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Review Paper

Biological Mechanisms Underlying Kyphosis: A Narrative Mini-review of Spinal Deformity Beyond Mechanics

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ABSTRACT

Kyphosis is traditionally described as excessive curvature of the thoracic spine, often attributed to structural and biomechanical abnormalities. However, emerging evidence suggests that biological and molecular mechanisms also contribute to its development and progression. This narrative mini-review explores the underlying biological factors associated with kyphosis, including alterations in vertebral growth plate, extracellular matrix remodeling, inflammatory pathways, and muscle degeneration. Understanding these mechanisms provides new insights into the pathophysiology of kyphotic deformities and may inform future therapeutic strategies.

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Introduction

Kyphosis, defined as excessive posterior curvature of the thoracic spine, is a common spinal deformity observed across different age groups, including adolescents with Scheuermann's disease and elderly individuals with degenerative spinal conditions. While the structural and biomechanical aspects of kyphosis have been extensively studied, growing attention has been directed toward the biological mechanisms that contribute to its development and progression. This shift reflects a broader understanding that spinal deformities are not solely mechanical but involve complex interactions between cellular, molecular, and tissue-level processes [1-3].

One of the key biological contributors to kyphosis is altered vertebral growth plate physiology, particularly in adolescent kyphosis. Abnormal endochondral ossification within the vertebral growth plates has been implicated in conditions, such as Scheuermann's kyphosis, in which irregular vertebral body growth leads to anterior wedging of the vertebrae [4]. Histological studies have demonstrated disorganization of chondrocyte columns, reduced proteoglycan content, and impaired matrix mineralization in affected vertebral endplates, suggesting that intrinsic defects in cartilage biology play a central role in the deformity [5, 6].

In addition to growth plate abnormalities, extracellular matrix (ECM) remodeling is critical in the progression of kyphosis. The structural integrity of vertebral bodies and intervertebral discs depends on the proper balance between matrix synthesis and degradation. Dysregulation of this balance, mediated by increased matrix metalloproteinase (MMP) activity and decreased collagen and proteoglycan synthesis, can weaken spinal structures and contribute to deformity progression [7, 8]. These changes are particularly evident in age-related kyphosis, where degenerative processes affect both bone and disc tissues.

Inflammation has also been implicated as a contributing factor in spinal deformities. Pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) can influence bone remodeling and cartilage metabolism, promoting catabolic processes within the spine [9, 10]. Chronic low-grade inflammation may alter the balance between osteoblast and osteoclast activity, leading to changes in vertebral bone density and structure that predispose individuals to kyphotic curvature. Furthermore, inflammatory pathways may interact with mechanical stress to exacerbate tissue degeneration.

Another important but often overlooked aspect of kyphosis is the role of paraspinal muscle degeneration. Muscle tissue surrounding the spine contributes significantly to postural stability and load distribution. In both adolescent and age-related kyphosis, fatty infiltration, muscle atrophy, and reduced regenerative capacity have been observed in paraspinal muscles [11, 12]. These changes may be linked to alterations in muscle stem cell function, mitochondrial dysfunction, and increased oxidative stress, all of which impair muscle performance and contribute to the progression of spinal curvature.

Recent advances in molecular biology have also highlighted the role of genetic and epigenetic factors in kyphosis. Variations in genes involved in cartilage formation, bone metabolism, and ECM organization have been associated with susceptibility to spinal deformities [13, 14]. Additionally, epigenetic mechanisms such as DNA methylation and microRNA regulation may influence gene expression patterns in spinal tissues, further contributing to disease development. Although research in this area remains limited, these findings suggest that kyphosis may have a significant biological basis beyond mechanical factors.

The interplay between mechanical loading and biological responses is central to the kyphosis pathogenesis. Abnormal spinal loading can alter cellular signaling pathways, including mechanotransduction mechanisms that regulate chondrocyte and osteoblast activity [2]. This interaction creates a feedback loop in which mechanical stress induces biological changes that further weaken spinal structures, ultimately exacerbating the deformity.

Overall, kyphosis should be understood as a multifactorial condition involving not only structural abnormalities but also complex biological processes, including growth plate dysfunction, ECM degradation, inflammation, and muscle degeneration. This integrated perspective may open new avenues for therapeutic interventions that target underlying biological pathways rather than solely correcting spinal alignment.

Conclusion

Kyphosis is a complex spinal deformity with significant biological underpinnings that extend beyond traditional mechanical explanations. Alterations in vertebral growth, ECM homeostasis, inflammatory signaling, and muscle integrity contribute to its development and progression. Advancing our understanding of these biological mechanisms may facilitate the development of novel therapeutic strategies to modify disease progression and improve clinical outcomes.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Authors' contributions

Conceptualization, investigation, writing the original draft preparation,visualization, and supervision: Alireza Shakeri Sefat; Project administration, funding acquisition, review and editing: All authors.

Conflict of interest

The authors declared no conflict of interest.

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