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Effect of the size and location of osteochondral defects in degenerative arthritis. A finite element simulation

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Abstract

Physiological studies have shown that focal articular surface defects in the human knee may progress to degenerative arthritis. Although the risk of this evolutive process is multifactorial, defect size is one of the most important factors. In order to determine the influence of osteochondral defect size and location on the stress and strain concentrations around the defect rim, a finite element model of the human knee was developed. From our results, it became clear that the size and location of cartilage defects drastically affect to those variables. No stress concentration appeared around the rim of small defects, being the stress distribution mainly controlled by the meniscus contact. On the contrary, important rim stress concentration was found for large osteochondral defects. This alteration of the stress distribution has important clinical implications regarding the long-term integrity of the cartilage adjacent to osteochondral defects.

Keywords: Human knee; Osteochondral defects; Cartilage damage; Defect size; Defect location; Finite element method

1. Introduction

Damage in articular cartilage is a problem that affects millions of people in the world [1]. An important cause of lesions or defects in articular cartilage, besides osteoarthrosis and osteochondrosis dissecans, is trauma. It plays a significant role, especially when dealing with young and active patients [2]. When the injury does not penetrate subchondral bone, biological healing is poor and leads to long-term degeneration of articular cartilage. Although recent studies have shown that focal chondral and osteochondral defects progress to osteoarthritis, this claim is difficult to prove in a prospective fashion, likely because of the inability to accurately diagnose articular surface injuries by noninvasive techniques and to follow them over long periods [3]. In addition, lesion progression is multifactorial. Lesion characteristics such as size, depth, location or patient depending factors such as age, level of activity, obesity and genetics strongly affect that progression [4].

There exist different approaches to surgically treat cartilage damage. Small defects ($< 1\,cm^2$) can be treated with a high ratio of success through drilling and stimulation techniques, while for larger lesions with important influence on the joint mechanics, transplantation techniques like osteochondral transplantations are considered the gold standard [1,2]. In young patients, small defects are often treated using marrow-stimulation techniques. In these procedures, the torn or compromised cartilage is removed to expose the underlying bone [5]. Clinical results suggest that the relative success of these procedures depends on the level of strain and stress experienced by the rim of the defect and the subsequent quality of the healing tissue during the early stages of healing [6].

Although it is clear the relation between the state of compressive and shear stresses within and around the chondral defect and its size and location, the relationship between defect size and mechanically induced degeneration due to local stress concentration has not been well documented. In animal studies, Huber-Betzer et al. [7] showed that strong gradients of contact pressure cause elevated shear stresses along the depth of the different cartilage layers. These two effects: high contact

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pressures and shear stresses adjacent to defects, may interfere with the cartilage normal function [8–12].

The establishment of a specific treatment for a certain defect size of cartilage lesions is not usually based on any firm biomechanical data [4]. Finite element simulations can help to compute the stress distribution in the human knee joint cartilage, and better understand the pathological degeneration produced in this articular joint by different defects. Three-dimensional finite element models can also be used to estimate the consequences of surgical treatments such as marrow-stimulation techniques. Some computational models of the human knee have already been constructed to predict its biomechanical behaviour. [13–20] computed joint contact stresses and stress distribution on the human healthy or meniscectomized knee joint under different conditions. To our knowledge, only Weiss et al. [6] considered the effect of cartilage defects. They developed a finite element model of the medial compartment of the knee to examine the effect of defect size on defect rim strains. They studied two healing defect sizes: 1.0 and 2.0 cm of diameter, on the medial condyle and at one location. They did not analyze the initial process nor considered the intermediate state corresponding to fully healed cartilage. Mansour [21] and Duda et al. [22] developed axisymmetrical finite element models of cartilage defects to investigate the influence of mechanical conditions on defect healing.

The main objective of the present study was to estimate contact pressures and stress distribution in the defect rim to investigate the effect of defect size and location on articular cartilage deformation. We used a finite element model of the human knee presented in a previous paper that included femur, tibia, cartilage layers, menisci and main ligaments. We studied different defect sizes located in the medial femoral condyle in low- and high-weight-bearing areas with and without meniscus. Also, we compared two different steps in the healing process, before cartilage regeneration and after partial healing of the defect.

2. Methods

2.1. Finite element model of the human knee joint

The finite element model used here was introduced in previous papers [19,23,24]. The geometrical data were obtained from magnetic resonance images (MRI) acquisition. The image block consisted of parallel digital images separated at intervals of 1.5 mm in the sagittal, coronal and axial planes. The procedure to obtain the geometrical model was described in detail in these references. With this geometry, a three-dimensional finite element model of the human tibio-femoral joint including femur, tibia, cartilage layers, menisci and principal ligaments was developed (Fig. 1). The general-purpose finite element code ABAQUS v.6.2 [25] was used to solve the associated stress analysis problem.

2.2. Model of cartilage defects

Four defect sizes were compared with respective areas of: 0.19, 0.78, 1.76 and 3.14 cm^2 (Fig. 2). Two different cases were studied: without any cartilage regeneration and after partial healing of the defects. In the first case, defects were sufficiently deep to fully penetrate subchondral bone while in the second situation, defects were filled with healed cartilage [6].

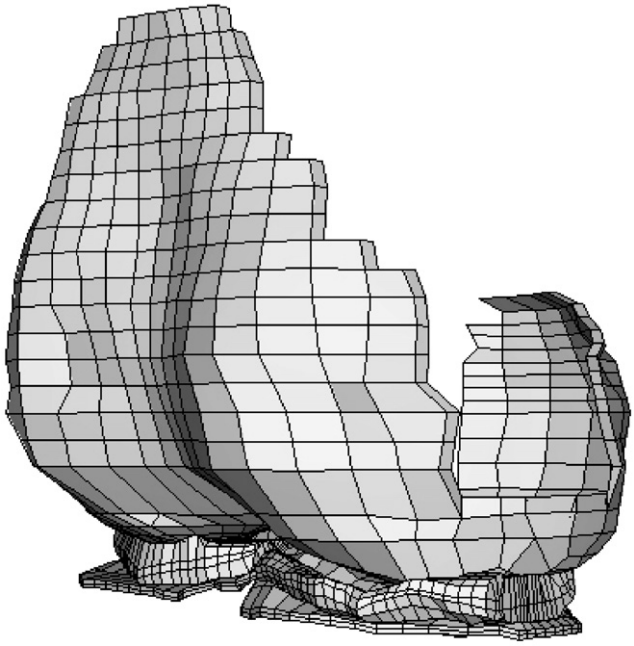

Fig. 1. Finite element model of the femorotibial contact region.

Two locations were considered, respectively, in low- and high-weight-bearing areas of the medial femoral cartilage at full extension, see Figs. 2 and 3, that corresponds to the most usually damaged condyle [26]. Analyses were carried out with and without the medial meniscus.

2.3. Material behaviour, loading and boundary conditions

The bony portions of the distal femur and proximal tibia were considered as fully rigid, due to their much higher stiffness with respect to the significant soft tissues. Considering that the loading time of interest corresponded to that of a single leg stance, and the viscoelastic time constant of cartilage approaches 1500 s [27], articular cartilages were modelled as linear elastic and isotropic with an elastic modulus of $E = 5$ MPa for the healthy cartilage [18] and $E = 1.5$ MPa for the healed cartilage [6] and with Poisson ratio of $\nu = 0.46$ for both, adapted from [28]. Menisci were also assumed to behave as a single-phase linear elastic and isotropic material with the following average properties: elastic modulus of $E = 20$ MPa and Poisson ratio of $\nu = 0.49$ [29]. Ligaments were modelled as non-linear hyperelastic fibered materials with their constitutive behaviour defined by a transversely isotropic strain-energy density function earlier proposed by Weiss [30] and expressed as

$$\Psi(\mathbf{C}) = \Psi_{\text{vol}}(J) + \Psi^{\text{m}}_{\text{iso}}(\bar{\mathbf{F}}) + \Psi^{\text{f}}_{\text{iso}}(\mathbf{a} \otimes \mathbf{a}), \quad (1)$$

where $\Psi^{\text{m}}_{\text{iso}}$ represents the deviatoric mechanical contribution of the tissue matrix, $\Psi^{\text{f}}_{\text{iso}}$ that of the fibers and Ψ_{vol} is a penalty function to force quasi-incompressibility, that in our case, took