OSTEOPHYTES DEVELOPMENT DURING OA - CONSIDERATION ANGIOGENESIS, MECHANICAL LOADING AND TISSUE MICROSTRUCTURE

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1. Introduction

In this paper the effects of mechanical loading, blood vessels development and tissue microstructure in degenerative joint disease (OA) is discussed. Osteoarthritis is one of the most common diseases that has become social as well as health issues. It predominantly affects the elderly but also sportsmen, obese people and those with curvature of the spine. It is a social issue. Although the issues is not yet fully understood mechanical aspects are crucial in the evolution of osteoarthritis [1, 2]. Mechanical overloading leads to chondrocytes apoptosis which increases generation of vascular endothelial growth factors [3]. Properly formulated mathematical model of cartilage degeneration and osteophytes development can significantly help to comprehend complexity of this process. Presented model reflects the most important aspects of the interaction between mechanical and biological factors crucial for osteoarthritis phenomenon.

2. Mathematical formulation

Assumptions:
• Porosity and permeability determine angiogenesis and osteogenesis process [4].
• Number of cells depends on the consumption of nutrients and the interactions between cells [5].
• Nutrients variations depend on consumption by cells and nutrients supply from capillaries.
• Blood vessels can grow only from pre-existing vascular network and angiogenic factors have biological and mechanical character.
• Stiffness of tissue depends on the numbers of bone cells and density of elastic strain energy.

System of equations:

Variation of bone cells number

$$\frac{\partial C(\mathbf{x},t)}{\partial t} = \eta(\mathbf{x},t)C(\mathbf{x},t) - \tau C^2(\mathbf{x},t) \quad , \tag{1}$$

Variation of nutrients concentration

$$\frac{\partial N(\mathbf{x},t)}{\partial t} = -a\eta(\mathbf{x},t)C(\mathbf{x},t) + f\int\limits_{\Omega} B(\boldsymbol{\zeta},t)e^{-\alpha r}d\Omega \quad , \tag{2}$$

Variation of blood vessels concentration

$$\frac{\partial B(\mathbf{x},t)}{\partial t} = b\int\limits_{\Omega} B(\boldsymbol{\zeta},t)e^{-\gamma r}d\Omega \int\limits_{\Omega} \Delta N(\boldsymbol{\zeta},t)e^{-\beta r}d\Omega + d\int\limits_{\Omega} B(\boldsymbol{\zeta},t)e^{-\vartheta r}d\Omega \int\limits_{\Omega} W_B(\boldsymbol{\zeta},t)e^{-\xi r}d\Omega \quad , \tag{3}$$

Variation of Young's modulus of tissue

$$\frac{\partial E(\mathbf{x},t)}{\partial t} = C(\mathbf{x},t) W_E(\mathbf{x},t) \quad , \tag{4}$$

3. Numerical example

The simple model of an interface between a bone tissue and a cartilage in a joint has been analysed in this example to follow the changes of blood vessels distribution, bone tissue formation and cartilage deterioration triggered by non-uniform mechanical load caused by imperfections of joint external shape. In Fig.1a the thick layer represents the domain occupied by a bone tissue. The thinner one represents cartilage. Due to imperfections in a joint in addition to the uniform pressure q this cartilage is loaded by additional horizontally oriented forces P over the two small sub-domains of the external surface. Bone and cartilage are perfectly connected to each other and the domain occupied by a bone tissue is supported at the left hand side edge. In Fig. 1b the selected results of numerical simulation are presented.

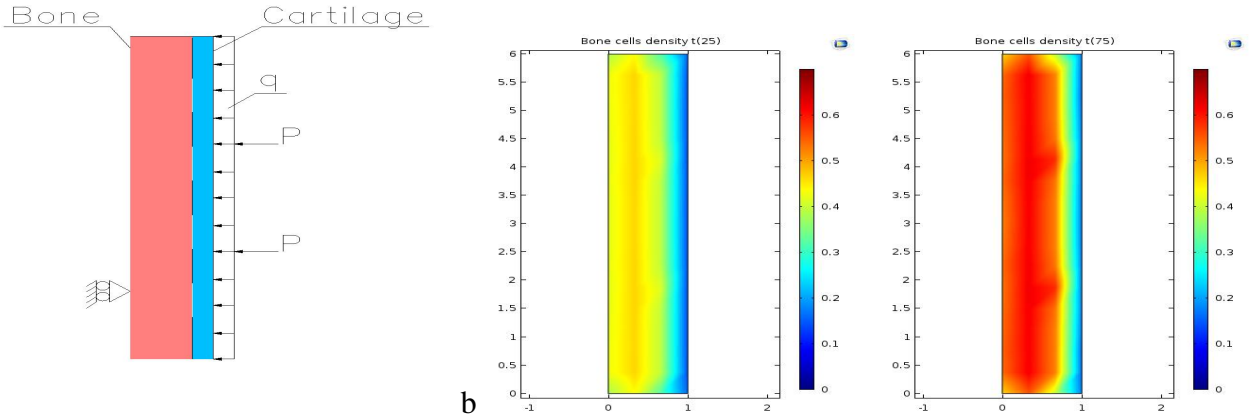

Figure 1. Considered schematic geometry and visualization of osteophytes development.

4. Conclusion

Presented calculations show alignment of numerical calculation results with available animal studies. Results confirm that correct assumptions were made. Blood vessels growth - angiogenesis initiates osteophytes development. Properties of microstructure determine rate and direction of joint structure modification. Nevertheless, non-uniform mechanical loading is the major factor in osteoarthitis disease.

5. References

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