Osteochondral Defects Markedly Alter Knee Joint Contact Stresses and Cartilage Strains: A Finite Element Study

[1]Shirazi, R; +[1]Shirazi-Adl, A
+[1]Ecole Polytechnique, Montreal, QC
abshir@meca.polymtl.ca

INTRODUCTION

Onset and progression of cartilage degeneration in the knee joint are likely associated with subchondral lesions. Defects as overgrowth of the subchondral plate (bone boss) and bone bruises are commonly detected in knee joints especially following ACL injury. In trauma or repetitive impact loads, local detachments of the cartilage at its calcified zone and/or deep collagen fibrillation have been observed. The effect of subchondral lesions on mechanics of both articular cartilage and neighboring bone remains however not yet quantified. This study aimed to compute the influence osteochondral defects on mechanics of articular cartilage and the entire joint. To do so, initially a validated knee joint model was refined and extended to incorporate proximal tibial bone. The short-term response of the joint lateral compartment while simulating various chondral lesions was subsequently investigated under axial compression force at full extension. Acute subchondral bone injuries, horizontal splitting and bone overgrowth and hardening were hypothesized to markedly alter chondral mechanics and, hence, to contribute to the initiation and development of joint degeneration.

METHODS

The mesh of an existing validated 3-D model of the human knee joint was modified by refinement of articular cartilage and addition of the calcified cartilage and proximal tibia (subchondral, trabecular and cortical bones) to a depth of ~16 mm (Fig. 1). The joint lateral tibiofemoral compartment alone was considered neglecting the medial compartment and ligaments. The cartilage and meniscus non-fibrillar matrices were modelled as incompressible isotropic hyperelastic solid while layer-wise fibrils networks were simulated either by membrane or continuum brick elements. Thickness of membrane elements in different regions of cartilage and menisci was computed based on the fibrils volume fraction in each zone. To study short-term (transient biphasic) response of the joint, an equivalent incompressible elastic response was employed. The drained modulus of cartilage solid matrix was depth-dependent increasing nonlinearly from 0.3 at the uppermost layer to 1.2 MPa at the lowermost layer at the calcified junction. Tibial calcified cartilage, subchondral bone, trabecular bone and cortical bone were assumed linear isotropic elastic. The transient response of the lateral compartment was studied at full extension under 1500 N compression applied via the femur while the tibia was fixed at the base.

To examine the effect of various lesions on the response of the intact joint (reference case a), a region of the calcified cartilage under the highly loaded area of the cartilage (15% or 116 mm^2 of the compartmental surface, Fig. 1) was initially demarcated. Local tibial bone boss overlying this region was modeled by increasing stiffness of solid matrix elements at the deep one (quarter thickness) or two (half thickness, case b) cartilage layers to that of the calcified cartilage (E=300 MPa). Bone damage at the defect zone to 7.1 mm depth was simulated by diminishing bone elastic modulus to 5 MPa. In additional cases, horizontal splitting at the cartilage-bone interface and local absence of vertical fibrils at the deep region in the defect zone were considered. The combined effects of base split, absence of vertical fibrils with underlying bone damage were also modeled (case c).

RESULTS

Tibial articular contact pressure markedly changed in both magnitude and pattern in presence of bone damage at the defect zone (1500 N, case c, Fig. 2 left). Joint axial translation increased (~11%) in the case with local bone damage. On the contrary, peak contact pressure increased (~9%) and the axial translation decreased by only 1% in presence of a bone boss of half-cartilage thick (case b, Fig. 2 left). Foregoing lesions at the tibial region influenced the contact pressure distribution on the femoral cartilage as well (Fig. 2 right); bone softening decreased contact pressure by up to 9% (case c) whereas the bone boss substantially increased this pressure by up to 19% (case b). The principal tensile strain in the cartilage matrix altered in both pattern and magnitude every where in case c with bone damage, at both lowermost (Fig. 3) and superficial layers. Bone damage also shifted the compression from the exposed cartilage away onto the unexposed cartilage via meniscus. The axial stress in the calcified cartilage, subchondral and trabecular bones was affected in case c with bone softening (damage).

DISCUSSION

Although the temporal sequence of events involving alterations in cartilage and periarticular bone remains yet controversial, ample evidence exists pointing to tight coupling between cartilage and bone conditions. Bone bruises are strongly associated with ACL rupture and/or meniscal tears. The bone modulus reduction algorithm, used in the current study to simulate local bone fractures, has been demonstrated to accurately model bone microdamages. Predictions confirmed the hypothesis on the marked influence of alterations in the subchondral bone on articular cartilage and joint mechanics. The peak contact pressures on tibial and femoral articular surfaces increased, respectively, by 9% and 19% when the bone boss was simulated at the tibial cartilage. The bone softening or damage, on the other hand, markedly altered the joint biomechanics as well as the pattern and magnitude of contact pressures, maximum tensile strains in cartilage, axial stresses in bony elements and load partitioning between exposed (cartilage-to-cartilage) and covered (meniscus-to-cartilage) areas. Comparatively, local bone softening (damage) appeared more detrimental in altering joint mechanical environment. Our predictions suggest that bone bruise and overgrowth adversely perturb the homeostatic balance in the mechanical environment of articular cartilage surrounding and opposing the lesion. As such, chondral defects have the potential to contribute to the initiation and development of post-traumatic osteoarthritis.

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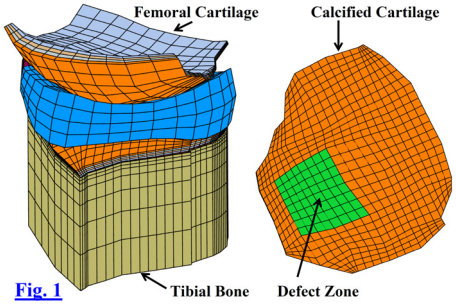

Fig. 1

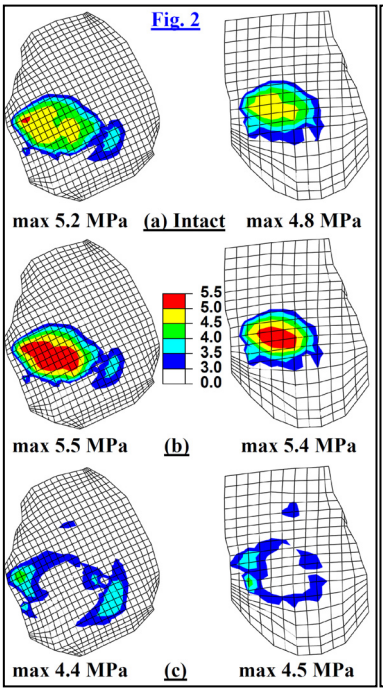

Fig. 2

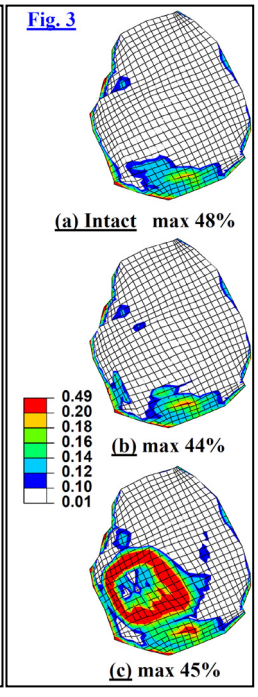

Fig. 3