

The Effects of Desferroxamine on Bone and Bone Graft Healing in Critical-size Bone Defects

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Abstract

Background: Desferroxamine (DFO) increases synthesis of VEGF by means of increased levels of “Hypoxia inducible factor-1” that triggers osteoblastic activity and stimulates osteoblastic differentiation (1-3).

The aim of this study is to evaluate the in vivo effects of DFO on bone graft healing and osteogenesis in critical-size bone defects.

Methods: Rat zygomatic arch critical-size bone defect model (5 mm.) was used as the experimental model. Thirty-two Sprague-Dawley rats (64 zygomatic arches) were divided into four groups (16 zygomatic arches in each). In Group 1, repair was done with the bone graft that was transferred from the other side. In this group, 200 microM / 300 microL dosage of DFO was injected at the zygomatic arch region starting at 7th day preoperatively and lasting 45th day postoperatively. Group 2 animals were assigned the control group of Group 1 and 0.9 %NaCl injection was applied. In Group 3 and 4 there was no repair after the formation of defects and rats were treated with DFO and 0.9 %NaCl for postoperative 45 days, respectively. Assessments were performed through histological (H&E, at the end of 1st, 6th and 12th weeks), radiological (CT, 2nd, 4th, 8th and 12th weeks) and biomechanical (3 point bending test, 12th week) tests.

Results: In radiological evaluation, decrease in the size of bone defect (Group 3 and 4) was statistically significant ($p<0.05$) at the 4th, 8th and 12th weeks. In Group 1 and 2, the evaluation of bone graft volume showed a statistical difference at all weeks ($p<0.05$). In the histological evaluation it was observed that there was an increase in osteoblast number in Groups 1 and 3 rats at all weeks and similarly vascularity rate was also high ($p<0.05$). At the end of 12 week, as a result of biomechanical evaluation of the subjects, we observed that the increased bone strength but no structural change in Group 1 animals.

Conclusion: In this study, it was shown that DFO treatment increased bone graft incorporation and healing in critical-size bone defects. In this aspect, we suggest that DFO can be used to increase graft incorporation in risky areas and reduce the defect size in patients who are not suitable for vascularized bone graft transfer.

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