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COLLAGEN AS THE BASIS OF BONE TISSUE AND ITS DEGRADATION IN INFLAMMATION

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Abstract:
Type I collagen forms the basis of the organic matrix of bone tissue and plays a key role in ensuring its strength, elasticity, and resistance to mechanical stress. In inflammatory processes, such as acute hematogenous osteomyelitis, the degradation of collagen fibers is activated, which disrupts the structural integrity of bone tissue. This article explores the structure and functions of type I collagen in bone tissue, as well as the mechanisms of its degradation during inflammation and the diagnostic significance of its degradation products.

Keywords: type I collagen, bone, osteomyelitis, inflammation, degradation, osteoclasts, markers of bone metabolism.

Introduction

Bone tissue is a unique specialized connective tissue in which organic and inorganic components interact closely to provide strength, elasticity, and regenerative potential. The main protein component of bone is type I collagen, which plays a crucial role in the formation of the organic matrix and contributes to mineralization. Disruptions in collagen metabolism can lead to the development of both congenital and acquired bone diseases. The processes of collagen degradation are particularly significant in inflammatory conditions such as osteomyelitis, where bone resorption is increased.

Collagen is a fibrous protein that forms the basis of connective tissue in humans and animals. It is the most abundant protein in the body, making up up to 25-30% of the total protein content. The function of collagen is to provide strength and elasticity to tissues, structural support to organs, wound regeneration and healing, and is also responsible for bone mineralization.

Today, there are over 28 types of collagen, with types I, II, III, IV, and V being the most significant. Type I collagen consists of three polypeptide chains (two α1 and one α2) that form a stable triple helix. These molecules are secreted by osteoblasts, form fibrils, and then organize into larger fibers, creating a framework for the deposition of mineral salts. In inflammatory bone diseases, including acute or chronic osteomyelitis, bone tissue metabolism is disrupted, the immune response system is activated, and pro-inflammatory cytokines (IL-1β, IL-6, and TNF-α) are released in high levels, stimulating osteoclastic activity and the production of enzymes that degrade the extracellular matrix. As a result of the action of enzymes (matrix metalloproteinases (MMP-1, MMP-8, MMP-13) and cathepsins (especially cathepsin K), collagen is degraded, which is accompanied by the release of its fragments into the blood or urine. These fragments can be used as markers of the intensity of bone resorption and inflammatory activity. Among them, CTX (C-terminal telopeptide of type I collagen) is the most popular, as it is the most sensitive marker of bone destruction. NTX (N-terminal telopeptide of type I collagen)- N-terminal telopeptide - a fragment from the opposite end of the collagen molecule. DPD (degoxipyridinoline) and PYD (pyridinoline)- stable cross-links between collagen molecules, released when mature collagen is destroyed. Reflects a slow but steady bone degradation. Especially useful in long-term monitoring. β-CrossLaps (β-CTx, Beta-isomerized C-terminal telopeptide of type I collagen) is a bone resorption marker that reflects the activity of osteoclasts. It is formed during the degradation of type I collagen and enters the bloodstream when the bone matrix is destroyed.

Thus, monitoring the levels of these markers allows for non-invasive assessment of the disease phase, bone metabolism activity, and the effectiveness of treatment, making these markers an important component of personalized diagnosis and prognosis of acute hematogenous osteomyelitis in children.

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