Medical Engineering and Physics 000 (2017) 1–10

Contents lists available at ScienceDirect

Medical Engineering and Physics

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

The impact of subchondral bone cysts on local bone stresses in the medial femoral condyle of the equine stifle joint☆

Lance L. Frazer[a], Elizabeth M. Santschi[b], Kenneth J. Fischer[a,c,*]

[a] Bioengineering Graduate Program, University of Kansas, Lawrence, KS, USA
[b] College of Veterinary Medicine, Department of Clinical Sciences, Kansas State University, Manhattan, KS, USA
[c] Department of Mechanical Engineering, University of Kansas, Lawrence, KS, USA

ARTICLE INFO

Article history:
Received 30 November 2016
Revised 6 June 2017
Accepted 14 June 2017
Available online xxx

Keywords:
Finite element analysis
Bone mechanics
Subchondral lucency
Subchondral lesion
Knee

ABSTRACT

Subchondral lucency (SCL), also referred to as subchondral bone cysts, can cause clinical problems in horses and humans. In humans, SCLs occur in youths and adolescents [1] due to mechanical factors (often related to athletics) and in skeletally mature individuals secondary to osteoarthritis (OA). In horses, SCL most commonly occurs in the medial femoral condyle (MFC) of growing horses (without OA), and causes lameness. The cause of equine SCL is debated, but bone trauma due to overload is the likely mechanism. Investigating the biomechanics of the healthy and cystic MFC is important to understand cyst growth and to provide a foundation for new treatment strategies. We hypothesize that SCL alters the mechanical environment of surrounding bone, which in the presence of continued loading, may lead to enlargement of the SCL. In this study, we developed and validated a finite element model of an equine stifle joint and investigated the stresses associated with varying sizes of SCL. We found substantial differences in tensile and shear stress at various stages of SCL development that suggest further bone damage leading to SCL enlargement. These data provide a first step in understanding of the altered mechanics of subchondral bone surrounding a SCL. Additional studies may provide the basis for improved treatment strategies for SCL in young horses, and may improve the understanding of SCL in humans.

1. Introduction

In humans, subchondral lucencies (SCLs) occur in the knee of youths and adolescents [1] due to mechanical factors (commonly related to athletics) and in skeletally mature individuals secondary to osteoarthritis (OA) [2]. SCL also occurs in young horses, most commonly in the medial femoral condyle (MFC) of the stifle joint, and causes lameness [3–6] (Fig. 1). SCL is most common in horses ≤2 years of age, but can occur in older horses. The occurrence in human youths and young horses are similar, and thus the equine stifle joint is a relevant and useful animal model.

The etiology of SCL is not well understood for horses or humans, however, trauma and osteochondrosis are most commonly implicated [3,5]. Serial radiographs of young horses that develop SCL first show trabecular bone sclerosis in the cranial MFC at the site of tibial contact in extension. The sclerotic area is typically 10–15 mm in width and depth at the joint surface and narrows as it extends 15–20 mm proximally. At the joint surface, the sclerosis is rapidly followed by flattening of the MFC and a slower progressive loss of sclerotic trabecular bone resulting in a SCL. As the void forms, the sclerosis becomes most apparent at the margins [7].

Histologic examination of SCL contents reveals fibroplasia and capillary proliferation as well as fibrous tissue, degenerated bone and cartilage and disorganized areas of granulation tissue and woven bone [8–10]. Biochemical analysis of the tissue lining the SCL removed arthroscopically reveals that inflammatory cytokines associated with bone resorption are present, and likely play a role in SCL formation and lameness [11,12].

Treatments of SCL are directed at reducing local inflammation and promoting bone and cartilage healing. Conservative therapies include reduced exercise and intra-articular injection of the femorotibial joint with corticosteroids [3,4]. Due to modest efficacy of conservative therapy, arthroscopic debridement of the SCL including the lining has been advocated as a treatment for many years [13–15]. After debridement, the resulting void was left open, or filled with cancellous bone, osteochondral grafts [16], or a combination of bone substitutes, growth factors, chondrocyte grafts and mesenchymal stem cells in fibrin glue [17]. The convalescent

☆ All authors have contributed to the design of the study, analysis of the data, and writing of this paper. Lance Frazer and Ken Fischer were responsible for generation of the finite element model and stress data. All authors have read and approved the final submitted manuscript.

* Corresponding author at: Department of Mechanical Engineering, University of Kansas, Lawrence, KS 66045-7609, USA.
E-mail address: fischer@ku.edu (K.J. Fischer).

http://dx.doi.org/10.1016/j.medengphy.2017.06.019
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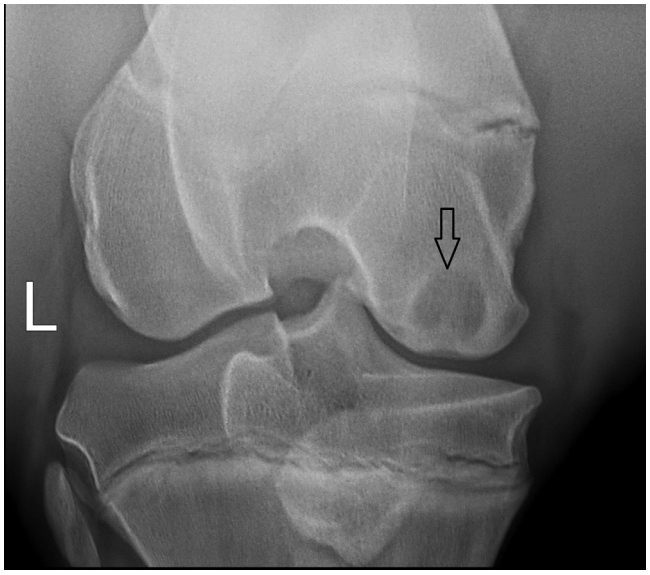

Fig. 1. Radiograph of a Grade 3 MFC SCL from a young Thoroughbred. The "L" denotes the lateral side of the stifle. The arrow indicates the proximal border of the bone cyst.

period for debridement averaged between 8 to 24 months [15]. Recently (2008), a publication indicated that injection of corticosteroids into SCL under arthroscopic control provided similar success rates as debridement and a shorter convalescence (6 months) [18]. For all therapies, clinical success rates range between 50% and 75%, with a tendency for surgery or injections to provide a somewhat better result than conservative treatment [4,6,9,13–18]. However, there is very little data about bone healing after treatment, and what is available suggests it occurs in less than 20% of patients [17].

Recently, treatment of MFC SCL using a transcondylar lag screw reported a rate at the high end of previous techniques for lameness resolution, a shorter (4 month) convalescence as compared to debridement, and consistent SCL healing [19]. The effect of the screw appears to be purely mechanical, thus, this clinical study suggests that biomechanics are important for understanding SCL development and treatment. However, very little is known about the stresses within the medial femoral condyle during cyst development.

The equine stifle is a complex biological system and direct collection of biomechanical data would be difficult, if not impossible. Computational modeling is a viable alternative to experimentation. Unfortunately, there is no direct data on an equine muscle forces and stifle joint loads in the stance phase. Though there is data available for human muscle forces and knee joint loads, prior models of human osteochondral defects have been quite simplistic in the boundary conditions [20,21]. Even so, computational biomechanics can provide a particularly powerful approach in comparative studies that examine changes in stress with different conditions [20]. Thus, the objective of this study was to evaluate and compare bone stresses in the medial femoral condyle of the normal stifle with stresses during the initiation phases and with three sizes of SCL using finite element analysis. The data can improve our understanding of stresses around SCL, allowing clinicians and engineers to develop and implement rational and effective treatment strategies.

2. Methods

The models in this study followed the radiographic progression of SCL as described clinically [19]. After creation of an anatomically accurate model of a healthy stifle joint, the model was modified to mimic stages of SCL progression and to analyze bone stresses in the stifle MFC and around SCLs. This study includes 11 models, separated into two model trees (flattened condyle and normal condyle) that represent clinically apparent aspects of SCL development.

2.1. Segmentation and meshing

Computerized tomography (CT) images were acquired from the extended stifle joint of a deceased yearling male Thoroughbred using an eight-slice helical scanner (kVp—140; filter—body; slice thickness—0.625 mm; X-ray tube current—170 mAs; exposure time—1320 s) (GE Medical Systems, Waukesha, Wisconsin, USA). Both bony and soft tissue structures were segmented from the images using ScanIP v7.0 (Simpleware, United Kingdom).

To segment soft tissues, visible boundaries in the CT scan were used, as well as anatomical information to separate adjacent soft tissues (e.g. cartilage and meniscus). It is reported that articular cartilage in the stifle joint is generally 2 mm thick [22]. Therefore, the segmented bone was dilated by 2 mm, and the original bone was subtracted from the dilated structure, creating an even articular cartilage surface around each bone (Fig. 2). The menisci could be segmented between the two articulating cartilages with visual aid from available cadaveric specimens. Ligaments were segmented by identifying the anatomical insertion points and following the corresponding tissue outlines in the CT image data.

Segmentation was guided by a professor of veterinary surgery with detailed emphasis on anatomical observations of the stifle and pathology. For instance, the cartilage near the MICET (axial) is 1.5 mm thicker than the abaxial cartilage that contacts the meniscus, and this anatomy was modeled as such (Fig. S1).

Structures included in the model were the femur, tibia, articular cartilage, menisci, patella, distal patellar ligaments, and the posterior cruciate ligament (PCL). This geometry was then discretized into 1,785,914 tetrahedral elements (C3D4) using the +FE Free meshing algorithm in ScanIP and subsequently exported into ABAQUS v6.14-2 (SIMULIA, Providence, RI) to complete and analyze the finite element model (Fig. 3). Cartilage and ligament structures shared nodes with bone, creating a multipart mesh as necessary.

Eleven geometries in total were developed for the implementation of this study. The first analysis was performed for normal MFC geometry, as described, followed by sclerosis in the ROI. Model defect sizes were chosen to depict conservative estimates of naturally occurring voids (Fig. 5). Grade 1 (G1) was a small central MFC depression ($\sim$0.03 cm^3) in subchondral bone, Grade 2 (G2) was a larger domed ($\sim$0.5 cm^3) defect with a large articular opening, and Grade 3 (G3) was a larger bone defect ($\sim$1.00 cm^3) with a smaller articular footprint (Fig. 6). The second set of analyses was performed with sclerosis and flattened MFC geometry (Fig. 4, Fig. S2) and with G1–G3 defects. All models maintained a similar number of elements.

Sclerosis is typically the first radiographic abnormality detected in young horses and was added to normal geometry by increasing bone modulus ($\sim$13%) of the ROI [23] (Fig. 5). Sclerosis was retained in the ROI for subsequent conditions, including G1, G2 and G3 defects to simulate regions of sclerotic bone that surround cysts (Fig. 5). MFC finite element models were tested on two geometries—normal and flattened. Flattening of the MFC is a very common radiographic abnormality seen in young horses [24] and precedes SCL formation [7]. The flattened area of the MFC corresponds to the contact area of the tibia in joint extension and includes the cranial and axial aspect of the MFC. A 10 mm diameter area was flattened by 1 mm to mimic the flattening observed radiographically and clinically (Fig. 4).

Please cite this article as: L.L. Frazer et al., The impact of subchondral bone cysts on local bone stresses in the medial femoral condyle of the equine stifle joint, Medical Engineering and Physics (2017), http://dx.doi.org/10.1016/j.medengphy.2017.06.019