Medical Engineering and Physics 37 (2015) 1125–1132

Contents lists available at ScienceDirect

Medical Engineering and Physics

journal homepage: www.elsevier.com/locate/medengphy

The formation and function of the sclerosis rim in the femoral head: A biomechanical point of view

ZhiPing Chen [a,*], Yong Xu [a], ZhenXi Qi [b], JinShui Zho [c]

[a] College of Mechanical and Electronic Engineering, Fujian Agriculture and Forestry University, Fuzhou 350002, PR China
[b] Institute of Medicine on Osteopathis, Fujian Academy of Integrative Medicine, Fuzhou 350108, PR China
[c] The Second Affiliated Hospital of Xiamen University, Fuzhou 350007, PR China

ARTICLE INFO

Article history:
Received 27 September 2014
Revised 19 August 2015
Accepted 10 September 2015

Keywords:
Finite element analysis
Bone remodeling
Sclerosis rim
Osteonecrosis of the femoral head

ABSTRACT

Sclerosis rim surrounding the necrotic area is commonly found in necrotic femoral head, but the biomechanical function of sclerosis rim has received relatively little attention. Little is known about the formation and natural history of sclerosis rim. In the present work, we assume that the necrotic change may trigger bone remodeling process in the femoral head, which took place according to Huiskes' bone remodeling model incorporated with the FE simulations as described earlier. We then investigate the function of sclerosis rim as a mechanical supporter in delaying further collapse of the femoral head based on our sclerotic rim model. The main tasks of this study are: (1) simulation of the density distribution in the necrotic femoral head after bone remodeling; (2) calculation of maximal von Mises stress in the subchondral bone of the weight-bearing area of the femoral head over the necrotic area before and after bone remodeling. Results show that the sclerotic rim is, from the biomechanical point of view, an adaptive response to the decrease in elastic modulus of the femoral head, and that the sclerotic rim that acts as a compensatory structural reinforcement can usually significantly reduce the maximal stress in the subchondral bone when the lesion is small, but not when the lesion is large.

1. Introduction

Osteonecrosis of the femoral head (ONFH) is a progressive disease leading to destruction of the hip joint [1]. There is a general agreement that the size and location of a necrotic lesion are important factors in predicting femoral head collapse [2–5]. However the role of the sclerosis rim (see Fig. 1) commonly found in necrotic femoral head in the natural history of osteonecrosis has not yet been given enough attention it deserves. A retrospective analysis of the sclerosis rim in patients with ONFH showed that the time from hip pain to joint replacement was 49 ± 11 months in patients with sclerosis rim, and 15 ± 2 months in patients without sclerosis rim; and there was a significant difference between these two groups ($P < 0.01$). This suggested that the sclerosis formation in the repair process of ONFH could delay the collapse process [6]. Lieberman showed that a sclerotic rim surrounding the necrotic bone would improve the effectiveness of core decompression for precollapse hips [7]. Bozic et al. [8] reviewed the long-term results of fifty-four hips treated with core decompression for ONFH. In their study, they used a modification of the Ficat and Arlet classification system along with Steinberg et al. classification system. They found that four of the thirteen stage-I hips, none of the seven stage-IIA sclerotic hips, thirteen of the sixteen stage-IIA cystic or sclerocystic hips, nine of the ten stage-IIB hips, and all eight stage-III hips were either radiographic failure, clinical failure, or both. Their findings suggested that core decompression was safe and effective in treating stage-I or stage-IIA sclerotic disease. They also noted the important difference between stage-IIA sclerotic disease and stage-IIA cystic or sclerocystic disease. The data available also suggested that core decompression for necrosis of the femoral head was more effective particularly for patients with small lesions and with a sclerotic rim surrounding the necrotic bone [7]. In contrast, some authors indicated that the progressive sclerosis in the femoral head might be the result of the accumulation of the necrotic lesions, while areas of the bone containing fractures might be weakened to the point of collapse [9]. Bullough and DiCarlo reported that the reason for collapse was the fracture of trabecular bone, which might occur on the necrotic side of the advancing sclerosis in the reparative front [10]. The exact mechanical mechanism of the sclerosis rim formation and its function for preventing the femoral head collapse is still unclear. Sclerosis surrounding an osteopenic area is a reactive bone remodeling at the necrotic-viable osseous junction [11]. Sclerosis represents trabecular hypertrophy

* Corresponding author. Tel.: +86 13599393878.
E-mail address: chenzhiping2008.09@163.com (Z. Chen).

http://dx.doi.org/10.1016/j.medengphy.2015.09.005
1350-4533/

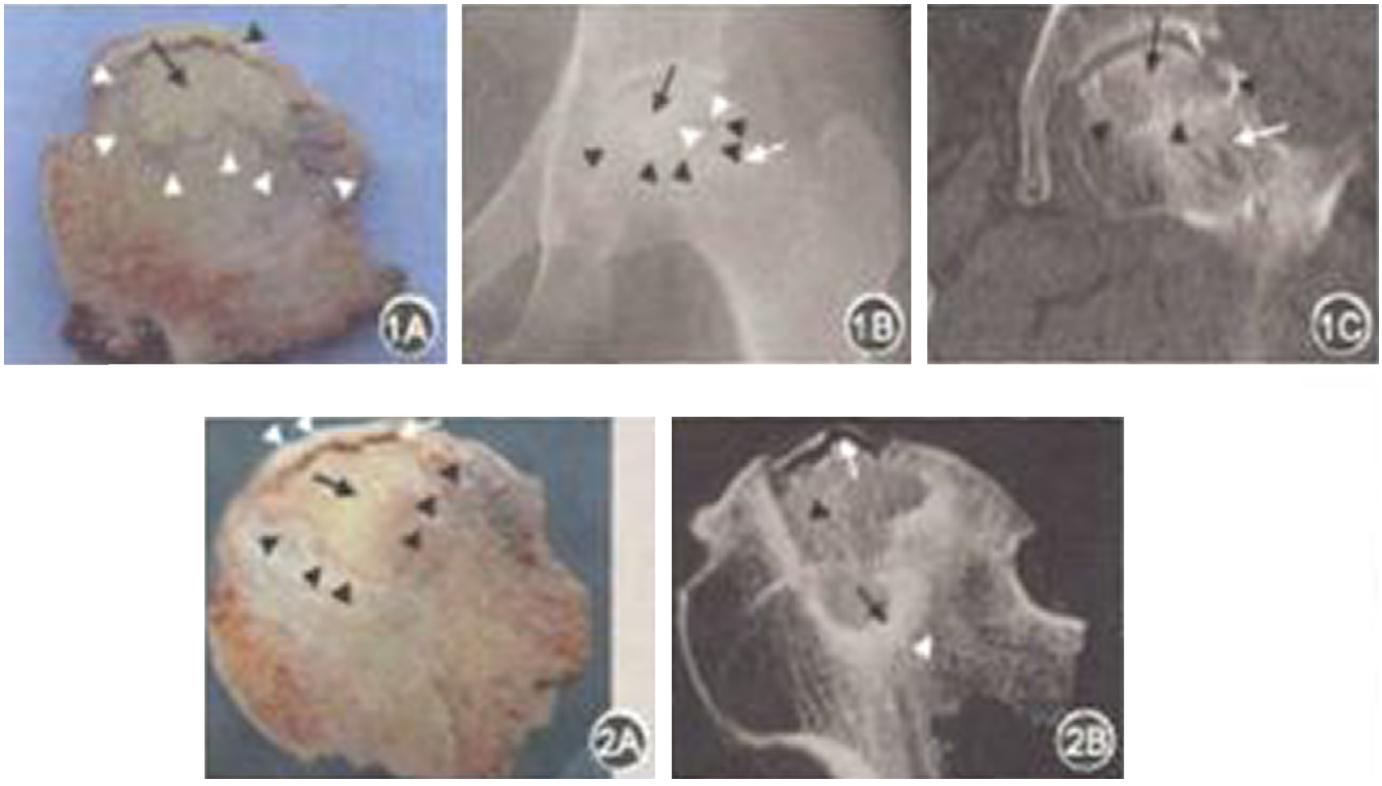

Fig. 1. Two cases of sclerosis rim [36]. In the specimen of necrotic femoral head (1A), the cartilage (short black arrow), the necrotic area (long black arrow), and the sclerosis rim (white arrow) are shown for patient one. In the antero-posterior radiograph of hip (1B) and coronal CT (1C), the necrotic area (long black arrow) and the sclerosis rim (short black arrow) are also shown for patient one. In the specimen of necrotic femoral head (2A), the cartilage (short white arrow) and the sclerosis rim (short black arrow) are shown for patient two. In the coronal CT (2B), the necrotic area (short black arrow) and the sclerosis rim (long black arrow) are shown for patient two.

[12]. The thickened trabeculae may maintain the mechanical strength during the repair process [13,14]. Glimcher and Kenzora [15,16] suggested that dead bone might retain adequate mechanical function before the of the femoral head collapse, which was induced by the structural property changes resulting from the activity of living cells during the repair process. The femoral head collapse might be prolonged by a decrease or halt of the repair process. Based on the above findings, we assume that the biomechanical factor may play an important role in regulating the sclerosis rim formation. Further, we assume that the sclerosis rim formation follows Huiskes' adaptive bone remodeling theory. According to Wolff's law and Huiskes' theory, the sclerosis rim is a reactive zone in the femoral head for maintaining a balance between stress and bone strength, we presume that the sclerotic rim may play an important role in supporting the necrotic femoral head in a compensatory manner and preventing the femoral head collapse. The objective of this study is to determine biomechanically the changes in the trabecular bone structure in the femoral head induced by the changes in mechanical properties of the necrotic bone, and finally the relationship between the sclerotic rim and the femoral head collapse. In the present work, the sclerotic rim are established using Huiskes et al.'s bone remodeling theory in combination with FE simulations. von Mises stress distribution in the femoral head with sclerotic rim is calculated using ANSYS finite element analysis (FEA).

2. Materials and methods

2.1. The finite element model

The necrotic lesions in all three models of the necrotic femoral heads are defined as spherical lesions. The diameter of these lesions can be 15 mm, 20 mm and 30 mm, which correspond, respectively, to Steinberg stage IIA (<15% of head involvement as seen on radiograph or MRI), Steinberg stage IIB (15–30% of head involvement) and Steinberg stage IIC (>30% of head involvement) [17]. These lesions are located under the weight-bearing surface of the femoral head, with distance of 2 mm between the upper margin of lesion and the lower margin of articular cartilage of the femoral head (see Fig. 2). The CT images (slice: 1.0 mm; Light Speed VCT (General Electric, Milwaukee, WI, USA)) used for building the solid 3-D FE model of the proximal femur were obtained from a male cadaver who was not having any bone-related diseases (informed consent was obtained from relatives). The femoral head neck angle of the cadaver is 130°, which is normal, and femoral head neck has neither coxa vara nor coxa valga. The CT data were then transferred to a computer to generate the models using a commercially available software package (Mimics; Materialise NV, Leuven, Belgium). The models were optimized with Materialise Magics v13 (see Fig. 3). ANSYS 12.0 (Canonsburg, PA) was used in this study. The finite element meshes used in the simulations are shown in Fig. 4. The models contained between 135,147 and 167,796 elements (190,454 and 235,652 nodes). The bone material is assumed to be isotropic and linearly elastic with a Poisson's ratio of 0.3. The simulation starts with a heterogeneous model of the femoral head. In particular, the heterogeneous mechanical properties are applied. The CT data are imported into Mimics to assign the density and elastic modulus distribution in the trabecular bone according to the relations between CT numbers, density and elastic modulus in human bone produced by Rho [18,19]. The moduli of necrotic lesions, in particular, are 0.124 GPa [20] and 0.7 GPa, respectively. The bone density distribution is updated for 47 iterations to allow convergence to a stable density distribution. The density of the cortical bone remains constant in the remodeling process. The physiologically relevant loading condition used in this study is provided by Simões et al. [21] (see Fig. 5). The simulations are performed with the distal end of the femur rigidly constrained and femoral head is horizontally constrained in constrained femoral head set-up. The models are loaded in one-legged stance. The loadings exerted on the femoral bone are