Spontaneous Rupture of the Patellar and Contralateral Quadriceps Tendons Associated with Secondary Hyperparathyroidism in a Patient Receiving Long-term Dialysis

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Although spontaneous rupture of the extensor tendon of the knee is more likely to occur in uremic patients with secondary hyperparathyroidism, simultaneous ruptures of bilateral knee extensor tendons is a rarely reported condition. We describe a 30-year-old man with uremia who underwent subtotal parathyroidectomy because of secondary hyperparathyroidism with very high serum parathyroid hormone (PTH) level (1940.4 pg/mL). Two weeks later, he complained of a sharp pain in both knees without trauma when he walked downstairs with his left knee forward and right knee behind. Spontaneous simultaneous ruptures of the right patellar tendon and the left quadriceps tendon were diagnosed and surgically repaired. The mechanism of spontaneous tendon rupture in uremic patients with secondary hyperparathyroidism seems to be related to high PTH level which results in osteolytic bone resorption at the tendon insertion site. Early surgical repair, control of secondary hyperparathyroidism, early use of vitamin D analogs, and total parathyroidectomy with or without autotransplantation of part of the parathyroid gland, can treat and prevent tendon rupture or re-rupture with satisfactory results. [J Formos Med Assoc 2006;105(11):941–945]

Key Words: hyperparathyroidism, parathyroid hormone, spontaneous rupture, uremia

Spontaneous tendon rupture is relatively uncommon but has been reported in uremic patients associated with hyperparathyroidism.¹⁻³ Simultaneous ruptures of the patellar and contralateral quadriceps tendons is a comparatively rare condition.⁴⁻⁵ The standard treatment for complete tendon rupture is surgical repair.⁵⁻⁷

We describe a patient receiving long-term hemodialysis in association with secondary hyperparathyroidism who was admitted because of spontaneous ruptures of his right patellar and contralateral quadriceps tendons. The ruptures were successfully treated with tendon repair and immobilization, and control of secondary hyperparathyroidism. The patient’s intact parathyroid hormone (iPTH) levels before and after tendon rupture were compared and the surgical findings in the tendons were assessed to determine why the spontaneous ruptures occurred.

Case Report

A 30-year-old man with uremia who had been receiving maintenance hemodialysis for 9 years was admitted to our hospital because of sudden...
onset of painful disability in both knees without traumatic history. The patient was 175 cm tall and weighed 80 kg. He had undergone total parathyroidectomy with autotransplantation because of secondary hyperparathyroidism 2 weeks before this admission. His serum iPTH level was 1940.4 pg/mL before parathyroidectomy (Table).

The patient visited our emergency department because of sharp pain in both knees when he walked downstairs and his left knee was in the front position. Physical examination revealed two large gaps, one below the right patella and one above the left patella (Figures 1A and 1B). Loss of the active extensor mechanisms of both knees was also noted. Radiographs and magnetic resonance

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<th>Table. Serum levels of parameters associated with tendon rupture in uremic patient with secondary hyperparathyroidism</th>
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<tr>
<td>Before rupture</td>
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<tr>
<td>2 yr 2 mo</td>
</tr>
<tr>
<td>iPTH*</td>
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<tr>
<td>Calcium†</td>
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<td>Phosphate‡</td>
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<td>Alkaline phosphatase§</td>
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*Normal range, 13.0–53.5 pg/mL; †normal range, 8.4–10.2 mg/dL; ‡normal range, 2.7–4.5 mg/dL; §normal range, 40–129 U/L.

iPTH = intact parathyroid hormone; NA = not available.

Figure 1. Large gaps: (A) below the right patella; (B) above the left patella. Sagittal T2-weighted magnetic resonance imaging of the (C) right knee and (D) left knee shows rupture at the proximal patellar tendo-osseous junction (arrow) and complete disruption at the distal quadriceps tendo-osseous junction (arrow), respectively. The interruption of cortical continuity with irregular and rough bone surface were also seen (asterisks).
imaging (MRI) revealed downward displacement of the left patella and upward displacement of the right patella. Sagittal T2-weighted MRI of the right knee showed a ruptured patellar tendon and also waviness and distal retraction of the tendon with avulsion of a bony fragment of patella (Figure 1C). Similarly, sagittal T2-weighted MRI of the left knee showed complete disruption of the quadriceps tendon with interrupted continuity of bony cortex (Figure 1D). Both MRIs depicted the hemorrhage and edematous swelling in the disrupted gaps and in the tissues surrounding the tendons with irregular bony surface over the cortex of the patella. Simultaneous rupture of the left quadriceps tendon and the right patellar tendon was impressed. Skull film also revealed the significant “salt-and-pepper” appearance.

Surgical repair was performed using tendo-osseous sutures and nonabsorbable, silicone-coated, braided polyester sutures (number 5 Ti-Cron). These were passed through multiple longitudinal holes 2.0 mm in diameter that were made in the bilateral patellae using a drill. Augmentation circumferential sutures (number 5 Ti-Cron) were then passed through transverse drill holes made in the center of both patellae and a right tibial entry point 1 cm posterior to the tibial tuberosity of the right knee (Figures 2A and 2B). During the operation, thin layers of cancellous bone attached to the ruptured ends of the patellar and quadriceps tendons were observed.

After surgery, we noted that the patient’s iPTH level had decreased from the level before parathyroidectomy to 409 pg/mL when the tendon ruptures occurred (Table), but not to the target level of iPTH after treatment, 150–300 pg/mL, suggested by the K/DOQI guidelines. Cylinder splints were positioned on both knees in full extension to provide immobilization for 10 days. Cylinder casts were then positioned on both knees in full extension to immobilize the right leg for a total of 6 weeks and the left leg for 8 weeks. After the casts were removed, progressively passive rehabilitation and then active rehabilitation of both knees was given. In addition, the patient’s uremic condition was regularly treated by our nephrologist.

Three months after surgical repair, range of motion of the knees increased to 0°/110° on the left and 0°/130° on the right. Two years after surgery, range of motion was 0°/140° in the right knee and 0°/130° in the left knee. Strength of the bilateral quadriceps muscles was 5/5. The patient’s knees were asymptomatic, and he was able to return to his full daily activities (Figures 2C and 2D). Serum iPTH had decreased to 12.9 pg/mL (normal range, 13.0–53.5 pg/mL) 2 years and 8 months after the operation (Table).

![Figure 2](image_url)

**Figure 2.** Surgical repair: (A) right knee; (B) left knee. Tendo-osseous and nonabsorbable sutures and Ti-Cron augmentation sutures, respectively, were passed through longitudinal and transverse drill holes in the patella. Lateral radiographs 2 years after repair show normal positions of the (C) right and (D) left patellae.
Surgical repair is the standard treatment of complete rupture of the knee extensor tendon. For this, many methods have been reported. In this patient, we used multiple teno-osseous and circumferential, nonabsorbable, augmentation sutures passed through transverse drill holes in the central patellae and tibial tuberosity. This is a newly designed technique to prevent late failure due to hardware fatigue and to avoid the need to remove hardware later. In patients with uremia and secondary hyperparathyroidism, aggressive treatment to control their hyperparathyroid state is needed to prevent further spontaneous tendon ruptures. The strict control of hyperphosphatemia, the early use of vitamin D analogs and, in recalcitrant cases, total parathyroidectomy with or without autotransplantation of part of the parathyroid gland are warranted to prevent these ruptures.

In conclusion, in cases of suspected partial or complete tear of the knee extensor tendon, detailed physical examination and proper imaging, such as radiology and MRI, can establish the diagnosis. The standard treatment of surgical repair with or without augmentation tendon–bone
sutures with external immobilization can achieve satisfactory results. Our new method of using nonabsorbable tendon–bone sutures with circumferential, nonabsorbable augmentation sutures passed through drill holes in the bone can prevent late metal failure and, thus, avoid the prospect of late metal removal. Patients with uremia and secondary hyperparathyroidism are at high risk for tendon rupture; therefore, aggressive control of their hyperparathyroid state is advised to prevent spontaneous tendon rupture or re-rupture.

References