The Role of Static Magnetic Healing Abutment in Osteoblastic Differentiation to Reduce Marginal Crestal Bone Loss

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Abstract

Dental implantation successfully can be made if the implant, bone, and soft tissue are integrated to increase patient satisfaction. Marginal crestal bone loss, if once occurring, will directly affect the longevity of dental implants; thus, it causes implant failure. Static magnetic field (SMF) has always been a great interest in dentistry as it has been used for various purposes in prosthodontics including prosthesis and overdentures. The use of SMF as healing abutment is under investigation.

This study investigated how SMF can reduce marginal crestal bone loss after dental implant placement. Functioning the static magnetic field (SMF), canonical Wnt ligands can be upregulated, and phosphorylation of GSK-3β and total β-catenin expression in osteoblasts are promptly stimulated. SMF activated the Wnt/β-catenin signaling pathway to invigorate the osteoblastic differentiation. SMF-activated Wnt-Fzd-LRP5/6 binding events can accumulate β-catenin intracellularly and phosphorylate GSK3, which activates MAPK and NF-κβ for osteoblastogenic gene and Runx2 transcription.

Therefore, SMFs positively affect bone healing and periodontal regeneration. Static magnetic healing abutment plays a role in increasing osteoblastic differentiation; hence, it reduces marginal crestal bone loss.

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Introduction

Marginal crestal bone loss after dental implant placement may represent a threat to implant longevity. It has occurred to 5-10% of patients. During the first year of dental implantation failure, peri-implant bone loss exceeds 1.0 mm. A year after, it is more than 0.2 mm.[1,2]

One factor that affects bone loss and determines the implant success is osseointegration in which the bone and soft tissue are integrated with the implant surface. Osseointegration is a sequential four phases of wound healing process which includes hemostasis, inflammatory phase, proliferative phase, and remodeling phase. The hemostasis occurs within minutes to hours, inflammatory phase in hours to days, proliferative phase in days to weeks, and the remodeling phase in about 3 weeks and even years.[3,4]

Mechanical bonding could trigger osseointegration with human bone as particles and ions crack and release on surrounding tissues. Titanium (Ti) particles, well-established biomaterials for dental implants, cause inflammation apart from progressive bone loss.[5] Recent research supports this finding by stating that titanium particles activate proinflammatory cytokines. Imbalanced cytokine levels released by the particles may resolute inflammation besides leading to alveolar bone and soft tissue loss.[6]

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Static magnetic field (SMF) is an effort in dentistry to find an alternative material to help bone growth and healing. In several animal studies, there are no adverse effects reported from magnetic implant placement.[7] Siadat et al.[8] mentioned that SMF can stabilize the implant and reduce bone loss as the bone healing occurs in some first weeks; while the implant stability quotient values obtained with SMF was greater than non-magnetic group.[9] This review aimed to investigate further the role of SMF in reducing marginal bone loss by increasing osteoblastic differentiation.

Reviews

With the static magnetic field (SMF), canonical Wnt ligands prominently increased. Similarly, the GSK-3β phosphorylate and β-catenin is expressed in osteoblasts. The osteoblastic differentiation also occurs due to the simulating SMF which also activates the Wnt/β-catenin signaling pathway.[10] The signaling molecule Wnt passes through both β-catenin-dependent canonical and independent noncanonical pathways when the osteoblast differentiation is going on.[11,12,13]

Two cytokines expressed by osteoblast-lineage cells are significant to osteoclast differentiation in addition to colony-stimulating factor-1 (CSF-1). The cytokines can bind with the receptor activator of nuclear factor κβ ligand (RANKL). Osteoblasts constitutively express CSF-1, while RANKL expression is inducible there, especially for bone resorption-stimulating factors e.g., 1α,25 dihydroxy vitamin D_3 ($1,25D_3$), interleukin (IL)-11, as well as parathyroid hormone. Like RANK (RANKL receptor), CSF-1 receptor (CSF-1r) is the product of osteoclast precursors which then diverge into osteoclasts. Osteoblasts can produce a soluble decoy receptor for RANKL, Osteoprotegerin (Opg) which blocks the RANKL–RANK interaction by inhibiting osteoclastogenesis.[11,14,15]

SMF stimulates the proliferation of mesenchymal stem cells, and in turn, activates Wnt/β-catenin signalling. Skeletal development and osteoblast differentiation are two processes affected by the pathway. Wnt binding to its receptors may inhibit GSK-3β, stabilize, and accumulate β-catenin. The β-catenin is then translocated to the nucleus. The members of the T-cell factor/lymphoid enhancer factor family of transcription factors interact with each other in the nucleus. This process activates Wnt downstream target genes.[11]

The MAPK and NF-κβ had an essential role in cell differentiation. SMF-activated Wnt-Fzd-LRP5/6 binding intracellularly accumulated β-catenin and phosphorylates GSK3. The phosphorylation activates MAPK and NF-κβ for osteoblastic gene and Runx2 transcription. Therefore, SMFs prompt good effects on bone healing, not to mention periodontal regeneration.[14,16]

In osteoclast genesis, NO mediating cell fusion of pre-osteoclasts could be generated due to inducible NO synthase expression. Cell differentiation is more vigorous as the NO pathway is being stimulated. Whether or not NO is involved needs to be investigated further, especially in the osteoclastogenic responses to SMF.

Conclusions

Static magnetic healing abutment is likely to increase osteoblastic differentiation; hence, it reduces marginal crestal bone loss.

Declaration of Interest

The authors declare that there are no conflicts of interest.

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