Traumatic Triple Tendon Rupture in Secondary Hyperparathyroidism

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Simultaneous multiple tendon ruptures have been reported previously in chronically ill patients. This article presents a case of multiple tendon rupture in a patient with chronic renal failure and hyperparathyroidism.

CASE REPORT

A 52-year-old man with chronic renal failure and secondary hyperparathyroidism presented with bilateral knee and left elbow pain after a fall. Chest pain, shortness of breath, and syncope were not reported. Medical history was significant for myocardial infarction, hypertension, asthma, and end-stage renal disease secondary to glomerulonephritis. He required hemodialysis for 16 years. Prior to his fall, he ambulated with a walker.

Initial evaluation revealed contusions and swelling of both knees and the left elbow, but no open wounds. The patient could not actively extend the left elbow or either knee. Tender, palpable defects were present inferior to both patellae and proximal to the left olecranon. Pedal pulses were present, and no evidence of neurologic deficit was noted.

Radiographs showed bilateral patella alta and an effusion of the left elbow with a small avulsion fragment off the olecranon (Figure 1). Following preoperative medical evaluation, including cardiac catheterization to assess coronary disease, the patient underwent tendon repair surgery.

For each patellar tendon repair, a longitudinal incision was made from the superior aspect of the patella to the tibial tuberosity. The epitendon was incised, the tendon exposed. Two Bunnell stitches were placed in each tendon with #5 Ethibond suture, and the sutures were passed through longitudinal drill holes in the patella. The retinaculum and epitendon were repaired and the skin closed.

For the triceps repair, a longitudinal incision was made over the olecranon, extending 5 cm proximally. The tendon was trimmed to a healthy edge. A rongeur was used to create a small trough of bleeding cancellous bone in the posterior aspect of the olecranon. Two drill holes were made in the trough, exiting on the dorsal surface of the ulna. A single Bunnell stitch of #5 Tycron was weaved through the triceps tendon and the ends were passed through the two drill holes and tied over the ulna.

Both lower extremities were placed in knee immobilizers in full extension, and the left upper extremity was held in extension in a custom-made splint. A rolling walker was used for weight bearing as tolerated with the knee immobilizers in place.

Six weeks postoperatively, radiographs revealed slight patella alta bilaterally. Slight displacement of a tiny avulsed bone fragment was noted on elbow radiograph. Both lower extremities remained immobilized in extension until 4 months postoperatively, at which time physical therapy began with emphasis on quadriceps strengthening. The left elbow was immobilized in extension for 10 weeks. Active motion was then started.

Evaluation 6 months postoperatively showed all incisions to be well healed. Both knees revealed passive extension to −2°, but the right and left knees lacked 40° and 30° of active extension, respectively (Figure 2). Both knees had active flexion to 100°. The left elbow exhibited active range of motion from 0°-135°. Knee radiographs revealed patella

Chronic renal failure is one common cause of hyperparathyroidism. Decreased glomerular filtration rate and failure of the renal tubules to excrete phosphorus causes hyperphosphotemia, which impairs the enzyme responsible for the synthesis of 1, 25 (OH)₂ vitamin D. Decreased levels of effective vitamin D reduce intestinal absorption of calcium, resulting in low serum calcium levels, leading to hyperparathyroidism.

Several potential effects of elevated parathyroid hormone on tendon function and integrity have been proposed. Increased osteoclastic activity caused by elevated parathyroid hormone increases bony resorption at the bony-tendon junction, predisposing to spontaneous tendon rupture at the insertion point. Preston and Adicoff proposed that in the hyperparathyroid state, dystrophic calcification and calcium deposits weaken tendons, predisposing them to rupture. Babini et al isolated calcium hydroxyapatite and urate crystals from ruptured tendons in patients with secondary hyperparathyroidism. Others have speculated that hyperparathyroidism results in elastin deposition in tendons, and dialysis-related amyloid deposition weakens tendons.

In addition to compromising tendon integrity, hyperparathyroidism alters the healing and functional results of tendon ruptures. Several authors have shown that the chronically ill patient who sustains multitudinous disruption is unlikely to have an excellent result. Preston and Adicoff noted that after prolonged physical therapy their patient required “slight support” to walk, climb stairs, and perform knee-bending exercises. Clark et al reported that within 2 months postoperatively the patient was ambulatory, but required a walker and a physical therapist. Wener and Schein noted that their patient required long braces for 6 months to walk. Our patient, despite cautious activity and motion restrictions, healed with bilateral extension lags and requires a walker to ambulate.

This case re-emphasizes the debilitating nature of simultaneous multiple tendon ruptures in the chronically ill population and emphasizes the need to identify and protect at-risk patients. Even with proper postoperative protection, these patients may have impaired ability to heal the tendon repairs.

### REFERENCES