

Spontaneous Quadriceps Femoris Tendon Rupture in a Patient with Chronic Kidney Disease and Secondary Hyperparathyroidism

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ABSTRACT

Background: Spontaneous tendon rupture is a recognised but uncommon complication of chronic kidney disease (CKD) and secondary hyperparathyroidism (SHPT). The vast majority of reported cases involve patients already receiving haemodialysis; reports in predialysis patients with advanced CKD remain exceedingly rare.

Case Presentation: A 57-year-old Japanese man presented to the Emergency Department with sudden-onset right lower-limb weakness. He had a background of advanced CKD (serum creatinine 8.97 mg/dL; estimated glomerular filtration rate [eGFR] 5.51 mL/min/1.73 m²) secondary to diabetic nephropathy, and had not yet commenced renal replacement therapy. Clinical examination demonstrated anterior distal-thigh swelling and a suprapatellar sulcus consistent with quadriceps tendon disruption. Preoperative magnetic resonance imaging (MRI) confirmed complete rupture of the right quadriceps tendon at the osteotendinous junction.

Results: Laboratory investigations revealed markedly elevated serum intact parathyroid hormone (PTH) at 675.5 pg/mL (reference range 10.3–65.9 pg/mL), hypocalcaemia (corrected calcium 8.2 mg/dL; reference 8.8–10.1 mg/dL), and hyperphosphataemia (5.3 mg/dL; reference 2.7–4.6 mg/dL). Ultrasonography identified enlargement of all four parathyroid glands. Computed tomography (CT) demonstrated extensive vascular calcification and a characteristic “rugger-jersey spine.” Surgical repair was performed and postoperative MRI confirmed satisfactory tendon continuity prior to discharge.

Conclusion: This case highlights the importance of considering SHPT-related tendinopathy in predialysis patients with advanced CKD presenting with unexplained tendon rupture. Proactive management of hyperparathyroidism in this population is essential to prevent spontaneous quadriceps tendon rupture (QTR) and its recurrence.

Keywords: chronic kidney disease; secondary hyperparathyroidism; quadriceps tendon rupture; predialysis; parathyroid hormone; tendinopathy; MRI; renal osteodystrophy

INTRODUCTION

Spontaneous quadriceps tendon rupture (QTR) is a well-recognised but uncommon musculoskeletal complication in patients with end-stage kidney disease receiving haemodialysis. Its pathogenesis is closely linked to the metabolic and hormonal disturbances accompanying chronic kidney disease (CKD), particularly secondary hyperparathyroidism (SHPT). Chronically elevated parathyroid hormone (PTH) is thought to promote osteoclast-mediated resorption at subtendinous bony insertion sites, predisposing the osteotendinous junction to weakening, dystrophic calcification, and ultimately spontaneous rupture.

Despite the well-established association between haemodialysis and tendon rupture, reports of spontaneous QTR in predialysis patients with advanced CKD and SHPT remain exceedingly scarce. The diagnostic challenge is compounded by the rarity of this presentation and the potential to overlook SHPT as the underlying aetiology. We report a case of spontaneous QTR in a predialysis patient with advanced CKD and severe SHPT, and discuss the pathophysiological mechanisms, diagnostic approach, and management implications.

CASE PRESENTATION

A 57-year-old Japanese man was transported to the Emergency Department by ambulance following sudden onset of right lower-limb weakness. He reported no antecedent trauma. His medical history was significant for advanced renal insufficiency (serum creatinine 8.97 mg/dL; estimated glomerular filtration rate [eGFR] 5.51 mL/min/1.73 m²) attributable to diabetic nephropathy; he had not yet commenced renal replacement therapy.

Physical examination of the lower limbs revealed anterior swelling over the distal right thigh and a palpable suprapatellar defect at the insertion of the quadriceps tendon, consistent with complete tendon disruption. After admission, magnetic resonance imaging (MRI) was performed.

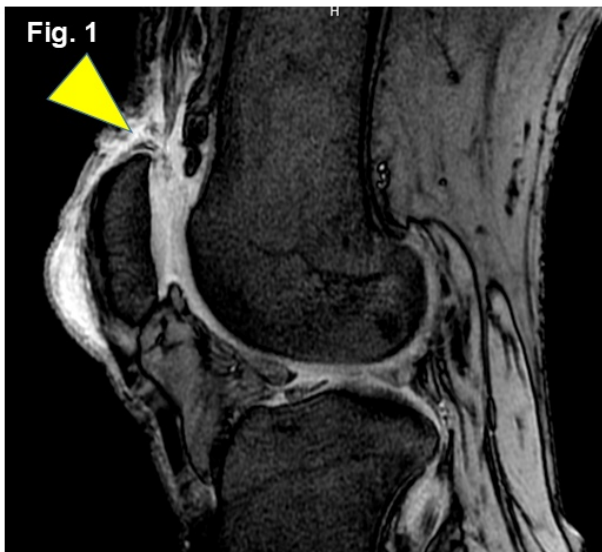


Figure 1. Preoperative T2-weighted sagittal MRI of the right knee demonstrating complete rupture of the quadriceps tendon at the osteotendinous junction (arrowhead).

Preoperative MRI confirmed complete rupture of the right quadriceps tendon at the osteotendinous junction (Figure 1). No intra-articular effusion or additional structural pathology was identified.

INVESTIGATIONS

Detailed laboratory evaluation revealed severe SHPT: serum intact PTH was markedly elevated at 675.5 pg/mL (reference range 10.3–65.9 pg/mL), serum corrected calcium was reduced at 8.2 mg/dL (reference 8.8–10.1 mg/dL), and serum phosphorus was elevated at 5.3 mg/dL (reference 2.7–4.6 mg/dL). These biochemical findings are summarised in Table 1.

Table 1. Summary of relevant biochemical investigations at presentation.

Parameter	Patient Value	Reference Range
Serum creatinine	8.97 mg/dL	0.6–1.2 mg/dL
eGFR	5.51 mL/min/1.73 m ²	≥60 mL/min/1.73 m ²
Intact PTH	675.5 pg/mL	10.3–65.9 pg/mL
Corrected calcium	8.2 mg/dL	8.8–10.1 mg/dL
Serum phosphorus	5.3 mg/dL	2.7–4.6 mg/dL

Ultrasonography demonstrated enlargement of all four parathyroid glands, consistent with diffuse parathyroid hyperplasia. Plain computed tomography (CT) identified extensive vascular calcification involving the coronary arteries and the thoracic and abdominal aorta. Spinal imaging revealed the characteristic “rugger-jersey spine” appearance — a finding pathognomonic of renal osteodystrophy attributable to alternating bands of osteosclerosis and osteoporosis on vertebral endplates.

A thorough medication review confirmed that the patient had received no fluoroquinolone antibiotics within the preceding 6–12 months and had not been exposed to systemic corticosteroids prior to the rupture. Current medications at the time of presentation included antihypertensive agents (amlodipine, losartan), a dipeptidyl peptidase-4 inhibitor for glycaemic management (linagliptin), a phosphate binder (lanthanum carbonate), and calcium carbonate supplementation.

MANAGEMENT AND OUTCOME

Surgical repair of the ruptured right quadriceps tendon was performed. Intraoperative assessment confirmed complete disruption between the quadriceps tendon and the patella at the osteotendinous junction (Figure 2). Primary end-to-end repair was undertaken with reinforcement sutures. The postoperative course was uneventful. Postoperative MRI performed prior to discharge demonstrated satisfactory continuity of the repaired tendon with no evidence of re-rupture or significant peritendinous complications.

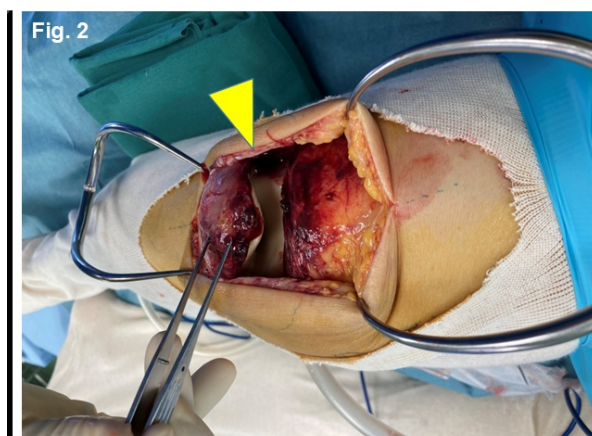


Figure 2.

Intraoperative photograph demonstrating complete disruption of the quadriceps tendon at the osteotendinous junction. The arrowhead indicates the superior proximal edge of the disrupted tendon; the exposed bony patella is visible inferiorly.

Concurrent management of SHPT was initiated in collaboration with the nephrology team. Optimisation of phosphate binders, introduction of activated vitamin D supplementation, and close monitoring of mineral metabolism parameters were undertaken. The patient was counselled regarding the importance of adherence to medical therapy and the risk of recurrent tendon injury in the setting of ongoing SHPT.

DISCUSSION

This case illustrates a rare but clinically significant presentation of spontaneous QTR in a predialysis patient with advanced CKD and severe SHPT. Although QTR has been previously documented in patients receiving haemodialysis, reports in those with advanced CKD who have not yet commenced renal replacement therapy are exceedingly uncommon, underscoring the importance of recognising this diagnosis outside the dialysis context.

The pathophysiological basis for tendon rupture in the setting of CKD-related SHPT is multifactorial. Advanced CKD produces a state of hypocalcaemia and phosphorus retention, which chronically stimulates PTH secretion from hyperplastic parathyroid glands. Persistently elevated PTH drives osteoclast activation at subtendinous bony insertion sites — particularly the osteotendinous junction — resulting in progressive resorption, mechanical weakening, and dystrophic calcification of peri-insertional tissue. Superimposed repetitive microtrauma at these structurally compromised insertion sites can culminate in complete tendon rupture, often with minimal or no precipitating trauma, as observed in the present case.

The biochemical profile in this patient — PTH 675.5 pg/mL, hypocalcaemia, and hyperphosphataemia — in conjunction with ultrasonic evidence of four-gland parathyroid hyperplasia and CT-demonstrated vascular calcification, represents a severe and longstanding SHPT phenotype. The “rugger-jersey spine” is a recognised radiological hallmark of advanced renal osteodystrophy, further attesting to the chronicity of the metabolic bone disease in this patient.

From a diagnostic standpoint, MRI is the preferred imaging modality for the characterisation of tendon rupture, offering superior soft-tissue contrast and precise delineation of the extent

and level of disruption compared with ultrasound or plain radiography. While the cost and availability of MRI may be limiting in resource-constrained settings, it provided critical preoperative information in this case and facilitated objective postoperative assessment of repair integrity.

Importantly, drug-related causes of tendinopathy were systematically excluded. Fluoroquinolone antibiotics and systemic corticosteroids are well-documented risk factors for tendon rupture; the absence of both in this patient’s medication history supports SHPT as the primary pathogenic mechanism. This exclusion is clinically meaningful, as attribution to a modifiable pharmacological cause could otherwise divert attention from the underlying metabolic disorder.

The present case emphasises that SHPT-related QTR is not confined to patients receiving dialysis. In any patient with advanced CKD presenting with unexplained tendon rupture or tendinitis, secondary — or tertiary — hyperparathyroidism should be actively considered in the differential diagnosis. Timely diagnosis enables appropriate surgical intervention and facilitates targeted medical management of the underlying mineral metabolism disorder to reduce the risk of recurrence.

CONCLUSION

Spontaneous quadriceps tendon rupture is an uncommon but potentially debilitating complication of advanced CKD and SHPT, and may occur in predialysis patients. Clinicians should maintain a high index of suspicion for this diagnosis in patients with advanced CKD presenting with lower-limb weakness or acute knee pathology. MRI is the investigation of choice for confirming the diagnosis and guiding surgical planning. Proactive and systematic treatment of SHPT is essential to prevent initial and recurrent tendon rupture in this high-risk population.

DECLARATIONS

Author Contributions

All authors contributed to the clinical care of the patient. T.M. performed the surgical procedure and was the primary author of the manuscript. K.F. provided nephrology consultation and critical revision of the manuscript. Y.T. and N.M. performed and interpreted the imaging investigations. T.H. contributed to perioperative management and manuscript review. All authors read and approved the final version of the manuscript.

Conflict of Interest

All authors declare no competing interests relevant to this publication.

Funding

No funding was received for the conduct of this study or the preparation of this manuscript.

Patient Consent

Written informed consent for publication of this case report and accompanying images was obtained from the patient’s legal surrogate. No patient-identifying information is included in this manuscript.

Acknowledgements

The authors thank Analisa Avila, MPH, ELS, of Edanz (<https://jp.edanz.com/ac>) for professional English language editing of a draft of this manuscript.

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