

SPONTANEOUS BILATERAL ACHILLES TENDON RUPTURE: A CASE REPORT AND LITERATURE REVIEW

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INTRODUCTION

The Achilles tendon is the thickest and strongest tendon in the body, arising from the confluence of the gastrocnemius and Soleus tendons.^{1,2} It can withstand forces up to 12 times body weight and accounts for 20% of all large tendon ruptures.² Most injuries of the Achilles tendon occur as a result of accidental trauma and athletic activities such as lunging and jumping.^{3,4} Mechanism of injury involves sudden and powerful contraction of the gastrocnemius and soleus muscles on a dorsiflexed foot, usually affecting men ages 30-50⁵.

Spontaneous bilateral Achilles tendon ruptures are a very uncommon occurrence.^{6,7-10, 6, 11, 1, 12, 13} Risk factors for these type of injuries include corticosteroid use¹², anabolic steroids,¹⁴ fluoroquinolones,¹⁵ chronic pain, and previous Achilles tendon rupture.¹⁶ To our knowledge, only 8 reports on spontaneous bilateral Achilles tendon tears have been published, most with satisfactory outcomes. The purpose of this case report is to illustrate a near-fatal complication of treatment of bilateral spontaneous Achilles tendon rupture and to review the current literature on the management of this condition.

CASE REPORT

DS is a 69 year old man with a history of chronic obstructive pulmonary disease, asthma, and obstructive sleep apnea. Two weeks prior to his admission, he was admitted to the hospital for a flare-up of his COPD. As an inpatient, he received intravenous corticosteroids and nebulizer treatments. He was discharged on Prednisone and a 10-day course of Levaquin. Over the next few weeks, he developed bilateral heel pain, and 3 days before his admission, he noted a “pop” from his right heel. He was transferred from an outside hospital to our medical center with the diagnosis of bilateral Achilles tendon ruptures.

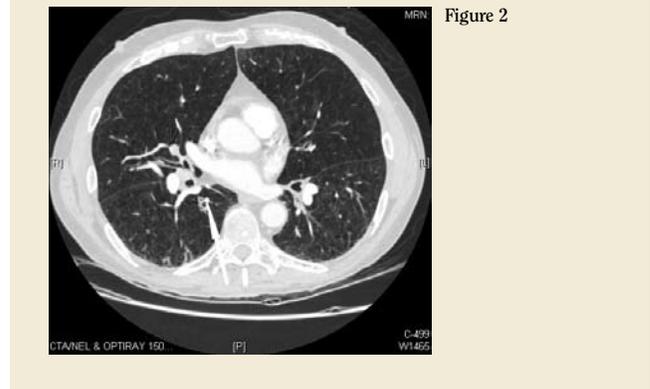
Physical examination on admission was notable for a bilateral positive Thompson’s test, and a palpable defect on

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the right side. Radiographs of both ankles were unremarkable. MRI of both ankles confirmed bilateral Achilles tendon ruptures (Figure 1). The patient’s baseline activity was treadmill walking. After considering his functional goals in conjunction with his medical comorbidities, he was thought not to be a candidate for surgical repair. We recommended bilateral cast braces for definitive treatment. The patients was subsequently discharged on Coumadin for thromboembolic prophylaxis.

One week after his discharge, DS visited his primary care physician who tapered his prednisone and recommended that he discontinue his Coumadin. One week after stopping his coumadin, DS presented with the abrupt onset of shortness of breath, cough, myalgias and fever. Vital signs revealed a temperature of 101.1°, heart rate 114, blood pressure 150/108,

respiratory rate 21, O₂ saturation 93% on room air. A CT angiogram was performed and revealed bilateral pulmonary emboli present in segmental branches going to both lower lobes, and into the right middle lobe zone (Figure 2). He was admitted and placed on a heparin drip. For his COPD exacerbation, he was given high-dose prednisone 60 mg orally for three days. He subsequently recovered and was discharged home.

DS progressed well with his braces. At 5 weeks follow-up, he still had a palpable defect on the right, but none on the left. His flexor strength was 3/5 bilaterally. At that time, he was advanced to custom anterior Achilles tendon splints to give him more mobility. At 10 weeks, his flexor strength had improved to 4/5 bilaterally and he reported no pain. He began progressive resisted exercises and his braces were discontinued.

By 7 months, DS was back to baseline level of activity; using a treadmill for 15-20 minutes a day. At that point, his plantar flexion was 4/5, but he was still unable to single or double heel raise. At the one year follow-up, the patient had increased his exercise tolerance to 200 feet. He was unable to toe walk and his Thompson's test remained positive. At the time of latest follow-up, DS had made a full recovery. He is pain free and not limited by his ankles.

ETIOLOGY

Achilles tendon ruptures, accounting for 20% of all large tendon ruptures,^{10, 9} occur in men ages 30-50. Tears usually occur either as a result of a large force, or a physiological force on a weakened tendon. The majority of tears occur in the watershed area, an area of structural weakness, located approximately 6 cm proximal to the tendon insertion on the calcaneus¹⁷.

Spontaneous tears account for about 1% of all Achilles tendon ruptures,^{9, 11} are much less common than traumatic tears, and much more likely to be associated with disease.^{18, 9, 11} These spontaneous ruptures are associated with an array of risk factors, both intrinsic and extrinsic.

Intrinsic risk factors include:

1. Prior trauma.
2. Degenerative changes of the tendon.
3. systemic disorders such as Cushing's disease, rheumatoid arthritis¹⁹, systemic lupus erythematosus²⁰, hyperthyroidism, and gout.

Extrinsic risk factors include pharmacological agents such as corticosteroids^{9, 7, 18, 21, 14, 22, 23} and fluoroquinolones^{24, 21, 25, 15}

Corticosteroid use is a factor in the vast majority of bilateral simultaneous Achilles tendon tears; present in approximately 90% of the cases¹². Currently, the biochemical mechanism is still unclear. Steroids have the ability to alter the collagen structure of tendons by contributing to dysplasia of collagen fibrils and thus reducing the tensile strength of the tendon¹¹. Corticosteroids act by interfering with collagen fiber cross-link-

ing, which as a result, disrupts the normal healing process of the tendon.^{11, 12, 18} Dose or duration of steroid treatment does not affect the susceptibility to spontaneous tendon rupture.¹⁸ The risk of spontaneous tears from local injections is controversial. One study in 1973 showed that local injection may increase the risk of spontaneous rupture.²⁶ Since then, there have not been any recent and rigorous studies validating the risk of Achilles tendon tears from local corticosteroid injection²⁷. In a review by Leppilahti et al, only about 2% of Achilles tendon tears had received local corticosteroid injections around the Achilles tendon.²⁸

Fluoroquinolones have been associated with spontaneous Achilles tendon tears.^{24, 21, 25, 15} This event is relatively rare, as a study in 1999 estimated that tendonitis occurred in approximately 15 cases per 100,000 exposure days.²⁹ The effect of fluoroquinolones on tendons does not seem to be dose-dependant, and tears have occurred in as little as 2 weeks after initial treatment with fluoroquinolones.³⁰ Incubation of canine tendon fibroblasts in ciprofloxacin showed a decline in cell proliferation compared with control cells. The researchers also found an increase in matrix-degrading protease activity from the fibroblasts and an inhibitory effect of fibroblast metabolism.¹ Some authors believe that tendonitis and subsequent rupture may be a direct result of tissue ischemia via a vasculitic mechanism induced by the fluoroquinolones.

DIAGNOSIS

Patients primarily complain of heel pain, but may show a constellation of symptoms that may sometimes mimic peripheral neuropathy⁸. The treating surgeon must have a high index of suspicion in debilitated and elderly patients, as most tears occur atraumatically and patients fail to describe hearing a "pop" before the onset of pain in the posterior aspect of the heel and ankle. Patients often describe weakness on plantar flexion, rendering them unable to stand on their toes.

In the acute clinical evaluation, a gap can be palpated. After time, however, edema can obliterate this, and palpation of the gap becomes unreliable. Several clinical tests including the Simmond's test, Thompson's test, and Copelands's sphygmomanometer test, have been described.²⁸ Thompson's test is the easiest to perform in the clinical setting, for it does not require special equipment, and is non-invasive. Patients lie prone on the exam table while the physician compresses the gastrocnemius muscle on the affected side. Failure of plantar flexion with compression of the gastrocnemius yields a positive test, confirming tendon rupture.

IMAGING STUDIES

Radiographs, ultrasonography, and magnetic resonance imaging (MRI) have been used to diagnose Achilles tendon tears.³² X-rays are routinely taken and can be useful in finding calcific deposits in the tendon, or evidence of avulsion fracture of the calcaneus. Ultrasonography is well described^{31, 32, 28, 33}, but is not widely used since its results are operator dependant and hardware is not readily available. MRI imaging is not necessary for diagnosis. MRI can be clinically helpful in the presence of

questionable diagnosis or for tear localization during preoperative planning.

TREATMENT

Achilles Tendon Tears can be treated either with immobilization or surgical repair. Most surgeons opt for non-operative management in older or chronically ill patients because of concern over perioperative risks. Surgical management is usually reserved for younger, active patients.³⁴

NON-OPERATIVE TREATMENT

Conservative treatment of Achilles tendon tears has been considered a reasonable form of treatment in poor surgical candidates. It has a relatively good outcome and the risk of wound breakdown is eliminated. It has been reported that the new mobile splints that allow early mobilization have outcomes comparable to surgical repair.³⁴⁻³⁶ In a retrospective cohort study published in 2003, Weber et al³⁷, suggests that non-operative treatment with an Equinus ankle cast and boot for 12 weeks was as effective as surgical treatment in return to sports and ultimate strength as operative treatment. Moreover, they also report that non-operative treated patients had a much faster subsidence of pain, return to unaided walking, and return to work. Wallace et al³⁶, showed similar outcomes between surgical and non-surgical treatment. Closed treatment had a lower minor complication rate.

Although conservative treatment may provide comparable functional outcomes to operative treatment, the risk of major complications such as deep venous thrombosis and re-rupture have been shown to be higher than in operative patients.^{38, 25, 39, 34} Cetti et al²⁵ in 1997 reported a re-rupture rate of 4.7% after conservative treatment. Other researchers such as Wong et al³⁴ and Wills et al³⁸ reported rates of re-rupture of 9.6% and 10% respectively. Incidence of deep venous thrombosis varies between authors, from 1.2% by Leppilahti and Orava²⁸ to 4% by Ingvar et al,⁴⁰ but most authors agree that this rate is higher than seen in operative cases. Other effects of “cast disease” may detour physicians from selecting conservative treatment as this may lead to stiffness and weakness due to muscle atrophy. New orthoses that allow full weight-bearing and early motion may limit these problems.

OPERATIVE TREATMENT

Operative treatment has been the treatment of choice in young patients, active or high-demand individuals, and patients with chronic ruptures^{34, 41} because it provides earlier motion, increased strength, and lower major complication rates than conservative treatment.^{42, 34, 43, 44} Several operating techniques have been described.^{34, 38, 25, 45, 39, 42, 44, 43, 46-48}

Open, end-to-end suture is the most commonly used surgical treatment. In several studies, re-rupture rates have been considerably lower than conservative treatment as well as return to activity has been considerably sooner.^{25, 49-52} Jacobs et al⁵² showed in 1978 that treated conservatively, seven of 32 patients had reruptures, while 0 of 26 patients receiving surgical treatment had reruptures. Plantar flexion strength was also shown to be higher in the operative group (75% of uninjured side) than the conservative group (65% of uninjured

side). More recently, Wong et al³⁴ conducted a meta-analysis of 125 papers (5056 total Achilles ruptures) and found that the re-rupture rate of patients treated conservatively was 9.8%, while the re-rupture rate of surgically treated tears was only 2.2%. A 2005 retrospective meta-analysis of twelve randomized controlled trials by Khan et al⁵³ showed that open operative treatment was associated with a lower risk of re-rupture compared with nonoperative treatment (relative risk, 0.27; 95% confidence interval, 0.11 to 0.64).

Surgical treatment has been associated with a large number of complications, ranging anywhere from 11.8%-21.6%^{25, 43} when compared to conservative treatment,^{38, 25, 39, 34, 52, 54, 49, 53} which ranged from 4%-10%. Therefore, surgical candidates must be screened thoroughly before a surgical decision is made. Wound sepsis is a high risk in operative patients and accounts for the largest number of post-surgical complications.⁵⁵ Other major complications reported were, chronic fistula, Deep-vein thrombosis, pulmonary embolism, and death. Minor complications included superficial infection, wound hematoma, delayed wound healing, skin necrosis, suture rupture and persistent pain.

In order to address wound problems associated with open repair, Ma and Griffith developed a percutaneous Achilles repair technique in 1977.⁵⁶ In this procedure, the suture is passed through both ends of the Achilles tendon percutaneously, thus allowing the repair with minimal incision. Numerous studies have looked at the efficacy of this procedure.^{57, 58, 53, 59, 47, 60-64} In the majority of these studies, percutaneous repair has been shown to have comparable results to open repair in terms of strength and return to activity. Moreover, these studies show a lower complication rate than open repair. Khan et al⁵³ found the relative risk of complications post- open repair compared to percutaneous repair to be 2.84 (95% CI 1.06-7.62). Cretnik et al⁴⁷ showed complications rates between open and percutaneous repair as 4.5% vs 12.4% respectively (p=0.013)

DISCUSSION

In this report we illustrate the case of patient DS, a classic example of patients at risk for bilateral spontaneous rupture of the Achilles tendon. DS received a high dose of intravenous steroids two weeks prior to his injury for exacerbation of chronic COPD. He had also just completed a 10-day course of Levaquin for an upper respiratory infection. He presented with heel pain and loss of plantar flexion, and his diagnosis was confirmed by Thompson's test and MRI.

Because of his extensive list of comorbidities and relatively low level of activity, we elected to treat DS non-operatively by casting him at first and then placing him in walker boots. He was discharged on coumadin for anticoagulation, which was unfortunately discontinued by another physician. Subsequently, he suffered a major complication—a near-fatal pulmonary embolism.

CONCLUSION

Bilateral Achilles tendon tears are rare, and usually occur in patients with chronic disease. Most cases are associated with corticosteroid use. Conservative treatment generally

effective for this population as it eliminates perioperative risks. However, immobilization does not guarantee a good result. Re-rupture rates are higher than operative repair and the gastrocnemius/soleus weakness is more pronounced. As this case

demonstrated, deep venous thrombosis and pulmonary emboli can be a devastating complication. Anticoagulants should be utilized in immobilized patients. Consideration should be given to functional Achilles boots that allow early motion.

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