Limited capsular release reduces flexion contracture in a rabbit model of arthrofibrosis

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ABSTRACT INTRODUCTION:

Joint contractures are a challenging and debilitating problem. The elbow joint is particularly sensitive to forming contracture. Many modern elbow contractures are post traumatic in origin. Despite early fixation and mobilization of elbow injuries, select patients still develop contractures. The exact etiology of these contractures is not well characterized.

In order to better evaluate the formation and biology of these contractures, our group has developed a contracture model in the rabbit1. This model has demonstrated formation of contracture over 8 weeks of immobilization following creation of cortical windows and hyperextension injury. Furthermore, this contracture has been shown to be quite stable, over as long as 16 weeks of remobilization. The contracture demonstrates biomechanical stiffness, as well as an increase in the number of myofibroblasts, a putative mechanism of contracture, in the capsule. By evaluating these parameters, a natural history of contracture is being developed.

Beyond being useful as a model of the pathology of joint contractures, our novel rabbit contracture model may be an effective model for evaluation of surgical and pharmacological interventions on contracture. Clinically, several different operative interventions are utilized, including open contracture release, arthroscopic contracture release, and interposition arthroplasty, as well as total joint arthroplasty1, 2. Open contracture release is often the initial surgical technique of choice. In spite of improving effectiveness of surgical release, patients often have residual limitation of motion postoperatively. Completing a surgical capsular release in our rabbit contracture model would allow us to further correlate the model with clinical experience, as well as create another opportunity for therapeutic intervention. We hypothesize that a limited capsular release will reduce, but not completely reverse, arthrofibrosis in this animal model.

METHODS:

Following institutional review board approval, twenty skeletally mature New Zealand White female rabbits were randomly divided into two study groups (Control and Limited Capsular Release). Rabbits in both groups underwent the same primary operation. Under appropriate anesthesia, they underwent lateral arthrotomy on the right knee. A 3 mm defect was created in the non cartilaginous portion of the femoral condyles. The joint was hyperextended to disrupt the posterior capsule. The joint was then be immobilized in full flexion with Kirschner wires.

Animals in each group underwent a second operation 8 weeks after the primary operation. This second operation was as follows:

Control: Removal of K-wire, manual lysis of bridging heterotopic ossification, sham capsular release

All animals were mobilized for 16 weeks. Following 16 weeks of remobilization, all animals were sacrificed under anesthesia. The joints were immediately tested using a custom made testing device. This device is validated using standardized weights, and has been documented in the literature by our group2. Briefly, the skin, muscle and tendons of the rabbit leg are dissected away, leaving tissue from the tibial tubercle up to approximately 10 mm proximal to the knee joint undisturbed. The tibia and femur are transected 7 cm from the joint line. The center of rotation of the joint is placed over the center of the torque cell. The position is confirmed fluoroscopically. This configuration is secured by intramedullary rods into the corresponding leg bones, which are attached to the torque cell. An extension torque is then applied through a pulley at 1 degree per second up to a maximum torque of 20 NCm. Flexion contracture was assessed by subtracting the angle at which the operative limb reached 20 NCm from the same parameter of the nonoperative limb.

Results are demonstrated as mean ± standard deviation. Statistical analysis was completed utilizing a Wilcoxon Rank Sum test. Significance was set at p<0.05.

RESULTS SECTION:

All animals survived both operations without operative complications. At the time of surgical release or sham surgery, the average flexion contracture was 129.2 ± 10.7 in the control group versus 29.6 ± 8.2 in the study group (p=0.0002).

Following sixteen weeks of remobilization, the average flexion contractures of the non-released and released animals were 49.0 ± 12.7 and 36.5 ± 14.2, respectively (p=0.035). One animal in the control group had signs of nerve injury (toe chewing), compared to four animals in the released group.

DISCUSSION:

In this animal model, a limited capsular release decreased flexion contracture immediately after surgery as well as following sixteen weeks of remobilization. This resembles clinical experience following contracture release. In addition, the large differences in flexion contracture that were documented at the time of release decreased significantly at final testing (99.6 degrees at time of release vs 12.5 degrees at final testing). This loss of motion is commonly documented after surgery.

There was an increased incidence of nerve injury signs in animals undergoing capsular release. Clinical experience with elbow contractures has demonstrated the importance of addressing the ulnar nerve at the time of contracture release. This correlation in the animal model is intriguing and warrants further investigation.

Limitations of this study include the fact that experiments are done on the rabbit knee rather than an elbow joint, which is the joint of most clinical interest. In addition, joint angle measurements made at the time of the operation were done utilizing fluoroscopic imaging at the time of operation, rather than using the custom testing device. This limits the depth of analysis possible on the postoperative flexion contracture measurements.

Overall, a limited capsular release decreased flexion contracture immediately after surgery as well as following sixteen weeks of remobilization. This experiment provides reference data for future testing of adjuvant treatment modalities for joint stiffness.

REFERENCES: