Degenerative Changes In The Biomechanical Properties Of Cartilage From Cam-type Femoroacetabular Impingement Deformities

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Introduction: Femoroacetabular impingement (FAI) results from abnormal contact between the femur and acetabulum and has been associated with osteoarthritis (OA) of the hip[1]. The most common cause of FAI is the cam-type deformity, a convexity at the anterior or anterosuperior femoral head-neck junction. Although a direct causal relationship remains controversial, recent studies have shown a relationship between the severity of cam-type deformities and degeneration[2]. Furthermore, cartilage tissue retrieved from the cam deformity exhibited proteoglycan depletion and altered cellular activity consistent with OA[3]. The goal of this study was to characterize the biomechanical and histological properties of the cartilage tissue from cam deformities. This will improve understanding of the contact mechanics and potential mechanical causes of hip OA associated with FAI.

Methods: Osteochondral specimens, 10 mm in diameter, were retrieved from the cam deformity of patients undergoing surgical correction of a symptomatic deformity (“Impingement” group, n=14). Control specimens were retrieved from the anterior femoral head of cadaver donors during autopsy (n=10). The study was approved by the institutional review board and surgical subjects gave informed consent prior to surgery. Indentation stress relaxation tests were performed on specimens in a saline bath with a single ramp to 10% of the cartilage thickness. Following the test, half the cartilage layer was removed for standard histological processing and sections were stained with Safranin-O. A portion of the remaining tissue was used to measure the water content from the wet and dry weights. A fibril-reinforced poro-hyperelastic finite element analysis was performed to determine the cartilage material parameters: modulus (E), Poisson’s ratio (ν) and permeability (k) of the non-fibrillar hyperelastic component, and the strain-independent (E₀) and -dependent (E₁) moduli of the collagen fibres[4]. The finite element simulation of the in vitro test was performed within a non-linear optimization to determine the parameters that best fit the stress relaxation test data. Relative proteoglycan content was quantified from the red intensity of microscopic images[5] and the mean red content in the superficial, middle, deep and full thickness was calculated. The equilibrium modulus (E_{eq}) was also calculated using the modified Hayes solution for comparison. Significant differences between Impingement and Control specimens were examined for each parameter using the student t-test, and linear regression was performed to assess the relationship between the non-fibrillar material parameters and the proteoglycan content.

Results: Significant differences were seen between Impingement specimens and Controls for E, ν, k (Figure 1) as well as E_{eq} and red content in all regions (p=0.0002 to 0.031) representing the proteoglycan component. No significant differences were seen in the water content nor fibril parameters E₀ or E₁ (p>0.1) although differences may be obscured by large variances. There was a strong linear relationship of E vs E_{eq} (R²=0.95, p<0.0001). However the slope was less than unity, indicating that E_{eq} overestimated the stiffness of the non-fibrillar component, likely due to the contribution of the collagen fibrils. A moderate correlation was seen between the red content and E (R²=0.53, p=0.0002; Figure 2), however correlations between red content and ν (R²=0.35, p=0.004) and k (R²=0.29, p=0.01) were weaker.
Discussion: Cartilage retrieved from symptomatic cam FAI deformities exhibited a non-fibrillar modulus 80% lower and permeability an order of magnitude higher than Control specimens. The non-fibrillar modulus correlated with the proteoglycan content. Specimens with complete depletion of proteoglycan exhibited a finite modulus and permeability, suggesting that collagen or other macromolecules play a role in compressive stiffness and fluid flow, although much less than the proteoglycan molecules. The differences in biomechanical properties of the cartilage from symptomatic cam deformities compared to Controls are consistent with osteoarthritic degeneration. This supports the hypothesis that abnormal contact of the deformity and acetabular rim is a cause of osteoarthritis[1].

Significance: This study has shown that cartilage from cam FAI deformities exhibits biomechanical properties consistent with
osteoarthritis. Identification and treatment of such deformities is likely to slow the disease process and delay or eliminate the need for eventual hip replacement surgery.

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