Small Chondral Defects Affect Tibiofemoral Contact Area and Stress: Should Treatment Algorithms Change?
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INTRODUCTION:
Full-thickness chondral defects of the knee can lead to pain and decreased function and have been implicated in the progression towards osteoarthritis.1 These lesions have minimal healing potential if left untreated, and little is known about the natural history of these lesions, particularly the asymptomatic ones2. While prior studies have discussed the role of defect size and location in larger lesions3, there is a lack of literature on the biomechanical effects of smaller defects on human knees. The purpose of this study is to examine how tibiofemoral contact pressures are affected by increasing defect size on the medial and lateral condyle at full knee extension using a human cadaveric knee model.

METHODS:
Isolated full-thickness, square chondral defects increasing from 0.09 cm2 to 1.0 cm2 were created on the medial and lateral femoral condyles of six human cadaveric knees with intact ligaments and menisci. Defects were created 1.0 cm from the femoral notch posteriorly. The knees were fixed to a uniaxial load frame (MTS 30/G machine) and loaded from 0N to 600N at full extension. Contact pressures between the femoral and tibial condyles were measured using flexible sensors. Distribution maps were recorded. In post-processing analysis, the peak value for contact pressure was defined as the highest value in the 2.54 mm2 area around the defect. The location of the peak contact pressure was determined anatomically with the center of the defect as reference. Full-thickness, square chondral defects were then created on the lateral femoral condyle 1.0 cm posterior to the femoral notch at the same size increments.

One-way repeated measures ANOVA was used to study the relationship between defect size and maximum contact pressures on both femoral condyles. Post-hoc analyses were performed using the Tukey HSD test. The relationship between the location of the location of the peak contact pressure point and size was cross-tabulated and assessed using Fisher's exact test.

RESULTS:
The average maximum contact pressures around the defect were 4.30MPa, 5.61MPa, 5.91MPa, and 6.91MPa on the medial femoral condyle at 0.09 cm2, 0.25 cm2, 0.49 cm2, and 1.0 cm2, respectively (Figure 1, top). Contact pressures were significantly different between defects by size (p < 0.01). Specifically, contact pressures were significantly different between 0.09 cm2and 1cm2 defects (p = 0.04) (Figure 2a). The location of the peak contact pressure point in the medial condyle was found posteromedially in defect sizes between 0.09 cm2(4 of 6 knees, 67% ) and 0.25 cm2 (3 of 6, 50%). The stress point shifted anterolaterally in defect sizes between 0.49cm2 (5 of 6, 83%) and 1.0 cm2 (6 of 6, 100%) (p < 0.01).

On the lateral femoral condyle, the average maximum contact pressures around the defect were 3.63MPa, 4.60MPa, 5.28MPa, and 5.81MPa at 0.09 cm2, 0.25 cm2, 0.49 cm2, and 1.0 cm2, respectively (Figure 1, bottom). Contact pressures around the defect were different between defect sizes (p = 0.02) (Figure 2b). Post-hoc analysis showed differences in contact pressures between 0.09 cm2and 1.0 cm2 defect sizes (p=0.02). The location of the peak contact pressure point was anterolateral to the center of defects between 0.09 cm2(3 of 6 knees, 50%) and 0.25 cm2 (4 of 6, 67%) in size. However, the stress point shifted posterolaterally for defect sizes between 0.49 cm2(4 of 6, 67%) and 1.0 cm2 (5 of 6, 83%) (p < 0.01).

DISCUSSION AND CONCLUSION:
As defect size increased on the lateral and medial femoral condyles, there were significant increases in contact pressure. Full-thickness chondral defects on both condyles led to greater cartilage stress after reaching a defect size of 1.0 cm2. Furthermore, the location of the maximum stress point also varied with increasing defect size. This point shifted anterolaterally from the center of the defect for sizes greater than 0.49 cm2 on medial condyle and posterolaterally for defect sizes greater than 0.49 cm2 on the lateral condyle. This effect may be evidence of force and contact area redistribution after the defect reaches 0.49 cm2 in size. These changes in contact area redistribution and cartilage stress in the 0.49 cm2 to 1.0 cm2 range may impact cartilage integrity around the defect in the long-term, as cartilage has a limited healing capacity.

Taken together, these findings suggest that size cutoffs exist earlier in the natural history of full-thickness chondral defects than previously studied. Although current algorithms for articular cartilage lesions recommend more aggressive interventions after lesions exceed 2-3 cm2, our study suggests a lower threshold over which the treatment of full-thickness chondral defects should change. These results may be used to better inform surgical treatment decision-making for the management of chondral defects.