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Perspective

In Joint Homeostasis, Osteocyte Dysfunction and Osteoarthritis

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INTRODUCTION

Osteoarthritis (OA), the most common type of arthritic illness, affects the load-bearing joints, such as the knee and hip. It's also known to be a leading cause of joint pain and dysfunction in the elderly, contributing to a lower quality of life. OA can impact articular cartilage, subchondral bone, and synovium, to name a few tissues. On plain x-ray, sclerosis, cyst, and osteophyte formation, as well as Bone Marrow Lesions (BMLs) on magnetic resonance imaging, are all radiological hallmarks of osteoarthritic subchondral bone that have been proven to highlight bone mineralization irregularities. Sclerosis and the formation of osteophytes are hypothesised to be caused by increased bone turnover with an increase in osteoblastic over osteoclastic activities.

Signaling anomalies in sclerostin, periostin, and Dentin Matrix Protein 1 (DMP-1) are hypothesised to be associated to BMLs and sclerosis. Meanwhile, cysts surrounded by less mineralized bone and osteoid growth uncoupled from mineralization may indicate that Wnt/catenin signalling and the OPG/RANKL/RANK pathway govern osteoblasts and osteoclasts differently depending on where they are located.

Bone cells regulate and precisely coordinate resorption and formation during the bone remodelling process to maintain the balance of adult bone metabolism. The cause of morphological and functional alterations in osteoarthritic bones is assumed to be primary osteoblastic dysfunction. The responses of osteoarthritic osteoblasts to cytokine stimulation were reduced, and the mineral-to-collagen ratio was aberrant. Meanwhile, OA is thought to be caused by abnormal osteocyte function. Differentiation of osteoblasts and osteoclasts Osteocytes, which make up 90-95 percent of bone cells, do mechano-transduction. Mechanical stimulation such as microcracks have been observed to cause osteoclasts to perish. Bone remodelling begins with the activation of osteoclastogenesis and bone resorption after osteocytes die. Osteoblast differentiation is regulated by the synthesis of sclerostin by osteoocytes. By blocking Wnt signalling, Sclerostin decreases osteoblastic activity and terminates the cycle of bone remodelling. By functioning as a fundamental regulator of bone remodelling, osteocytes appear to play a critical role in maintaining bone homeostasis and integrity. The role of subchondral bone in the progression of OA is well understood. Subchondral bone abnormalities are defined by abnormal bone form at the tissue level, irregular osteocyte activities at the cellular level, and altered protein expressions at the molecular level. The Wnt/- catenin pathway, which is critical for osteogenesis and bone remodelling, is prevalent in osteoarthritic subchondral bones. Sclerostin, a want inhibitor, is found in calcified cartilage and subchondral bone. Its absence is usually connected to the onset of OA, most likely as a result of Wnt signalling activation.

Subchondral Bone Marrow Lesions (BMLs) are sclerotic bones with decreased mineralization that are hypothesised to predict the progression of OA. According to our findings, the inorganic concentration of bone plugs from knee OA specimens was also significantly reduced. BMLs were confirmed by the presence of osteoid growth surrounding trabeculae or in the marrow cavity of OA bone. Abnormal mineralization is most likely associated to DMP-1. DMP-1 is responsible for the mineralization of collagen content since it is essential for mineral nucleation. Osteoarthritis should be treated on an individual basis, with a combination of treatment approaches most likely. The treatment should be adjusted based on the reaction. Unfortunately, almost all of the treatments that have been tested and employed are either medicines or surgery, or both. In a recent meta-analysis, for example, 60% of trials looked at the effect of medication treatment and 26% looked at surgical techniques. The absence of research examining rehabilitation treatments, such as bracing and other self-management strategies, has been dubbed "research agenda bias," and is partly due to lucrative drug development potential. Osteoarthritis should be treated on an individual basis, with a combination of treatment approaches most likely. The treatment should be adjusted based on the reaction. Unfortunately, almost all of the treatments that have been tested and employed are either medicines or surgery, or both. In a recent meta-analysis, for example, 60% of trials looked at the effect of medication treatment and 26% looked at surgical techniques. The absence of research examining rehabilitation treatments, such as bracing and other self-management strategies, has been dubbed "research agenda bias," and is partly due to lucrative drug development potential. Osteoarthritis is caused by a combination of abnormal local mechanical forces and systemic vulnerability. Increased age, female sex, and perhaps dietary deficits are all systemic variables that raise the joint's vulnerability to osteoarthritis. While epidemiological studies have revealed a significant polygenic genetic component to risk, the genes responsible have yet to be identified. Local mechanical variables including misalignment, muscular weakness, or changes in the structural integrity of the joint environment (such as meniscal injury) help the condition progress in persons who are at risk. Obesity and joint damage, both of which can raise the risk of developing or progressing osteoarthritis, can have an impact on loading.

REFERENCES

1. Callahan CM, Drake BG, Heck DA, Dittus RS. Patient outcomes following tricompartmental total knee replacement: a meta-analysis. Jama. 1994;271(17):1349-1357.
2. Nuki G. Osteoarthritis: a problem of joint failure. Zeitschrift für Rheumatologie. 1999;58(3):142-147.
3. Peach CA, Carr AJ, Loughlin J. Recent advances in the genetic investigation of osteoarthritis. Trends Mol Med 2005;11:186-191.

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