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Biologically Enhanced Hamstring Tendon Transfer for Treatment of Acute Rupture of Posterior Tibialis Tendon in an Athlete: Case Report

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ABSTRACT

We report the case of a 32-year-old basketball player who presented with an acute flatfoot deformity after performing a unipodal power jump. Rupture of the posterior tibial tendon within the foot was diagnosed and then treated by hamstring tendon transfer combined with application of autologous biologic preparations. The functional outcome at 18 months was good. To our knowledge, this is the first reported case of posterior tibial tendon rupture treated by hamstring tendon transfer.

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Rupture of the posterior tibial tendon (PTT) inside the foot can arise not only when the tendon is inflamed, which is the first stage of PTT insufficiency and the most common scenario (1), but also when the tendon is normal (2,3). Acute rupture of an otherwise normal PTT has been described in association with ankle fracture (4). The typical mechanism of injury is pronation and external rotation of the foot leading to medial malleolus fracture, as described by the Lauge-Hansen classification system (5,6). Primary end-to-end suture is generally advised, and outcomes of this approach are generally good (7–9). However, there are cases in which primary suture is not possible because the edges of the tendon are severely damaged or the ends are retracted. In such cases, tendon transfer is clearly indicated (10). This is especially true in patients with a history of medial ankle pain or flatfoot deformity. Flexor digitorum longus (FDL) transfer is a well-known option in the treatment of stage 2 PTT dysfunction, which is characterized by flexible acquired flatfoot deformity, as per the Meyerson classification (11,12). FDL transfer with good outcomes has been described in the acute setting (10,13). Other tendons have been used for replacement or augmentation of the PTT. However, all of them involved the foot (14,15).

Biologic solutions, especially administration of autologous biologic preparations, are gaining importance in the treatment of musculoskeletal injuries (16). Platelet-rich plasma (PRP) and stem cells have been widely

used to encourage soft tissue regeneration, and results with respect to tendon regeneration have been promising (17). We describe semitendinosus tendon transfer combined with administration of a mixture of autologous PRP and adipose-derived stem cells (ASCs) and the outcome of this therapy at 18 months in a fairly young athlete who suffered acute PTT rupture while being monitored for medial ankle pain.

Case Report

The patient was a 32-year-old professional basketball player who visited us after a championship game in which he heard a crack in his right ankle after performing a unipodal power jump. He had been unable to continue the game. The initial physical examination revealed pain and tenderness behind the right medial malleolus, complete loss of the right foot arch, and the “too many toes” sign. He was unable to actively invert his right foot or perform a single right leg heel rise. We noted that the team physician had been monitoring the patient for nearly 2 years because he had suffered medial right ankle pain, which had led to a diagnosis of PTT dysfunction. The dysfunction had been refractory to conservative treatment, including rehabilitation and use of orthotics, and the foot and ankle disability index score (sports module) remained at 59.4. Corticosteroid had been injected into the retromalleolar region 15 days before the accident. No bony lesions were seen on radiographs of the right ankle obtained on the patient's presentation to us. Rather, both severe tendinosis and a high-grade 5-cm interstitial tear extending from the distal portion of the tendon to the level of the medial malleolus

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Fig. 1. Magnetic resonance images of the posterior tibialis tendon (PTT) obtained on the patient's presentation to us. (A) Axial short tau inversion recovery image shows severe tendinosis indicated by abnormally increased signal intensity and diffuse thickening, associated with mild surrounding fluid related to mild tenosynovitis (red arrows). (B) Sagittal short tau inversion recovery image shows an extensive interstitial tear involving a long segment of the PTT. Loss of continuity and some fiber redundancy are indicative of a full-thickness/subtotal tear (blue arrows). The appearance of the flexor digitorum longus (red arrow) just posterior to the PTT is normal (red arrow).

were seen on magnetic resonance (MR) images. There was no evidence of tendon retraction (Fig. 1).

Surgery, by retromalleolar approach, was planned and executed. Intraoperative exploration of the tendon revealed a tear in the

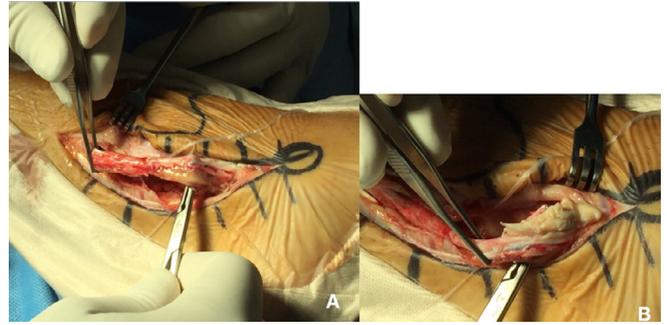


Fig. 2. Intraoperative view of the damaged tibialis posterior tendon. (A) About 4 cm of lacerated, nonfunctional fibers render both edges of the tendon unsuitable for direct repair. (B) The lesion has a typical “chewing gum” appearance.

retromalleolar region and a bone spur, which was removed with a rongeur. Some lacerated, nonfunctional fibers stretched across the separation between the 2 ends of the torn tendon, but the ends were 4 cm apart; thus, direct repair was not feasible (Fig. 2A). Close inspection of the tendon confirmed tendinosis, manifested as a typical “chewing gum” lesion (Fig. 2B). At this point, we resected the lacerated fibers and sent them to the pathology laboratory, where ischemic changes in the tendon and chronic synovitis were later identified (Figs. 3 and 4). We decided to fill the 4-cm gap with an ipsilateral semitendinosus graft (Fig. 5A). A 3-cm horizontal incision was made medial to the tibial tubercle at the level of the hamstring tendon insertion, the fascia was opened, and the semitendinosus tendon was identified and pulled out gradually from the wound after cutting its expansions. We used a no. 11 blade to cut the tendon completely 8 cm above its tibial insertion; then the tendon was doubled and sutured according to the Bunnell-Meyer suture pattern to the 2 free ends of the PTT under slight inversion of the foot (Fig. 5B).

We decided to add autologous fat and PRP to promote healing. Fat was harvested from the patient's abdominal wall and prepared by means of the nanofat technique, through which ASCs are obtained by means of mechanical lysis (18). PRP was obtained by double centrifugation of the patient's own blood (10 mL) with the use of a GLO PRP system (Glofinn Oy, Salo, Finland). We obtained, in 3 syringes, a total of 3 mL of autologous biologic solution comprising 30% PRP and 70% ASCs, and the solution was injected into the proximal and distal ends of the tendon at the junction between the tendon and the graft (Fig. 5C).

The ankle was fitted with a non-weightbearing cast, which, at 3 weeks, was removed along with the stitches. The ankle was then fitted with a foam walker (Aircast; DJO Global Inc., Vista, CA) for partial weightbearing to allow early mobilization and strengthening of the triceps, tibialis anterior, and peroneal tendons. At 6 weeks, the patient was allowed full weightbearing and began dynamic exercises of the tibialis posterior (TP) using an elastic band and a leg/ankle exerciser in addition to pool exercises. At 8 weeks, full strength and proprioceptive exercises of all ankle muscles were begun. The patient's basketball shoes were fitted with a special orthosis, and he was instructed to practice unipodal jumps and progressively resume training with the team. At 3 months, he resumed competitive play. On clinical examination at 3 months, he was pain free, the right ankle showed a full range of motion, and the arch was fully restored. The TP was shown, by heel inversion and single leg heel rise test, to have good function. MR imaging at 1 year showed an intact and continuous PTT. A linear, minimally hyperintense signal in its distal segment just before its insertion was seen and assumed to reflect the suture site (Fig. 6). The foot and ankle disability index score at the final follow-up examination, which was 18 months after the surgical repair, was 90.6, up 31.2 points from the preoperative score of 59.4.

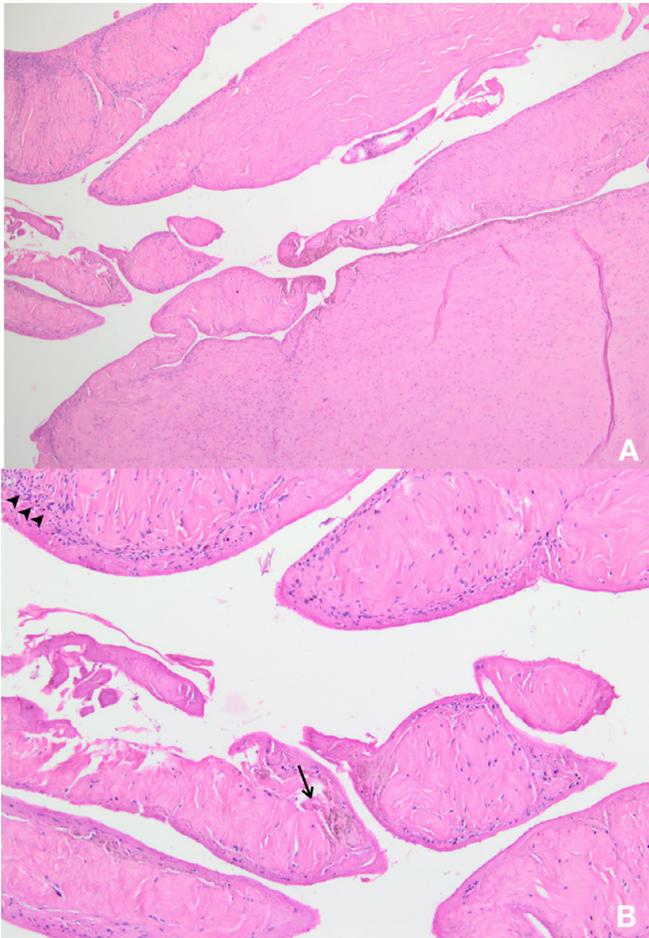


Fig. 3. Microscopic views of a hematoxylin-eosin–stained histologic section of the excised lacerated tendon. (A) Under magnification $\times 40$, partial ischemic tendinous changes associated with chronic synovitis are seen. (B) Under magnification $\times 100$, lymphocytic infiltration associated with the chronic synovitis (black arrow) and small lacerations associated with hemorrhagic change (black arrowheads) are evident.

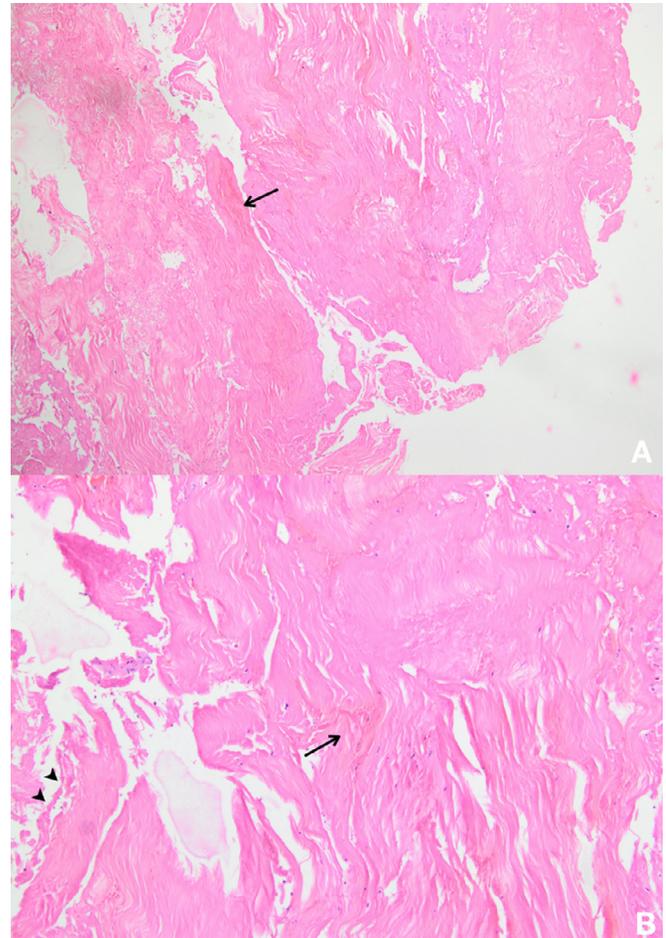


Fig. 4. Microscopic views of another hematoxylin-eosin–stained histologic section of the excised lacerated tendon. (A) Under magnification $\times 40$, partial ischemic tendinous changes (black arrow) associated with lacerations are seen. (B) Under magnification $\times 100$, evidence of lacerations (black arrowheads) associated with hemorrhagic and ischemic changes are seen (black arrow).

Discussion

Traumatic rupture of the PTT against a background of medial ankle pain is often misdiagnosed; accurate diagnosis requires a high index of suspicion. Unrecognized, traumatic rupture of the PTT can lead to progressive and painful pes planus deformity owing to lack of medial arch support (3). Untreated, traumatic rupture of the PTT can lead to severe osteoarthritis of the ankle and foot (19). Many authors acknowledge that the rupture is often identified only during surgery (3,4,8,20–22). Indirect signs can suggest the correct diagnosis. Medial bone flakes from distal tibia on a plain radiograph, or more specifically on a computerized tomography image, are suggestive of PTT rupture (3). Failure of closed reduction suggests soft tissue interposition resulting from a deltoid ligament injury or even from a PTT injury (9). Once rupture of the PTT is truly suspected, tendon evaluation should follow. Ultrasonography is considered the reference imaging modality for evaluation of the tendon itself, and it is the imaging tool most sensitive to intratendinous tears (23). We chose MR imaging to evaluate all ankle ligaments and tendons. Our patient was already being monitored and treated for chronic TP tendinopathy, and the sudden appearance of flatfoot was a strong indicator of acute tendon rupture.

The PTT is the largest and anteriormost tendon in the medial ankle. It is a powerful invertor of the ankle, attributable to its multiple insertions on the bones of the hindfoot and midfoot (with the exception of the talus

and the first and fifth metatarsals). It is blocked by the retinaculum behind the medial malleolus where it shifts almost 90° in direction to terminate in the foot (24). According to Petersen et al (25), a relatively avascular portion corresponds to the region where the TP wraps around the medial malleolus. This avascular portion consists of fibrocartilaginous tissue, which is known to have a poor healing response, and this tissue is subject to repetitive compressive and shear stress in sport activities. This renders the region at risk for degeneration and spontaneous rupture, especially the segment of tendon distal to medial malleolus (3,25).

This segment of the tendon is also the segment in which rupture secondary to trauma is most often reported (5). Such a condition was noted intraoperatively in our case. However, in cases associated with medial malleolus fracture, rupture often occurs proximal to the fracture line (7). Mallick and Faleme (26) focused on the retromalleolar anatomic situation of the PTT. This part, being the least mobile owing to passage under the retinaculum, is subject to rupture with the occurrence of medial malleolus fracture. The authors noted that low-level medial malleolus fracture in particular gives rise to tendon rupture. West et al (4) also noted that chronic repetitive low-energy trauma will generally cause the PTT to fail within its midsubstance, distal to the medial malleolus, whereas acute high-energy trauma will cause rupture of the TP at the musculotendinous junction, proximal to the medial malleolus, especially if the trauma is associated with an avulsion fracture.

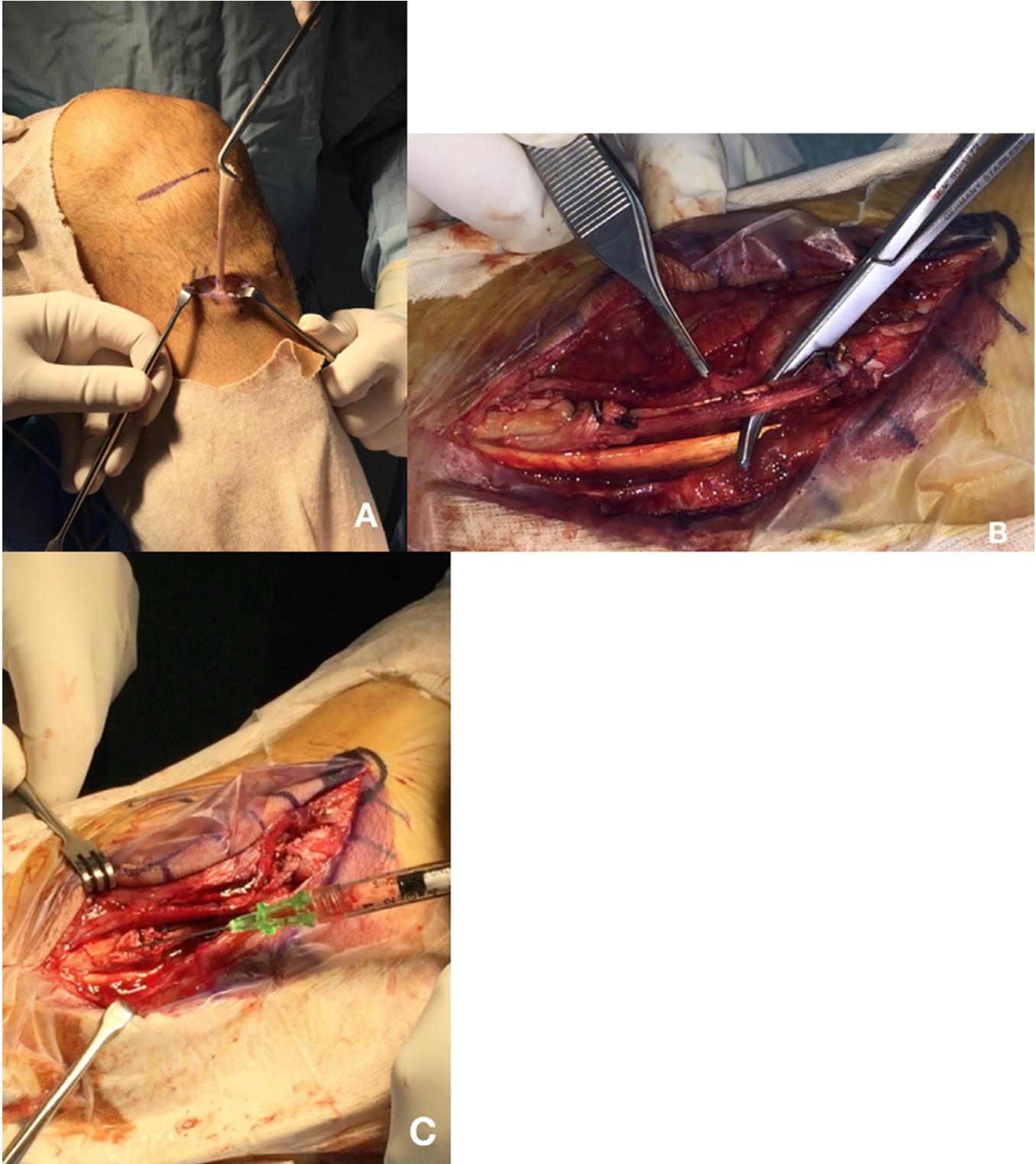


Fig. 5. Intraoperative views of the hamstring tendon transfer procedure. (A) Ipsilateral semitendinosus graft harvest, grasped by the Kelly clamp from its proximally tenotomized portion. (B) The harvested semitendinosus graft, pointed by the Kelly clamp, transferred and attached to both edges of the tibialis posterior by application of Bunnell-Meyer suture patterns. (C) Nanofat and platelet-rich plasma solution being injected into the proximal suture site.

Once acute spontaneous rupture of the PTT is diagnosed, secondary causes should be ruled out. These include rheumatoid arthritis, diabetes mellitus, and other degenerative connective tissue diseases (4). Our patient did not suffer from any of these conditions.

Monto et al (27) were the first to describe acute rupture in the absence of ankle fracture. Martinelli et al (28) reported a similar acute rupture in a previously asymptomatic foot. However, in that case, 1 month after the traumatic event, surgery was performed for persistent

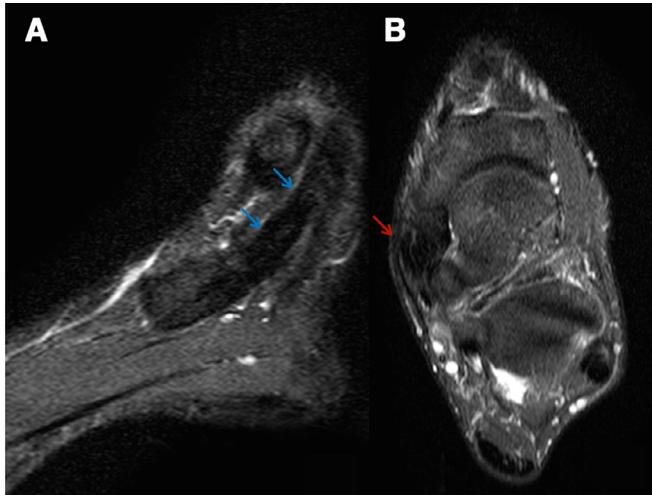


Fig. 6. Magnetic resonance images of the posterior tibial tendon (PTT) obtained 1 year after surgery. (A) Sagittal short tau inversion recovery image shows the PTT to be intact and continuous (blue arrow). (B) Axial short tau inversion recovery image shows a residual linear minimally hyperintense signal in the distal segment of the PTT just before its insertion. This hyperintensity is most likely related to the suture site (red arrow). There is no evidence of tenosynovitis.

flatfoot deformity, and signs of degeneration were observed during the operation. The condition was judged to be that of stage 2 PTT dysfunction, and the authors decided on medial displacement calcaneal osteotomy with FDL transfer. The clinical results were good. Our case was similar, except our patient was being followed up for refractory TP tendinopathy. We treated the case as that of an acute rupture and decided not to perform any corrective bony procedure. Of particular interest is the fact that our patient had been given a local steroid injection 2 weeks before the acute rupture. It cannot be said that ruptures such as the one in our case are directly related to corticosteroid injection, but it is of note that spontaneous tendon ruptures after local steroid injection have been reported, especially ruptures in the hand and foot (29,30). Local injection of steroids is commonly practiced by some orthopedic surgeons because it provides acute relief of symptoms, but such steroid injections can have a deleterious effect on the tendon, especially if the patient unintentionally resumes activities that place an excessive load on the body, which is often the case among professional athletes. This happened with our patient, who continued participating in competitive sports when he should have continued resting. Unfortunately, he paid attention only to the fact that he felt well as a result of the steroid therapy. Therefore, we advise avoidance of local steroid injections as therapy for chronic tendon conditions, particularly if the foot and ankle are involved; otherwise, we advise that clinicians carefully instruct their patients on the importance of limiting their activities for 1 or 2 months, regardless of whether their symptoms decrease or even disappear.

Early surgical treatment of acute PTT rupture is necessary to avoid progression to flatfoot and secondary osteoarthritis (19,31). All previous reports have confirmed good outcomes after primary suture (3,4,7–9) and after FDL transfer (10,13,28).

To the best of our knowledge, transfer of a tendon from outside the foot to the PTT has not been described previously. Although such tendon transfer adds donor site morbidity, we believe that the transfer is warranted because preservation of all tendons of the foot and ankle prevents potential future instability. This is especially true when the FDL tendon is involved, because this tendon plays an important role in the biomechanical function of the foot (15). Hamstring tendon grafts have been widely used for replacement of torn anterior cruciate ligaments and torn patellar tendons, and they have also been used for

reconstruction of the Achilles tendon (32,33). In addition, they have been used to reconstruct ankle ligaments and to repair upper extremity ligament defects (34,35). Moreover, hamstring harvest is considered a safe and relatively easy procedure. Hamstring harvesting may cause knee flexion weakness, but the clinical and functional outcomes are similar compared with those of unaffected knees (36). Thus, we decided to harvest our patient's semitendinosus tendon to replace the ruptured PTT.

The literature remains inconclusive concerning the effects of PRP on tendon and soft tissue healing. The physiopathology of tendon regeneration is becoming clearer. The inflammatory cascade that was previously targeted to treat tendinopathy is now understood to be the cornerstone of the tendon healing process (37).

PRP solutions are obtained from centrifugation of whole blood and by extracting the platelet-active fraction. Therefore, they contain a high concentration of growth factors derived from platelets (38). These preparations can be used for primary or adjuvant therapy (37,38), such as by application over a repaired tendon, as was done in our case, to promote healing of the tendon at the edges. Although our patient was healthy and did not suffer from any peripheral neurovascular disorder that could have altered the tendon healing process, we decided to use PRP because tendon healing does not occur as easily as does bone or soft tissue healing, and especially because our patient suffered from a chronic degenerative condition and the defect involved a relatively avascular area.

Application of PRP has shown promise in terms of clinical outcomes, especially in cases of lateral epicondyle extensor tendinopathy and patellar tendinopathy (17,39,40). However, strong clinical evidence is lacking, and the current literature is inconclusive concerning the indications and optimum protocol for PRP administration. In fact, authors of most systematic reviews have concluded that PRP preparation methods need to be standardized so that robust comparative studies can be performed on which strong evidence-based conclusions can be drawn (38,41). One benefit is that injection of autologous biologic preparations does not result in the systemic or local secondary effects that result from injection of corticosteroids, which is the reason behind their widespread use in the treatment of various pathologies.

The effect of PRP on the healing process after acute tendon rupture has been studied in animal models. In a dog model, the Achilles tendon was intentionally torn and then treated by primary suture with or without PRP as adjuvant therapy (42). Better function and better vascular and fibroblast proliferation resulted in the PRP-treated dogs. Improved neovascularization may explain the long-lasting effect of PRP on tendon healing (43).

Combination biologic therapy—that is, mesenchymal stem cells, PRP, and dermal allograft constructs—applied in combination on a single tendon has shown promise, especially in cases of rotator cuff tear (44,45). Outcomes have been good under a strict rehabilitation protocol.

Of particular interest, one such combination therapy is PRP injection combined with fat grafting. Autologous fat is easy to obtain in large quantities, and fat grafting is a relatively safe procedure. A disadvantage to using fat has been the high resorption rate, but PRP has recently been shown to enhance fat graft survival (46). Theoretically, multipotent stem cells have the ability to differentiate into any kind of mesenchymal cells, including tenocytes. Remarkably, they are in rich supply in adipose tissue in comparison to the numbers found in bone marrow and other sources (47). Nanofat grafting is a recently described technique by which ASCs are isolated without viable adipocytes (18), processed, and then delivered by superficial intradermal or subdermal injection into patients requiring skin rejuvenation. Good results have been attributed to the presence of stem cells rather than the fat grafting itself. Li et al (48) studied the effect of different volume fractions of PRP combined with ASCs on fat graft survival. The addition of PRP improved ASC

proliferation, and the PRP dosage influenced the viability of the adipose grafts. The greatest synergistic effects were seen with the 20% and 30% PRP volume fractions, with no significant difference between them. The addition of PRP to ASCs resulted not only in greater adipocyte proliferation but also in more neovascularization and less vacuolization. For our patient, we chose combination adjuvant therapy because we wanted to guarantee tendon healing and obtain a good clinical outcome, especially because we were dealing with a tendon grafting procedure and not a primary repair. PRP at 30% was used to augment the nanofat grafting.

In conclusion, PTT rupture is uncommon and often misdiagnosed. If not associated with medial malleolus fracture or secondary flatfoot deformity, PTT rupture is easily missed, and the long-term consequences can be devastating owing to the inevitable evolution into an acquired disabling flatfoot deformity. Thus, early surgical repair of PTT rupture is mandatory. Primary repair should always be attempted in cases of acute rupture. However, the surgeon can encounter loss of substance or a degenerated tendon, as is often the case in chronic PTT insufficiency syndrome. Local tendon transfer is a viable option, with good outcomes reported mostly in cases of FDL transfer, but local transfer may alter the biomechanical properties of the foot. Our case confirms that excellent results can be expected after hamstring transfer and gives surgeons another feasible option when facing a PTT rupture for which primary end-to-end suture is not possible. We believe this to be the first report of the previously described nanofat technique (18) applied in combination with PRP to a tendon injury. A wide spectrum of autologous biologic preparations is emerging, and PRP mixed with ASCs shows promise for soft tissue reconstruction. It seems to us that such combination therapy could become a primary rather than an adjuvant therapy for tendon injuries.

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