Treatment of Achilles Tendon Partial Tear with Lipoaspirate (Lipogems®)

Brady Saray-Logan, Christopher J Rogers MD, RMSK
San Diego Orthobiologics Medical Group, Carlsbad, CA

ABSTRACT

Elite and recreational athletes commonly present with Achilles tendinopathy and partial tears. The tendon pathology demonstrates a failed healing response rather than an inflammatory process. The source of pain in tendinopathy is typically related to the neurovascular ingrowth seen in the tendon's response to injury [3]. Conservative medical treatments include rest, bracing, eccentric strength training, low energy shock wave therapy, topical glyceryl trinitrate [6]. More recently, minimally invasive ultrasound guided treatments such as tendon fenestration, tendon scraping and hydro-dissection of the paratenon have been reviewed. Surgical intervention is rarely indicated.

Recent studies have demonstrated that lipoaspirate contains and produces growth-factors, such as platelet-derived growth-factor (PDGF), fibroblast growth-factor (FGF), transforming growth-factor beta (TGF-β), and vascular endothelial growth-factor (VEGF) [1]. These growth factors are known to play an important regulatory role in cellular proliferation, cellular migration, matrix synthesis and angiogenesis. The Lipogems® system can produce a non-expanded, rinsed, and microfragmented adipose tissue graft that maintains an intact stromal vascular niche and contains cellular elements with mesenchymal stem cell and pericyte characteristics [11].

We present the case of a 16 year old girl who developed Achilles tendinopathy and partial tear that was non-responsive to conservative care. She was treated with ultrasound guided lipoaspirate (Lipogems®) injection. We report her significant improvement in pain and physical function at six month follow-up. Future clinical trials of lipoaspirate for the treatment of chronic Achilles tendinopathy and tear are warranted.

INTRODUCTION

Chronic degenerative tendon disorders (tendinopathy) occur frequently and are difficult to treat. Tendon injuries are often associated with significant physical dysfunction and disability, due to the limited self-repair capacity and propensity for scar formation [1]. Non-insertional Achilles tendinopathy is a common cause of ankle pain and typically occurs 2 - 6 cm proximal to the tendon insertion on the calcaneus.

Athletes, whether elite or recreational, are the most common group to present with Achilles tendinopathy, but it is also found in people with advanced age, obesity, diabetes or hypertension. Other risk factors for Achilles tendinopathy include previous tendon injury, decreased muscle strength, altered gait kinematics, limited ankle dorsiflexion, training errors, and the use of steroids or fluoroquinolones.

The degenerative process is essentially a failed healing response in the tendon [2]. Fibromatous degeneration is the most frequent finding and is related to the relative hypovascularity of the critical zone. Mucoid degeneration is the second most common type of degeneration with large mucoid patches and vacuoles found between thinned degenerated tendon fibers. Early episodes are asymptomatic, however symptoms develop when the vacuoles and lacunae coalesce to form an interstitial tear. Histological degenerative changes include loss of the normal collagenous architecture, replacement with amorphous mucinous material, hypercellularity, increased glycosaminoglycan content and neovascularization. Less commonly, tenolipomatosis or calcifying tendinopathy with tendon calcification may occur [3].

Patients often present with focal swelling and tenderness to palpation on the posteromedial aspect of the tendon. Both ultrasound and magnetic resonance imaging (MRI) assist in the diagnosis of Achilles tendinopathy, but ultrasound has a higher degree of accuracy and is more cost effective [4]. Ultrasound imaging may reveal a fusiform tendon with peritendineum thickening, hypoechogenic foci, intrasubstance tendon tears, decreased tendon gliding and intratendinous and peritendinous vascularity on color Doppler [5].

Conservative care of Achilles tendinopathy includes removal of precipitating factors, rest, training routine modifications and the use of orthotics to correct foot or ankle malalignment. Decreased myotendinous flexibility may be treated with stretching. Muscle weakness is treated with progressive eccentric strengthening exercises twice a day for up to 12 weeks using either the Alfredson or Silbernagel protocol. Combining these protocols with low-energy shock-wave therapy (ESWT) may improve outcomes [6].

The combination of autologous platelets and a fibrin matrix in a sheep tendinopathy model has demonstrated increased tenocyte proliferation, synthesis of type-I collagen and angiogenic factors (VEGF, HGF) promoting neovascularization [7]. In a prospective clinical case series of 14 patients with chronic non-insertional Achilles tendinopathy, leukocyte-rich platelet rich plasma (LR-PRP) demonstrated significant improvement in the American Orthopedic Foot and Ankle Society (AOFAS) scale, the Victorian Institute of Sport Assessment – Achilles (VISA-A) scale and ultrasound imaging results at 18 month followup [5]. A well-designed RCT study of chronic Achilles tendinopathy found no significant difference in pain or activity level 24 weeks after receiving an ultrasound guided LR-PRP injection combined with an eccentric stretching program [8]. Yet, in a systematic review and meta-analysis, LR-PRP demonstrated a strongly positive effect when compared with LP-PRP, suggesting that what is injected and how it is injected may influence outcomes [9].

Up to 33% of patients will fail conservative care and consider surgical excision of the pathologic tissue with or without tendon transfer augmentation. Open surgery is associated with an overall complication rate of 11% and success rate ranging from 50-100%. Minimally-invasive percutaneous therapies designed to strip the paratenon from the tendon with high-volume fluid injection or ventral tendon scraping have shown promise in relieving symptoms. Percutaneous tendon fenestration (tenotomy) performed under ultrasound guidance has also demonstrated good outcomes.

Adipose tissue is a connective tissue derived from embryonic mesoderm, consisting of a heterogeneous population of cells such as adipocytes, preadipocytes, smooth muscle cells, endothelial cells, mast cells and fibroblast immune cells. From adipose tissue manipulation it is possible to isolate these so called stromal vascular fraction (SVF) which contains, among others, mesenchymal stem cells (MSC) [10]. When compared with bone marrow, adipose tissue contains several times the number of pluripotent stem cells [12, 13].

CASE REVIEW

Our patient was a 16 year old female student athlete who presented with a two year history of progressive right heel pain. She notes history of several injuries to the ankle while playing basketball. One particular day, she noticed a "pop" associated with increased Achilles tendon pain. She had pain with many activities, but mostly when walking around campus or climbing stairs. She was a basketball player, but had been unable to practice or exercise for more than one year. She was using a CAM walker boot for almost a year while walking or performing activities of daily living. She had been diagnosed with chronic non-insertional Achilles tendinopathy on the basis of MRI and clinical presentation. She had received more than 7 months of physical therapy which included eccentric calf strengthening, soft tissue mobilization, and modalities. She did not use oral analgesics, NSAIDs, or topical medications. She had not received any form of injection therapy. She was using acetaminophen 500mg and tramadol 50mg for pain intermittently. She did not take any other medications. She denied the use of antibiotics in the past. She denied any allergies or other medical conditions. She has a family history of arthritis, but denied any family history of autoimmune disease, connective tissue disease or seronegative arthritis.

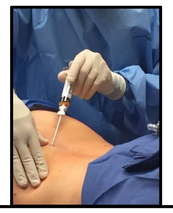
On physical examination she presented wearing her CAM walker boot. She had significant focal swelling of the right Achilles tendon which was tender to light palpation. The calf squeeze test was negative. A 4 cm palpable, firm and tender mass was located from 2 cm proximal to the tendon insertion. Ankle range of motion was limited to 20 degrees dorsiflexion and 20 degrees plantarflexion. She was able to actively move the ankle, but was unable to hop, squat or perform a single heel raise. Her light touch sensation was intact and the dorsalis pedis and posterior tibial pulses were intact in the lower extremity. She was noted to have moderate pes planus bilaterally, but normal subtalar joint motion, leg length and triceps surae muscle bulk.

Her Foot and Ankle Disability Index Score (FADI) was 65.4 and her average Visual Analog Score (VAS) was 7 out of 10.

Magnetic resonance imaging (MRI) of the right ankle revealed Achilles tendinosis with a 3.3 cm longitudinal split tear, extending from 2.7 cm to 6 cm proximal to the tendon insertion.

Musculoskeletal Ultrasound (MSK US) Examination of the right ankle was performed using a 8-12 MHz linear probe. Examination revealed fusiform thickening, diffuse hypoechoic defects and hypoechoic disorganization of the tendon fibrillar pattern from 2 to 6 cm proximal to the Achilles tendon insertion on the calcaneus. Edema was evident in Kager's fat pad anterior to the Achilles tendon. Mobility of the Achilles tendon with ankle motion was limited. The retrocalcaneal bursa, plantaris tendon and FHL tendon were unremarkable.

Given the radiographic findings, duration of symptoms and level of functional limitations, we elected to treat with lipoaspirate injection (Lipogems®) of the Achilles tendon. The Lipogems® system can produce a non-expanded, rinsed, and microfragmented adipose tissue graft that maintains an intact stromal vascular niche and contains cellular elements with mesenchymal stem cell and pericyte characteristics. We hypothesized that these cells would express a phenotype that could encourage tendon neovascularization, tenocyte proliferation and type-I collagen synthesis. Platelet rich plasma has also demonstrated increased tenocyte proliferation, synthesis of type-I collagen and promotion of neovascularization. However, in this case, we believed that the degree of tissue degeneration and the duration of symptoms warranted the addition of adipose derived cells to augment any benefit provided by platelet rich plasma. Studies to inform the physician as to the number or type of cells required to heal such an injury do not currently exist. We were limited by the volume that can be injected into a degenerated tendon and adjusted our treatment plan accordingly.



Performing lipoaspiration with 13 G cannula and VacLok syringe.

Preparation of the lipoapirate using the Lipogems® system.

PROCEDURE

The patient was informed of all other possible treatment options and informed consent was obtained from the patient and her mother to proceed with PRP / lipoaspirate injection. The patient was advised to avoid the use of aspirin or NSAIDs for one week prior to the procedure. On the day of the procedure, she was given Ancef 1 gm IVP 60 minutes prior to the procedure. Fifty-two milliliters of blood was obtained from the antecubital fossa to prepare 3 ml of leukocyte poor PRP using the 1% hematocrit setting on the PRP processing centrifuge (Arthrex Angel, Naples FL). We did not determine the baseline serum platelet level or assay the PRP sample to determine platelet content.

The patient was placed in the prone position in our procedure suite and monitored with blood pressure, pulse oximetry and electrocardiographic monitoring. The skin over her lateral flank was prepped with Hibiclens and sterilely draped. The skin was anesthetized with 5 ml 1% lidocaine. A 2 mm stab incision was made with an #11 blade. Tumescent anesthesia was performed with 96 ml Klein's solution and a 17Gx185 mm anesthetic infiltration cannula. Klein's solution had been prepared with 500 ml normal saline, 20 ml of 2% lidocaine and 0.5 ml 1:1000 epinephrine. After waiting 12 minutes for detumescence, lipoaspiration was performed with a 13Gx185 mm cannula and VacLok syringe to produce 20 ml lipoaspirate. At this time, the Lipogems® method was used to process the lipoaspirate. In brief, the Lipogems® method allows for a minimal enzyme-free manipulation in a sealed, sterile device to minimize trauma to the cellular content. The device contained normal saline and filters which gradually reduce the adipose clusters and eliminates oil, hematic residue and tumescent anesthesia. We obtained 8ml of washed and rinsed lipoaspirate (Lipogems®) for injection. The patient's wound was cleansed and dressed.

The ankle was prepped with Hibiclens and sterilely draped. The skin over the medial aspect of the Achilles tendon was anesthetized with 1% lidocaine at two locations. The tendon and paratenon were not anesthetized. The tendon was visualized with ultrasound and a sterilely draped 12 MHz linear probe. An 18 g needle was advanced into the tendon to perform tenotomy at multiple locations with special attention to treat the hypoechoic aspects of the tendon until the consistency of the tendon was softened. Using the 18 g needle and 1ml syringe containing 0.5 ml of Lipogems® was injected at 8 locations throughout the tendon without resistance. Lipogems® was also injected anterior and medial to the tendon. After injection of the Lipogems®, 3 ml of LP-PRP was injected at several locations within the tendon. The injection site was cleansed and bandaged. The patient presented to our recovery room and was discharged within 20 minutes. She was advised to wear her CAM walker boot and to use ice, tylenol or Tramadol as needed for up to three days. She was given an abdominal binder and prescription for cephalexin 250 mg QID for 5 days.

RECOVERY

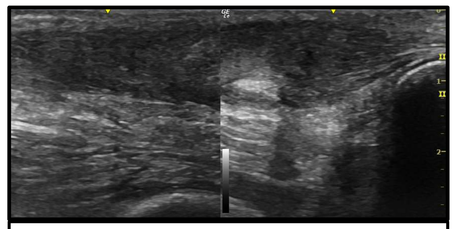
The patient presented to our clinic for followup one week after her procedure. She denied any complications, side effects or significant pain. Her incision site had produced clear discharge for one day and was healed in three days. She did not have any bruising of the adipose harvest site. Her ankle was stiff and swollen, but not red or significantly tender. She was able to walk short distances with the CAM walker boot. She was instructed to start physical therapy which included passive/active range of motion, edema management, soft tissue mobilization and progression of strength training as tolerated. She was allowed to bear weight as tolerated.

At six weeks followup, our patient noticed a significant reduction in her pain at rest and with ambulating short distances. She was not compliant with physical therapy, choosing to stretch and ambulate on her own schedule. She had developed mild kinesiophobia due to the duration of her injury. On physical examination, the tendon swelling has reduced and the tendon was less tender to palpation. Ultrasound examination revealed an improvement in the hyperechoic fibrillar pattern of the tendon, however the tendon thickening and hypoechoic defects remained. The patient was encouraged to initiate the formal physical therapy program.

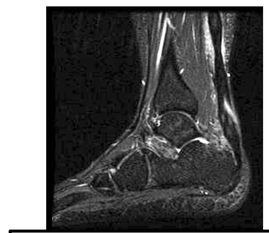
At three month followup, our patient noticed substantial reduction in pain and improved physical activity. Her repeat FADI score was 97.1. Her average VAS was zero out of ten. She could walk, climb and perform heel raises and squats without pain. On physical examination, she had full dorsiflexion and normal plantar flexion strength. The tendon was not tender to palpation. Ultrasound examination revealed significant improvement in the hyperechoic fibrillar pattern of the tendon with near complete resolution of the hypoechoic defects. She was compliant with an eccentric strengthening program in physical therapy twice a week and at home twice a week.

At six month followup, our patient is able to walk long distances and climb stairs without pain. She can run up to 30 minutes at a time and performs all strengthening exercises without pain. Although not required, we elected to repeat her ankle MRI which revealed resolution of the high intensity linear band and diffuse speckled signal consistent with resolution of her tendinopathy and partial tears. The tendon thickness and Kager's fat pad edema was improved as well.

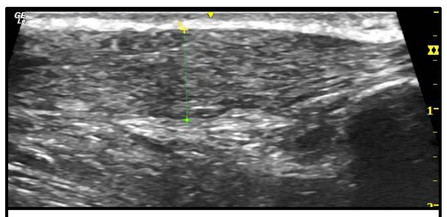
At this time, she takes no medication and does not wear a CAM walker boot. But she does have plans to attend her high school prom and wear high heels.



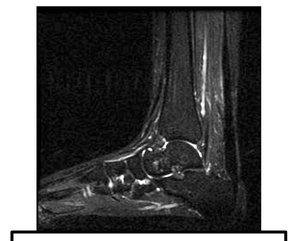
Sagittal ultrasound imaging reveals fusiform thickening, hypoechoic defects, and disorganization of fibrillar pattern in the Achilles tendon.



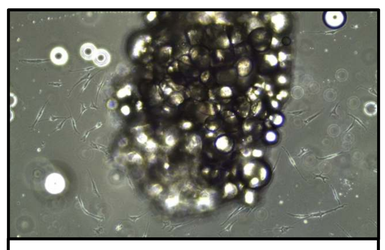
Sagittal (STIR) MRI image shows 3.3 cm longitudinal partial tear in Achilles tendon.



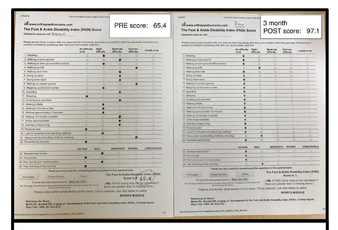
Sagittal ultrasound imaging reveals improved fusiform thickening, resolution of hypoechoic defects and increased hyperechoic fibrillar pattern of the Achilles tendon 6 months after treatment



Sagittal (STIR) MRI image reveals resolution of partial tear of the Achilles tendon 6 months after treatment



Mesenchymal stem cell (MSC) emerging from lipoaspirate



The Foot & Ankle Disability Index (FADI) Scores before (65.4) and 3 months after treatment (97.1)

SUMMARY

We were able to successfully treat a 16 year old girl who had developed functionally limiting chronic Achilles tendinopathy and partial tear that were non-responsive to conservative care. She was treated with ultrasound guided lipoaspirate (Lipogems®) and LP-PRP injection combined with a progressive strengthening program. She achieved a resolution of her pain and restoration of physical function at the six month follow-up. Ultrasound and MRI imaging revealed a diffuse fibrillar pattern consistent with regenerated tendon fibers that had replaced the tendinopathic and partially torn tendon regions.

Tendon engineering is an interesting challenge for the orthopedic medicine physician. Lipoaspirate and LP-PRP have the potential to stimulate tenocyte proliferation, matrix synthesis and angiogenesis which can lead to regeneration of a degenerated Achilles tendon. Future clinical trials using lipoaspirate for the treatment of chronic Achilles tendinopathy and partial tears are warranted.

Contact

Christopher J. Rogers MD
6125 Paseo Del Norte, Suite 100
Carlsbad, CA 92011

Email: Rogers@SDOMG.com
Website: SDOMG.com
Phone: 760-909-2355

San Diego Orthobiologics Medical Group

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