

B 会 場

IO-01～06

国際セッション口演

(B会場)

5月25日(土) B会場 11:10～12:10

IO-01

Prevention of buccal bone resorption using a pamidronate-loaded collagen matrix following tooth extraction

Jae-Kook Cha

Keywords: Tooth extraction, Ridge preservation, Bisphosphonate, Histology

Aim: the aim of this *in vivo* investigation was to assess the anti-resorptive effect of low concentration pamidronate on the buccal plate in fresh extraction sockets.

Materials and methods: The distal roots of the third premolars were extracted in 6 beagle dogs bilaterally. A collagen matrix loaded with either 15mg/ml pamidronate (test group) or saline (control group) was positioned on the outer surface of buccal bone immediately after tooth extraction and subsequently covered with a coronally advanced flap. Histological and histometrical outcomes were evaluated 12 weeks later.

Results: The histologic healing pattern clearly differed between the test and control groups. The mean vertical distance between the buccal and lingual bone crest differed significantly between the test and control groups (0.52 ± 0.43 and 2.21 ± 1.15 mm, respectively; $p=0.037$). The width of the buccal bone 1mm below the crest was significantly wider in the test group than the control group (4.68 ± 0.68 vs 3.44 ± 0.60 mm, $p<0.001$).

Conclusions: Local administration of pamidronate onto a collagen matrix may reduce the dimensional changes of the buccal bone plate both vertically and horizontally.

IO-03

Periodontal Inflammation Results In Decreased Renal Function in Patients With Chronic Kidney Disease

Praveen Sharma

Keywords: Periodontitis, CKD, Inflammation, Oxidative stress, Structural equation model

Objectives: Chronic kidney disease (CKD) is associated with increased morbidity and mortality, largely due to cardiovascular disease. Periodontitis may impact on CKD-associated morbidity/mortality by contributing to systemic inflammation and oxidative stress burden.

The aim of this study was to quantify the effects of periodontal inflammation on renal function using structural equation models (SEM).

Methods: We recruited 770 patients with stage 3-5 pre-dialysis CKD. Periodontal inflammation was expressed using the periodontal inflamed surface area (PISA) score. Renal function was assessed using estimated glomerular filtration rate (eGFR).

SEMs were created to unravel potential pathways by which periodontal inflammation may influence renal function or vice-versa.

Results: The mean age of participants was 63 years, 61% were male, 48% never-smokers and 37% had diabetes. Path analysis using SEM revealed an indirect effect of increase in PISA score, via oxidative stress, on decreasing renal function such that a 10% increase in PISA score equated to a 3% decrease in eGFR (95% CI 0.4-4.6% $p=0.021$). There was no significant effect of eGFR on PISA score.

Conclusion: We confirm, using SEM, our causal hypothesis that, a decline in periodontal health results in a decline in kidney function via an increase in oxidative stress burden.

IO-02

Perio Systemic Relationship: Where do we stand?

Gurparkash Singh Chahal

Keywords: Periodontitis, Association, Non alcoholic fatty liver disease, PTLBW, Biomarker

Objectives: To explore the association between periodontal disease and nonalcoholic fatty liver disease (NAFLD), Pre-term Low Birth Weight (PTLBW) and to correlate levels of Cardiovascular disorder associated biomarkers (MCP-1, sCD40L).

Materials and method: In three independent studies, clinical periodontal parameters, respective systemic markers for 50 NAFLD patients (A), 74 pregnant females (positive history of previous PTLBW) (B) and 103 age, gender matched healthy controls were recorded. In a set of 70 chronic periodontitis patients MCP-1 and sCD40L levels in GCF, serum and saliva were also recorded following periodontal therapy.

Results: Positive correlation ($p=0.008$) between bleeding on probing- serum TNF- α (A), serum levels of PGE2 - gingival index (B) and statistically significant difference in periodontal status parameters was observed. sCD40L and MCP-1 levels in GCF were strongly correlated with increasing severity of periodontal disease and levels in serum. In post-treatment, the levels decreased significantly ($p \leq 0.001$).

Conclusions: Results point to a plausible mediating link of periodontal and systemic health in NAFLD patients and adverse pregnancy outcomes. MCP-1 and SCD40L were found to be reliable markers indicating severity of periodontal destruction, further strengthening the evidence of the impact of periodontal disease on systemic disease.

IO-04

The inflammatory-resistant property of gingival stem/progenitor cells under porphyromonas gingivalis lipopolysaccharides stimulation

Li-li Zhou

Keywords: gingival mesenchymal stem/progenitor cells, LPS, inflammation

Aim: This study investigates for the first time the effect of Porphyromonas gingivalis lipopolysaccharides (Pg-LPS) on proliferative/regenerative aptitudes of gingival stem/progenitor cells (G-MSCs).

Materials and methods: G-MSCs ($n=5$) were treated by 0, 10ng/ml, 100ng/ml, 1 μ g/ml or 10 μ g/ml Pg-LPS. At 1 hour, Toll-like receptor 4 (TLR-4) expression and NF- κ B and Wnt/ β -catenin signalling pathways were examined. Colony-forming unit assay was conducted at day 12. At 24 and 48 hours, MTT test, ALP activity, mRNA for tumour necrosis factor- α (TNF- α), interleukin-6, collagen-I (Col-I), collagen-III, RUNX-2, alkaline phosphatase (ALP), osteonectin and protein expression of interleukin-6 and TNF- α were analysed.

Results: With increasing Pg-LPS, TLR-4 was upregulated, pNF- κ B-p65 rose from median (Q25/Q75) 6.56% (4.19/7.90) to 13.02% (8.90/16.50; $p=0.002$) and pNF- κ B-p65/ tNF- κ B-p65 from 0.14 (0.10/0.17) to 0.30 (0.21/0.42; $p=0.002$). β -Catenin, t β -catenin and p β -catenin/ t β -catenin showed no differences. Increasing Pg-LPS concentration increased cell numbers from 288.00 (72.98/484.32) to 861.39 (540.41/1599.94; $p=0.002$), ALP mRNA from 0.00 (0.00/0.01) to 0.56 (0.00/1.90; $p=0.004$) and TNF- α from 32.47 (12.11/38.57) to 45.32 (28.68/48.65; $p=0.036$). Over time, ALP activity increased from 0.89 (0.78/0.95) to 1.90 (1.83/2.09; $p<0.001$), mRNA for TNF- α from 0.00 (0.00/0.12) to 0.01 (0.00/0.06; $p=0.007$), mRNA for Col-I from 82.70 (0.03/171.50) to 124.00 (52.85/232.50; $p=0.019$), while mRNA for RUNX-2 decreased from 1.73 (0.92/3.20) to 0.84 (0.48/1.47; $p=0.005$).

Conclusion: Pg-LPS upregulated G-MSCs' proliferation, without attenuation of their regenerative potential. The effects were NF- κ B, but not Wnt/ β -catenin, pathway dependent.

Keywords: Antimicrobial, metabolite, KetoC, periodontitis

Objectives: Periodontitis defined as a chronic inflammation initiated by bacterial dysbiosis that predominated by periodontopathogens, resulting a bone loss. Although bioactive metabolite (KetoC) has been reported to show various beneficial effects, the antimicrobial function remains unclear. In this present study, we investigated the effect of KetoC in periodontitis mice model and explored the underlying mechanism.

Materials and methods: Eight-week-old male C57BL/6N mice were given a daily oral gavage of KetoC (15mg/mL) or vehicle for 2 weeks. At 7th day, 5-0 silk ligature was ligated on the second maxillary left molar to induce periodontitis. In addition, *P. gingivalis* W83 (109 CFU) suspension was given orally to the mice every 3 days for the last 7 days. At 14th day, all mice were euthanized. Bone destruction was measured; amount of *P. gingivalis* was quantified by real-time polymerase chain reaction (RT-PCR). In vitro, the antimicrobial effect of KetoC on *P. gingivalis* was explored.

Results: *In vivo*, KetoC attenuated alveolar bone destruction and suppressed the elevated *P. gingivalis* amount in the periodontitis-induced group. *In vitro*, KetoC down-regulated *P. gingivalis* proliferation in a dose-dependent manner.

Conclusions: KetoC diminished alveolar bone destruction in periodontitis model via its direct antimicrobial function.

Keywords: Cytokines, Experimental periodontitis

Objective: The aim of this study was to investigate the alteration to the dynamics of periodontal destruction in ligature-induced experimental periodontitis in rats.

Material and Methods: To induce experimental periodontitis, a 3-0 silk ligatures were placed around maxillary first molars bilaterally. Male Wistar rats (n=20) were equally divided into 4 groups: non-ligature for 7 days (NL-7), ligature for 7 days (L-7), non-ligature for 14 days (NL-14) and ligature for 14 days (L-14). Samples were collected in each time point. Micro-CT was used to measure bone resorption in the left side of the maxilla, and qPCR was used to measure the expression of *IL-6*, *Mmp-9*, *Rankl*, *Opg*, and the *Rankl/Opg* ratio in gingival tissue. With tissue from the right side of the maxilla, H-E staining was used for histological analysis, and TRAP staining was used to observe the number of osteoclasts.

Results: The L-7 group showed lower bone volume fraction ratio and bone mineral density than L-14 group. On the other hand, the L-7 group had more numbers of inflammatory cells infiltration and more numbers of TRAP-positive osteoclasts lining on the bone surface than L-14 group. Correspondingly, qPCR analyses also showed that the gene expressions of *IL-6*, *Rankl*, *Mmp-9*, but not of *Opg* were upregulated in L-7 group but stabilized in L-14 group.

Conclusion: These findings suggest that in ligature-induced experimental periodontitis, bone resorption value increased and inflammatory infiltration observed at 7 days and decreased noticeably at 14 days during ligation placement