High-Energy Pilon Fractures Result in Significant Chondrocyte Cell Death on Both the Tibia and Talar Side of Injury

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Purpose: Posttraumatic osteoarthritis (PTOA) development after intra-articular fracture (IAF) of the tibial plafond is multifactorial and poorly understood. Chondrocyte cell death from impaction of the talus into the tibia may contribute significantly to this process. The purpose of this study was to compare chondrocyte viability in low-energy versus high-energy tibial plafond fractures in a porcine model at both the tibial plafond and the unfractured talus.

Methods: Testing was performed on 15 porcine hind limbs using a validated large-animal IAF model. Three groups were tested: 3 control pigs (CP) (18 chondral samples), 6 low-energy fracture pigs (LEP; 34.2 J) (36 chondral samples), and 6 high-energy fracture pigs (HEP; 73.9 J) (36 chondral samples). Chondral samples were taken from tibia adjacent to fracture (Tib-F), tibia at 5 mm away from fracture, talus near fracture (Tal-F), and talus 5 mm away from fracture. Samples were incubated at 37°C in culture medium for 48 hours and then stained using Calcein AM (live cells) and ethidium homodimer-2 (dead cells). Confocal microscopy evaluation was performed on all samples to determine: (1) ratio of live cells to dead cells and (2) the adjusted fractional cell death. Cell counting was performed in a blinded manner using Imaris software with specifications held constant throughout this process.

Results: Chondrocyte cell death was markedly higher comparing the HEP cohort to the LEP cohort at all locations. HEP Tib-F fractional cell death was 41.92% (standard deviation [SD] 3.5) compared to LEP Tib-F 28.8% (SD 4.32) ($P<0.0001$) and HEP Tal-F fractional cell death was 40.41% (SD 4.1) compared to LEP Tal-F cell death of 25.91% (SD 4.28) ($P = 0.0018$). Similarly, the fractional cell death of chondral samples near fracture was significantly greater than the cell death 5 mm away from fracture in both the LEP and HEP (all $P<0.0001$). Finally, chondrocyte cell death near fracture was not significantly different between the tibia and the talus when comparing LEP and HEP to each other (cell death HEP Tib-F 41.92% vs HEP Tal-F 40.41%, $P = 0.29$; cell death LEP Tib-F 28.80% vs LEP Tal-F 25.91%, $P = 0.16$).

Conclusion: Most prior research on cartilage damage following articular fractures has focused on the fractured side of the joint. This study demonstrates that chondrocyte injury occurs on both sides of the joint during IAF, which may explain the whole joint degeneration that occurs with PTOA. Future therapeutics in PTOA mitigation should be directed at the entire joint as opposed to just the fractured portion.