TENDON MICROTEARS IN AN ANIMAL MODEL OF EPICONDYLITIS CAUSED BY CYCLICAL LOADING

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INTRODUCTION:

Epicondylitis is a tendon disorder that can occur among athletes and workers who performing repetitive and forceful hand motions. Although it is associated with a high level of disability, the mechanisms of injury are not completely understood (1,2). It is commonly assumed that microtears are the initiating event of tendon injury, and microtears have been observed in tendon biopsies and sonograms from patients with overuse injuries (3,4). However, the size and distribution of microtears, and their relationship to loading are not well characterized. Knowledge of these factors may be useful in developing primary and secondary preventive measures.

Our laboratory has developed an in vivo rabbit model of epicondylits that involves repetitive loading of the flexor digitorum profundus (FDP) muscle. The purpose of this study was to determine whether the size and density of microtears in the FDP tendon were different in a limb exposed to repetitive finger loading in comparison to a control limb.

METHODS:

Seven female, young adult New Zealand White Rabbits were studied. Under general anesthesia, the FDP muscle of one limb was electrically stimulated to contract repetitively for 2 hours per day, 3 days a week, for 10 weeks (60h of cumulative loading). The contralateral limb served as the control. The study was approved by the University's Committee on Animal Research.

Each session of repetitive loading involved inducing anesthesia with Isoflurane with the rabbit in a supine position and the forearms loosely secured to supports. The muscle stimulation needle (33G) was inserted subcutaneously in the mid-forearm region over the central region of the FDP and the needle tip was pushed back through the skin in order to not injure the muscle. A needle was similarly inserted in the contralateral limb, but was not stimulated. A brass, fingertip glove was slipped over digit 3 and connected to a load cell to measure flexion force of the finger about the metacarpophalangeal joint. The muscle was stimulated (Grass-Telefactor, West Warwick, RI) with a train of pulses at 1Hz, with a train duration of 200ms and a pulse rate of 100 pulses/s. The stimulation voltage was adjusted [6-12V] to maintain a peak fingertip force of 0.42N (15% of P_o). After two hours of repetitive loading, the stimulation electrode and fingertip glove were removed.

At 10wks, the animals were sacrificed and the epicondyles were dissected with the FDP tendon and muscle attached. Samples were fixed, decalcified, sectioned, and serial sections were Trichrome stained. After white balancing, digitial photomicrographs were made at 200 X of the central region of the tendon approximately 1500 μm distal to the enthesis. Custom image analysis software (LabView, National Instruments, Austin, TX) automatically identified and measured every contiguous non-staining area (e.g., tear) of greater than 10 μm^2 in size. Distribution of number of tears by size and tear density were compared between the exposed and control limbs using the paired t-test. Preparation of tissues and photomicrographs were performed blinded.

RESULTS:

Examinations of the wrist, forearm and elbow revealed no limping, reduction in gross claw flexion strength, or skin breaks. Necropsy evaluation of the subcutaneous region of the stimulation needle insertion site revealed minimal scar tissue localized within 5 mm of the insertion site; the scar tissue did not extend near the FDP tendon.

The histogram distribution of tear sizes followed a power-log function. Summary measures of morphologic characteristics of the tears are presented in Table 1. The mean of the median size of the tears in the exposed limbs was 40.1 μm^2 (± 2.3) which was significantly larger than the control limbs value of 32.2 μm^2 (± 9.4) (paired t-test; p=0.04). There was a trend toward an increase in the ratio of the total tear area relative to evaluated total tissue area 0.16 (± 0.09) in the exposed limb in comparison to the control limb 0.07 (± 0.06) but the difference did not reach statistical significance (paired t-test, p=0.07). There was no significant difference between limbs in the number of tears per μm^2 0.0009 (± 0.0002) vs. 0.0007 (± 0.0002) (paired t-test, p=0.13).

	# of tears	Median tear size (μm^2)	Total tear area (μm^2)	Total image area (μm^2)	Ratio of tear area to image area
Exposed mean	1068	40.1	197,016	1,187,657	0.16
Exposed S.D.	303	2.3	122,914	147,222	0.09
Control mean	814	32.2	85,129	1,219,602	0.07
Control S.D.	274	9.4	72,457	98,624	0.06

Table 1. Mean values of tear morphology characteristics of tendons exposed to cyclical loading and tendons from control limbs (N=7).

DISCUSSION:

This *in vivo* model demonstrates that repetitive loading of the FDP muscle leads to an increase in the median size of microtears in the FDP tendon at the epicondyle in comparison to the control limb. The change was also associated with an increase in the total tear area. There was no significant difference in the density of tears. The findings suggest that microtears exist in normal tendons and that repetitive loading increases the size of existing microtears, but not the number of tears

The limitations of the study included the small number of animals studied, the biomechanical exposure pattern, the effects of histologic preparation, and the use of rabbits in the model. The load applied to the finger was well within the physiologic range of the muscle and the number repetitions and the duration of loading are less than that experienced by athletes and workers who perform repeated tasks (1). In addition, the rabbit forearm biomechanics and musculature are not identical to the human; therefore, generalization of these findings to humans should be carried out with caution.

REFERENCES:

(1) *Work-Related Musculoskeletal Disorders.* National Academy Press, Washington, D.C., 1999, p 98-115. (2) Archambault JM, Wiley JP, Bray RC. Sports Med. 1995; **20**:77-89. (3) Jozsa JM, Jarvinen KP, Kvist M, Leadbetter W. Scan J Med Sci Sports. 1997; **7**:86-95. (4) Gibbon WW, Cooper JR, Radcliffe GS. Br J Sports Med 1999; **33**:129-130.

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