A rehabilitation program for a 48 year old male with spontaneous bilateral patellar tendon ruptures

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Abstract

Introduction: Simultaneous bilateral patellar tendon ruptures are an extremely rare occurrence. Reported cases have been associated with known systemic disease processes such as systemic lupus erythematosus and rheumatoid arthritis. Rehabilitation post surgery is a long process and no standard protocol exists.

Case Description: A 48 year old male who simultaneously ruptured bilateral patellar tendons while playing dodge ball is presented. The patient's extensive history of substance abuse over a few decades offers a new possible causative factor to rupturing bilateral tendons.

Intervention: The intervention reported here includes an intense program of manual therapy, therapeutic exercise and modalities.

Outcome: The patient progressed through rehabilitation with no problems. He was able to return to work quickly as well as to some extracurricular activities. He was unable to progress to running or plyometric activities before his medical insurance stopped paying for his physical therapy treatments.

Discussion: This case report describes a detailed outline of a rehabilitation program for someone with bilaterally repaired patellar tendons. Further research and discussion is needed to create a standard protocol for rehabilitation. In addition, more research should focus on histological changes in the tendons as a result of recreational drugs, alcohol, and some medicinal drugs.

Introduction

The power of human knee extension in the lower limb is transmitted through the patellar tendon in the knee. It is responsible for generating the power used in everyday activities, such as walking, running, and jumping. Tension in this extensor mechanism can approach 17.5 times one's body weight during athletic activity.[1] This remarkable attribute is due to the patellar tendons strength, reported as the second strongest tendon in the body.[2] The weakest component of this mechanism is the patella itself, which often ends up taking the brunt of the forces through the knee, resulting in a transverse fracture.[1] Quadriceps tendon ruptures occur twice as frequently when compared to the patellar tendon ruptures.[1] Ruptures to the patellar tendon are infrequent, but catastrophic to function when they do occur. The patellar tendon also merges with the medial and lateral retinacula, which are usually torn during the injury as well. This causes further dysfunction of the extension mechanism due to the retinaculum's attachment to the vastus lateralis and vastus medialis muscles. All cases are characterized by the inability to extend the leg, and inability to actively flex the leg after it's passively extended.[3] Most ruptures occur in those with systemic disease processes including and not limited to metabolic diseases, those with corticosteroid injections, use of Fluoroquinolones, after total knee replacements, in those who use the central third of the patellar tendon for anterior cruciate ligament repairs, in athletes with repetitive trauma (jumper's knee), and poor soft tissue quality.[4]

Ruptures sites are frequently seen at the tendon's bony insertion, rather than the mid-section of the tendon.[1] The end regions, or bony insertions are under three to four times more stress than the midsection of the tendon when under peak loads.[5] Mechanism of injury are reported as an acute force, such as a rapid force shift, overwhelming a weakened structure

beyond its inherent tensile strenght.[2] An eccentric quadriceps force, or deceleration mechanism, with slight knee flexion is often seen with sport participants.[1]

Simultaneous bilateral patellar tendon ruptures are an even rarer occurrence than a single rupture. Due to the low occurrence rate there is a minimal amount of literature about these tears and the rehabilitation process. Those cases that have been reported are described in association with a systemic disease process such as rheumatoid arthritis, connective tissue diseases, hyperparathyroidism, diabetes, and systemic lupus erythematosus.[1] It is believed that the simultaneous rupture occurs during less strenuous, nonathletic activity secondary to the weakening and disorganization of the collagen structures of the knee from the disease process.[3] This is amplified by the repetitive microtrauma over time.

Several studies have been performed investigating the histological changes proceeding spontaneous ruptures of tendons. Kannus reported degenerative changes associated with ruptures including hypoxic degenerative tendinopathy, mucoid degeneration, and calcifying tendinopathy.[6] In the early stages of degenerative transformations, there are changes in the size and shape of the mitochondria and nuclei, and intracytoplasmic or mitochondrial calcification.[6] The later phases are attributed to hypoxic or lipid vacuoles, and occasional necrosis.[6] The collagen fiber changes include longitudinal splitting, disintegration, angulation, and abnormal variations in diameter.[6] The patellar tendon is 90% type I fibers, and fewer than 10% are type III.[5]

There is also an increase in tendolipomatosis in the patellar tendon when compared to other tendons such as extensor pollicis longus tendons. Tendolipomatosis are invasive soft tissue tumors that cause a decrease in the tensile strength of the tendon, as adjacent structures also become thinner and disrupted.[6] Another important aspect in degeneration include the

decline in water content found with aging, as well as the collagen turnover rate, and tenoblastic activity.[6]

The patella receives blood supply from the infrapatellar fat pad, and retinacular structures.[5] The infrapatellar fat pad contributes to the posterior aspect of the tendon, and the retinaculum supplies the anterior aspect of the tendon. However, the proximal and distal portions of the tendon have been found to be avascular.[5] These avascular areas are frequently the sites of patellar tendon rupture.[5]

Histological abnormalities in the musculoskeletal system haven been discovered in those abusing alcohol and drugs. Studies have estimated that 1/3 to 2/3 of alcoholics show chronic skeletal muscle myelopathy, including chronic atrophy and weakness.[7] York, et al. completed a study comparing strength among alcoholics and non-alcoholics.[7] Their findings revealed that alcoholics have decreased isokinetic strength in elbow, knee extensors and flexors, and ankle plantarflexor muscle groups.[7] In particular measurements of isometric strength of quadriceps revealed significant deficits in alcoholics at all hip angles studied.[7] Other studies have revealed alcoholics show Type IIb fast contracting fiber atrophy, a mild increase in fibrous tissue, mitochondrial changes such, and fraying myofibrils.[8] Alcoholics also have decreased protein synthesis in muscles. Another study has also correlated the degree of changes with the extent of alcohol abuse.[9]

It's been hypothesized that cocaine-induced muscle damage is the result of spasm and vasoconstriction of muscle associated vasculature, similar to the toxic effects seen elsewhere in the body causing heart attacks and cerebral vascular accidents.[10] In addition, cocaine abusers also have experienced rhabdomyolysis and/or muscle damage. Decreased blood flow can cause ischemia eventually leading to free radical formation in the muscles.[10]

Furthermore toxicity in tissues may also be the result of chemical reactions in the body producing increased creatine kinase levels, hydrogen peroxide, and other cocaine metabolites.[10] Since cocaine is metabolized in the liver by several enzymes, the increased serum creatine kinase levels combined with lipid peroxidation suggests damage is caused by the formation of free radicals. These free radicals further damage the muscle membranes, which further increase cytocolic enzyme release.[10] It is also possible that the effects of cocaine have an additive effect of repeated doses. Reported cocaine abusers with rhabdomyolysis are usually chronic users.[10]

The use of statin medications to lower cholesterol levels has also been associated with tendinopathies, which may predispose one to ruptures.[11] Achilles ruptures and statin use have been reported in literature in otherwise healthy subjects. One particular subject ruptured bilateral Achilles tendons, and had no tenderness or pain prior to the injury. Its believed that statins increase the risk of tendon rupture by altering matrix metalloproteases.[11] A high percentage of Achilles ruptures (83%) are associated with high lipoprotein levels.[11] Another study reported bilateral bicep tendon ruptures with usage of statins to lower cholesterol. In 2006, the Food and Drug Administration reported of 247 tendon ruptures associated with statin use.[12]

Surgery is the only option for individuals with ruptured patella tendons to optimally return to all functional activities. Studies have found that the only factor to relate to clinical outcomes is the length of time between the injury and surgical repair.[5] Those who delay surgery are at an increased risk of contractures, and inability to gain back full knee range of motion, quadriceps atrophy, and strength.[5] This is due to fibrous adhesions between the

patella and femur, retraction of the distal end of the patellar tendon on the tibial tuberosity within scar tissue, and/or calcific deposits.[5]

Progression of rehabilitation progress is physician dependent because of different post-op protocols. However, general rehabilitation includes range of motion, open kinetic chain isometric and isotonic strengthening exercises, closed chain exercises, mobilizations, and manual and active stretching activities. Many protocols call for immobilization lasting about six weeks in a plaster cast or brace.[4] Unfortunately this increases the stiffness of the knee.[4] Usually partial weight bearing with crutches is permitted; however someone with bilateral ruptures is typically forced to use a walker, weight bearing as tolerated on the foot while walking. Isometrics are begun the first day, and active and passive range of motion is initiated about two weeks later.[5] Functional electric stimulation can also used for quadriceps recruitment. Several different types of stretching techniques have been reported to decrease stiffness of the knee, and increase range of motion.[13] Cross friction massage to the patellar tendon is used to increase the blood flow to the tendon, facilitating healing.[14] Manual therapy consisting of joint mobilizations involve accessory glides of the joint or a physiologic movement of the joint to increase range of motion, and decrease pain.[15] A closed kinetic chain strengthening program is initiated, with further advancement to sport specific rehabilitation. Resuming recreational activities are allowed when a patient demonstrates full range of motion and 85% to 90% of strength compared to the contralateral leg.[5] Again, this poses a challenge for the physical therapist to monitor progress with bilateral ruptures.

The complicated and challenging rehabilitation process patients with bilateral patellar tendon ruptures go through is not well documented in the literature, indicating a lack of a

gold standard rehabilitation protocol. The purpose of this study is to identify an effective rehabilitation program for an individual recovering from surgery after tearing bilateral infrapatellar tendons. The research question is, what is the effectiveness of a mobilization, manual stretching, and therapeutic exercise program for someone recovering from surgically repaired patellar tendon rupture? This case was approved by the International Review Board at The Sage Colleges in Troy, NY.

Case Description

This patient is a 48 year old male who completely ruptured bilateral infrapatellar tendons with extensions into vastus lateralis and vastus medialis, while playing dodge ball with a youth group. He also sprained the right medial collateral ligament, and tore the lateral patellar retinaculum bilaterally. The patient states he was running and stopped quickly when one knee gave out, followed by the other. He immediately went to the emergency room with excruciating pain, tenderness, increasing edema, high riding patellas, and inability to bear weight on either leg. Upon examination he was also unable to perform a straight leg raise bilaterally. He had surgery four days later to repair the ruptures. There were no complications with the surgery. His doctors suspected he did have an infection on the distal region of the scar on the right leg, where the skin is the thinnest over the tibial tuberosity. The patient initiated outpatient physical therapy after receiving therapy in a rehab setting, as well as home therapy.

The patient is five foot eleven inches, and weighs 230 pounds. Current medical history includes hypertension, hyperlipidemia, and hypothyroidism. All of these are under the control with the use of medications. Those medications include 80mg Simvastatin, 0.125mg

Synthroid, and 20mg Prinivil. Past medical history is unremarkable, and reports no previous injuries to either knees. The patient's only reported surgery was a septoplasty. The only problem with the patellar tendon reconstruction occurred post surgery. He had acquired an infection near the distal end of the scar on the right leg, near the tibial tuberosity. It was treated with antibiotics and healed with no further complications. The left scar healed with no problems.

The patient reported at the initial examination that he is a recovered substance abuser. He was involved with this activity for several years, which may have predisposed him to histological changes in his tendons. He stated that he started drinking alcohol, using Marijuana, lysergic acid diethylamide (LSD), methamphetamine, qualudes and cocaine in 1978. He stopped using drugs in 1986, and resumed again in 1990. Drugs included Stadol nasal spray, extra strength Vicodin, Loratab, and Darvocet. He stopped using those in 1997. In 2000 he started using Marijuana, Cocaine, Oxycontin, Methadone and Xanax. His sobriety date for drugs and alcohol is August 17, 2005.

At this time the patient is unable to work as a medical device sales representative, and is unable to drive at the time of the initial evaluation. He is divorced and has two children who don't live with him. There are three levels in his house which have been an obstacle. The patient reported he would like to get back to work as soon as possible, golfing, and other recreational activities such as jet skiing, and running.

Examination

During the initial examination the patient presented ambulating with a wheeled walker and was wearing bilateral knee immobilizers. His chief complaint at the time was

significant knee stiffness, swelling, and weakness. He denied any significant amount of pain at this time.

The patient reports increased difficulty ambulating long distances as well as on stairs. He states he recently walked two to three miles using a walker, but was very uncomfortable doing so secondary to the immobilizers. The patient states that both ascending and descending stairs is difficult for him because he feels weak and unstable. He was wearing the immobilizers at all times, including night. He scored a 12/80 on The Lower Extremity Functional Scale at the initial visit, suggesting significantly altered functional ability.[16] The patient stated that his pain is 0/10 on the Borg Pain Scale when at rest.[17] He reported his pain increases bilaterally to an 8/10 on the Borg Pain Scale with certain activities. His pain was located mainly posterior to the knee cap.

The patient's scar was 14 cm long and was completely closed, but still in the process of healing. It was a healthy healing purple color. Girth measurements at the joint line were 41.8cm on the left, and 43cm on the right. Girth measurements 15 cm proximal to patella was 49.5 cm on the left, and 50 cm on the right. The patient's active range of motion for extension was 2° on the left and 3° on the right. Active range of motion for flexion was not tested at the initial examination as per rehabilitation prescription for passive range only at the time, by the patient's doctors orders. Passive range of motion for extension was 0° bilaterally, and 40° of flexion on the left, and 32° of flexion on the right.[16] The patient exhibited a negative Homan's test bilaterally. The patient's manual muscle testing revealed 3+/5 bilaterally of hip flexors, 4/5 hip abductors on the left, 4-/5 hip abductors on the right, 4/5 bilaterally ankle plantarflexors (seated), 4/5 ankle dorsiflexors on the left, and 4-/5 ankle

dorsiflexors on the right.[18] Hamstring and quadriceps flexibility was poor bilaterally. Knee flexion or extension strength was not tested secondary to doctor's orders.

Evaluation:

The patient was experiencing limited range of motion, strength, increased swelling, decreased flexibility, and functional ability at this time secondary to complete bilateral patellar tendon ruptures and surgical repair. The patient would benefit from physical therapy to increase knee range of motion, hip, knee and ankle strength, decrease pain, and swelling, and increase functional ability, as well as return to work and recreational activities.

Diagnosis:

The Preferred Practice Pattern this patient fits under is 4I: Impaired joint mobility, motor function, muscle performance, and range of motion associated with bony or soft tissue surgery in the Guide To Physical Therapy Practice, outlined by the American Physical Therapy Association.[19] This is appropriate due to his reconstructive surgery after completely rupturing bilateral patellar tendons and his current impairments.

Prognosis:

The patient was progressing as expected since surgery, as he followed the quadriceps tendon repair protocol. Over the course of three months the patient was expected to progress further increasing his strength, range of motion, flexibility, decrease swelling, and pain, and to improve functional activity tolerance, and return to work and leisure activities. Due to the

patient's motivation and enthusiasm to participate with all activities, it was predicted that the patient will achieve all goals and expected outcomes planned for him.

Short term goals included increasing passive range of motion of bilateral knees to 0-115° in 6 visits; increase knee stability and control to be able to walk on uneven surfaces such as sand or grass while using a single point cane without bilateral knee immobilizers in 6 visits; improve ability to stand for 30 minutes at a time and ambulate over 500ft distances in 6 visits to be able to return to work; the patient will report a 0/10 on the Borg Pain Scale to be able to perform all exercises in therapy and while performing his home exercise program; and the patient will also verbalize understanding and demonstrate independence with a home exercise program within the first 6 visits.

Long term goals include the patient to restore active and passive range of motion in bilateral knees to 0-130° without symptoms at discharge to be able to squat and ambulate stairs with no report of difficulty; report a feeling of control and stability of bilateral lower extremities while ambulating on uneven terrain without use of any assistive devices to be able to play golf and walk on the beach; increase strength to 5/5 by discharge to be able to participate in recreational activities with no report of discomfort; ambulate a full flight of stairs with no report of feeling unsteady or weak to be able to ascend and descend stairs at work; as well as improve strength and control of quad extensions at discharge to be able to run and return to sports at discharge.

Plan of Care:

The patient was seen in outpatient physical therapy two or three times a week, for 10 weeks. Treatment included a home exercise program to emphasize exercises performed at

therapy, therapeutic exercises to improve strength and range of motion, gait training without an assistive device as per doctor's prescription. The patient also benefited from strengthening hips and ankles bilaterally to prevent any loss of strength in those muscle groups due to decreased use. All physical therapy sessions also included manual therapy by a physical therapist. Manual stretching techniques, joint mobilizations, scar massage, manual resistance exercises and modalities as needed for pain and inflammation were used.

Intervention:

The patient participated in all physical therapy sessions in outpatient physical therapy. Interventions incorporated therapeutic exercises including strengthening and flexibility exercises of the knee musculature, gait training, and manual therapy. Modalities such as neuromuscular electrical stimulation, and ice were also used. The patient's doctor informed the physical therapists when to begin the next phases of therapy.

Neuromuscular electrical stimulation was used bilaterally to achieve an involuntary contraction of the quadriceps.[15] This was used at the beginning of each physical therapy session. The pads were placed over the vastus medialis and mid-rectus femoris for fifteen minutes which were on for 10 seconds, and off for 50 seconds. The patient reported when he felt a comfortable contraction.

Therapeutic exercises ranged from strengthening exercises to exercises to improve flexibility and range of motion. Documented in Table 1 are the exercises this patient performed, including when they were started, and when they progressed the exercise by increasing the weight. Strengthening exercises were focused on not only the quadriceps but the other muscles of the lower extremities that stabilize and coordinated with the quadriceps.

This will also prevent any weakness and misuse injuries from occurring to those muscle groups. The hamstrings, hip flexors, ankle dorsiflexors, and plantar flexors were also strengthened. The standing straight leg raise targeted the hip flexors, and the lateral leg raises and posterior leg raises targeted the gluteal muscles. The straight leg raise, lateral leg raise, and posterior leg raise were also performed. Quad sets, and adductor sets were initiated at the first visit as well. Finally a blue TheraBand® was used to strengthen and maintain the ankle muscle groups.

At about the fifth week of therapy the doctor allowed quadriceps strengthening exercises to begin. Using a black TheraBand®, the patient hooked a fastened TheraBand® around the proximal calf. With the resistance provided by the TheraBand®, the patient performed knee extension. The patient also initiated seated knee extensions, minisquats, leg curls, hip flexion, and hip extension using machines at this time. Around the 7th week of therapy the patient was able to perform a full rotation on the stationary bike. Up until that point he was rocking back and forth to facilitate the movement and stretch.

Flexibility exercises targeted the same groups, especially the quadriceps to increase range of motion of the knee joints. Most stretches were held for 30 seconds, and 5 sets were performed.[20] The Standing Lunge Stretch was initiated at the first visit to increase range of motion while applying some weight on the joint. The patient determined how much he could tolerate when applying the stretch. The patient also performed a hamstring stretch. While the patient stood on the side of a plinth, he stretched the leg closest to the table by resting it on top of the plinth, with an extended knee.

After one week of therapy the patient was able to perform additional range of motion exercises. A prone knee flexion stretch was started. While prone the patient hooked one leg

in front of the other, and gently flexed the knees to the tolerated flexed position. Heel slides, and a seated knee flexion stretch was also started at that time.

Scar massage was done for about five minutes prior to mobilizations and manual stretches.[21] It was applied transversely across scar, and superiorly and inferiorly to inhibit any adhesions to the deep tissue. A cross friction massage was performed when the patient reported increased tightness of the iliotibial band near the insertion to the knee.

Joint mobilizations were administered to increase range of motion, and decrease pain.[22] Grade three Kaltenborn mobilizations were carried out to increase flexion and extension of the knee, as well as patellar mobility. Patellar mobilizations were performed mobilizing it in all four planes: medially, laterally, superiorly and inferiorly. Knee extension mobilizations were performed with posterior to anterior glides of the proximal tibia, while stabilizing the femur, as well as performing anterior to posterior glides of the femur on the tibia, which is stabilized with a towel roll under the mid calf. The patient was in long sitting while these mobilizations were performed. Flexion mobilizations were performed with the patient in supine position, and the knee in maximum flexion. Anterior to posterior glides of the proximal tibia and fibula were performed with the knee in flexion. An inferior glide of patella was also performed with the knee in the flexed position. After several oscillations the patients knee was gradually flexed more until the maximum barrier was reached.[22]

Manual stretching techniques were used after mobilizations were done.[13] With the patient in supine, the hip and knee were positioned into 90° of flexion. The knee flexion was slowly increased to patients maximum toleration or tissue barrier. This stretch was held for 2 sets of 20 seconds, and then held for 30 seconds, for another 2 sets. With the patient in prone lying, the knee was flexed to maximum toleration for 3 sets of 30 seconds. Hamstring

stretches were performed with the patient supine, while passively bringing up the leg. These were held for 3 sets of 30 seconds.

Finally manual resistive exercises were initiated on the 8th week. With the patient seated at edge of the plinth, the patient extended the knee against manual resistance provided by physical therapist. Three sets of 10 repetitions were completed with a 30 second rest in between each set.

At the end of each physical therapy session ice was applied bilaterally to the patient's knees for 10 minutes for pain, and inflammation.[15] A home exercise program was also given to the patient on the first visit. Exercises included quadriceps and adductor sets, hip lateral leg raise, straight leg raises, posterior leg raises, ankle dorsieflexion and plantarflexion using a blue theraband. The patient was also informed that exercises he learned at therapy as he progressed should also be incorporated into his home program such as heel slides, minisquats, and seated knee extensions.

Outcome:

The patient's left knee immobilizer was removed about one week after initiating therapy on 5/26/09, and the right immobilizer was gradually weaned off about one week later, as per doctor's orders. The right knee's progress was about one to two weeks behind secondary to starting range of motion exercises later because of the infection, and more extensive damage in that knee. The patient began using bilateral single point canes the second week of therapy, and no longer needed them about a week and half later. He was driving within a couple of weeks after initiating therapy with no problems. He also was able to return to work, attending surgeries which required him to stand for long periods of time.

He was able to perform a step over step pattern on the stairs while using railing within the first month as well. The patient attempted to play golf the 6th week into therapy, but was limited due to soreness and decreased walking efficiency. All short term goals were achieved with his quick progress with no problem.

Half way through treatment The Lower Extremity Functional Scale was given again and showed progress with a score of 28/80 compared to the 12/80 at initiation of treatment.[23]

The patient's treatment was cut off due to insurance reimbursement. At his last treatment session the patient reported a 70-75% return to all activities he wants to be able to do. He returned to work, and was playing golf again, and reported no discomfort with either activity which was one of his goals he did achieve. He was able to ambulate easier over grass and uneven surfaces. However, he reports that he doesn't feel like he is walking "normally" yet, and notes that he is still unable to walk long distances without some discomfort. He still had a wide base of support with a decreased stance time on the right. The patient was unable to progress to running or plyometric activities before his medical insurance stopped paying for his physical therapy treatments. He reported he occasionally has some discomfort when descending stairs, but otherwise reports no problems ascending and descending a full flight of stairs. He needs to use elevators when faced with ascending and descending several flights of stairs in hospitals, and different work environments. The patient reports minimal to no pain with all activities. The most discomfort he feels is in the infrapatellar region of the knee with some exercises, and when he is over exerting himself.

The patient didn't achieve his long term goal for knee flexion (130°), but came within several degrees of achieving this goal. Table 2 outlines the progression of his range of motion. Active range of motion is 125° on the left, and 119° on the right for knee flexion.

Active knee extension is -2° on the left and 0° on the right. Passive knee flexion range of motion on the left is 125°, and 122° on the right. Prone passive knee flexion range of motion is 122° on the left, and 116° on the right. The patient's goal for flexibility was partly achieved. His hamstring flexibility was fair at discharge, and his quadriceps flexibility was good/normal.

The patient didn't achieve his strength goal of a manual muscle testing grade of 5/5 for knee extension (quadriceps muscles) and flexion (hamstring muscle group). These were tested as 4-/5, but hip flexion and abduction strength was measured as 5/5 bilaterally.

Discussion

The rehabilitation process is a difficult and long process for someone with repaired bilateral patellar tendon ruptures. This patient responded well as predicted to the therapy interventions used. Unfortunately the funding by his medical insurance for physical therapy was cut off before he was able to start higher functioning, sport specific rehabilitation such as running, and jumping. The patient was back to work, playing golf, and most functional activities with no problem. He was pleased with his progression and was patient with the rehabilitation progress as suspected.

Various manual therapy techniques were used to increase the patient's range of motion, and to relieve pain. Knee mobilizations, cross friction massage, and manual stretching were used for each treatment. Cross friction massage of the patellar tendon was used at each treatment, and massage to the iliotibial band was utilized when the patient reported increased pain and tightness near the lateral knee. This was performed to address abnormal scar tissue secondary to chronically inflamed tissue, mobilizing the skin and

underlying deep tissue.[14] The patient responded well to this with no reported problems or discomfort. Joint mobilizations attributed to increasing his range of motion, and decreasing pain through neurophysiologic effects.[15] Mobilization ranges were brought to the patient's physiologic and tolerated end point. Stretching followed to address soft tissue tightness surrounding the knee point, inhibiting any contractures from developing. At the end of his physical therapy treatment the patient's range of motion (-2-128° on the left, and 0-122° on the right) was improving towards normal according to McGee, who reports normal range of motion for the knee is 0-135°. However, the end feel of the left knee was due to tissue approximation, and therefore would not progress much further. McGee reports that functional range of motion necessary for everyday activities such as squatting to tie a shoe, sitting in a chair, and climbing stairs needs only 0-117°. This suggests that the patient will be able to perform all functional activities without any problems, and the remaining degrees of motion he is lacking will not hinder his functional status. His right knee lagged behind the left secondary to starting rehab on that leg later due to the infection after the surgery, but it was progressing at the same rate as the left knee.

Therapeutic exercises were tolerated well throughout the plan of care. An exercise program consisting of aerobic exercises, various strengthening exercises, as well as self-stretches were used throughout his plan of care. More vigorous strengthening exercises truly began about one month after initiating outpatient physical therapy as the patient's doctor ordered. These targeted his quadriceps, as well as hip flexors, hamstrings, and gastrocnemius muscles. The patient reported the pain and discomfort he felt doing the exercises dissipated quickly after each session. His strength was improving, but didn't fully return to optimal strength at the end of his treatment. The patient reported improving ease with walking, but

his decreased strength and muscle tightness of the lower extremity presented in an abnormal gait pattern at his last visit. He also reported that ascending and descending stairs was no longer a problem as it was previously, apparent by improved motor control of his quadriceps and surrounding muscle groups. His decreased strength was also the reason he was unable to begin running and plyometric activities. Manual resistive exercises for knee extension were started the week before he was discharged. He was progressing well with these exercises just as he was with the other strengthening exercises. With the expectation that he continues his home exercise program and the exercises given in therapy, the patient should be able to begin running and jumping at some point in the future.

The use of knee cryocuffs were used at the end of the treatments to decrease skin and joint temperature, and decreases blood flow, therefore acting as an analgesic and to decrease worsening of any present inflammation.[15] The patient responded well to this. The use of bilateral neuromuscular electrical stimulation was also tolerated well. This was used to achieve an involuntary contraction of the quadriceps, facilitating motor control and prevented further atrophy of the quadriceps from disuse.

In a study by Kannus, several patients with tendon ruptures report increased tenderness, stiffness, pain or discomfort before the ruptures occur, suggesting a possible inflammatory or degenerative process.[6] However, Kannus reported that two thirds of his participants did not have any proceeding symptoms before the ruptures.[6] The patient in this study reported no previous injuries, pain or tenderness to patellar tendons prior to the injury. This suggests that the degeneration process could also be asymptomatic.

While no definitive reason was given for this particular patient simultaneously rupturing both patellar tendons, several assumptions can be made. The patient wasn't taking

any steroids or fluoroquinolones, which have all been linked to simultaneous ruptures,[11,12] but he was taking a statin for lowering cholesterol. Many studies have exposed the musculoskeletal side effects this drug can cause. Simultaneous bilateral Achilles, and bicep brachii ruptures with no previous symptoms have been identified in literature, associated with statin use to decrease cholesterol levels.[11] It's also possible the number of ruptures associated with statins are under reported secondary to health professionals unawareness to the possible effects on tendon metabolism and tendinopathy.[11]

It is also suspected that the heavy use of drugs and alcohol for many years in this patient's past could have predisposed him to this injury secondary to histological changes in tissues. Many of the histological changes that have been researched are in muscles. However, for a joint and tendon to withstand the loads placed on it from the muscle, muscular control and tone need to be functioning properly as a unit. Patellar tendon ruptures have not been identified in the literature as being a result of drug or alcohol abuse; however Achilles tendon ruptures in drug abusers have been identified. This offers a new perspective to the literature, with a possible link to tendinopathies and/or systemic process resulting in bilateral ruptures.

Some limitations to the present study is the lack of literature pertaining to the cause and rehabilitation process of someone with bilateral ruptures. The protocol followed was determined by the patient's doctor rather than a gold standard in a rehabilitation setting. Some other limits are in the outcome measures. Most of this patient's progress was verbally reported by the patient, and his progress was also dependent on his perception. This decreases the generalizability of the program to others. It's suspected that not everyone will respond equally as well to some of the treatments as this patient had. The patient's visits

were unexpectedly cut off by insurance, therefore his final outcomes documented and used in this study weren't fully assessed as discharge outcomes. Previous outcome measures were used in the outcome assessment for this study. The patient was also unable to progress to higher functioning tasks and rehabilitation such as running, and his strength had not returned to normal.

The exact pathogenesis of tendonopathies and ruptures of the patellar tendon is a complex mixture of intrinsic and extrinsic factors.[6] Whether the histological changes occur from recreational drugs, medications, disease processes, degenerative changes associated with aging and/or overuse, or a combination, the changes are often asymptomatic until an acute rupture occurs. The cause of this patient's bilateral tendon ruptures is unknown, but is most likely due to the lengthy abuse of recreational drugs, medications, alcohol, and prescribed usage of statins.

The literature on simultaneous bilateral patella tendon ruptures is lacking due to the rarity of the injury, and the literature on a proper rehabilitation program is also lacking. A program consisting of several therapeutic exercises, various manual therapy techniques to focus on increasing range of motion, modalities to maintain and facilitate muscle contractions, and decrease pain resulted in favorable outcomes for this patient. The program reported here resulted in a 70-75% return to all activities of daily living, most recreational activities, and the patients return to work.

Future research should focus on simultaneous bilateral tendon ruptures, of not only the patella, but of other main tendons such as the Achilles, and biceps brachii tendons. Several factors have been identified and reported as causing bilateral ruptures but many are inconclusive. More patient specific drug usage, either recreational or medicinal, chronic

alcoholism, and disease processes should all be considered as possible factors increasing the susceptibility of bilateral tendon ruptures, especially the combination of several factors. A more standardized protocol for patellar tendon ruptures is also needed for use in a physical therapy setting to improve and optimize patient outcomes by creating a more standard plan of care. More studies on the effectiveness on plyometric exercises should also be utilized to further increase strength and control of the quadriceps. These should be carried out with a great deal of caution to prevent re-injury and re-rupture of the repaired tendon.

Conclusion:

The current study sought to look at an intense range of exercises, manual therapy techniques including mobilizations and manual stretching, and modalities in a rehabilitation program for a patient with bilateral patellar tendon ruptures who had a history of drug abuse. This program allowed a 48 year old man to return to work and most recreational activities within a few months of surgical repair. He is unable to run and jump but is expected to do so with time as he further increases his strength and fine motor control of muscles surrounding the knee joint. Further research should focus on creating a standard protocol for rehabilitating repaired patellar tendons. In addition, several studies have reported that the cause of bilateral ruptures are due to systematic disease processes, but additional research should focus on histological changes in the tendons as a result of recreational drugs and alcohol, as well as medicinal drugs.

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Table 1. Therapeutic Exercises for Bilateral Patellar Tendon Rupture Rehabilitation											
	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9	Week 10	Week 11
Standing Lunge stretch	X	X	X	X	X	X	X	X	X	X	X
SLR	X	X	X	X	X	X	X	X	X	X	X
Quad sets	X	X	X	X	X	X	X	X	X	X	X
Adductor sets	X	X	X	X	X	X	X	X	X	X	X
Hamstring stretch	X	X	X	X	X	X	X	X	X	X	X
TheraBand ®: DF, PF	X	X	X	X	X	X	X	X	X	↑	X
LLR	X	X	X	X							
PLR	X	X	X	X							
Standing Hip Exercises	X	X	X	X							
Prone knee flexion stretch			X	X	X	X	X	X	X	X	X
Heel slides			X	X	X	X	X	X	X	X	X
Seated knee flexion stretch			X	X	X	X					
Seated knee extension					X	X	↑	X	↑	↑	↑
Terminal knee extension					X	X	X	X	X	X	X
Mini-squats					X	X	X	X	X	X	X
Resisted Multi hip flexion					X	↑	↑	X	X	X	X
Resisted Multi hip extension					X	↑	↑	X	↑	↑	X
Leg curls					X	↑	↑	X	X	↑	X
Bike							X	X	X	X	X

*SLR(straight leg raise); LLR(lateral leg raise); PLR(posterior leg raise); TB (Theraband); DF(dorsiflexion); PF(plantar flexion); ↑(increased weights used)

Table 2. Bilateral Knee Measurements by degrees										
	12-May/Wk 1		4-Jun/Wk 4		15-Jun/Wk 6		20-Jul/Wk 10		24-Jul/Wk 11	
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
Flexion PROM	32°	40°	78°	95°	105°	119°	122°	125°	122°	125°
Extension PROM	0°	0°	0°	0°	0°	0°	0°	-2°	0°	-2°
Flexion AROM	NA	NA	75°	90°	100°	116°	119°	125°	120°	128°
Extension AROM	3°	2°	1°	1°	0°	0°	0°	-2°	0°	-2°
Flexion Strength	NA	NA	3/5	3/5	3+/5	3+/5	4/5	4/5	4/5	4/5
Extension Strength	NA	NA	NA	NA	3+/5	3+/5	4-/5	4-/5	4-/5	4-/5