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original research

Medial Gastrocnemius Myotendinous Junction Displacement and Plantar-Flexion Strength in Patients Treated With Immediate Rehabilitation After Achilles Tendon Repair

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Context: Pathologic plantar flexion frequently occurs after operative repair of the Achilles tendon (AT) because of immobilization and non–weight bearing in the first weeks of traditional rehabilitation. Novel rehabilitation strategies that apply mobilization and weight bearing have been proposed, but their effects on medial gastrocnemius myotendinous junction displacement (MJD) and isometric plantar-flexion strength (PFS) are unknown.

Objective: To compare the effects of 12 weeks of immediate versus traditional rehabilitation on MJD and PFS in patients with percutaneous AT repair and to compare AT rupture scores (ATRSs) during follow-up.

Design: Controlled laboratory study.

Setting: Human performance laboratory.

Patients or Other Participants: A total of 26 amateur soccer players (age = 42.3 ± 9.7 years, body mass index = 29.5 ± 3.9 kg/m^2) with percutaneous AT repair.

Intervention(s): Athletes were randomly divided into 2 groups: an immediate group, given physical therapy from day 1 to day 84, and a traditional group, given physical therapy from day 29 to day 84. We used repeated-measures analysis of variance to compare the data.

Main Outcome Measure(s): We measured MJD and PFS at days 28 (fourth week), 56 (eighth week), and 84 (12th week) after AT repair.

Results: After 12 weeks of rehabilitation, we observed a large clinically meaningful effect and statistical difference between groups. At day 28, the immediate group showed higher values for PFS ($P = .002$), MJD ($P = .02$), and ATRS ($P = .002$) than the traditional group. At day 56, the immediate group presented higher values for MJD ($P = .02$) and ATRS ($P = .009$). At day 84, the immediate group registered more MJD ($P = .001$).

Conclusions: Compared with traditional rehabilitation, 12 weeks of immediate rehabilitation after percutaneous AT repair resulted in better MJD, PFS, and ATRS after 4 weeks; better MJD and ATRS after 8 weeks; and better MJD after 12 weeks.

Key Words: percutaneous technique, physiotherapy, lower extremity

Key Points

- Immediate rehabilitation after percutaneous Achilles tendon repair promoted more myotendinous junction displacement than did traditional rehabilitation.
- These results will help clinicians and physical therapists critically evaluate the effects of immediate rehabilitation on myotendinous junction displacement in patients with percutaneous Achilles tendon repair.

An Achilles tendon (AT) rupture is the most frequent tendon injury in the lower limb, with an incidence of 18 per 100 000 individuals each year,[1–5] most of whom are middle-aged[5–7] male amateur athletes.[1,8] Tendinous overload[6,9] and a nonuniform distribution of stress[10] promoted by neuromuscular imbalance[11] or pathologic muscular conditions[12] are mechanical factors that contribute to an AT rupture.[6,9,10] Rupture directly compromises motor tasks that require propulsion and takeoff actions, such as gait, running, and jumping,[11,13,14] and appropriate rehabilitation is required to improve the alterations, such as strength loss, that frequently develop after AT surgery.[13,15]

Different models have been proposed for rehabilitation after operative repair of the AT[3,4,6,16–21] and can be classified into 2 main groups based on the timing of mobilization and weight bearing. Rehabilitation programs

Table 1. Baseline Participant Characteristics, Mean ± SD (95% Confidence Interval)

Characteristic	Rehabilitation Group	
	Traditional (n = 13)	Immediate (n = 13)
Age, y	41.8 ± 11.5 (37.8, 47.9)	42.7 ± 7.8 (37.1, 45.8)
Height, m	1.7 ± 0.1 (1.7, 1.8)	1.7 ± 0.1 (1.7, 1.7)
Mass, kg	89.8 ± 15.3 (80.6, 98.9)	82.5 ± 13.0 (74.6, 90.3)
Body mass index, kg/m^2	30.8 ± 4.4 (28.1, 33.4)	28.2 ± 3.3 (26.2, 30.2)
Calcaneus-metatarsal length, cm	19.4 ± 1.9 (18.3, 20.6)	19.0 ± 1.9 (17.8, 20.2)
Achilles tendon rupture score	95.5 ± 12.1 (88.1, 100.0)	98.6 ± 3.7 (96.4, 100.0)
Injury-to-surgery interval, d	6.8 ± 3.0 (4.9, 8.6)	6.5 ± 2.6 (4.9, 8.1)
Uninjured plantar-flexion strength, N	629.8 ± 149.4 (539.6, 720.1)	652.1 ± 158.1 (556.6, 747.6)

that begin 4 weeks after AT repair represent the traditional rehabilitation model, which is standard practice.[21] Conversely, treatments beginning within the first 4 weeks after AT repair constitute the early rehabilitation model.[21] Two alternative early rehabilitation models exist. One was defined by Kearney et al[3] as *immediate rehabilitation* and starts in the first week after surgery. The other begins with some degree of immobilization or non–weight bearing in the first 4 weeks after AT repair, normally via a rest period during the first 2 weeks after surgery.[21]

Currently, managing AT rehabilitation after operative repair remains controversial[1,2,4,5,8,16,22] because of the unknown mechanical tolerances of different operative techniques[4,6] for preventing clinical failure[22]; the comorbidities associated with traditional management,[3–6,8,14,16,22] such as muscle atrophy, adherences, delayed collagen remodeling, thromboembolism, and high rerupture rates[2–6,22,23]; and the risks and complications that can occur during early rehabilitation,[3,4,6,15,21,22] such as tendinous elongation and early reruptures.[3,4,6,15,22] However, most treatments include recommendations for primary reattachment of the damaged ends to achieve appropriate mechanical stability, a percutaneous approach to avoid excessive tissue damage,[1,2,4,8] and early rehabilitation[3,5,6,8,10,16,21–23] to promote better clinical outcomes than are typical with open or traditional rehabilitation programs.[3,5,6,8,10,16,22,23]

Traditional models do not appear to provide the tissue and neuromuscular benefits that early models provide.[3,6,10,16,21–23] Indeed, they result in pathologic adaptations to plantar-flexion tasks.[12] For example, using velocity-encoded cine phase-contrast magnetic resonance images, Finni et al[12] found greater velocity and displacement of the medial gastrocnemius about the deep plantar flexors during submaximal isometric voluntary contractions 13 weeks after AT repair in patients treated with traditional rehabilitation compared with healthy control participants. Therefore, measuring myotendinous junction displacement (MJD) appears to be a useful tool for exploring muscle adaptations to plantar-flexion tasks in immediate rehabilitation models. Moreover, with their mechanical model, Muramatsu et al[24] demonstrated more MJD due to shortening of the medial gastrocnemius contractile units that displace the deep and superficial aponeurosis. Similarly, Duclay et al[25] found a linear relationship between MJD and internal shortening of the fascicle in the medial gastrocnemius when measured at 50% and 100% of maximal voluntary isometric contraction (MVIC) in a control group and a group participating in a 7-week eccentric strengthening program for the plantar-flexor muscles.

Researchers[14] have also proposed that the main neuromechanical adaptations of the plantar flexors may occur within the first 12 weeks after AT repair. However, it is unclear if immediate rehabilitation beginning the first day after operative repair and lasting for 12 weeks could improve MJD and plantar-flexion strength (PFS) during isometric plantar-flexion tests. Therefore, the primary purpose of our study was to compare the effects of 12 weeks of immediate rehabilitation versus traditional rehabilitation on MJD and PFS in patients with percutaneous AT repair. Moreover, AT rupture scores (ATRSs) were compared during patient follow-ups. We hypothesized that 12 weeks of immediate rehabilitation would result in better MJD, greater PFS, and a higher ATRS than traditional rehabilitation after percutaneous AT repair.

METHODS

Study Design

This randomized, controlled laboratory study was performed in the biomechanical unit of Instituto Traumatológico from June 2014 to March 2015. The MJD and PFS were assessed in amateur soccer players 28 (fourth week), 56 (eighth week), and 84 (12th week) days after percutaneous repair of an acute[3] midsubstance AT rupture.[26] These variables were assessed in patients rehabilitated from days 1 to 84 after surgery (immediate group; n = 13) and from days 29 to 84 after surgery (traditional group; n = 13).

Participants

We studied 26 amateur soccer players with percutaneous AT repair.[26] Simple randomization placed each patient in 1 of 2 standardized rehabilitation groups (ratio 1 : 1), with 13 patients in each group (Table 1). Random patient numbers were generated electronically by the MATLAB software (version 2010a; The Mathworks Inc, Natick, MA), such that the immediate group comprised patients 1, 4, 5, 7, 10, 11, 14, 15, 17, 20, 21, 23, and 26, and the traditional group comprised patients 2, 3, 6, 8, 9, 12, 13, 16, 18, 19, 22, 24, and 25.

Patients' baseline characteristics were recorded before operative intervention (Table 1). The same physical therapist (C.D.) recorded age, height, mass, body mass index, ATRS[7] before injury, injury-to-surgery interval, distance from the third metatarsal head to the calcaneal tuberosity (calcaneus-metatarsal length), and uninjured isometric PFS (Table 1). The MVIC was determined using

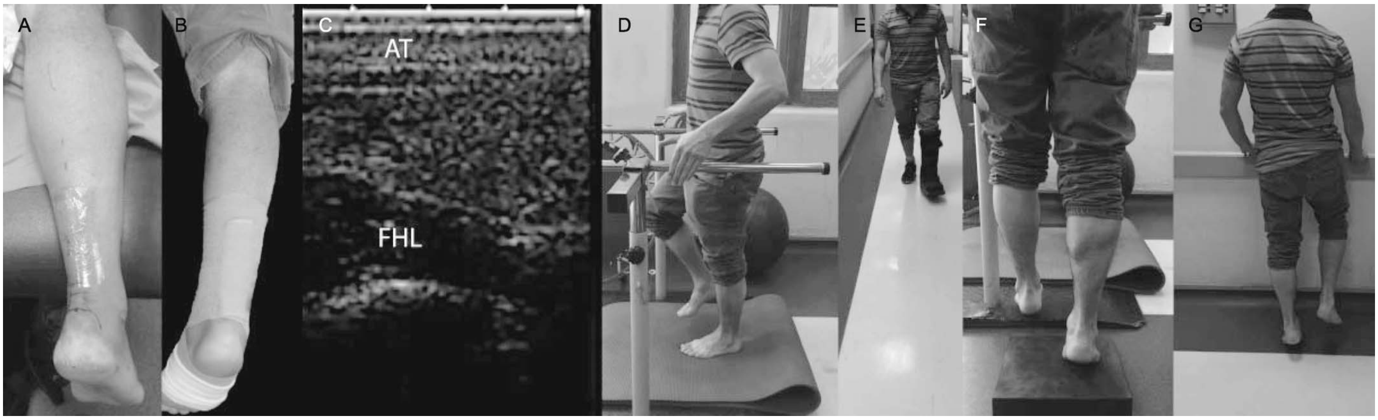

Figure 1. Immediate rehabilitation. The first phase consisted of A, wound protection, B, wound compression, and C, clinical failure monitoring (Achilles tendon [AT]) and deep flexor feedback (flexor hallucis longus [FHL]). The second phase consisted of D, 1-legged balance exercise, and E, full weight bearing. The third phase consisted of F, plyometric exercises in plantar-flexion range, and G, 1-legged heel-raise capacity at week 12.

the uninjured limb because of the impossibility of performing the strength test on the injured limb.

An a priori analysis was conducted using G*Power software (version 3.1.9.2; Universität Düsseldorf, Düsseldorf, Germany), which yielded a sample size of 12 patients per group with a statistical power of 80% and a type I error rate of 5% for a mixed repeated-measures analysis of variance.

Inclusion criteria were (1) men aged 20 to 55 years; (2) *amateur soccer players*, defined as participating in 1 or 2 practice sessions per week; (3) first midsubstance AT rupture; (4) injury-to-surgery interval $\leq$10 days; (5) percutaneous AT repair performed according to Amlang et al[26] using FiberWire No. 2 (Arthrex Inc, Naples, FL)[2]; and (6) written informed consent. Exclusion criteria were (1) autoimmune disease; (2) previous tendinous injury of the lower limb; (3) history of trauma; (4) orthopaedic alterations of the lower limb; (5) history of chronic steroid therapy; (6) inability to receive postoperative treatment at our facility; (7) postoperative rehabilitation dropout[11]; (8) *lack of consistent attendance*, defined as a greater than 50% absence rate from the total scheduled sessions; (9) thrombotic disease development[11]; (10) cognitive alterations; or (11) any condition that would alter the assessed variables. The study was approved by the comité de ética del Instituto Traumatológico.

We did not include dropouts in this study but did exclude 2 patients. One patient in the immediate group developed deep venous thromboembolism of the treated limb less than 6 hours after surgery and was referred to the emergency department for medical treatment. One patient from the traditional group was excluded after a rerupture at week 8 postrepair that required a subsequent operation.

Interventions

Each patient participated in 1 of 2 standardized physical therapy programs that differed for only the first 28 days. These programs were structured in 3 main phases. The first phase (days 1–28) consisted of principles of inpatient education, wound and tendon protection, and controlled mobilization and weight bearing. The second phase (days 29–56) involved principles of inpatient education, tendon protection, controlled stretching, and isometric and concentric strengthening. The third phase (days 57–84) incorporated principles of inpatient education, stretching, and controlled eccentric and plyometric strengthening. Patients advanced from the first to the second phase if they could tolerate gait without crutches, could perform active plantar-flexion exercises, did not have a local infection, and had had the wound suture removed. Patients progressed from the second phase to the third phase if they could tolerate full weight bearing without crutches and a 1-legged plantigrade position.

The immediate group participated in physical therapy for 1.5 hours 3 times per week from days 1 to 84 after surgery.[12] During the first phase, the immediate group was treated by the same physical therapist (C.D.). In turn, the traditional group remained immobilized and non–weight bearing for the first 28 days after surgery. From days 29 to 84, all patients received rehabilitation treatment for 1.5 hours 3 times per week.[12] During the second and third phases, all patients were treated by a second physical therapist (R.P.).

Wound Treatment. To protect the surgical wound after percutaneous AT repair, patients used transparent film dressings (Tegaderm; 3M, St Paul, MN) that were replaced weekly during the first phase. The wound was compressed (pressure therapy) by a 2 $\times$ 10-cm rectangular unit of ethyl vinyl acetate within a therapeutic bandage using self-adherent wrap (Coban; 3M). This bandage was replaced after each physical therapy session (Figure 1A and B).

Plantar-Flexion Exercises. To stimulate the plantar flexors, patients performed active concentric contractions of the plantar flexors.

During the first phase, patients in the immediate group lay supine and performed 120 cycles per week (4 series with 10 repetitions 3 times per week) between full plantar flexion and –15° of dorsiflexion without resistance on days 1 to 7, 120 cycles (4 series with 10 repetitions 3 times per week) between full plantar flexion and –15° of dorsiflexion against a yellow elastic band (Theraband, Akron, OH) placed at the level of the metatarsals on days 8 to 14, 180 cycles (4 series with 15 repetitions 3 times per week) between full plantar flexion and –7° of dorsiflexion against a red elastic band (Theraband) placed at the level of the metatarsals on days 15 to 21, and 180 cycles (4 series with

15 repetitions 3 times per week) between full plantar flexion and 0° of dorsiflexion against a red elastic band placed at the level of the metatarsals on days 22 to 28. Patients were monitored for the first 2 weeks using a B-mode ultrasound (Soundmed Inc, Miami, FL) equipped with a 7.5-MHz linear transducer to determine if tendon gapping occurred during exercise. Separation of the tendon ends of more than 5 mm was considered a clinical failure according to Orishimo et al.[22]

For the second phase, immediate and traditional group patients performed 240 cycles per week (4 series with 20 repetitions 3 times per week) between full plantar flexion and 0° of dorsiflexion against a blue elastic band (Theraband).

During the third phase, both groups performed 300 cycles per week (4 series with 25 repetitions 3 times per week) between full plantar flexion and full dorsiflexion against a gray elastic band (Theraband).

Deep Plantar-Flexion Exercises. To encourage tendon gliding of the deep plantar flexors about the AT and to prevent adherence processes during the first phase, patients performed 360 cycles per week (4 series with 30 repetitions 3 times per week) of active flexor hallucis longus and flexor digitorum longus contractions without resistance in a supine position. Patients were trained through feedback and monitored for the first 14 days using a B-mode ultrasound equipped with a 7.5-MHz linear transducer to determine if tendon gapping occurred during exercise (Figure 1C).

Heel-Raise Exercises. To strengthen the plantar flexors, patients performed 3 types of heel-raise exercises: a sustained 2-legged heel raise, a 2-legged heel raise, and a 1-legged heel raise. For the first phase (days 15–28), patients maintained a 2-legged heel raise with full plantar flexion (2 series with 10 repetitions of 10 seconds 3 times per week) and the upper extremities supported by parallel bars. For the second phase, patients performed 120 2-legged heel raises per week (3 series with 20 repetitions 3 times per week) between 0° of dorsiflexion and full plantar flexion with the upper extremities supported by parallel bars to assist with posture. For the third phase, patients performed 300 one-legged heel raises per week (4 series with 25 repetitions 3 times per week) between 0° of dorsiflexion and full plantar flexion with the upper extremities supported by parallel bars to assist with posture and to emphasize a slow, controlled heel drop.

Balance Exercises. To promote postural control, patients performed plantigrade exercises. For the first phase, patients maintained a vertical posture in a 2-legged plantigrade position on an exercise mat (Theraband) with alternating closed and open eyes in front of a mirror (2 series with 10 repetitions of 20 seconds 3 times per week). During the second and third phases, all patients maintained a vertical posture in a 1-legged plantigrade position on an exercise mat with alternating closed and open eyes in front of a mirror (2 series with 10 repetitions of 20 seconds 3 times per week; Figure 1D).

Controlled Mobilization and Weight Bearing. To protect the AT against overload and to diminish the risk of rerupture and elongation, patients used an articulated walking boot (DJO Global Inc, Vista, CA; Figure 1E).

During the first phase, the immediate group performed a mobilization progression of –15° of dorsiflexion on days 1 to 14, –7° of dorsiflexion on days 15 to 21, and 0° of dorsiflexion on days 22 to 28. Patients also progressed gait, walking with 10 kg of maximum weight bearing and 2 crutches on days 1 to 7; 25 kg of maximum weight bearing and 1 crutch on days 8 to 14; 40 kg of maximum weight bearing without crutches on days 15 to 21; and full weight bearing on days 22 to 28.

Stretching. Patients performed closed chain plantigrade dorsiflexion during the second and third phases to recover the same range of motion as the uninjured limb.

Plyometric Exercises. Patients enhanced the plyometric capacity of the plantar flexors during days 57 to 70 of the third phase. They performed 300 two-legged heel raises per week between –15° of dorsiflexion and full plantar flexion (4 series with 25 repetitions 3 times per week) with the upper extremities supported by parallel bars to assist with posture and to emphasize a fast heel raise and a slow, controlled heel drop. The dorsiflexion range was limited by an inclined plane (Figure 1F). For days 71 to 84, patients performed 300 one-legged heel raises per week (4 series with 25 repetitions 3 times per week) between 0° of dorsiflexion and full plantar flexion with the upper extremities supported by parallel bars to assist with posture and to emphasize a fast heel raise and a slow, controlled heel drop (Figure 1G).

Takeoff Exercises. On days 71 to 84 of the third phase, patients performed 180 running takeoffs per week, starting against a vertical wall (4 series with 15 repetitions 3 times per week), to enhance the takeoff capacity of the plantar flexors.

Controlled Running Exercise. To improve the eccentric capacity of the plantar flexors, patients performed 5 minutes of slow running with metatarsal support (3 times per week) on days 71 to 84 of the third phase.

Achilles Tendon Rupture Score

The ATRS was calculated by the same physical therapist (R.P.) according to the methods of Nilsson-Helander et al.[7] Minimal clinically important differences were defined when scores were equal to or greater than 10 points.[7]

Isometric PFS

To determine PFS, each patient sat in 90° of hip flexion, with vertical alignment of the trunk, and 90° of knee flexion that was fixed by a compressive load that limited the angular change and segmental shift, and with the ankle at 0° (Figure 2).[2] After patients adopted the testing position, they performed 3 maximal attempts guided by pain perception of equal to or less than 8 of 10 during each evaluation. Pain perception was determined using a verbal analogue scale, ranging from 0 (*absence of pain*) to 10 (*maximal pain*). Patients rested for 3 minutes between attempts. The mean of the 3 maximal attempts was used for statistical analyses. Patients were aware of their strength level, which was visually projected in real time through a performance curve of strength (N) minus time (seconds; delay $\leq$0.001 seconds). We quantified PFS using an S-beam load cell (Intertechnology Inc, Toronto, ON, Canada) placed at the level of the metatarsals (third metatarsal head) and a foot plate[14,27] adapted to the plantar surface. Contact with the foot plate was ensured through rigid fixtures on the forefoot, midfoot, and ankle (Figure 2). Minimal clinically

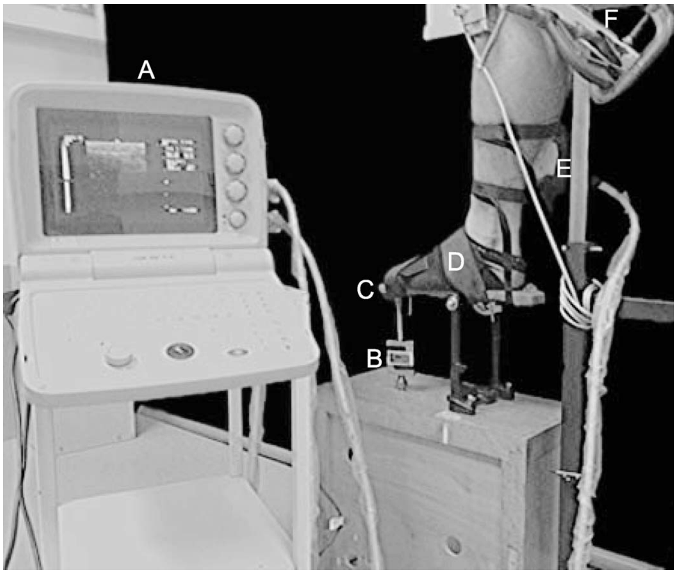

Figure 2. Experimental design for isometric plantar-flexion strength. A, B-mode ultrasound. B, S-beam load cell. C, Foot plate. D, Rigid fixtures for the foot and ankle. E, A 7.5-MHz linear transducer. F, Rigid fixtures for the knee.

important differences were defined when changes were greater than 10%.[28]

Medial Gastrocnemius MJD

The MJD was quantified arithmetically as the mean difference between the position of the medial gastrocnemius myotendinous junction at maximal contraction and its position at rest. Three maximal attempts were performed, as described for PFS. The myotendinous junction was assessed using a B-mode ultrasound equipped with a 7.5-MHz linear transducer[29] placed according to Kay and Blazevich.[30] Raw images similar to those reported by Muraoka et al[31] were obtained (Figure 3).

We measured the MJD in a protected position with the patient on 1 side, mechanically disadvantaging the triceps surae[32,33] and creating lower levels of tension over the repaired AT.[22,33] This position was adapted during MVICs to prevent any damage to the patient, such as an AT rupture due to excessive tension. Furthermore, this position was used to generate a motor task that would deform the medial gastrocnemius aponeurosis, as in the study of Arampatzis et al,[27] who demonstrated displacement of the plantar flexors in uninjured participants during MVIC. In addition, Muramatsu et al[24] proposed that the underlying mechanism of aponeurosis deformation of the medial gastrocnemius is the shortening of muscle fibers during normal muscle contraction. For the testing position, we took measurements during normal muscle contractions using electromyograms, and fascicle-length graphs indicated that the medial gastrocnemius was activated.[33] Therefore, we hypothesized that better rehabilitation, as reflected by this behavior, would be detected through MJD exploration.

Minimal clinically important differences were defined as changes equal to or greater than 2 mm. This value was based on the degree of MJD found at 40%, 60%, and 80% for MVIC[27] and at 30% and 60% for maximal anisometric muscle contractions.[34]

Data Analysis

Data are expressed as mean ± standard deviation. The relative rate (%) between the interventions is expressed with a 90% confidence interval.[35] Normality and homoscedasticity of the data were verified with Shapiro-Wilk and Levene tests, respectively, using SPSS (version 19; IBM Corp, Armonk, NY). Each variable was assessed using the Mauchly test of sphericity with no need for a Greenhouse-

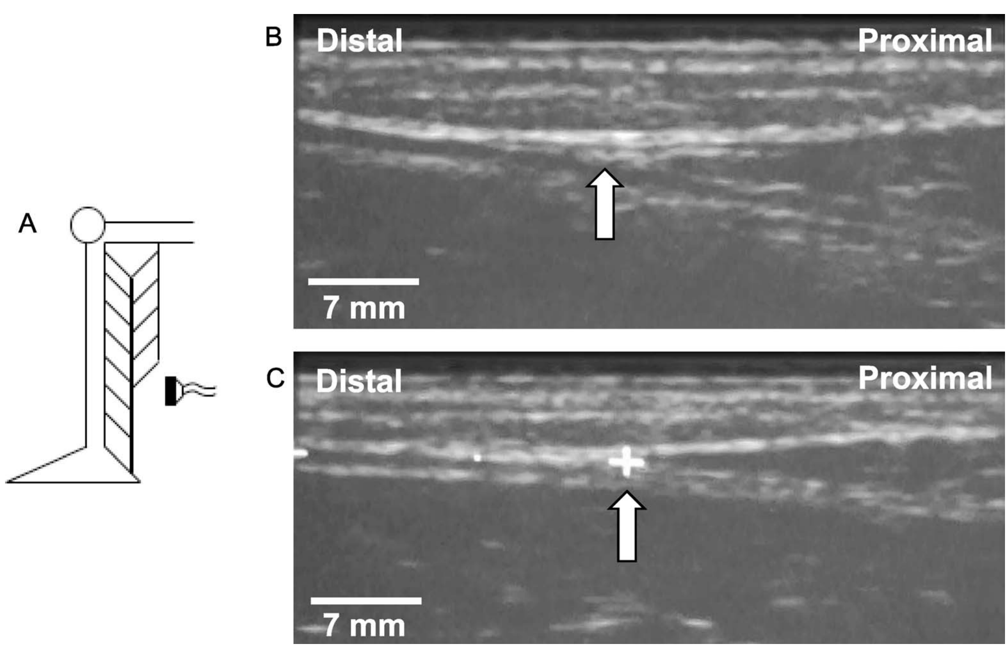

Figure 3. A, Location of the longitudinal transducer on the medial gastrocnemius myotendinous junction. Sagittal-plane ultrasound image of the medial gastrocnemius myotendinous junction (white arrow) for patient 1 at week 4 after percutaneous Achilles tendon repair shows myotendinous junction displacement, B, at rest and, C, at maximal voluntary isometric contraction.

Table 2. Results of Patient Follow-Up

Variable	Week	Rehabilitation Group, Mean ± SD		P Value	Effect Size or Standardized Difference (95% Confidence Interval)	Magnitude Interpretation	Chance of Effect, %			Minimal Clinically Important Difference
		Traditional (n = 13)	Immediate (n = 13)				Better	Trivial	Weaker	
Isometric plantar-flexion strength, N	4	162.5 ± 64.8[a]	281.3 ± 103.3	.002	1.38 (0.62, 2.05)	Large	99.6[b]	0.4	0.0	Important
	8	345.7 ± 141.1	418.0 ± 140.4	.20	0.51 (−0.16, 1.15)	Moderate	71.4[c]	25.8	2.8	Important
	12	455.0 ± 127.3	490.6 ± 112.2	.46	0.30 (−0.36, 0.94)	Small	51.0[c]	41.2	7.8	Not important
Myotendinous junction displacement, mm	4	4.1 ± 2.5[a]	6.1 ± 1.7	.02	0.94 (0.23, 1.58)	Large	16.9[d]	83.1	0.0	Important
	8	7.5 ± 2.0[a]	9.2 ± 1.5	.02	0.96 (0.25, 1.61)	Large	34.8[d]	65.2	0.0	Important
	12	8.1 ± 1.4[a]	10.2 ± 1.4	.001	1.47 (0.70, 2.14)	Large	96.2[e]	3.8	0.0	Important
Achilles tendon rupture score	4	18.6 ± 14.0[a]	42.7 ± 12.2	.002	1.72 (0.92, 2.42)	Large	99.7[b]	0.3	0.0	Important
	8	42.7 ± 14.6[a]	60.4 ± 14.9	.009	1.20 (0.46, 1.86)	Large	98.2[e]	1.7	0.1	Important
	12	64.4 ± 19.4	74.2 ± 14.2	.21	0.58 (−0.10, 1.22)	Moderate	74.2[c]	22.4	3.4	Important

[a] Indicates differences between groups ($P < .05$).
[b] Indicates almost certain clinical benefit.
[c] Indicates possible clinical benefit.
[d] Indicates unlikely clinical benefit.
[e] Indicates very likely clinical benefit.

Geisser adjustment for $P < .05$. The effects of the intervention were evaluated using a 2×3 mixed repeated-measures analysis of variance with the α level set at .05 to describe the interaction of intervention $\times$ time. Confidence and P values were adjusted with a Bonferroni correction. When an F value was different, a post hoc Bonferroni analysis was performed to determine the difference between means.

In addition to the null-hypothesis method, data were assessed using the magnitude-of-change model[35,36] and the clinically meaningful calculations spreadsheet published by Hopkins.[37] Standardized differences or effect sizes for each variable were calculated using a pooled standard deviation.[38] Threshold values for the magnitude evaluation were described as *small* (>0.2), *moderate* (0.5), and *extensive* (>0.8).[38] Quantitative changes for a beneficial/better or negative/poor effect were qualitatively assessed as *almost certainly not* (<1%), *very unlikely* (1%–5%), *unlikely* (5%–25%), *possible* (25%–75%), *likely* (75%–95%), *very likely* (95%–99%), and *almost certain* (>99%).[39] If beneficial/better or negative/poor effects were both greater than 10%, the true difference was assessed as unclear.[36]

RESULTS

A normal distribution was obtained for all assessed variables ($P > .05$). Groups showed similarities in all baseline characteristics ($P > .05$; Table 1). The findings are summarized in Table 2.

DISCUSSION

Our principal finding was that immediate rehabilitation[37,40] resulted in better MJD at the end of intervention. Given the model of clinically meaningful effect size,[37,40] this finding implied that a high chance of generating a very likely clinical benefit with a minimal clinically important difference may exist if 12 weeks of immediate rehabilitation are applied after percutaneous AT repair (Table 2). In contrast, PFS and ATRS measurements were not different among treatments (Table 2). These results are in accordance with Finni et al,[12] who reported that patients immobilized after AT repair developed a pathologic plantar-flexor synergy that resulted in less muscle displacement of the medial gastrocnemius and upper muscle displacement of the deep plantar flexors. However, debate still exists about the cause of less muscle displacement of the medial gastrocnemius. This displacement may be due to neural factors of plantar-flexor disuse in uninjured participants,[41] pathologic mechanical factors (eg, adherences) after AT repair (as described in a case report),[42] or a combination of these factors. New research models are needed to fully understand this interaction.

After surgery, a repaired AT has less tensile resistance during healing.[5,22] This critical resistance persists for 6 to 8 weeks after surgery, according to the histologic repair process,[10] and if high deformation is induced over the repaired zone too soon during healing, a risk for a rerupture exists. Given these factors, the traditional rehabilitation model, which is based on non–weight bearing and immobilization, remains standard practice after AT repair.[3–6,8,14,16,21] However, the negative changes to plantar flexors[11,12] may be modified, as our findings suggested. Moreover, magnetic resonance imaging revealed that an organized scar forms over the AT during the 4 weeks after surgery in early rehabilitation models,[6] and no differences in rerupture rates between early and traditional models have been described in systematic reviews or meta-analyses.[3,16] These studies support our observation that immediate rehabilitation beginning the first day after percutaneous AT repair would result in positive clinical benefits over traditional rehabilitation.

Measurements of PFS at the end of treatment demonstrated that patients achieved maximal isometric plantar flexion, with no differences between groups. Moreover, no clear clinical benefits or minimal clinically important differences were identified between groups at the end of treatment. Wang et al[14] found that less agonist activation was needed to generate explosive contractions in the first 12 months after AT repair, which could explain the similar PFS levels between groups at week 12 in our study. This suggests that primary alterations to joint strength could manifest through explosive generation and not in an isometric test, such as that used to measure PFS in our study. Identifying and improving these deficits is of great clinical importance when we consider that 65% to 80% of plantar-flexor torque is provided by the superficial plantar-flexor mechanism,[12] which involves the medial gastrocnemius.

Importantly, week 4 PFS differed between the immediate and traditional groups, almost certainly provided clinical benefits, and produced a minimal clinically important difference. We believe this finding is clinically relevant for preventing patient injury. Given that traditional treatment establishes at least 4 weeks of immobility and non–weight bearing, these patients may be more susceptible to injuries at the start of physical interventions. Therefore, from a clinical perspective, traditionally treated patients should be provided with bolstered protective strategies, such as detailed patient education and the extended use of an articulated walking boot. Silbernagel et al[15] reported a 10% to 30% difference in long-term PFS between repaired and uninjured limbs. For long-term PFS, we observed minimal clinically important differences for the immediate (24.8%) and traditional (27.8%) groups. To advance to the sport-reintegration phase, patients should receive additional strength and neuromuscular training.[11]

At week 12, the immediate group's MJD was 10.2 $\pm$ 1.4 mm, which is within the range of physiologic values reported for maximal plantar flexion in uninjured participants by authors of in vivo studies, including Magnusson et al[43] (12.9 $\pm$ 1.4 mm), Muraoka et al[31] (10.8 $\pm$ 2.4 mm), and Maganaris et al[29] (10.3 $\pm$ 2 mm). This range was different from that of the traditional group: 8.1 $\pm$ 1.4 mm at week 12. Therefore, immediate rehabilitation for 12 weeks after percutaneous AT repair may promote isometric plantar flexion that is closer to physiologic muscle synergy.[12]

The lower MJD in the traditional group has clinical implications. Specifically, isometric plantar flexion performed with decreased MJD suggests the acquisition of pathologic neuromuscular plasticity processes (ie, motor adaptations). This phenomenon has been described in uninjured individuals with plantar-flexor disuse after 4 weeks of immobilization and non–weight bearing[3,41] and in animal models treated with immobilization for the first 21 days after AT repair.[44] In the latter study, the levels of neural growth proteins CRMP2 and gelsolin isoform-b were lower in animals treated with immobilization than in those treated with immediate rehabilitation.[44] This hypothesis is further supported by Clark et al,[41] who reported that neural factors were the greatest (48%) contributor to the loss of joint strength during 4 weeks of plantar-flexor disuse. In our study, just 4 weeks of immobilization and non–weight bearing resulted in poor MJD. Nevertheless, electrophysiologic corroboration of this hypothesis is needed.

Another clinical implication of lowered MJD is alterations in muscle synergy, such as pathologic coactivation or pathologic muscular timings as shown on integrated electromyography. In turn, these alterations may result in a predisposition to tendinous overload, a factor in degenerative pathologic conditions and AT rupture.[9,10] A greater chance of tendinous overload exists from week 12 after percutaneous AT repair because of a greater number of takeoff activities that increase repetitive stress on the AT; however, these tasks are needed to advance in performing daily activities and in sport reintegration. Therefore, improving the restoration of physiologic plantar-flexion synergy is relevant in preventing new injuries that result from the pathologic actions of the musculoskeletal system[11,13] and is especially pertinent before starting exercises that place high levels of repetitive stress on the repaired AT. Considering this, clinicians must not only incorporate an early intervention plan of mobility or weight bearing after the AT repair but must also consider an appropriate neuromuscular plan, such as that proposed by Kearney et al,[3] Don et al,[11] and Finni et al.[12]

The final ATRS of the immediate group revealed fewer symptoms of autoperception with minimal clinically important differences compared with the traditional group after percutaneous AT repair.

Complications of AT repair included deep venous thromboembolism and rerupture.[3,16] For early rehabilitation models, researchers have reported that the rate of rerupture is approximately 4%[16] and the rate of deep venous thromboembolism is approximately 6%.[16] We observed 1 rerupture (7%; 1 of 14) in the traditional group and 1 case (7%; 1 of 14) of deep venous thromboembolism in the immediate group.

The sample population of patients in our study has been described as "weekend warriors," middle-aged men with a high risk for an AT rupture or rerupture due to the intermittent practice of competitive sports.[4,8,10,23] Hypoxia- and mechanically induced degenerative changes can arise from this behavior; histologic findings[5,6,10] have demonstrated that spontaneous AT ruptures occur in these patients, with nonuniform distribution of stress resulting in vascular injury due to ischemic torque placed on the AT. Therefore, in addition to an adequate postoperative intervention plan, these patients require lessons in appropriate techniques for sport reintegration and regular sport practice.

Some limitations of our study were the lack of a multicenter design and of electromyographic and dynamic motor-task measurements. Nevertheless, our research approach presented several strengths, including the use of validated methods and measurement instruments, study of a randomized and homogeneous sample, and the inclusion of patients with a high risk of AT rupture. Our results are a valuable addition to the currently limited knowledge on the effects of immediate rehabilitation after percutaneous AT repair.

In the future, authors need to identify the neuromuscular causes of muscle synergy changes after percutaneous AT repair and the neuromuscular benefits of different exercises during rehabilitation. In addition, more operative techniques are needed to demonstrate appropriate clinical failure tolerances to prevent tendon lengthening, the results of which could be used to refine immediate rehabilitation models.

CONCLUSIONS

Our findings provided novel evidence that 12 weeks of immediate rehabilitation promoted better MJD than traditional rehabilitation after percutaneous AT repair, without resulting in differences in PFS or ATRS at the end of rehabilitation.

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