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Effects of local simvastatin on periosteal distraction osteogenesis in rabbits☆

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Abstract

Our aim was to evaluate the effect of local simvastatin on the formation of new bone using a new design of periosteal distractor. The distractors were placed between the periosteum and bone at the inferior border of the mandible of 20 New Zealand rabbits. In the first group ($n = 10$) simvastatin was applied locally to the distraction zone. The other 10 rabbits served as controls. The formation of new bone was evaluated with digital direct radiography, computed tomography (CT), and histomorphometric analyses. New bone formed in all rabbits, but more formed in the experimental group according to histomorphometric variables. However, other measurements did not differ significantly between the groups. The new design of the periosteal distraction device was successful in causing new bone to form. Local simvastatin made no significant contribution to the procedure.

Keywords: Periosteal distraction; Simvastatin; Bone augmentation; Osteogenesis

Introduction

The "gold standard" for the reconstruction of bony defects remains autogenous grafting,[1] but morbidity at the donor site and the limited quantity of bone available are the main disadvantages.[2–7] Distraction osteogenesis has the potential to overcome these disadvantages, and in addition can provide lengthening of soft tissue together with new bone.[5–7] Various techniques of distraction osteogenesis have been described with reasonable success rates, and in recent years the idea of osteogenesis by periosteal distraction for the treatment of bone deficiencies has also been suggested.[1,2,8,9]

The highly vascularised internal osteoblastic layer of periosteum plays a part in distraction osteogenesis. It is composed of mesencymal stem cells,[5] and it has therefore been suggested that it is more important than endosteum in distraction osteogenesis.[9,10]

Statins are effective lipid-lowering drugs that are widely used to reduce the risk of cardiovascular disease given their ability to inhibit the 3-hydroxy-3-methylglutaryl-coenzyme.[11] They are extensively bound to plasma proteins and predominantly metabolised by the cytochrome P450 family of enzymes, and their main route of elimination is through bile after being metabolised by the liver.[12] Several studies have shown that both systemic and local simvastatin contribute to bony regeneration.[11–15] Although the exact mechanism is not known some hypotheses have been

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