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Biomechanical Cadaveric Evaluation of Partial Acute Peroneal Tendon Tears

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Abstract

Background: No clear guideline or solid evidence exists for peroneal tendon tears to determine when to repair, resect, or perform a tenodesis on the damaged tendon. The objective of this study was to analyze the mechanical behavior of cadaveric peroneal tendons artificially damaged and tested in a cyclic and failure mode. The hypothesis was that no failure would be observed in the cyclic phase.

Methods: Eight cadaveric long leg specimens were tested on a specially designed frame. A longitudinal full thickness tendon defect was created, 3 cm in length, behind the tip of the fibula, compromising 66% of the visible width of the peroneal tendons. Cyclic testing was initially performed between 50 and 200 N, followed by a load-to-failure test. Tendon elongation and load to rupture were measured.

Results: No tendon failed or lengthened during cyclic testing. The mean load to failure for peroneus brevis was 416 N (95% confidence interval, 351–481 N) and for the peroneus longus was 723 N (95% confidence interval, 578–868 N). All failures were at the level of the defect created.

Conclusion: In a cadaveric model of peroneal tendon tears, 33% of remaining peroneal tendon could resist high tensile forces, above the physiologic threshold.

Clinical Relevance: Some peroneal tendon tears can be treated conservatively without risking spontaneous ruptures. When surgically treating a symptomatic peroneal tendon tear, increased efforts may be undertaken to repair tears previously considered irreparable.

Keywords: peroneal tendon tears, rupture, 50% rule, tenodesis

Peroneal tendon pathology is a common source of lateral hindfoot pain. Peroneal tendon pathology can be divided into peroneal tendinitis, peroneal instability, and peroneal tendon tears.[14] The etiology of peroneal tendon tears is not well understood, although chronic damage and acute trauma in sports have been proposed. Different theories exist, one for the peroneus brevis consisting of subluxation of the peroneal tendon due to laxity of the superior peroneal retinaculum and consequent attritional damage against the peroneal sulcus, and another considering compression of the peroneus brevis by the peroneus longus inside the retromalleolar groove.[14] Peroneus longus tears are much less common than peroneus brevis tears, and classically they are reported to occur at the tip of the distal fibula, at the peroneal tubercle, or at the os peroneum.[3,12]

Treatment of peroneal tendon tears is generally determined by the size of the tear; for example, if the cross-sectional area of the tear is greater than 50%, tenodesis of the peroneus brevis to the longus has been recommended,[2,7,12,16] as it is believed that spontaneous ruptures may occur.[9] All articles refer to this last mentioned study, which reported basic experimental studies on rabbit Achilles tendons showing that some tendons would rupture in an in vitro animal model after 50% of their visible width had been severed, but no tendon failed in an in vivo animal study after 75% Achilles tendon damage had been inflicted.

Because of the scarcity of information relative to how to treat peroneal tendon tears, we performed this biomechanical study. The aim was to analyze the mechanical properties of human cadaveric peroneus brevis and longus tendons

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Figure 1. Picture of the special frame designed for the study. The specimen is held against an inclined plane through an endomedullary rod. The actuator pulls the peroneal tendons in line with the tendon excursion.

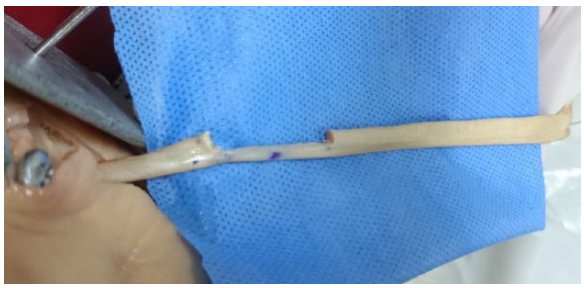

Figure 2. Picture of damaged peroneus longus tendon. Every defect created measured 3 cm in length and compromised 66% of the visible tendon width, as measured with a calibrated caliper.

subjected to artificially induced damage, compromising 66% of their visible width, tested in a cyclic mode and load to failure on a tensile testing machine. Our hypothesis was that the tendons would resist high tensile forces before rupturing under cyclic or failure mode testing.

Methods

Eight frozen cadaveric specimens of the lower leg were used, less than 65 years of age without previous operations or visible pathology. They were thawed at room temperature for 16 hours before testing. All manipulation, storage, and disposal of the specimens were performed according to approved protocols of our local anatomy department, following international standards.

The proximal end of each specimen was carefully dissected to identify the peroneus brevis and longus tendon. The proximal tibia was fixed to an intramedullary rod inserted into the bone, and stability was checked through manual testing. If deemed necessary, methyl methacrylate was used to further fix the rod to the tibia. Distally, at the level of the lateral aspect of the calcaneus, superficial dissection was performed until the distal insertion of the peroneus brevis was identified, and passage of the peroneus longus through the cuboid tunnel was also identified. The approach was used to expose the tendons distally and introduce artificial damage to each tendon. No further dissection on the peroneal tendons or retinaculum was performed. The specimens were then mounted on a specific frame to fix the tibia in line with the testing actuator (Kinetecnics, Santiago, Chile). A continuous 800-N load was applied to the intramedullary rod and controlled by a load cell, holding the specimen against the distal end of the frame where a foot plate was installed in 20 degrees of plantarflexion, to simulate an inclined plane where the foot rested (Figure 1). This amount of plantarflexion was chosen because it has been reported to correspond to the angulation at which the peroneal tendons are more prone to damage.[1]

One healthy specimen was tested without artificial damage to test the peroneus brevis tensile failure. To do this, the proximal end of the peroneus brevis tendon was reinforced with Fiberwire #2 (Arthrex, Naples, FL) and attached to a special clamp designed for tendon attachment. The clamp was secured to the testing machine, and load to failure testing was performed by pulling the tendon proximally until failure of the tendon or its attachments was noted. Eight specimens were then tested sequentially, in which a longitudinal full-thickness tendon defect was created on every peroneus longus and brevis tendon. This defect was centered behind the tip of the fibula and measured 3 cm in length and compromised 66% of the visible tendon width as measured by a calibrated caliper (Figure 2). All tendons were tested first in a cyclic fashion using 100 repetitions between 50 and 200 N. All specimens were retrieved distally and evaluated visually after this cyclic loading, measuring changes in the defect length. This evaluation was performed twice by 2 different observers. If no visual change or tendon failure was observed after the initial testing, a load-to-failure test was performed until tendon rupture, specimen damage, or fixation failure was observed. This procedure was performed for every peroneal tendon. The sequence of testing started with the cyclic test of the peroneus brevis, then the cyclic test of the peroneus longus, then the test to failure for the peroneus brevis, and finally the test to failure for the peroneus longus. Two peroneus brevis tendons were not included in the testing, because of iatrogenic damage sustained during the study setup. One lower leg specimen initially considered to be used as a healthy specimen to test the experimental setup presented a split rupture of the peroneus brevis tendon. This specimen was used to test the general setup with respect to frame size, actuator alignment, and clamp attachments to the tendons.

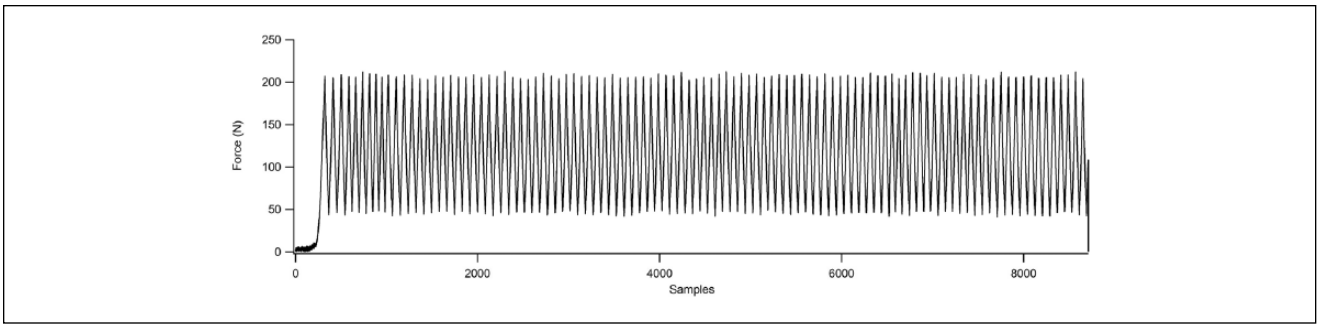

Figure 3. Diagram illustrating cyclic testing of 1 of the cadaveric tendons. One hundred cycles were performed for every tested tendon, between 50 and 200 N, as shown in the diagram. No mechanical change was observed for any tendon in this phase, as demonstrated by a symmetric tension curve.

Table 1. Load to Failure of Peroneal Tendons.

Tendon	Load to Failure (N)
PB #1	416.7
PB #2	416.7
PB #3	309.5
PB #4	485.3
PB #5	403.3
PB #6	468.5
PL #1	723.5
PL #2	667.9
PL #3	476.3
PL #4	622.3
PL #5	741.5
PL #6	849.3
PL #7	647.5
PL #8	1059.9

Abbreviations: PB, peroneus brevis; PL, peroneus longus.

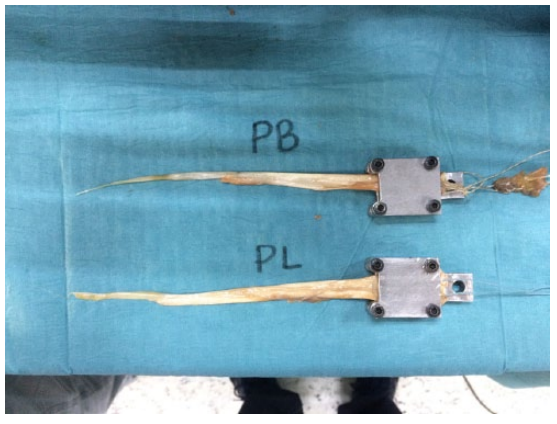

Figure 4. Picture showing a peroneus brevis and longus tendon after the failure phase. The clamp is seen at the far right of the picture. Every failure occurred through the defect created.

All results were analyzed using commercially available software, obtaining deformation values and tension in cyclic and failure testing. Statistical analysis was performed using SPSS software (SPSS, Chicago, IL).

Results

No tendon failed during the cyclic testing (Figure 3). Every tendon was examined, and no change in defect length was noted, as measured with the calibrated caliper.

In the load-to-failure testing, the peroneus brevis tendons failed at a mean of 416 ± 25 N. The peroneus longus tendons failed at a mean of 723 ± 173 N (see Table 1). All tendons failed at the defect site (Figure 4). The 95% confidence intervals for the load-to-failure testing were 351 to 481 N and 578 to 868 N for the peroneus brevis and peroneus longus tendons, respectively (Table 2). All data were distributed normally ($P > .50$, Shapiro-Wilk test). The coefficient of variation was 0.14 for the peroneus brevis tendons and 0.23 for the peroneus longus tendons.

Discussion

Peroneal tendon tears are relatively frequent, as they are present on cadaver dissections in up to 37%, and when treating ankle instability, they can be found in up to 30% of cases.[16] Ankle instability may be an important contributing factor, as it predisposes the tendons to subluxate, specifically the peroneus brevis, which may sustain attritional damage against the edge of the fibular groove, where most of the tears are located. The tears of the peroneus brevis generally present as splits centered behind the lateral malleolus, measuring 3.3 cm in length.[15] Recommendations for treatment appeared in the literature after the lesion was initially described by Meyer in 1924.[10] Subsequent descriptions identified the lesion,[1,8,13] but McMaster[9] was the first to suggest that spontaneous tendinous ruptures could occur when 50% of the tendon was damaged, inferred from data

Table 2. Load to Failure of Peroneal Tendons.

Tendon	Observations	Mean	Standard Error	95% Confidence Interval
Peroneus brevis	6	416.7	25.2	351.9–481.4
Peroneus longus	8	723.5	61.2	578.9–868.2

obtained by damaging rabbit Achilles tendons in an in vitro study in which spontaneous ruptures could occur after imposing that condition. Interestingly, in an in vivo study done concurrently, McMaster noted that when damaging 75% of the Achilles tendon width, allowing animal movement as tolerated, no tendon rupture occurred. Later work has used McMaster's study to propose treatment algorithms, such as Krause and Brodsky[7] in 1998, in which operative repair was proposed for peroneus brevis tears if damage was less than 50% of the tendon width, and a tenodesis was proposed if damage surpassed the 50% rule. Later studies and review articles have maintained the same proposed algorithm,[2,12] although no consensus exists, with wide variation in treatment decision making and rehabilitation.[4]

In our study, we showed consistently that no peroneal tendon failed in cyclic mode testing, with a defect compromising 66% of its visible width. Considering that the normal force to which the peroneal tendons are subjected is 18 N for the peroneus longus and 28 N for the peroneus brevis,[11] our cyclic testing considered high tensile forces (50–200 N), above the normal force transmitted to the tendons in the stance phase of gait. Considering that there is a trend toward shorter immobilization times and earlier onset of range of motion exercises,[18] the inherent resistance of the remaining peroneal tendon becomes more important when considering early weight bearing, especially considering the possibility of repairing tendon tears, and not performing transfers or tenodesis. Analyzing the load to failure test, the 95% confidence interval for the peroneus brevis tendon was 351 to 481 N and for the peroneus longus tendon was 578 to 868 N. These values are to be considered important for early range-of-motion exercises, when high torque can be expected to occur in the plantarflexor tendons. The Achilles tendon maximal force has been estimated to be 1924 N,[6] and if we consider the relative muscle strengths of the calf, the peroneus brevis and longus would be able to produce 217 N,[5] load that would be resisted with no rupture risk by the peroneal tendons, as per our study. Although the previous analysis provides useful information relative to the intrinsic resistance the peroneal tendons may have, other considerations must be taken into account when allowing early rehabilitation, as concomitant superior peroneal retinaculum repair, ankle ligament repair, and ankle muscular imbalance may render impossible an aggressive early rehabilitation.

Our study had various limitations, as the number of tendons tested was small, and this could have influenced the results. The coefficient of variation was low for both groups, 0.14 for the peroneus brevis group and 0.23 for the peroneus longus group. This suggests that the variation among tendons was low and therefore that it is a representative sample. It can be argued that the tendons used were healthy, and therefore the results and conclusions would apply only to nontendinopathic tendons. Although one specimen did present a peroneus brevis rupture, indicating the presence of tendinopathy, it is not possible to ascertain that tendinosis was equally present in the specimens tested compared with clinical patients, which certainly is a limitation. We did not perform histologic analysis searching for tendinopathy, which is an issue that could be investigated in the future. Our methodology was an attempt to represent a specific condition, fixing the ankle to 20 degrees of plantarflexion, not allowing any motion, and thus not representing a real clinical situation. However, there are no published cadaveric models for peroneal tendon tears, and therefore some assumptions and conditions had to be applied. An articulated model that could allow ankle motion is attractive and could be considered in a subsequent study. Relative to the tendon tears themselves, they were created as a rectangular defect compromising 66% of the visible tendon width. This damage does not occur in vivo, and therefore the resistance tested may not correlate with real clinical conditions. We decided to perform the described damage to standardize it and objectively test the resistance of 33% of the tendon substance remaining. The damage was created considering the visible width and was measured with a calibrated caliper. In these measurements, human error is present and therefore is a source of variation. We tried to diminish this error checking twice the measured damage, and indeed we discarded 2 specimens that were improperly performed. Finally, the cyclic testing considered 100 cycles between 50 and 200 N, which were cycles and loads arbitrarily assigned, with no validated model to follow. It can be argued that the number of cycles was too low compared with a normal loading amount. However, considering that in a postoperative situation the number of steps can be reduced to 1000 per day,[17] and the load applied to the peroneal tendons in midstance phase of gait is 28 N for the peroneus brevis and 18 N for the peroneus longus,[11] the total load (number of cycles multiplied by peak load) is approximately 23 kN. Our cyclic test considered 100 cycles with a peak load of 200 N, which amounts to a total load of 20 kN, which is similar to the estimated load in a postoperative situation.

We believe that peroneal tendon tears will be more and more frequent because of the increased physical activity of

older patients. Contributing factors such as peroneal tendon instability and ankle instability will have to be considered and ruled out when treating these disorders. When dealing with peroneal tendon tears, in symptomatic patients unresponsive to conservative treatment, it is important to decide which tear should be repaired and which could be treated with a tenodesis. On the basis of our findings, we suggest that it is safe to leave up to 33% of remaining tendon without risking spontaneous rupture, therefore not needing a tenodesis, which is the current indication. For high-demand patients, we strongly recommend repairing the rupture, either to itself (in case of a split rupture) or with a tendon autograft or allograft. Further studies are needed to determine the resistance of repaired tendons and whether the tendons benefit from grafting.

In conclusion, we believe that the 50% rule for peroneal tendon tears to decide between repair and tenodesis can be challenged. We suggest that some peroneal tendon tears can be treated conservatively without risking spontaneous ruptures, especially if the tear is mildly symptomatic or asymptomatic and when other correctable factors are present, such as ankle instability or a varus hindfoot. When operating on a symptomatic peroneal tendon tear, increased efforts may be undertaken to repair previously considered irreparable tears. Finally, we can also suggest that when rehabilitating a repaired peroneal tendon tear, earlier weight bearing and/or motion can be safely indicated without risking increased damage to the tendon, especially if there are no additional structures involved in the repair.

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