LOAD SHARING BETWEEN MEDIAL STRUCTURES OF THE KNEE IS AFFECTED BY ISOLATED INJURY TO THE MEDIAL COLLATERAL LIGAMENT AND SUBSEQUENT JOINT IMMOBILIZATION

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INTRODUCTION

Biomechanical assessment of ligaments during the healing process requires the use of animal models. Despite the fact that these are *in vivo* models of ligament healing, mechanical properties are typically measured using *in vitro* tests. *In vitro* testing necessitates dissection to isolate the ligament of interest; however, dissection artifact is a concern because isolating the structures that carry load is challenging at early healing intervals and after immobilization. The load sharing between joint structures may be different in a joint healing from an isolated ligament injury compared to the load sharing in a normal un-injured joint. The first purpose of measuring load sharing is to avoid dissection artifacts, ensuring that load-carrying tissue is not removed during dissection. The second purpose of measuring load sharing is to consider the whole joint response to injury rather than only considering the response of the isolated structure that was injured.

In a model of healing isolated injury to the rabbit medial collateral ligament (MCL), isolated MCL injury affected the load sharing between the MCL and the medial structures (medial capsule plus MCL) of the joint at early healing intervals [1]. For normal joints without injury, the MCL was the dominant load-carrying structure of the medial structures of the joint: the ratio of the failure load of the isolated MCL to the failure load of the combined medial structures (medial capsule plus MCL) was 89%. Following isolated MCL injury and subsequent healing, the load sharing between these medial structures was altered. At 3 weeks of healing, the load-sharing ratio of the failure load of the isolated MCL to the combined medial structures was only 24%; thus, a capsule-dominated response. By 6 weeks of healing, the response returned to a MCL-dominated response, demonstrated by a 90% load-sharing ratio (absolute values of failure loads remained decreased compared to normal). What remains unknown is whether load sharing is affected at later healing intervals if the joint has been immobilized following isolated ligament injury.

Our purpose in this study was to determine the effect of immobilization on the structural strength of the isolated MCL and the combined medial structures (medial capsule plus MCL) following isolated injury to the MCL. Our hypothesis was that the failure load of the combined medial structures, following isolated MCL injury, would be decreased with immobilization. Likewise, the failure load of the healing isolated MCL would be decreased with immobilization. Furthermore, the load sharing between the medial structures would be altered comparing immobilized joints to normal joints. Load sharing is characterized in this study by the ratio of the failure load of the isolated MCL (MCL alone) to the failure load of the combined medial structures (medial capsule plus MCL). If this ratio is less for immobilized joints than for normal joints, load sharing is affected and dissection artifact is a concern at later healing intervals when immobilization is performed following isolated injury to the MCL.

MATERIALS AND METHODS

Twenty-nine female New Zealand White rabbits had an acute 4 mm gap created in the midsubstance of both hindlimb MCLs using a protocol approved by the institutional animal care committee. Right hindlimbs were pin-immobilized in full flexion and left (contralateral) hindlimbs remained non-immobilized. Eighteen animals were designated for the "combined medial structures" group at three healing intervals: 3 (n=6), 6 (n=6), and 14 (n=6) weeks. Eleven animals were designated for the "isolated MCL" group at the two later healing intervals: 6 (n=5) and 14 (n=6) weeks.

For the "isolated MCL" group, dissection removed all muscle and fascia from the hindlimb, leaving the ligaments and menisci intact. After mounting in the Instron test machine, the lateral collateral ligament, anterior and posterior cruciate ligaments, and lateral and medial menisci were removed, isolating the MCL. For the "combined medial structures" group, dissection removed all tissues proximal to the bony ridge of the femoral groove and distal to the bony ridge of the tibial tuberosity. All fascia and capsule, including the MCL, from the anterior centre of the knee moving medially towards the posterior centre of the knee remained intact. After mounting in the Instron, the lateral collateral ligament, anterior and posterior cruciate ligaments, and lateral meniscus were removed, leaving only medial structures intact.

Both groups underwent the same mechanical testing protocol [2]. After joints were mounted at 70 degrees of flexion in the Instron, two cycles between 5 N compression and 2 N tension at 1 mm/min were performed, determining the position where the medial tissues first resisted tensile distraction. At this position, the Instron crosshead was zeroed, providing a consistent start point for all tests. Thirty cycles between the zero position and a displacement of 0.7 mm were performed at 10 mm/min. At the peak of cycle 31, the crosshead was held at 0.7 mm for 20 minutes. The crosshead was returned to the zero position for 5 minutes before the complex was elongated to failure at 20 mm/min.

Since only one hindlimb per animal can be immobilized, the ratio of the failure load of the isolated MCL to failure load of the combined medial structures for immobilized joints cannot be calculated between paired limbs and must be calculated using group means. The failure load of the isolated MCL was compared to that of the combined medial structures using Student's t-tests (alpha=0.05). Within the "combined medial structures" and "isolated MCL" groups, the failure load of the immobilized limb was compared to the contralateral non-immobilized limb for each animal using paired t-tests.

RESULTS

The failure load of the combined medial structures of the rabbit knee joint following isolated MCL injury was significantly decreased comparing the immobilized limb to the non-immobilized limb at 6 and 14 weeks ($p<0.005$; Figure 1a). The failure load of the immobilized healing isolated MCL was also decreased compared to the non-immobilized healing isolated MCL at 6 and 14 weeks ($p<0.01$; Figure 1b). The failure loads of the immobilized complexes (isolated MCL and combined medial structures) did not increase with healing. Only the non-immobilized joints that were contralateral to these immobilized joints had failure properties that improved with healing. The failure load of the combined medial structures of the non-immobilized joints was increased at 14 weeks compared to 3 weeks ($p=0.006$; Figure 1a). The failure load of the healing isolated MCL from non-immobilized joints was increased at 14 weeks compared to 6 weeks ($p=0.003$; Figure 1b).

A contralateral effect of immobilization was analyzed by comparing the failure loads of the non-immobilized joints that were contralateral to immobilized joints, as measured in the current study, to the failure loads when no immobilization was performed on either limb, as measured in the previous study [1]. At 3 weeks of healing, the failure load of the combined medial structures was lower when the contralateral limb was immobilized ($p=0.04$). At 6 weeks of healing, the failure load of the healing isolated MCL was lower when the contralateral limb was immobilized ($p=0.05$).

With immobilization, the failure load of the healing isolated MCL was significantly less than that of the combined medial structures at 6 and 14 weeks ($p<0.004$). For immobilized joints following isolated MCL injury, the load-sharing ratio of the failure load of the isolated MCL to the failure load of the combined medial structures was 16% at 6 weeks and 8% at 14 weeks. From the previous study [1], this load-sharing ratio was 89% for normal joints and, for non-immobilized joints following isolated MCL injury, the ratio increased from 24% at 3 weeks to 90% at 6 weeks.

The contribution of the capsule can be estimated as the difference between the failure load of the combined medial structures (medial capsule plus MCL) and the isolated MCL (MCL alone). For the immobilized joints, the capsule contributed 59 N at 6 weeks and 73 N at 14 weeks. In non-immobilized joints from the previous study [1], the capsular contribution was 130 N at 3 weeks, similar to normal values (105 N), but was reduced to 11 N at 6 weeks.

DISCUSSION

These results revealed several interesting findings. First, immobilization decreased the failure strength of both the healing isolated MCL and combined medial structures at 6 and 14 weeks. Second, immobilization affected the failure properties of the contralateral non-immobilized limb compared to when neither limb was immobilized. A significant effect of immobilization was likely not observed at 3 weeks because of this contralateral effect. Third, the load-sharing ratio of the failure load of the isolated MCL to the combined medial structures for the healing immobilized joints was not equivalent to that for normal joints at or before 14 weeks, unlike healing non-immobilized joints when this occurred between 3 and 6 weeks. This suggests that, for immobilized joints with isolated MCL injury, the capsular contribution is significant and dissection artifacts are a concern at later healing intervals. Immobilization delayed the transition from a capsule-dominated response to a MCL-dominated response in this model.

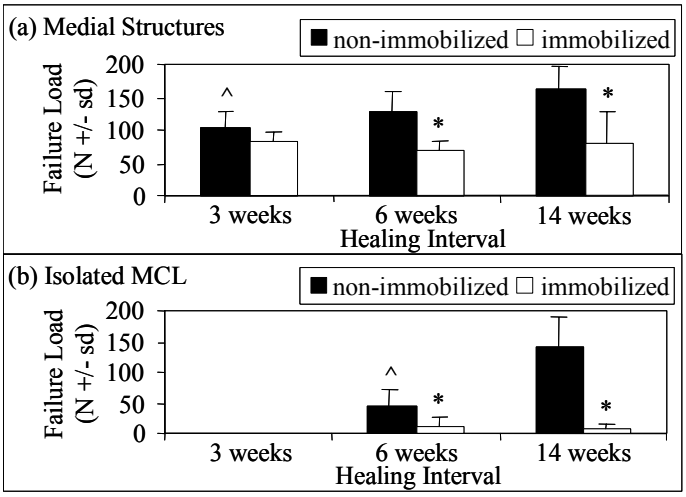

Figure 1. Failure loads of (a) combined medial structures (medial capsule plus MCL) and (b) isolated MCL following isolated MCL injury
*** less than non-immobilized ($p<0.01$)**
^ less than 14 weeks ($p<0.006$)

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