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Research Article

OSTEOCALCIN-A HOVER OR HYPERLINK IN PERIODONTITIS

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A B S T R A C T

Osteocalcin levels have been postulated as a marker of inhibition of bone formation. Serum osteocalcin is presently considered a valid marker of bone turnover when resorption and formation are coupled, and a specific marker of bone formation, when formation and resorption are uncoupled. It may be involved in recruiting osteoclasts to sites of newly formed bone and thus may function as a negative regulator. In periodontitis, osteocalcin has been suggested to be a marker of bone formation where bone resorption is greater than formation, and GCF osteocalcin levels are more revealing than serum or saliva levels regarding bone turnover in periodontium.This paper highlights the role of osteocalcin as a potential marker of bone turnover in periodontal disease.

INTRODUCTION

Periodontitis is an inflammatory disease usually leading to loss of bone. In periodontitis, there is an increased turnover of alveolar bone although there may be a dominance of bone resorption over bone formation, leading to alveolar bone loss and loss of attachment.[1] Chronic periodontitis (CP) and aggressive periodontitis (AP), forms of inflammatory periodontal disease, differ from each other in terms of the magnitude, sequel and control of the response.[2]Bone homeostasis maintains by a coupled process of resorption followed by formation which reflect a change in bone turnover.[3] Markers of bone formation are proteins revealing osteoblast activity and are by-products of collagen synthesis, matrix proteins or osteoblastic enzymes.[4]

Osteocalcin is a small (5.4 kDa), calcium-binding protein of bone accounting for 10–20% of the non-collagenous protein in bone matrix. It has three residues of a calcium-binding amino acid, gamma-carboxyglutamic acid (Gla), that allow specific conformational changes enabling its binding to hydroxyapatite and later accumulation in bone matrix .[5] This vitamin K- and D-dependent protein produced by mature osteoblasts, osteocytes and odontoblasts, is found, in the extracellular mineralized matrix of bone and in the serum of circulating blood.[6] It may be involved in regulation of osteoblast function, regulation of bone turnover and/or mineralization. Markers of bone resorption, which reflect osteoclastic activity are mostly the breakdown products of type I bone collagen, the main component of the organic bone matrix .[4]

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Vitamin D plays a critical role in mediating calcium absorption and regulating musculoskeletal health.[7] It has also been demonstrated to function in the regulation of cardiovascular health, immune responses, wound healing and cancer prevention. Endogenous synthesis of vitamin D occurs in the skin and is induced via ultraviolet radiation.[8] Vitamin D deficiency may also play a role in periodontal disease and tooth loss, and insufficient vitamin D status is common among pregnant women. Vitamin D insufficiency (serum 25[OH]D <75 nmol/l) is associated with maternal periodontal disease during pregnancy. Vitamin D supplementation represents a potential therapeutic strategy to improve maternal oral health.[9] Several studies have investigated the relationship between GCF osteocalcin levels and periodontal disease

Osteocalcin and Periodontal disease

Kazushi Kunimatsu *et al* (1993)[10] investigated the levels of osteocalcin, a bone specific matrix protein, in gingival crevicular fluid (GCF) from periodontal disease patients. They found that in gingivitis patients, no significant amounts of osteocalcin were detected and in periodontitis patients, on the other hand, osteocalcin levels were detected, ranging between 0 and 540 pg/tube and positively correlated with the clinical parameters ($P < 0.01$). Therefore, strongly suggested that in addition to the presence of GCF osteocalcin, the levels of osteocalcin may reflect the degree of the periodontal inflammation at the sampled sites.

Sema Becerika *et al* (2011)[11] investigated the gingival crevicular fluid (GCF) calprotectin, osteocalcin and cross-linked N-terminal telopeptide (NTx) levels in health along with different periodontal diseases. The results revealed that the CP, G-AgP and gingivitis groups had higher GCF

calprotectin total amount compared to healthy subjects ($p < 0.008$). CP and G-AgP groups had similar, but higher levels compared to gingivitis groups ($p < 0.008$). CP and G-AgP groups had lower GCF osteocalcin total amount compared to gingivitis and healthy groups ($p < 0.008$). CP group had higher GCF NTx but lower osteocalcin total amount and osteocalcin/NTx ratio than the G-AgP group ($p < 0.008$) and suggested that elevated GCF calprotectin levels play a role as a reliable inflammatory marker in the pathogenesis of periodontal disease. Fluctuating GCF levels of osteocalcin and NTx might point out to the abnormal bone turn over in periodontitis.

Sabri Fatih Kurşunlu *et al* (2013)[12] conducted a cross-sectional study to investigate gingival crevicular fluid (GCF) osteocalcin levels in chronic periodontits (CP) and periodontal healthy in elderly subjects. The results showed that CP had higher GCF osteocalcin levels compared to healthy groups ($P < 0.05$) and suggested that osteocalcin plays a role on periodontal disease pathogenesis. Fluctuating GCF levels of osteocalcin might point out to the abnormal bone turnover in periodontitis.

Osteocalcin and Osteoporosis

Pedro Bullon *et al* (2005)[13] assessed plasma, saliva, and gingival crevicular fluid (GCF) levels of osteocalcin and correlated them with periodontitis and osteoporosis .The results showed that GCF osteocalcin concentrations were significantly higher in the PG women than in the NPG group ($P = 0.008$). Mean probing depth correlated significantly with GCF osteocalcin concentrations ($r = 0.35$; $P = 0.002$). Hence, concluded that osteocalcin levels in GCF correlates with periodontal but not with osteoporosis status.

Osteocalcin and Smoking

Shimae Nafarzadeh *et al* (2015)[14]compared the salivary osteocalcin (OC) level among smokers and non-smoker patients with chronic periodontitis and showed that smoking and periodontal diseases are associated.

Lubaba A. Abdul Ameer *et al* (2015)[15]evaluated the effect of smoking on the salivary alkaline phosphatase and osteocalcin in subjects with chronic periodontitis compared to control subjects.They found that the osteocalcin levels were lower in smoker chronic periodontitis group (0.13±0.20) than non-smoker chronic periodontitis group (1.09±2.26) with significant difference ($0.05 \geq P > 0.01$). Mean of alkaline phosphatase level was lower in smoker chronic periodontitis (11.14±4.53) than non-smoker chronic periodontitis (11.45±4.17) with a non-significant difference, while there was a significant difference in alkaline phosphatase concentrations between smoker and non-smoker control groups. Hence, a conclusion was drawn that the suppression of salivary osteocalcin levels by smoking and slight increase in alkaline phosphatase in smokers groups, may explain the deleterious effects of smoking on periodontal health status.

Osteocalcin and Vitamin D

Assad Assad Amara Matouga *et al* (2014)[16] evaluated the level of 25-Hydroxy vitamin D3 and osteocalcin in GCF and serum before and after scaling and root planning in chronic periodontitis patients .They showed that the respective local osteocalcin level were significantly dropped from baseline to six weeks after (SRP) (9.56ng/ml versus 7.38ng/ml, P=0.001). The respective systemic osteocalcin level significantly increased after six weeks from SRP (10.85ng/ml versus 17.74 ng/ml, P=0.001) . The respective local 25-Hydroxy vitamin D3 level were significantly increased from baseline to six weeks after (SRP) (3.41ng/ml versus 4.57ng/ml, P=0.001). The respective systemic 25-Hydroxy vitamin D3 level significantly increased after six weeks from SRP (39.88ng/ml versus 41.48 ng/ml, P=0.001). Hence from the above results, it was concluded that 25-hydroxy vitamin D3 might have an important role in the pathogenesis of periodontal disease and could be used as adjunctive therapeutic modality for the prevention and treatment of different types of periodontitis and osteocalcin could be used as a potential diagnostic marker for periodontal disease activity in both serum and gingival crevicular fluid.

Osteocalcin and Diabetic Mellitus

Amina Hamed Ahmed *et al* (2016) [17]evaluated the correlation among salivary and serum levels of cystatin C, osteocalcin, and fibronectin in subjects with diabetes and periodontitis alone or in combination compared with control. The results showed that there was a significant correlation between serum and salivary cystatin, osteocalcin and fibronectin in control group, periodontitis group, diabetic group and periodontitis subjects with diabetes. In addition, in diabetic group, there was a significant correlation between serum osteocalcin and serum cystatin; serum osteocalcin and serum fibronectin; salivary osteocalcin and salivary fibronectin. While in those with diabetes and periodontitis, there was a significant correlation between serum osteocalcin and salivary cystatin; salivary osteocalcin and serum cystatin; salivary osteocalcin and salivary fibronectin. The conclusion drawn was the patterns of significant correlation are the same in control and periodontitis groups, while it is different in diabetic group and periodontitis with diabetes.

Zina Ali Daily *et al* (2017)[18] assessed the osteocalcin levels in saliva of diabetic patients and systemically healthy persons. The results revealed that the patients had chronic periodontitis with poorly controlled type 2 diabetes mellitus (CP+pT2DM) demonstrated the highest median values of all clinical periodontal parameters and highest increase in levels of salivary OC followed by CP+wT2M group then CP and control groups. From the results, they concluded that the patients with poor glycemic control had more severe periodontal tissue break down with increase in levels of OC than well controlled type 2 diabetic patients and non-diabetic patients all of them with chronic periodontitis.

CONCLUSION

Osteocalcin is one of the greatest plentiful matrix proteins found in bones and the only matrix protein synthesized in the bone. Small osteocalcin fragments are found in regions of bone remodelling and are in fact degradation products of the bone matrix, that is released outside cells into the gingival crevicular fluid (GCF) and saliva after destruction of periodontal tissue during periodontitis.Osteocalcin, incorporated into the bone matrix, is released into the circulation from the matrix during bone resorption and, hence, is considered a marker of bone turnover, rather than a specific marker of bone formation.

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