LUMEN ET VIRTUS

Osteoarthritis: Evolutionary consequence?

https://doi.org/10.56238/levv15n39-128

Milena Alves Santana[1], Maria Luísa Ginuino Carvalho[2], Isabella Schettine Quintela[3], Vivaldo Palma Lima Filho[4], Ana Flávia Oliveira Ribeiro[5], Karllos Adryano Priscinotte Rodrigues Lima[6], Pedro Pereira Barbosa[7], Adeni Ferreira dos Santos Junior[8], Tássia Rita Uchiyama Dinelli

ABSTRACT

Osteoarthritis is a degenerative disease that affects the joints and is a multifactorial disease. This article aims to analyze its evolutionary consequences and the factors that influence the phenotypic diversity of the disease. Phenotypic variation among living things is crucial to enable natural selection, but this variation needs to be limited to avoid the collapse of the vital functions of the organic system. Skeletal tissue cell types have their origins in aquatic animals and have maintained their characteristics and signaling pathways during the transition to terrestrial life, with conserved epigenetic modifications. Phenotypic diversity in the osteochondral unit occurs due to processes of ontogeny, remodeling, phenotypic variation, adaptation, and environmental sensitivity. These processes involve internal and external stressors, orderly transitions of cell development, changes in the timing of development, rapid adaptation, and epigenetic modifications.

Keywords: Osteoarthritis, Bone Lesions, Evolution.

[1] Graduated in Medicine
University Center of Várzea Grande (UNIVAG)
E-mail: mylenasantana2016@hotmail.com
[2] Medical Student
UNICEPLAC
E-mail: maria.carvalho@medicina.uniceplac.edu.br
[3] Graduated in Medicine
Vale do Rio Doce University - UNIVALE
Email: isaschettine@gmail.com
[4] Graduated in Medicine
José do Rosário Vellano University - UNIFENAS - BH
E-mail: dinho1408@hotmail.com
[5] Medical Student
UNIFENAS BH
E-mail: nanaflaviaribeiro@hotmail.com
[6] Medical Resident in Orthopedics and Traumatology at the Orthopedic Institute of Goiânia (IOG)
Orthopedic Institute of Goiânia (IOG)
Email: karllospriscinotte@hotmail.com
[7] Medical Resident in Orthopedics and Traumatology at the Orthopedic Institute of Goiânia (IOG)
Orthopedic Institute of Goiânia (IOG)
E-mail: pedropb14@icloud.com
[8] Doctor in Orthopedics and Traumatology
E-mail: ferreiraadeni@gmail.com

INTRODUCTION

Osteoarthritis is a multifactorial degenerative condition that affects the joints, its pathophysiological basis is based on interactions of microenvironments in the osteochondral unit, basically the stimulation of the mechanoreceptors trigger the release of inflammatory cytokines that lead to the degradation of the extracellular matrix. During the process of ontogenesis, after the stressor stimulus and the response to it, there is the process of adaptation to maintain functionality. Phenotypic diversity occurs due to various remodeling or adaptation processes that are influenced by orderly cell cycle transitions, epigenetic modifications and adaptation development, i.e., the adaptive response of the osteochondral unit can be either by the environment or by genetic mutations or by evolutionary lineage.

Osteoarthritis is characterized by the complex interaction between several microenvironments in the osteochondral unit. In this context, articular cartilage and its extracellular matrix play a key role in triggering this process. The phenotypic diversity observed in the osteochondral unit is the result of the interaction of several factors, including ontogeny, remodeling, phenotypic variation, adaptation, and environmental influence. These processes are driven by both internal factors, such as biomechanical stresses, and external factors. In addition, coordinated transitions during cell development, along with temporal changes, contribute to the complexity of this process. The ability to quickly adapt also plays a crucial role, allowing adjustments in response to changing conditions.

It is important to note that epigenetic modifications are also involved in this scenario. They play a significant role in regulating gene expression and responding to stimuli from the environment. Therefore, the pathophysiology of osteoarthritis is an interaction of several factors, which culminates in the dysfunction of the osteochondral unit and the development of the disease.

METHODOLOGY

This is a literature review, whose databases were taken from the SciELO and PubMed data platforms. The research period was from July 2023, meeting the inclusion criteria, which were articles from the years 2000 to 2023, in Portuguese and English, online texts, and in full texts. The following health descriptors (DeCS) were used as strategies for better evaluation of the texts: "Osteoarthritis", "Evolution" and "Evolutionary diseases".

DISCUSSION

Osteoarthritis is a degenerative disease that affects the joints and is caused by several factors. Some of these factors are non-modifiable, such as aging and genetics, while others are modifiable, such as obesity, repetitive mechanical stress, and muscle imbalances.[1] Skeletal tissue cell types have their origins in aquatic animals and have retained their characteristics and signaling pathways during

the transition to terrestrial life, with conserved epigenetic modifications.[4] Phenotypic variation among living things is crucial to enable natural selection, but this variation needs to be limited to avoid the collapse of the vital functions of the organic system.1

The pathophysiology of osteoarthritis involves the interaction of different microenvironments in the osteochondral unit, with articular cartilage and its extracellular matrix playing an important role in the process.[1] Mechanoreceptors, such as integrins, respond to mechanical stress and trigger a signaling cascade that leads to the production of inflammatory and catabolic cytokines.[2] This results in degradation of the extracellular matrix and imbalance between the processes of However, proper balance, with a moderate and intermittent load, is necessary for cartilage health and homeostasis.[1]

The vertebral joint has the evolutionary model called osteochondral unit, and the morphogenesis of this unit includes the same mesenchymal stem cells that give rise to chondrocytes in articular cartilage and chondrocytes in the growth plate.[4] Next, a process of controlled differentiation and phenotypic expression occurs at specific moments of development, this process is called ontogenesis.[4] During ontogeny, all bone structures are subject to mechanical stress, and microscopic and macroscopic responses to the stressor mechanism lead to an adaptive remodeling process to maintain bone and joint function.[4] The microecological environment of the osteochondral unit is influenced by several factors: nutrition, mechanical load, oxygen availability, temperature, hydration, and spontaneous genetic mutation.[5] The stability of the phenotype of a fluctuating micro or macroenvironment is called channeling, which is an attribute conserved in all organisms.[5]

There are 3 epigenetic mechanisms that participate in joint health and disease, namely: DNA methylation, histone modification, and non-coding RNA.[1] Over time, vertebrates have acquired new families of miRNAs, enabling morphological novelties.[1] miRNAs perform channeling through incomplete bonds to the target mRNA, limiting translation to a specific threshold.[1]

Phenotypic diversity in the osteochondral unit occurs due to processes of ontogeny, remodeling, phenotypic variation, adaptation, and environmental sensitivity.[9] These processes involve internal and external stressors, orderly transitions of cell development, changes in the timing of development, rapid adaptation, and epigenetic modifications.[9] The osteochondral unit can adapt and remodel in response to environmental stimuli, without relying solely on genetic mutations or the evolution of new signaling pathways over time.[9]

FINAL CONSIDERATIONS

Thus, it is important to understand that osteoarthritis has an evolutionary history, with osteochondral adaptation and remodeling, resulting from evolutions of signaling pathways, epigenetics, and environmental stimuli. However, even though phenotypic variation is important throughout natural selection, there must also be a phenotypic conservation inherent to evolution to

prevent the organism from collapsing. Therefore, it is possible to analyze the evolution of osteoarthritis throughout history, but with characteristics inherent to time.

REFERENCES

Ratneswaran A, Kapoor M. Osteoarthritis year in review: genetics, genomics, epigenetics. Osteoarthritis Cartilage. 2021 Feb;29(2):151-160. doi: 10.1016/j.joca.2020.11.003. Epub 2020 Nov 21. PMID: 33227439.

Alves, Stephane Mirella Costa et al. Emplastro de lidocaína para tratamento da dor na osteoartrite de joelho: revisão sistemática: Lidocaine emplaster for treatment of pain in knee osteoarthritis: systematic review. Brazilian Journal of Health Review, v. 5, n. 6, p. 24552-24564, 2022.

DOS SANTOS, Jean Douglas Moura et al. Effectiveness of therapeutic ultrasound in the treatment of pain and return to function in knee osteoarthritis-an integrative review Efetividade do ultrassom terapêutico no tratamento da dor e retorno da função na osteoartrite de joelho-uma revisão integrativa. Brazilian Journal of Health Review, v. 5, n. 3, p. 9281-9296, 2022.

Hutton CW. Generalised osteoarthritis: an evolutionary problem? Lancet. 1987 Jun 27;1(8548):1463-5. doi: 10.1016/s0140-6736(87)92209-4. PMID: 2885455.

Tangredi BP, Lawler DF. Osteoarthritis from evolutionary and mechanistic perspectives. Anat Rec (Hoboken). 2020 Dec;303(12):2967-2976. doi: 10.1002/ar.24339. Epub 2019 Dec 27. PMID: 31854144.

Im GI, Choi YJ. Epigenetics in osteoarthritis and its implication for future therapeutics. Expert Opin Biol Ther. 2013 May;13(5):713-21. doi: 10.1517/14712598.2013.764410. Epub 2013 Feb 15. PMID: 23410522.

Berenbaum F, Wallace IJ, Lieberman DE, Felson DT. Modern-day environmental factors in the pathogenesis of osteoarthritis. Nat Rev Rheumatol. 2018 Nov;14(11):674-681. doi: 10.1038/s41584-018-0073-x. PMID: 30209413.

Goldring SR, Goldring MB. Changes in the osteochondral unit during osteoarthritis: structure, function and cartilage-bone crosstalk. Nat Rev Rheumatol. 2016 Nov;12(11):632-644. doi: 10.1038/nrrheum.2016.148. Epub 2016 Sep 22. PMID: 27652499.

van der Kraan PM, van den Berg WB. Osteoarthritis in the context of ageing and evolution. Loss of chondrocyte differentiation block during ageing. Ageing Res Rev. 2008 Apr;7(2):106-13. doi: 10.1016/j.arr.2007.10.001. Epub 2007 Oct 23. PMID: 18054526.

Fernández-Moreno M, Rego I, Blanco FJ. Genética en la osteoartritis [Genetics in osteoarthritis]. Reumatol Clin. 2007 Oct;3 Suppl 3:S13-8. Spanish. doi: 10.1016/S1699-258X(07)73649-5. Epub 2008 Nov 13. PMID: 21794474.

Sulzbacher I. Arthrose--Histologie und pathogenetische Ansätze [Arthrosis--histology and pathogenetic approaches]. Radiologe. 2000 Dec;40(12):1126-33. German. doi: 10.1007/s001170050896. PMID: 11197930.

Geyer M, Schönfeld C. Novel Insights into the Pathogenesis of Osteoarthritis. Curr Rheumatol Rev. 2018;14(2):98-107. doi: 10.2174/1573397113666170807122312. PMID: 28782470.

King LK. Osteoarthritis and comorbidity: time for action. Osteoarthritis Cartilage. 2023 Apr;31(4):423-424. doi: 10.1016/j.joca.2023.01.007. Epub 2023 Jan 21. PMID: 36693559.