between psychosocial factors and periodontal diseases. The importance of subjective factors in oral infections has been demonstrated when psychosocial features were considered in patients with destructive periodontitis (Genco, R.J. et al 1999). Prospective investigations have also demonstrated the importance of psychological disturbances on periodontitis progression (Linden, G.J. et al 1996) and on periodontal therapy response.

1.1 Mechanistic Links Between Depression and Chronic Periodontitis

Few biological and psychosocial hypothesis explaining the relationship between depression and periodontal health exist.

a. Negligence of oral health care behavior:

This hypothesis is based on the assumption that depressed patients neglect oral hygiene and professional regular dental care due to reduced motivation and interest (Kurer, J.R. et al 1995). Depression is also associated with unhealthy habits such as smoking (Breslau, N. et al 1993) and alcohol dependence (Zucker, R.A. 1986), two factors that are also known to increase one’s risk for chronic periodontitis. Table 1 shows studies demonstrating association between depression and oral hygiene habits.
### Table 1
Studies Demonstrating Association between Depression and Oral Hygiene Habits

<table>
<thead>
<tr>
<th>Author/ Year of publication</th>
<th>Study methodology</th>
<th>Plaque and Gingival score</th>
<th>Measurement of depression or depressive symptoms</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kurer et al 1995</td>
<td>Prospective cohort study</td>
<td>Turesky’s modification of Quigley &amp; Heines Plaque index - B&amp;L in incisors, premolars, molars/ Gingival index (GI) by Loe &amp; Silness(1963) - 4 sites per tooth</td>
<td>Hospital Anxiety Depression Scale (HADS)</td>
<td>Mean depression score was related with plaque accumulation</td>
</tr>
<tr>
<td>Monteiro Da Silva et al 1998</td>
<td>Case control</td>
<td>Plaque index (PI) by Silness &amp; Loe (1964) - 4 sites in tooth # 3,6,12,19,23, 28</td>
<td>Hospital Anxiety and Depression Scale (HADS) - Depression is not predictor for plaque accumulation</td>
<td></td>
</tr>
<tr>
<td>Klages et al 2005</td>
<td>Cross-sectional</td>
<td>Approximal Plaque Index(API) by Lange (1975) - Oral aspect in 1st &amp; 3rd quad. Buccal aspect in 2nd &amp; 4th Quad. Sulcus Bleeding Index(SBI) by Muhlemann &amp; Sohn (1971) – Oral aspect in 1st &amp; 3rd quad, Buccal aspect in 2nd &amp; 4th quad</td>
<td>Symptom Check List 90- R(SCL-90R) - Depression subscale(Self-report scale)</td>
<td>Subjects with more depression showed same plaque accumulation but more gingival inflammation</td>
</tr>
<tr>
<td>Saletu et al 2005</td>
<td>Case control</td>
<td>Approximal Plaque Index(API) by Lange (1975) - 6 sites per tooth/ Papilla bleeding Index(PBI) by Saxer &amp; Muhlemann (1975) - 6 sites per tooth</td>
<td>Hamilton Depression(HAMD) (Observer-rating scale) Zung Self-rating Depression Scale(ZSDS) (self-rating scale)</td>
<td>API &amp; PBI showed no significant correlations with psychometric variables including depression</td>
</tr>
<tr>
<td>Johanssen et al 2007</td>
<td>Case control</td>
<td>Plaque (Presence/Absence) - 6 sites Statistical per tooth/ Gingival index(GI) by Loe gingival Silness(1963)- 6 sites per tooth/ mean Bleeding on probing (%) - 6 sites per Tooth</td>
<td>Diagnostic &amp; Manual of mental disorder 4th edition (DSM-IV)</td>
<td>The depressed patient had significantly higher inflammation and deeper pocket</td>
</tr>
</tbody>
</table>
b. Alterations in Host Immune Response:

Depression enhances the production of pro-inflammatory cytokines, including IL-6 (Zorilla, E.P. et al. 2001). Significantly, both depressive symptoms and syndromal depression are associated with heightened plasma IL-6 levels. Following successful pharmacological treatment, elevated IL-6 levels decline in patients with a major depression diagnosis. Moreover, both physical and psychological stressors can provoke transient increases in proinflammatory cytokines, in animal models, both stress and administration of epinephrine elevated plasma IL-6, consistent with evidence that IL-6 production is stimulated through β-adrenergic receptors, among other pathways (Papanicolaou DA et al. 1998). Thus IL-6 and pro-inflammatory cytokines are directly stimulated by negative emotions and stressful experiences, providing one direct pathway.

Overproduction of proinflammatory cytokines may lead to maladaptive immune and endocrine changes subsequently. IL-6 is a potent stimulator of corticotrophin releasing hormone (CRH) production, a mechanism that leads to heightened HPA activity, including elevated levels of plasma ACTH, followed by increased cortisol levels (Dentino AN et al. 1999); elevations in ACTH and cortisol can provoke multiple adverse immunological changes.

When the levels of cortisol rise, they can initiate, perpetuate or aggravate syndromal depression, depression-like behaviors, and depressive symptoms such as anxiety, insomnia and poor memory. Thus, negative emotions that dysregulate IL-6 secretion may also promote adverse neuroendocrine alterations.

Indeed, in addition to their association with enhanced secretion of pro-inflammatory cytokines, depression also can have direct adverse effects on NK cell activity and also decreased cellular and humoral immune responses with sympathetic effector mechanisms (Friedman, E.M. & Irwin, M. 2001).

Depressed patients also have elevated levels of circulating catecholamines that result in the suppression of NK cells and cellular immune responses (Sanders, V.M. & Straub, R.H. 2002).

### Table 2

**Studies Demonstrating Relationship between Periodontitis and Depressive Symptoms**

<table>
<thead>
<tr>
<th>Author/Year of publication</th>
<th>Study methodology</th>
<th>Periodontal measurement</th>
<th>Measurement of depression or depressive symptoms</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moss et al 1996</td>
<td>Case control study</td>
<td>PD, CAL, Serum Ab titer for 3 pathogens (Bf, Pg, Aa)</td>
<td>Brief symptom Inventory (BSI) – Depression subscale (Self-report scale)</td>
<td>Positive association between periodontitis and Bf Aa, which is varied by depression score</td>
</tr>
<tr>
<td>Monteiro da Silva et al 1996</td>
<td>Case control study</td>
<td>PD, CAL, Radiographic alveolar bone loss assessment</td>
<td>Hospital Anxiety and Depression Scale (HADS) - Depression subscale</td>
<td>RPP group showed significantly increased depression compared to RCAP and control group</td>
</tr>
<tr>
<td>Genco et al 1999</td>
<td>Cross-sectional study</td>
<td>PD, CAL, ACH, Microbiological test</td>
<td>Brief symptom Inventory (BSI) – Depression subscale (Self-report scale)</td>
<td>An increase in CAL was significantly associated with depression. Depression and financial strain were significant predictor of CAL</td>
</tr>
<tr>
<td>Elter et al 1999</td>
<td>Prospective cohort study</td>
<td>PD, CAL – Measured from sequential examination over years</td>
<td>Center for Epidemiologic Studies Depression Scale (CES-D) (Self-report scale)</td>
<td>Subjects with more depressive symptom among whites had a higher rate of attachment loss</td>
</tr>
<tr>
<td>Saletu et al 2005</td>
<td>Case control study</td>
<td>PD, CAL, Radiographic alveolar bone loss level based on orthopantomogram</td>
<td>Hamilton Depression Scale (HAMD) (Observer-rating scale), Zung Self-rating Depression Scale (SDS) (Self-rating scale)</td>
<td>Subjects with periodontitis showed significantly higher self-rated &amp; observer-rated score</td>
</tr>
</tbody>
</table>
### References


<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Study Type</th>
<th>Measurement</th>
<th>Diagnosis</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ng et al, 2006</td>
<td>Cross-sectional study</td>
<td>PD.GR, CAL</td>
<td>Symptom Check List 90- R(SCL-90R) – Depression subscale(Self-report scale) ; Depression Anxiety Stress Scales-State (DASS-S) - Depression subscale (Self report scale). Depression Anxiety Stress Scales -Trait (DASS-T) - Depression subscale(Self-report scale)</td>
<td>Subjects with more severe CAL had a greater depression score than the periodontally healthy subjects</td>
<td></td>
</tr>
<tr>
<td>Johanssen et al 2007</td>
<td>Cross-sectional study</td>
<td>PD.CAL</td>
<td>DSM-IV</td>
<td>Depressed patients had significantly higher gingival inflammation and deeper pockets</td>
<td></td>
</tr>
</tbody>
</table>

2. Conclusion

Depression directly prompts immune dysregulation, and these processes may lead to subsequent maladaptive immune and endocrine changes. Production of IL-6 and other proinflammatory cytokines can be directly stimulated by depression providing one direct pathway. In addition, depression and stress may also contribute to prolonged infection or delayed wound healing, processes that fuel sustained pro-inflammatory cytokine production. Hence, research that addresses the dysregulation of the immune and endocrine systems associated with depression could substantially enhance our understanding of psychological influences on health, particularly among the elderly.

Evidences from literature have shown that clinically relevant depression might affect the success of periodontal therapy, which has to be proven by future studies involving clinical, neuro-physiological, neuro-endocrinological and psycho-pharmacological investigations. Such studies could elucidate the question regarding the extent to which the obviously related biochemical and behavioral factors are responsible for the pathogenesis of periodontitis. Therefore, factors related to the social environment, which may provoke changes in host defenses and modify health behavior, should be taken into account. This suggests that one must go further than microbiological and immunological markers to understand the pathogenic mechanisms that result in periodontal destruction. Furthermore clinical evidences suggest that psychological factors such as stress and depression represents an important part of overall periodontal maintenance and may also prevent oral inflammation from developing into systemic inflammation in susceptible individuals. Thus the use of simple stress profile and depression scale could provide valuable information on the psychological status of a patient and may be valuable tools in a modern periodontal practice that emphasizes individualized diagnosis, treatment planning and maintenance.

Aa – A. actionmycetemomitis, Ab- Antibody, ACH- Interproximal Alveolar Crestal Height, Bf- B.forsythus, CAL- Clinical Attachment Loss, PD- Pocket Probing Depth, Pg-P. gingivalis, REC- Gingival Recession


