MORPHOFUNCTIONAL CRITERIA FOR EVALUATING ANGIOGENESIS AND OSSEOINTEGRATION IN JAW BONE DEFECT RECONSTRUCTION USING VARIOUS BIOMATERIALS

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ABSTRACT: Successful jaw bone reconstruction and dental implantology rely on rapid vascularization and harmonious osseointegration of implanted biomaterials. Insufficient angiogenesis leads to graft failure, prolonged inflammation, and fibrous encapsulation. The objective of this study was to establish morphofunctional criteria for evaluating early vascularization and subsequent osseointegration using xenogeneic, allogeneic, and synthetic bone graft substitutes in jaw defects.

KEYWORDS: Bone augmentation, angiogenesis, osseointegration, VEGF, CD31, xenograft, biomaterials, histomorphometry, jaw defect.

Reconstruction of maxillofacial bone defects remains a major challenge in oral and maxillofacial surgery. The clinical success of bone graft substitutes depends not only on their scaffold properties, but primarily on their ability to stimulate rapid capillary ingrowth. Early angiogenesis is a mandatory prerequisite for osteogenesis, as newly formed microvessels transport oxygen, essential nutrients, and progenitor cells required for osteoblast differentiation. When vascularization is delayed, biomaterial integration fails, resulting in ischemic graft failure or persistent fibrotic repair.

The experimental results demonstrated distinct tissue responses among the investigated materials. During the early healing phase at day fourteen, immunohistochemical analysis revealed strong expression of vascular endothelial growth factor and cluster of differentiation thirty-one at the periphery and within the pores of the deproteinized bovine bone scaffold. This intense angiogenic activity provided an optimal microenvironment for osteoprogenitor recruitment. In contrast, synthetic calcium phosphate ceramics induced a delayed vascular response with localized macrophage accumulation and restricted capillary penetration. In the allogeneic group, early vascularization occurred, but rapid enzymatic degradation of the matrix caused structural volume loss before stable osteoid formation was achieved.

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