

CHARACTERISTICS OF OSTEOGENESIS IN CLOSED FEMORAL DIAPHYSEAL FRACTURES UNDER EXPERIMENTAL METABOLIC SYNDROME CONDITIONS

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Abstract

Metabolic syndrome is a multifactorial pathological condition characterized by obesity, insulin resistance, dyslipidemia, and arterial hypertension. Recent studies have demonstrated that metabolic syndrome negatively influences bone metabolism and tissue regeneration. The present study investigates the characteristics of osteogenesis in closed femoral diaphyseal fractures under experimental metabolic syndrome conditions. Experimental observations revealed delayed callus formation, reduced osteoblastic activity, impaired angiogenesis, and slower bone remodeling. Histological and radiological examinations indicated significant differences between the control and experimental groups during the fracture healing process. The findings suggest that metabolic syndrome adversely affects the biological mechanisms responsible for bone repair and regeneration. Understanding these changes may contribute to the development of effective therapeutic strategies for improving fracture healing outcomes in patients with metabolic disorders.

Keywords: Metabolic syndrome, osteogenesis, femoral fracture, bone regeneration, fracture healing, osteoblasts, bone remodeling.

Introduction

Bone tissue possesses a remarkable regenerative capacity that enables the restoration of structural integrity following injury. Fracture healing is a complex biological process involving inflammation, soft callus formation, hard callus development, and remodeling. Successful bone repair requires coordinated interactions among osteoblasts, osteoclasts, chondrocytes, vascular endothelial cells, and various growth factors.

Metabolic syndrome has become one of the most common health problems worldwide. It is characterized by a combination of obesity, insulin resistance, hyperglycemia, dyslipidemia, and hypertension. These metabolic abnormalities contribute to chronic inflammation, oxidative stress, and vascular dysfunction, all of which may negatively affect bone metabolism and fracture healing.

Several studies have reported that patients with metabolic syndrome experience delayed bone regeneration and increased risk of fracture-related complications. However, the precise mechanisms underlying impaired osteogenesis remain insufficiently understood. Therefore, the present study aims to evaluate the specific characteristics of osteogenesis in closed femoral diaphyseal fractures under experimental metabolic syndrome conditions.

Materials and Methods

The experimental study was conducted using laboratory rats divided into two groups: a control group and an experimental group with induced metabolic syndrome. Metabolic syndrome was established through long-term administration of a high-fat and high-carbohydrate diet combined with reduced physical activity. After confirmation of metabolic syndrome through biochemical and anthropometric assessments, standardized closed femoral diaphyseal fractures were induced under general anesthesia. Fractures were stabilized using appropriate fixation techniques to ensure comparable healing conditions.

Animals were observed for 4, 8, and 12 weeks after fracture induction. Radiological examinations were performed to evaluate fracture consolidation and callus formation. Histological analyses were conducted using hematoxylin-eosin staining to assess cellular activity, vascularization, and bone matrix formation. Parameters such as osteoblast density, trabecular organization, callus size, and degree of mineralization were evaluated and compared between the groups.

Results and Discussion

The results demonstrated significant differences in fracture healing between the control and metabolic syndrome groups. During the early stages of repair,

inflammatory responses persisted longer in animals with metabolic syndrome. Soft callus formation was delayed, and the transition to mineralized tissue occurred more slowly than in the control group.

Histological examination revealed decreased osteoblastic activity and reduced synthesis of bone matrix proteins. The number of newly formed blood vessels within the fracture site was significantly lower, indicating impaired angiogenesis. Since vascularization is essential for nutrient delivery and cellular migration, reduced blood vessel formation likely contributed to delayed healing. Radiological findings showed larger fracture gaps and lower bone density in the experimental group. At twelve weeks post-fracture, complete consolidation was observed in most control animals, whereas incomplete union remained evident in several animals with metabolic syndrome.

Chronic inflammation associated with metabolic syndrome appears to interfere with normal osteogenic signaling pathways. Elevated levels of pro-inflammatory cytokines may inhibit osteoblast differentiation and promote bone resorption. Furthermore, insulin resistance can impair cellular metabolism and reduce the anabolic effects of insulin on bone tissue.

These findings are consistent with previous research indicating that metabolic syndrome compromises bone quality and regenerative potential. The observed reduction in osteogenesis highlights the importance of metabolic regulation in fracture management.

Conclusion

Experimental metabolic syndrome significantly alters the process of osteogenesis during healing of closed femoral diaphyseal fractures. Delayed callus formation, decreased osteoblastic activity, impaired angiogenesis, and prolonged remodeling were identified as major pathological features. These alterations contribute to slower fracture healing and increased risk of complications. Future research should focus on therapeutic interventions aimed at improving bone regeneration in individuals affected by metabolic syndrome. Effective control of

metabolic abnormalities may enhance osteogenesis and promote successful fracture repair.

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