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The mechanics of focal chondral defects in the hip

Brenden J. Klennert [a,b], Benjamin J. Ellis [a,b], Travis G. Maak [c], Ashley L. Kapron [c], Jeffrey A. Weiss [a,b,c,*]

[a] Department of Bioengineering, University of Utah, 36 S. Wasatch Drive, Rm. 3100, Salt Lake City, UT 84112, United States
[b] Scientific Computing and Imaging Institute, University of Utah, 72 S. Central Campus Drive, Salt Lake City, UT 84112, United States
[c] Department of Orthopaedics, University of Utah, 590 Wakara Way, Salt Lake City, UT 84108, United States

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ABSTRACT

There is a mean incidence of osteoarthritis (OA) of the hip in 8% of the overall population. In the presence of focal chondral defects, defined as localized damage to the articular cartilage, there is an increased risk of symptomatic progression toward OA. This relationship between chondral defects and subsequent development of OA has led to substantial efforts to develop effective procedures for surgical cartilage repair. This study examined the effects of chondral defects and labral delamination on cartilage mechanics in the dysplastic hip during the gait cycle using subject-specific finite element analysis. Models were generated from volumetric CT data and analyzed with simulated chondral defects at the chondrolabral junction on the posterior acetabulum during five distinct points in the gait cycle. Focal chondral defects increased maximum shear stress on the osteochondral surface of the acetabular cartilage, when compared to the intact case. This effect was amplified with labral delamination. Additionally, chondral defects increased the first principal Lagrange strain on the articular surface of the acetabular cartilage and labrum. Labral delamination relieved some of this tensile strain. As defect size was increased, contact stress increased in the medial zone of the acetabulum, while it decreased anteriorly. The results suggest that in the presence of chondral defects and labral delamination the cartilage experiences elevated tensile strains and shear and contact stress, which could lead to further damage of the cartilage, and subsequent arthritic progression. The framework presented here will serve as the procedure for future finite element studies on cartilage mechanics in hips with varying disease states with simulated chondral defects and labral tears.

1. Introduction

With a mean incidence of 8% in the overall population, osteoarthritis (OA) of the hip is a major clinical problem that produces a significant economic burden (Dagenais et al., 2009). Localized damage to the articular cartilage, referred to herein as a chondral defect, occurs due to sudden mechanical overload (Fig. 1). Joints with abnormal shape, articulation or instability, such as dysplasia or femoroacetabular impingement, are especially susceptible to this localized damage to the articular surface (McCarthy and Lee, 2002; Tannast et al., 2008). If left untreated, chondral defects result in a significantly increased risk of symptomatic progression toward OA (Ding et al., 2005).

The recognition of the relationship between chondral defects and subsequent development of OA has led to substantial efforts to develop effective procedures for surgical cartilage repair. For the knee, there is a large body of research on the mechanical effects of chondral lesions (Brown et al., 1991; Guettler et al., 2004) and the treatment prognoses of defects of varying size, depth and location (Hjelle et al., 2002). These data have supported the development of clear guidelines for deciding between nonsurgical treatment and various treatment options, and have resulted in the postponement or, in some cases, complete avoidance of total knee replacement for patients with chondral defects in the knee (See (Bedi et al., 2010; Mall et al., 2015) for reviews).

In contrast to the knee, surgical treatment of chondral defects in the hip is rarely attempted due to the paucity of research on their mechanical effects and prognosis and the difficulty in accessing many of the defects via arthroscopy. Open surgical dislocation is required to access many areas of cartilage in the hip, and this procedure is associated with significant morbidity. With the increasing popularity of hip arthroscopy over the last 10 years, arthroscopic techniques for cartilage restoration in the knee, including microfracture, chondroplasty, and cartilage transplantation, are slowly being translated to the hip (Emre et al., 2012;

* Corresponding author at: Department of Bioengineering, University of Utah, 36 S. Wasatch Drive, Rm. 3100, Salt Lake City, UT 84112, United States. Fax: +801 585 5361.
E-mail address: jeff.weiss@utah.edu (J.A. Weiss).

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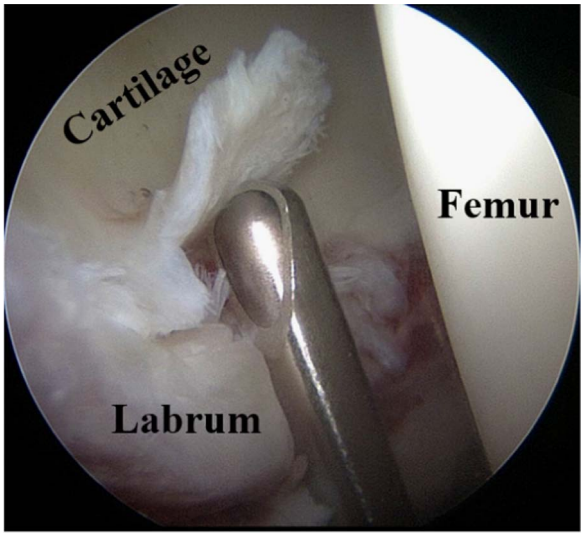

Fig. 1. Arthroscopic image of a focal chondral defect at the chondrolabral junction of the articular surface of the acetabulum in the hip. The cartilage has delaminated from the pelvis and the surgeon has lifted the resulting cartilage flap for display. This flap will be removed, creating a focal chondral defect.

Fontana, 2012; Fontana et al., 2012; Karthikeyan et al., 2012). However, substantial differences between the mechanics of the hip and knee, particularly in curvature and loading, as well as the confounding factor of labral damage commonly accompanying the cartilage defects, limit the direct translation of previous knee data to support treatment guidelines in the hip. A clear understanding of the mechanical effect of defects on the surrounding cartilage in the hip would help to separate stable defects that will not expand and can potentially be treated conservatively (non-surgically) from defects that are likely to expand and thus should be treated more aggressively (i.e,. with arthroscopic or even open surgical intervention). This will help to establish a foundation for an evidence-based prognosis and a patient-specific cartilage treatment algorithm.

Focal chondral defects are an especially common finding in patients with dysplasia (Kaya et al., 2014; McCarthy and Lee, 2004), which is a hip pathomorphology characterized by a shallow acetabulum with lack of coverage of the femoral head. Dysplasia causes a 4.3-fold increased risk for radiographic hip OA (Reijman et al., 2005). The link between acetabular dysplasia and hip OA is thought to be altered chondrolabral mechanics (Cooperman, 2013; Kosuge et al., 2013). In acetabular dysplasia, chronic shear stress is produced by the instability and anterolateral migration of the femoral head at the acetabular margin. As this continues, the labrum can become delaminated from the acetabular rim leading to chondral-labral dissociation and extensive full-thickness chondral defects (Bogunovic et al., 2014). An estimated 20% of all hip OA is secondary to mild to moderate acetabular dysplasia (Solomon, 1976).

The finite element (FE) method provides a valuable approach to study the contact mechanics of the hip on a patient-specific basis. In our previous research, we developed, validated and applied a patient-specific modeling pipeline to study contact stresses in normal, dysplastic and retroverted hips (Anderson et al., 2008; Harris et al., 2012; Henak et al., 2013a; Henak et al., 2013d; Henak et al., 2013e). With careful attention to mesh zoning and the choice of constitutive model, reliable predictions of transchondral (through the thickness of the articular cartilage) stress and strain can be obtained (Henak et al., 2013b; Henak et al., 2013c; Maas et al., 2016). Accurate predictions of stress and strain at the articular surface, through the articular layers of cartilage, and at the osteochondral interface are important for predicting mechanical overload and overt failure of the solid matrix in articular cartilage (Beck et al., 2005; Flachsmann et al., 1995). Our previous study examined cartilage and labrum mechanics in human hips with acetabular dysplasia. Although finite element based approaches have been applied to study repair strategies for chondral defects in the knee (Mononen et al., 2011; Pena et al., 2007; Wu et al., 2002), there have been no studies to date focusing on chondral defects in the hip. The aim of this study was to determine the effects of chondral defects and labral delamination on mechanical overload of the surrounding articular cartilage and labrum in the dysplastic hip. We hypothesized that the presence of a focal chondral defect would increase the maximum shear stress at the osteochondral interface and the first principal strain in the surrounding cartilage and labrum, and that labral delamination and increasing defect size would amplify this effect.

2. Methods

A subject-specific FE model of a dysplastic hip was used to study the mechanics of simulated focal chondral defects and labral delamination. The subject was selected from a cohort of ten patients with acetabular dysplasia (three male and seven female, BMI $23.4 \pm 5.9\ \mathrm{kg\,m^{-2}}$, age 26 ± 6 years, center edge angle [CEA] (Wiberg, 1939) $14.8 \pm 4.6°$, acetabular index [AI] (Lequesne, 1963) $18.1 \pm 6.9°$). The subjects were recruited and imaged with IRB approval (University of Utah IRB #10983). These patients, including the subject selected for this study, were used in a previous study of chondrolabral mechanics in the dysplastic hip (Henak et al., 2013a). The selected patient was a 25 year old, 70-kg female with a dysplastic left hip characterized by a CEA of 19.6° and an AI of 16.4°.

The FE model was generated from volumetric CT arthrography data (Henak et al., 2014), which was segmented using the Amira software (Visage Imaging, Inc., San Diego, CA), with a combination of thresholding and manual techniques described in previous studies (Harris et al., 2012; Henak et al., 2013a; Henak et al., 2011). The model included the left hemipelvis, proximal end of the left femur, femoral cartilage, acetabular cartilage and labrum (Fig. 2A). To adequately resolve stresses through the thickness of the articular cartilage and around the focal chondral defects, we generated new quadratic tetrahedral FE meshes with local refinement for the femoral and acetabular cartilage layers and labrum using ANSA (BETA CAE Systems SA, Thessaloniki, Greece). For the model of the intact cartilage, mesh refinement was performed in the areas of the acetabular cartilage with the highest contact stress during the gait cycle (Fig. 2B). For the models with chondral defects, further refinement was performed around the area of the defect and the labrum-bone interface where labral delamination was simulated.

Focal chondral defects were simulated as semicircular holes at the chondrolabral boundary in the posterolateral region of the acetabulum. The choices of shape and location were based on clinical reports of the location, incidence and shape of chondral defects in the hip (Ganz et al., 2003; Kaya et al., 2014; McCarthy and Lee, 2004). For the model in this study, the defect was placed in an area of high shear and contact stresses in the acetabular cartilage. In addition to simulations of the intact joint, three defect sizes were analyzed: 3.5-mm in diameter, 7.0-mm in diameter, and 15-mm in diameter (Fig. 2C). The effect of labral delamination was examined for all defect sizes by allowing a sliding contact boundary condition at the labrum-bone interface just above the defect. This allowed for the labrum to slide up the acetabular rim of the pelvis as the joint was loaded.

Preprocessing, analysis and postprocessing were performed using the FEBio software suite (www.febio.org) (Maas et al., 2012). The pelvis and femur were modeled as rigid bodies. The acetabular and femoral cartilage were modeled with a continuous fiber distribution constitutive model in combination with a nearly incompressible neo-Hookean ground matrix (shear modulus $\mu = 1.82$ MPa, bulk modulus $K = 1860$ MPa). The fiber strain energy integrates contributions from fibers along all directions emanating from a point. An initially spherical distribution was chosen, representing an initially isotropic contribution that produces strain-induced anisotropy. The fiber strain energy was represented using a power law ("fiber-exponential-power-law-uncoupled" material in FEBio, initial modulus $\xi_1 = 9.19$ MPa, fiber power coefficient $\beta = 4$). Fibers supported load only in tension. With the chosen material parameters, this constitutive model exhibits strain-induced anisotropy, strain-stiffening and tension-compression nonlinearity (Ateshian et al., 2009). The chosen material coefficients provided the best fit to our experimental data for unconfined compression of human acetabular cartilage (Henak et al., 2013c). The labrum was represented as a transversely isotropic hyperelastic material consisting of a fiber family embedded in a neo-Hookean matrix ("trans iso Mooney-Rivlin" material in FEBio, $\mu = 2.8$ MPa, $K = 1000$ MPa, exponential toe region coefficients $C_3 = 0.05$ MPa and $C_4 = 36$, straightened fiber modulus $C_5 = 66$ MPa, fiber stretch for straightened fibers $\lambda = 1.103$) (Quapp and Weiss, 1998), with the direction of fiber reinforcement following the circumferential orientation of the labrum, as per our previous study (Henak et al., 2011).

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