Simultaneous Patellar, Quadriceps, and Triceps Tendon Ruptures in a Professional Male Bodybuilder After Low-Impact Trauma

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ABSTRACT

BACKGROUND: Simultaneous rupture of multiple major tendons is exceedingly rare and is typically associated with high-energy trauma or underlying systemic disease. This report describes the management and outcome of an unusual low-energy, three-tendon rupture involving the quadriceps, patellar, and triceps tendons in a professional bodybuilder.

CASE: A 36-year-old male professional bodybuilder presented after slipping on ice with acute loss of bilateral knee extension and right elbow extension. Physical examination and imaging confirmed complete rupture of the left quadriceps tendon, right patellar tendon, and right triceps tendon. The patient underwent single-stage surgical repair of all three tendons, utilizing transosseous fixation for both knee tendons and a suture anchor construct for the triceps tendon, followed by a tailored rehabilitation protocol. Functional and radiographic outcomes were assessed over one year. At one year, the left quadriceps and right triceps demonstrated a full range of motion and strength, allowing the patient to return to work without limitation. Imaging of the right patellar tendon demonstrated proximal tendon discontinuity and patella alta, consistent with repair failure. Despite this, the patient maintained 5/5 quadriceps strength, intact straight leg raise, and reported no limitations in daily activities, likely due to compensatory stabilization from an intact retinaculum. Revision surgery was recommended but deferred.

CONCLUSION: This case demonstrates that simultaneous multi-tendon rupture can occur after low-energy trauma in the absence of identifiable systemic disease. Early clinical suspicion supported by imaging and coordinated surgical and rehabilitative management can yield meaningful functional recovery, even in the presence of radiographic failure.

LEVEL OF EVIDENCE: IV

KEYWORDS: Quadriceps tendon rupture; Patellar tendon rupture; Triceps tendon rupture; Low-energy trauma; Tendon repair

INTRODUCTION

Tendon ruptures are typically isolated injuries associated with age, activity, or trauma severity. Quadriceps tendon ruptures most commonly occur in patients over 40 years of age and are often associated with underlying degenerative changes.[1-3] In contrast, patellar tendon ruptures are more common in younger active individuals during sports activities involving eccentric loading of the flexed knee, such as jumping.[1,2,4] Triceps tendon ruptures are rare and most often occur following a fall on an outstretched hand or via direct elbow trauma.[5-7] While isolated ruptures of these tendons are well-described, simultaneous rupture of multiple tendons is exceedingly rare and generally requires high-energy trauma. The occurrence of combined upper and lower extremity tendon ruptures following low-energy mechanisms is particularly unusual and has rarely been reported in the literature. We present a rare case of grade 3 ruptures of the left quadriceps, right patellar, and right triceps tendons in a 36-year-old male professional bodybuilder following a low-energy fall.

Extensive case history exists on anabolic-androgenic steroid (AAS) users suffering tendon ruptures, dating back to 1995.[8,9] A recent report was also published on an AAS-using bodybuilder who suffered a triceps tendon rupture.[10] However, the patient had no reported prior history of systemic illness, pharmacologic risk factors, or steroid use, making the injuries unusual. This report highlights surgical decision-making and rehabilitation strategies required for multi-tendon injuries.

Informed consent was obtained from the patient for the publication of this case report.

CASE DESCRIPTION

Pre-operative Course

A 36-year-old male professional bodybuilder (height: 6′0″, weight: 236 lbs., BMI: 32) presented to the emergency department following a low-energy fall on ice, sustaining sudden eccentric contraction of his right leg and direct impact to his right upper extremity. The patient reported immediate inability to bear weight. Physical examination revealed complete loss of active knee extension bilaterally, with the left knee demonstrating a 3 cm palpable gap proximal to the patella and the right knee exhibiting patella alta. The right elbow displayed a 2 cm retracted mass at the triceps

Figure 1. Sagittal T2-weighted fat-suppressed MRI of the right knee with discontinuity of the proximal patellar tendon and periosteal avulsion from the inferior pole of the patella. The retracted tendon is visualized proximal to the patella with associated effusion and soft-tissue edema. [STUDY: RIGHT KNEE; SERIES: T2 FS SAG]

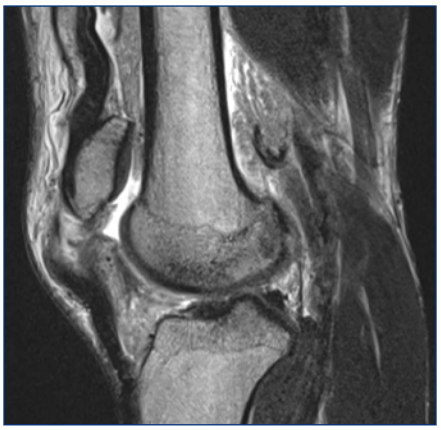


Figure 2. Sagittal T2-weighted MRI of the right elbow demonstrating complete distal triceps tendon rupture. The tendon is retracted proximally from the olecranon, with high signal fluid interposed at the expected insertion site, and no bridging fibers identified. These findings are consistent with a full-thickness avulsion of the triceps tendon.
[STUDY: RIGHT ELBOW; SERIES: T2 FS SAG]

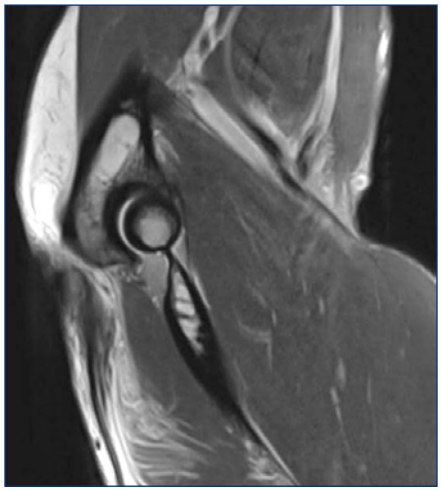
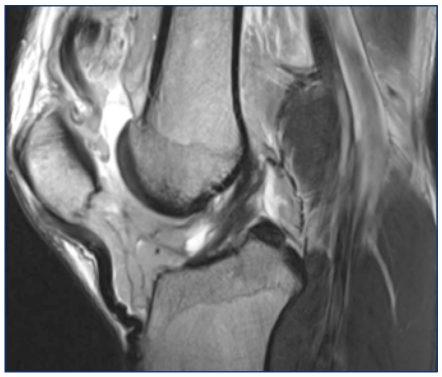


Figure 3. Sagittal T2-weighted fat-suppressed MRI of the left knee showing full-thickness quadriceps tendon rupture. The tendon is retracted proximally from its patellar insertion, with high T2 signal intensity occupying the gap, and no intact bridging fibers seen. Associated soft- tissue edema and suprapatellar effusion are present.
[STUDY: LEFT KNEE; SERIES: T2 FS SAG]



insertion with absent active extension against gravity. The patient denied any prodromal symptoms.

Radiographs revealed a left patella baja, a right patella alta, and no fractures. The right knee MRI showed complete patellar tendon avulsion from the inferior pole of the patella with medial retinaculum laxity [**Figure 1**]. Radiograph of the right elbow revealed tissue swelling over the olecranon, and MRI demonstrated a full-thickness triceps tendon tear with 2-3 mm diastasis [**Figure 2**]. MRI also demonstrated a 3.8 cm retracted left quadriceps tendon tear with partial vastus lateralis injury [**Figure 3**]. Laboratory investigations, including a complete blood count [**Table 1**] and a comprehensive metabolic panel [**Table 2**], were not indicative of steroid usage. The patient was admitted for orthopaedic consultation and pre-operative optimization. Indications for surgery were functional deficits from complete tendon rupture (ambulation compromised from bilateral knee extension loss and upper extremity function compromised from right elbow extension loss). Due to decreased right arm function, it was determined that restoring upper extremity strength would be beneficial for mobility during lower extremity recovery.

Intra-operative Course

The patient underwent single-stage repair of all three tendons. Anterior midline approach identified complete avulsion of the left quadriceps tendon with a 2 cm retracted periosteal sleeve from the superior patellar pole. The quadriceps tendon tissue demonstrated no gross calcification, degeneration, or features suggestive of chronic tendinopathy. After tendon debridement, a transosseous repair was performed using #5 FiberWire sutures (Arthrex, Naples, FL) passed through three drill holes in the patella (medial, central, lateral). The retinaculum was concurrently repaired with a locking epitendinous stitch. An anterior longitudinal incision exposed a complete avulsion of the right patellar tendon from the inferior pole with significant periosteal stripping. The patellar tendon tissue demonstrated no intraoperative evidence of degeneration, calcific deposition, or chronic attritional tearing. An identical transosseous technique was employed, securing the tendon to its anatomic insertion. The right triceps tendon was addressed via a posterior elbow approach, revealing a full-thickness tear at the olecranon insertion. The right triceps tendon tissue appeared robust and consistent with an acute avulsion injury without evidence of degenerative changes or poor tissue quality. This was reconstructed using the SpeedBridge system with two PEEK SwiveLock anchors (Arthrex, Naples, FL), providing biomechanical stability while avoiding excessive soft-tissue dissection.

Post-operative Course

Post-operatively, an immobilization protocol was required. Bilateral hinged knee braces were locked in extension to permit brace-protected weight-bearing as tolerated, and the right elbow was splinted at 60° of flexion with non-weight-bearing precautions. To enable upright mobility, the patient ambulated with a front-wheeled walker outfitted with a right forearm platform. Pain was managed with scheduled acetaminophen and oxycodone.

Rehabilitation from weeks 0–6 emphasized strict immobilization, with passive range of motion initiated at four weeks under therapist supervision. At weeks 6–12, hinged braces

Table 1. Pre-Operative Complete Blood Count (CBC) Panel Data with Differential

Component	Reference Range & Units	Result
WBC	3.50–11.0 10*3/uL	9.7
RBC	4.20–5.50 10*6/uL	**4.15**
Hemoglobin	13.5–16.0 g/dL	**12.3**
Hematocrit	37.0–47.0 %	37.9
MCV	80.0–98.0 fL	91.4
MCH	26.0–34.0 pg	29.7
MCHC	32.0–36.0 g/dL	32.5
RDW	11.5–14.5 %	**16.3**
Platelets	150–400 x10*3/uL	326
MPV	7.4–10.4 fL	7.9
Neutrophils (Relative)	%	61.0
Lymphocytes (Relative)	%	24.0
Monocytes (Relative)	%	13.0
Eosinophils (Relative)	%	2.0
Basophils (Relative)	%	0.3
Neutrophils (Absolute)	1.5–7.5 10*3/uL	5.9
Lymphocytes (Absolute)	1.0–4.0 10*3/uL	2.3
Monocytes (Absolute)	0.2–0.8 10*3/uL	**1.3**
Eosinophils (Absolute)	0.0–0.5 10*3/uL	0.2
Basophils (Absolute)	0.0–0.2 10*3/uL	0.0
RBC Morphology	—	**Abnormal**
Hypochromia	—	1+
Polychromasia	—	1+
Ovalocytes	—	1+
Eosinophils (Absolute)	0.0 - 0.5 10*3/uL	0.2

Abbreviations: WBC = White Blood Cell, RBC = Red Blood Cell, MCV = Mean Corpuscular Volume, MCH = Mean Corpuscular Hemoglobin, MCHC = Mean Corpuscular Hemoglobin Concentration, RDW = Red Blood Cell Distribution Width, MPV = Mean Platelet Volume
Bold indicates out-of-range values

were progressively unlocked to allow controlled motion (0–90° for the knees, 30–90° for the elbow). By 12 weeks, the patient began outpatient physical therapy focused on gait training and progressive strengthening.

Right Triceps Tendon: One month post-op, the right elbow achieved 0–90° active/passive range of motion (ROM) without palpable defects and with antigravity strength. By three months, ROM improved to 0–115°, and the brace was discontinued. At six months, ROM (0–135°) was attained with functional extension strength. At one year, elbow function remained intact without daily limitations.

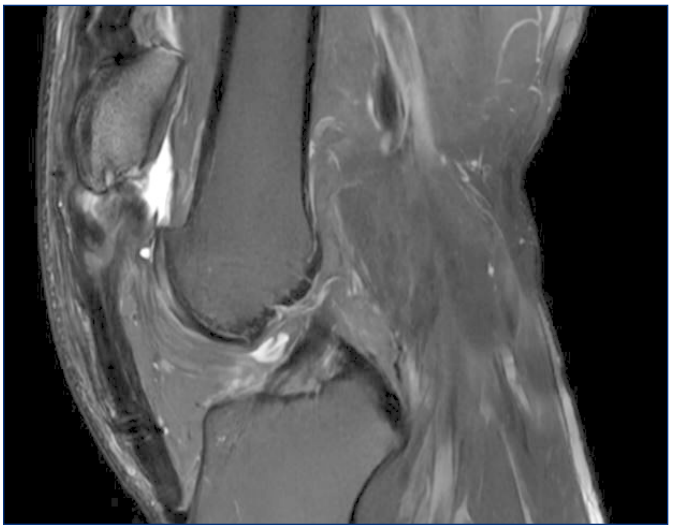
Left Quadriceps Tendon: At one month, the left knee had 0–60° ROM without extensor lag and could support weight-bearing in a brace. By three months, ROM expanded to 0–110° with full passive extension, and the patient demonstrated normal quadriceps activation. Strength continued to

Table 2. Pre-Operative Comprehensive Metabolic Panel (CMP) Data

Component	Reference Range & Units	Result
Sodium	136–145 mmol/L	138
Potassium	3.5–5.3 mmol/L	3.8
Chloride	98–110 mmol/L	103
CO_2	20.0–31.0 mmol/L	31.0
BUN	6.0–24.0 mg/dL	19.0
Creatinine	0.6–1.2 mg/dL	1.1
Glucose	70–140 mg/dL	125
Calcium	8.6–10.4 mg/dL	**8.3**
AST	10–36 U/L	**100**
ALT	9–46 U/L	**123**
Alkaline Phosphatase	40–115 U/L	64
Total Protein	6.4–8.4 g/dL	6.9
Albumin	3.6–5.1 g/dL	**3.1**
Total Bilirubin	0.2–1.2 mg/dL	0.2
GFR	>60.0 mL/min/1.73m^2	>60.0
Anion Gap	6–19 mmol/L	**4.0**

Abbreviations: BUN = Blood Urea Nitrogen, AST = Aspartate aminotransferase, ALT = Alanine aminotransferase, GFR = Glomerular Filtration Rate
Bold indicates out-of-range values

Figure 4. Sagittal T2-weighted fat-suppressed MRI of the right knee at follow-up showing irregular morphology of the proximal patellar tendon with heterogeneous signal, interposed fluid, and surrounding soft-tissue edema. A gap at the tendon origin with proximal retraction further supports the mechanical failure of the previous repair.
[STUDY: RIGHT KNEE; SERIES: T2 FS SAG]



improve, allowing return to work as a personal trainer at six months with full functional ROM (0–125°), which was sustained at one year (0–130° ROM), with no residual weakness affecting mobility.

Right Patellar Tendon: At one month, ROM was 0–60° with intact but limited strength. By three months, ROM

improved to 10–110°, and straight leg raise was intact. At six months, the patient ambulated independently and began light exercise, though mild swelling persisted. At one year, examination of the right knee revealed active and passive ROM from 3° to 125° and the ability to obtain and maintain a straight leg raise without extensor lag. Strength testing revealed 5/5 quadriceps strength. Despite preserved extensor mechanism function and activity modification, the patient reported, and an orthopaedic examination demonstrated moderate swelling of the right knee. Given the chronicity of the effusion and persistent symptoms despite conservative management, an MRI of the right knee was obtained to further evaluate the extensor mechanism. MRI of the right knee showed signal heterogeneity and irregularity at the patellar tendon origin with mild patella alta and a stable effusion, suggestive of surgical failure [**Figure 4**]. Despite this, the patient reported no limitations with daily activities. Revision with Achilles allograft was recommended but deferred until after the patient's out-of-country wedding, after which he did not return for follow-up and remained unreachable despite repeated attempts at contact by the clinical team.

DISCUSSION

This case represents a rare instance of simultaneous quadriceps, patellar, and triceps tendon ruptures following low-energy trauma. Previous reports of a patient with Ehlers-Danlos syndrome who sustained a bilateral patellar tendon rupture and another patient with systemic lupus erythematosus who sustained a bilateral Achilles tendon and patellar tendon rupture address patients with known etiologies.[11,12] The occurrence of this simultaneous tri-tendinous rupture after a low-energy fall highlights the importance of considering atypical etiologies even in "healthy" adults. Metabolic workup was pursued to identify potential underlying causes; however, potential factors such as hyperparathyroidism, inflammatory arthritis, and collagen disorders were ruled out by serum studies. Additionally, the patient denied fluoroquinolone or corticosteroid use.[13-15] Family history was unremarkable, with no reported instances of spontaneous ruptures or hypermobility disorders. While no definitive etiology was identified, the negative workup itself is instructive, suggesting that subclinical tendon degeneration or biomechanical factors may predispose certain individuals to significant impairments after seemingly trivial traumas. In this case, his occupation suggests that chronic repetitive loading during exercise may have caused subclinical tendon degeneration, thereby increasing the likelihood of mechanical failure. Notably, a possible history of unreported usage of AAS or substances that do not appear on standard panels could have compromised tendon integrity. Mechanistically, animal and human studies suggest that AAS may alter tendon collagen metabolism, increase tendon stiffness, and reduce compliance, potentially predisposing to rupture.[16–18] The patient's muscular physique and low-energy injury mechanism make unreported AAS exposure a consideration. If so, this case would corroborate Kanayama et al's findings on how steroid-utilizing bodybuilders had a higher incidence of tendon ruptures, often outside of weightlifting.[15] A high index of suspicion for additional complications should also be maintained when the source of trauma is less overt or not indicative of the presented injury; this was how the triceps tendon rupture was discovered by the orthopaedic team during the initial consult examination before imaging studies.

While the patient adhered to rehabilitation protocol, having two compromised lower extremities can make it difficult to follow non-weight-bearing guidelines on both extremities; this may have contributed to the right patellar tendon never fully healing. When the patient began light resistance weight training exercise six months post-op, he may have inadvertently pushed himself too hard, possibly contributing to a reinjury. In cases of failed primary patellar tendon repair, revision surgery may require reconstructive techniques depending on tendon retraction, scarring, and tissue quality. Reconstructive strategies include hamstring autograft or synthetic augmentation, and bone-patellar tendon-bone or Achilles tendon allograft reconstruction. Published series report restoration of extensor mechanism function with improvements in quadriceps strength, knee range of motion, and reduction in extensor lag.[19]

CONCLUSION

This case highlights the rare occurrence of simultaneous ruptures of the quadriceps, patellar, and triceps tendons in a bodybuilder following a low-energy mechanism. Early clinical suspicion supported by multimodal imaging ensured a timely diagnosis. The patient underwent single-stage surgical repair and careful rehabilitation to regain functional independence at one-year follow-up.

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