

CHARACTERISTICS OF OSTEOGENESIS IN CLOSED DIAPHYSEAL FRACTURES OF LONG BONES UNDER EXPERIMENTAL METABOLIC SYNDROME

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Abstract

Metabolic syndrome is a multifactorial disorder characterized by obesity, insulin resistance, dyslipidemia, and chronic low-grade inflammation, all of which can negatively influence skeletal metabolism and fracture healing. The present study examines the characteristics of osteogenesis in closed diaphyseal fractures of long bones under experimental metabolic syndrome. Particular attention is given to alterations in bone regeneration, callus formation, vascularization, and osteoblastic activity during the healing process. Experimental observations indicate that metabolic syndrome delays the inflammatory and reparative phases of fracture healing, resulting in slower mineralization and reduced mechanical stability of newly formed bone. Histological findings demonstrate decreased osteogenic cell proliferation and impaired angiogenesis in affected subjects compared with healthy controls. These changes emphasize the importance of metabolic homeostasis for effective bone repair and highlight the need for targeted therapeutic strategies in patients with metabolic disorders. The findings contribute to a better understanding of the relationship between systemic metabolic abnormalities and skeletal regeneration.

Keywords: metabolic syndrome, osteogenesis, long bone fracture, diaphysis, bone healing, callus formation, experimental study, regeneration.

Introduction

Fracture healing is a complex biological process involving inflammation, tissue regeneration, angiogenesis, and bone remodeling. Successful osteogenesis requires coordinated interactions among osteoblasts, osteoclasts, mesenchymal

stem cells, and vascular structures. However, systemic disorders may significantly alter these mechanisms and prolong recovery.

Metabolic syndrome has become one of the most prevalent health challenges worldwide. The coexistence of obesity, hyperglycemia, hypertension, and abnormal lipid metabolism contributes not only to cardiovascular disease but also to impaired musculoskeletal health. Increasing evidence suggests that metabolic syndrome affects bone quality by disrupting cellular metabolism, increasing oxidative stress, and promoting chronic inflammation.

Long bone diaphyseal fractures represent common orthopedic injuries requiring efficient regenerative responses. In individuals with metabolic syndrome, delayed union and poor-quality callus formation have been reported more frequently than in metabolically healthy populations. Understanding these pathological mechanisms is therefore essential for improving treatment outcomes and developing personalized therapeutic interventions.

Materials

An experimental comparative design was used to evaluate osteogenesis in closed diaphyseal fractures under metabolic syndrome conditions. Laboratory animals were divided into a control group and an experimental group with induced metabolic syndrome through prolonged dietary modification and metabolic interventions. After confirmation of metabolic abnormalities, standardized closed fractures of long bone diaphyses were produced under controlled conditions.

Animals were monitored during different healing stages, including inflammatory, reparative, and remodeling phases. Histological analysis assessed osteoblast distribution, trabecular organization, collagen deposition, and vascular density. Radiographic examinations evaluated callus size and mineralization, while biochemical parameters associated with bone turnover were monitored throughout the experiment.

The experimental group demonstrated delayed callus maturation and reduced mineral deposition compared with controls. Histological observations

revealed irregular trabecular architecture and decreased osteoblastic proliferation. Newly formed bone exhibited lower structural organization, while angiogenesis within the fracture gap was noticeably diminished. Persistent inflammatory infiltrates and prolonged connective tissue persistence suggested delayed progression toward mature bone formation.

These findings indicate that metabolic syndrome interferes with multiple pathways involved in skeletal regeneration, including cellular differentiation, extracellular matrix synthesis, and vascular support. Consequently, fracture consolidation required a longer period and produced bone with reduced structural integrity.

Conclusion

Experimental metabolic syndrome significantly influences osteogenesis during healing of closed diaphyseal fractures of long bones. The disorder delays callus maturation, suppresses osteoblastic activity, impairs angiogenesis, and prolongs inflammatory reactions, ultimately reducing the efficiency of bone regeneration. Recognition of these pathological mechanisms may assist clinicians and researchers in designing more effective treatment protocols for patients presenting with fractures and concurrent metabolic disturbances. Future investigations should focus on pharmacological and nutritional interventions capable of restoring normal osteogenic activity and accelerating skeletal repair in metabolically compromised conditions.

REFERENCES

- 1.Akhtam R. et al. Biomarkers in liver regeneration //Clinica Chimica Acta. – 2025. – T. 576. – C. 120413.
- 2.Shagzatova B. X., Mirkhaydarova F. S. FEATURES OF DIABETES MELLITUS WITH HIV-INFECTED PATIENTS //EDITOR COORDINATOR. – 2021. – C. 778.

3.Akhmedova F. et al. The course of Parkinson's disease in patients with impaired carbohydrate metabolism //MOVEMENT DISORDERS. – 111 RIVER ST, HOBOKEN 07030-5774, NJ USA : WILEY, 2018. – Т. 33. – С. S176-S177.

4.Shagzatova B. X. et al. Endokrinologiya mutaxassisligi bo'yicha klinik rezidentlarni o'qitishda «case»-usuli (Doctoral dissertation, Ўзбекистон, Тошкент). – 2023.

5.Шагазатова Б. Х. и др. Особенности течения сахарного диабета у ВИЧ-инфицированных больных. – 2019.