Single-Condyle Surface Incongruity in the Rabbit Knee Causes Whole-Joint Degeneration

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Purposes: Osteoarticular injuries including intraarticular fractures can cause persistent joint incongruity that eventually leads to whole-joint cartilage degeneration (i.e., post-traumatic OA). The pathomechanisms of chronic joint degeneration associated with local joint incongruity were studied using a rabbit knee model of focal osteochondral defect. The goal of this study was to document the effect of hemi-compartmental joint incongruity on the whole-joint cartilage in the rabbit knee. It was hypothesized that a focal osteochondral defect created on the medial femoral condyle would cause whole-joint degeneration in the rabbit knee.

Methods: Forty New Zealand white rabbits were subjected to a surgical insult in the left knee. In half of these animals, a 2 mm osteochondral defect was created in the maximally weight bearing area of the medial femoral condyle. The others received a sham surgery (arthrotomy control). In each group, half of the animals were sacrificed at 8 weeks and the other half at 16 weeks. At the end of test period, the insulted knees were tested for AP drawer stability, using a custom loading device.[1] The joints were then prepared for histomorphological evaluation. The femoral and tibial surfaces in both the medial and lateral compartments were rated individually using Mankin’s Histological Histochemical Grading Scale (HHGS, 14 points max)[2], by two blinded observers. In the case of the medial femoral condyle with an osteochondral defect introduced, changes in the adjacent cartilage were evaluated. Statistical significance was taken as p < 0.05.

Results: In the knees introduced an osteochondral defect, significant (p<0.001) elevation of HHGS occurred in the medial compartment, on both the femoral and tibial surfaces. The HHGS score in the lateral femoral surface also significantly increased. (p = 0.002) (Figure 2) The most degenerative changes were observed in the medial tibial surface. In addition, when degeneration was classified as mild (3 ≤ HHGS <6), moderate (6 ≤ HHGS <9) or severe (HHGS ≥9), the number of joints with severe degeneration in the medial tibial surface increased with time, from 1 out of 10 at 8 weeks to 4 out of 10 at 16 weeks. On the medial femoral surface, while only mild degeneration occurred at 8 weeks, moderate to severe degeneration was identified in three joints at 16 weeks. (Figure 3) Moreover, the knees introduced a focal osteochondral defect were accompanied by modestly increased AP laxity, as indicated by decrease of anterior drawer stiffness and increase of neutral-zone length. (p=0.067 and 0.017, respectively) (Figure 4)

Discussion: A focal osteochondral defect on the medial femoral surface in the maximally weight bearing area caused cartilage degeneration in the medial compartment on both the femoral and tibial surfaces. Although mild, cartilage degeneration was identified in the lateral compartment, implying potential interaction in articular contact mechanics between medial and lateral compartments. Local biochemical factors (e.g. inflammatory cytokines released in the synovial fluid) also might have influenced the whole-joint cartilage metabolism. The most severe degeneration occurred on the medial tibial surface, at the opposite side of the introduced. There was a trend that medial tibial degeneration developed with time, suggesting chronic aberrations in the contact mechanics in the medial compartment. The mild disease development in the defect animals from 8 weeks to 16 weeks, the medial defect model is potentially useful for piloting new treatments for chronic OA.

Conclusion: Presumably, local joint incongruity can caused whole – joint cartilage degeneration.

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Figure 1: The posteromedial approach to expose the maximally weight-bearing area, located posteriorly on the medial femoral condyle (A). Histology of medial femoral condyle of the rabbit knee showed the thickest cartilage on the maximally weight-bearing area posteriorly (B).

Figure 2: cartilage degeneration on each articular surface (HHGS score 0-14)

Figure 3: Number of animals with the various ranges of degeneration on each articular surface at 8 and 16 weeks time periods

Figure 4: Anterior drawer stiffness(A) and Neutral-Zone length(B) changes between control and defect groups