Investigation of Tibiofemoral Cartilage Damage Following Anterior Cruciate Ligament Failure From Impact Compression Of Cadaveric Knee Join

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Introduction: Rapid and high impact activities can cause cartilage damage, which may develop to secondary osteoarthritis (OA). This study sought to determine the extent and distribution of tibiofemoral cartilage damage in the event of an anterior cruciate ligament (ACL) failure during impact joint compression.

Materials and Methods: Five cadaveric knee joints (age: 35-60), with intact soft tissues, were potted in dental cement, fixed to potting cups and mounted onto the Material Testing System at 70° flexion to simulate a landing posture. The tibia was limited to axial displacement and rotation; the femur was restricted to transverse displacements. Passive markers were placed on the cups to monitor rotational and translational motions of the tibia and femur using a motion-capture system. Each specimen was compressed at a single haversine of 10-Hz frequency [1] using displacement control to simulate a landing impact. The compression trial was successively repeated with increasing actuator displacement. The test was ended when a significant compressive force drop was observed; ACL failure was confirmed via magnetic resonance (MR) imaging and dissection. Pre- and post-test MR scans were used to estimate % reduction in tibial cartilage thickness. Tibiofemoral osteocartilage explants (4-mm diameter) were extracted from the post-impact specimens and fixed in 10% buffered formalin, followed by decalcification in 30% formic acid. They were then dehydrated in aqueous ethanol, further cleared in toluene and embedded in paraffin. These explants were sectioned at 10 micron thickness and placed on glass slides. The sections were deparaffinized, stained in Hematoxylin & Eosin (H&E) and Safranin-O/Fast Green (Saf-O) and coverslipped. These sections were scored in series using the modified Mankin grading system [2] to assess the status of cartilage lesion.

Results: All specimens underwent ACL failure. For a post-impact specimen HK1R, % reduction in tibial cartilage thickness was high (13.5-16.1%) at AM/CM sites and minimal at CL/AM/BM sites; there is a weak correlation (R²=0.23) between % reduction in cartilage thickness and Mankin scores across the explant sites (Fig not shown). High damage scores were observed at the posterior regions of both femoral condyles and at the exterior and interior halves of the medial and lateral tibial plateau respectively (Fig 1). The damage types included surface irregularities, delamination, fraying, clefts (yellow arrow) and tidemark disruption (yellow circle) (Fig 2). Applying t-test to the mean explant Mankin scores (n=5) across all specimens showed significant damages at the posterior tibial plateau and at the exterior, posterior and interior femoral condyles (Fig 3).

Discussion: Percentage reduction in cartilage thickness may not be an adequate damage indicator, especially for microscopic damages. Bone bruises due to ACL injury were reported to be localized at the posterior tibial plateau and middle regions of both femoral condyles [3,4]. Moreover, Kleemann [5] reported a Mankin score of 3.2 for mild OA. Therefore, the mean Mankin scores of 4.33–4.60 at posterior tibiocartilaginous compartments in the current study indicated these sites to likely at an early OA stage. During joint compression, the impact loads that result in ACL failure can also potentially inflict cartilage damage, leading to a possible accelerated risk of joint degeneration. Reducing knee joint compression can be a possible brace design consideration for attenuating cartilage damage and lowering the risk of OA in athletes.


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