

THE ROLE OF THE BONE STRENGTH ON THE CYST GROWTH IN THE MANDIBLE

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1. Introduction

Regarding the etiology of bone cysts, different theories have been proposed in order to describe the growth mechanism. One of them states that pressurized fluid enters the bone through a rupture and fractures trabeculae, thereby causing an area of disappearance of bone tissue (osteolysis). Although there is not much evidence indicating that fluid collecting inside the cyst fractures trabeculae, loading conditions of the surrounding bone may indeed cause decreasing of bone density and therefore bone loss. This may be described by a mechanoregulated bone adaptation theory [6]. According to another theory, pressurized fluid may decrease perfusion and oxygen supply for tissue, thereby leading to osteocytes death and further osteolysis. The basic assumption for both hypotheses is that pressurized fluid plays a crucial role in the development of bone cysts. Intracystic pressure may change during the cyst growth, as it is regulated by various factors such as osmotic tension of the fluid, the elasticity of the cyst wall, the permeability and the blood pressure of the capillaries in the cystic wall [2]. In this study we use a computational bone remodelling model to evaluate load conditions during the cyst growth.

2. Method

The computational model is linked to the remodelling model [4,5] that describes the change in the bone density in relation to remodelling stimulus based on the strain energy density as well as mass density of a tissue.

$$\frac{d\rho}{dt} = \begin{cases} B(\psi - K_{\min}), & \psi < K_{\min} \\ 0, & K_{\min} \leq \psi \leq K_{\max} \\ D(\psi - K_{\text{over}})^2 + K_w, & \psi > K_{\max} \end{cases}$$

We mainly focus on the situation when stimulus exceeds the threshold value K_d indicating bone loss due to overload. We use the finite element model (Fig.1) to predict cyst growth and bone architecture changes in response to pressure induced by the presence of the fluid. The model consists of two materials of different properties, namely inner trabecular and outer cortical bone assumed as linear, isotropic and homogenous. It is noted that there are various relations between density and bone Young's modulus E available in the literature. In this paper, following equation is adopted: $E = c \rho^3$ [3], where c is a constant and equals $3790 \text{ [MPa} \cdot \text{cm}^9/\text{g}^3]$. At each element of the FE model the value of the stimulus is calculated as a strain energy density divided by bone mass density. If the stimulus exceeds threshold value, the element is removed from the mesh.

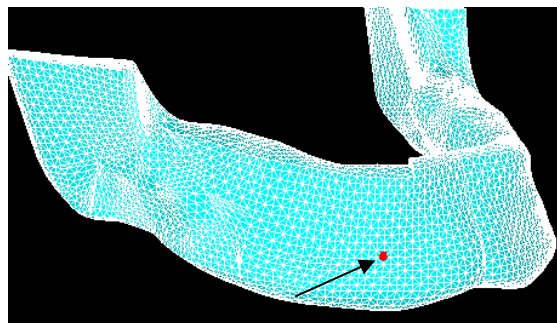


Fig.1. Finite element model of the mandible with the cyst.

With this volume change the boundary load conditions are updated to spread the pressure action over newly created areas. The surface of the cyst is loaded statically with normal pressure [1]. This resorption initiating value depends strongly on the bone elastic modulus. The stronger the bone is, the higher value of the pressure is necessary to start the process of cyst growth strictly connected with bone resorption. The choice of the applied load is not straightforward, since reported cancellous bone density and thus Young's modulus values cover a wide range. However, in this study the exact applied load values are not that important since, the results are evaluated qualitatively and not quantitatively.

3. Results

Bone resorption in response to pressure inside the cyst leads to growth of the cavity, which takes an irregular shape (Fig.2). The cyst growth and its shape depend on the Young modulus of the surrounding bone. After the initial stage, decreasing pressure maintains further expansion of the cyst in the mandible. However, the regulatory mechanisms of intracystic pressure and growth of cyst under lower pressure are unclear and remain speculative. Further studies are required to clarify them.

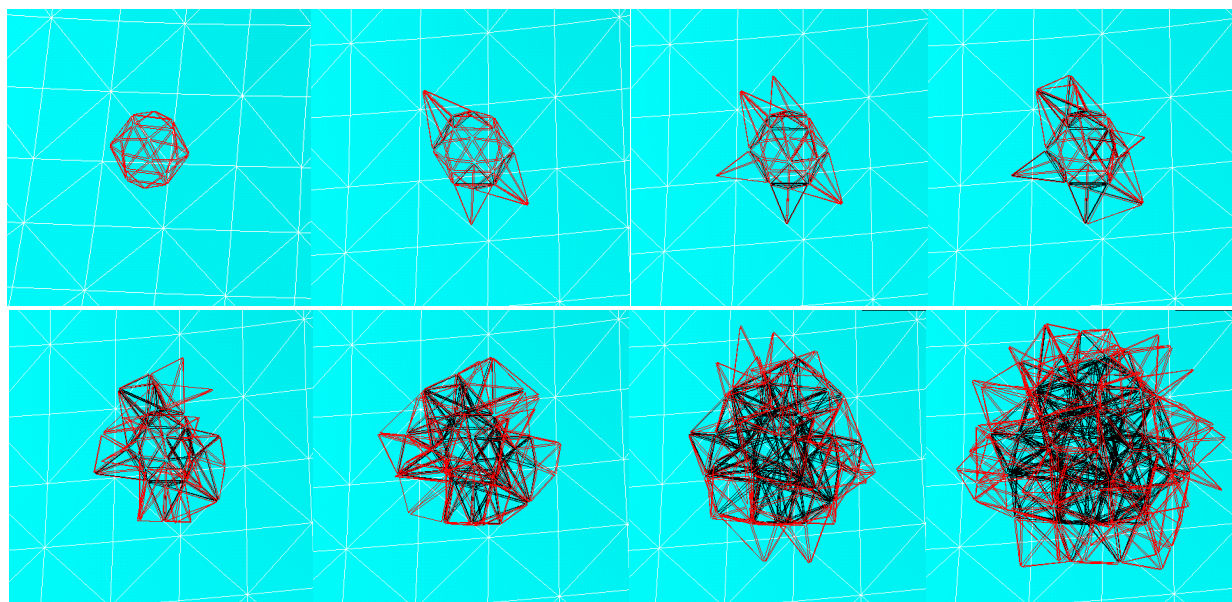


Fig.2. Subsequent stages of the cyst growth.

4. References

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