Novel Fracture-Based Porcine Model of Acute Compartment Syndrome Creates Elevated Intra-Compartmental Pressure Using Only a Simulated Blast Injury

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INTRODUCTION:

Existing animal models of compartment syndrome poorly mimic the clinical scenarios that result in acute extremity compartment syndrome (ACS) for two reasons. First, existing models require exogenous manipulation (e.g., tourniquet, intra-compartmental infusion, intra-compartmental balloon) of intra-compartmental pressure (ICP) in order to create ACS. Second, existing models do not commonly include significant osseous or soft tissue injuries. No existing animal model creates a musculoskeletal injury which endogenously elevates ICP. The purpose of the current study was to evaluate the ability of a novel porcine model of ACS, which relies on a simulated blast injury, to endogenously create elevated ICPs consistent with ACS.

METHODS:

The hindlimb of juvenile Landrace pigs were first injured by creating a diaphyseal tibia fracture using a three-point bending mechanism. The fractured limb was then subjected to seven blasts of highly compressed air at 100-110 PSI to mimic a blast injury. Following injury creation, both the injured and the contralateral, uninjured control leg were subject to continuous pressure monitoring using an arterial line placed intra-compartmentally. In a subset of injured animals, the tibial shaft fracture was stabilized using plate and screws followed by imbrication of the anterior compartment fascia at 4 hours post-injury. A pressure measurement was made after operative fixation. Select pigs were harvested between 48-72 hours post-injury and an additional pressure measurement was made at the time of harvest. A second subset of pigs underwent operative fixation followed by fasciotomy. A pressure measurement was made to evaluate the effects of the fasciotomy. ICP data was then compared between control, fasciotomy, preoperative experimental, postoperative experimental, and 48-72 hours post-injury experimental groups.

RESULTS:

At 4 hours post-injury, the novel model of compartment syndrome created significantly elevated ICP (45.9 ± 12.6 mmHg; N=14) compared to control limbs (16.9 ± 7.1 mmHg; N=12; $p<0.001$). In injured extremities, operative fixation and anterior compartment fascial imbrication further increased the ICP (85.0 ± 42.2 mmHg, N=8) relative to the preoperative state ($p=0.042$). Fasciotomy resulted in marked decreases in anterior compartment pressure following operative fixation (Mean: 14.8 ± 2.8 mmHg, n=4) which was equivalent to control limbs ($p=0.686$). Five fracture and fixation pigs were maintained for 2-3 days post-injury for a separate study. Pressure measurements at the time of harvest demonstrated that elevated ICP persisted following injury, though the response between individuals was less predictable between individuals (Mean: 87.2 ± 41.8 mmHg, N=5).

DISCUSSION AND CONCLUSION:

This novel porcine model of ACS elevates ICP comparable to existing models of compartment syndrome using a musculoskeletal injury without exogenous ICP manipulation. ICP elevation is relieved with fasciotomy, consistent with clinical ACS. ICP remained elevated at 48-72 hours but with wide variability. Compared to existing models of compartment syndrome, the current model is mechanistically more similar to clinical ACS and provides the opportunity to study the effects of elevated ICP on severely injured muscle. Furthermore, because this model results in persistently elevated ICP days after injury, it provides a unique means of testing new therapy adjuncts for the ACS.