J Korean Radiol Soc 2001;45:507 - 511

Bilateral Simultaneous Quadriceps Tendon Rupture in a Patient with Secondary Hyperparathyroidism: A Case Report[1]

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Simultaneous bilateral rupture of the quadriceps tendon without a significant history of trauma may occur in association with chronic metabolic disorders such as chronic renal failure and secondary hyperparathyroidism, though has rarely been reported. We describe a case of spontaneous bilateral quadriceps tendon rupture in a 36-year-old female patient with secondary hyperparathyroidism.

Index words : Knee, MR
Tendons, injuries
Tendons, MR

Simultaneous bilateral rupture of the quadriceps tendon is very rare, usually occurring in the elderly or in patients suffering from a chronic illness such as gout, collagen vascular disease, diabetes mellitus, hyperparathyroidism, or chronic renal failure (1, 2). Quadriceps tendon rupture is diagnosed clinically, but where there is no history of trauma or a hematoma present at physical examination masks the defect, spontaneous rupture may be difficult to diagnose. Bilateral rupture is even more difficult to detect, since its appearance at physical examination may be symmetric. Magnetic resonance imaging (MRI) is the most accurate imaging modality for assessing tendon rupture and for preoperative planning, though its findings have not been reported in the Korean medical literature. We report a case of simultaneous bilateral quadriceps tendon rupture in a patient with chronic renal failure and secondary hyperparathyroidism, and describe the radiologic findings, including those of plain film and MRI.

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Received February 12, 2001; Accepted August 20, 2001
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Case Report

A 36-year-old woman presented with bilateral knee pain and swelling immediately after falling over on a sidewalk. She was unable to ambulate without assistance. According to her past medical history, she had experienced chronic renal failure secondary to chronic glomerulonephritis caused by IgA nephropathy diagnosed 12 years ago, and had been on maintenance hemodialysis for five years. Physical examination revealed bilateral, painful knee distension. After needle aspiration of approximately 60 cc of bloody fluid from both knee joints, physical examination revealed an indistinct palpable defect at the suprapatellar region of the knees. The abnormal laboratory findings were as follows: serum creatinine, 10.5 mg/dL; serum alkaline phosphatase, 2992 IU/L; serum calcium, 8.3 mg/dL; BUN, 55.8 mg/dL; total protein, 5.6 g/dL; albumin, 3.3 g/dL; hemoglobin, 8.2 g/dL; hematocrit, 25.9%. Electromyographic studies showed no electrodiagnostic findings of peripheral neuropathy or lower motor neuron denervation. A radiographic survey of the entire skeleton demonstrated characteristic findings of secondary hyperparathyrodism including the salt-and -pepper ap-

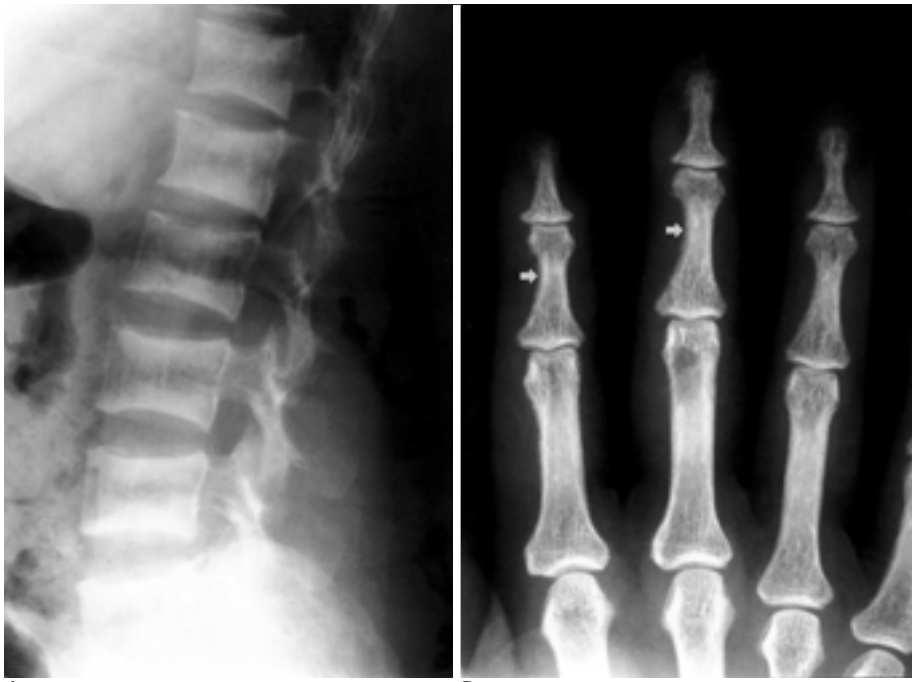
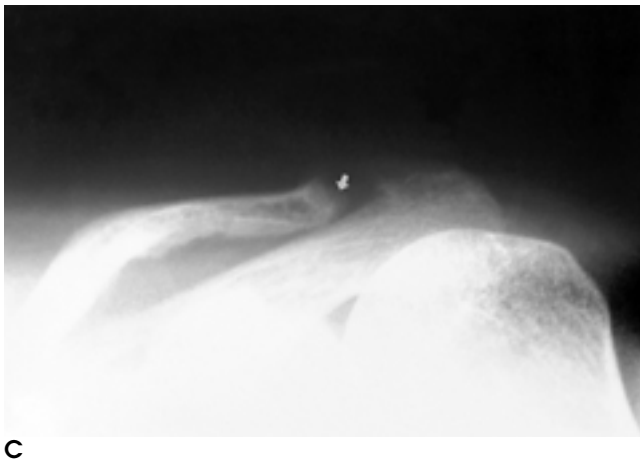


Fig. 1. Plain radiographic findings representing secondary hyperparathyroidism
A. Bandlike sclerosis in the superior and inferior margins of the vertebral bodies is seen.
B. Subperiosteal resorptions (arrows) including brown tumor (arrowhead) are seen in the phalanges.
C. Subchondral resoprtion (arrow) is noted in the distal clavicle.



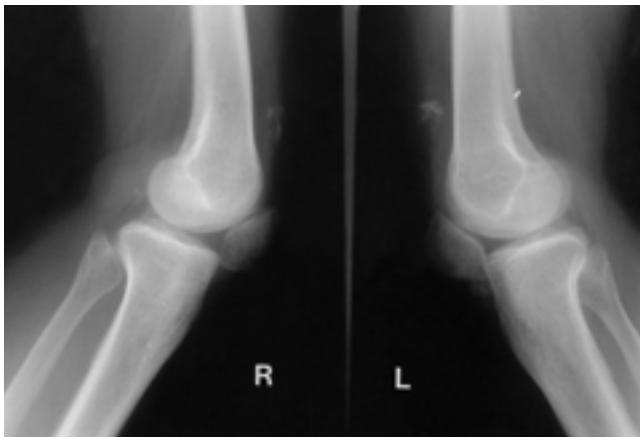


Fig. 2. Lateral radiographs of the both knees demonstrate low lying patellae with irregular shaped calcifications in the suprapatellar soft tissues. The soft tissue planes demarcating the distal quadriceps tendons are indistinct.

pearance of the skull, a rugger-jersey spine, and subchondral bone resorption of the hand and acromioclavicular joints (Fig. 1). Plain radiographs of both knees revealed low-lying patellae bilaterally, and irregularly-shaped calcified densities in the suprapatellar soft tissue (Fig. 2). To further evaluate these findings, MR T1- and T2- weighted imaging of both knees was performed. Sagittal T1- and T2- weighted MR images of the knee joints revealed complete detachment of the quadriceps tendon from the superior pole of the patella and proximal retraction of the ruptured tendon. Hypointense T1 and hyperintense T2 signal intensities were noted between the patella and detached tendon in both knees, where multiple, focal, bony fragments of high T1 and intermediate T2 signal intensity were present in the retracted quadriceps tendons (Fig. 3). Surgical repair was undertaken and the MRI findings were confirmed. Both quadriceps tendons were completely disrupted just proximal to the superior pole of the patella and old hemorrhage with scarred fibrous tissues was found between the ruptured tendon and patella. In both knees, some ossified fragments and focal subperiosteal erosion of the patella were also noted.

Discussion

A normal quadriceps tendon is one of the strongest tendons in the body, being able to withstand a load of 15 to 30 kg/mm (3). In patients with underlying disease,

spontaneous bilateral rupture of the quadriceps tendon may occur during ordinary daily activities such as walking or even stepping from a car (3). The microscopic findings include fatty or myxoid degeneration, calcification within the tendon, cystic softening, and decreased collagen with marked loss of nuclei (3, 4). A similar pathology is seen in old age, when obesity, diabetes, atherosclerosis, chronic renal failure and inflammatory reactions such as those accompaning gout, tuberculosis, syphilis, and acute bacterial infections may occur (3).

The pathogenesis of bilateral quadriceps tendon rupture in chronic renal failure is uncelar, and more than one factor may be involved. Changes in collagen and the ground substance in patients with renal failure include ischemia and dystrophic calcification. Systemic acidosis, as well as the direct effects of parathyroid hormone and subperiosteal bone resorption, with subsequent weakening of the tendon-bone interface, have been implicat-

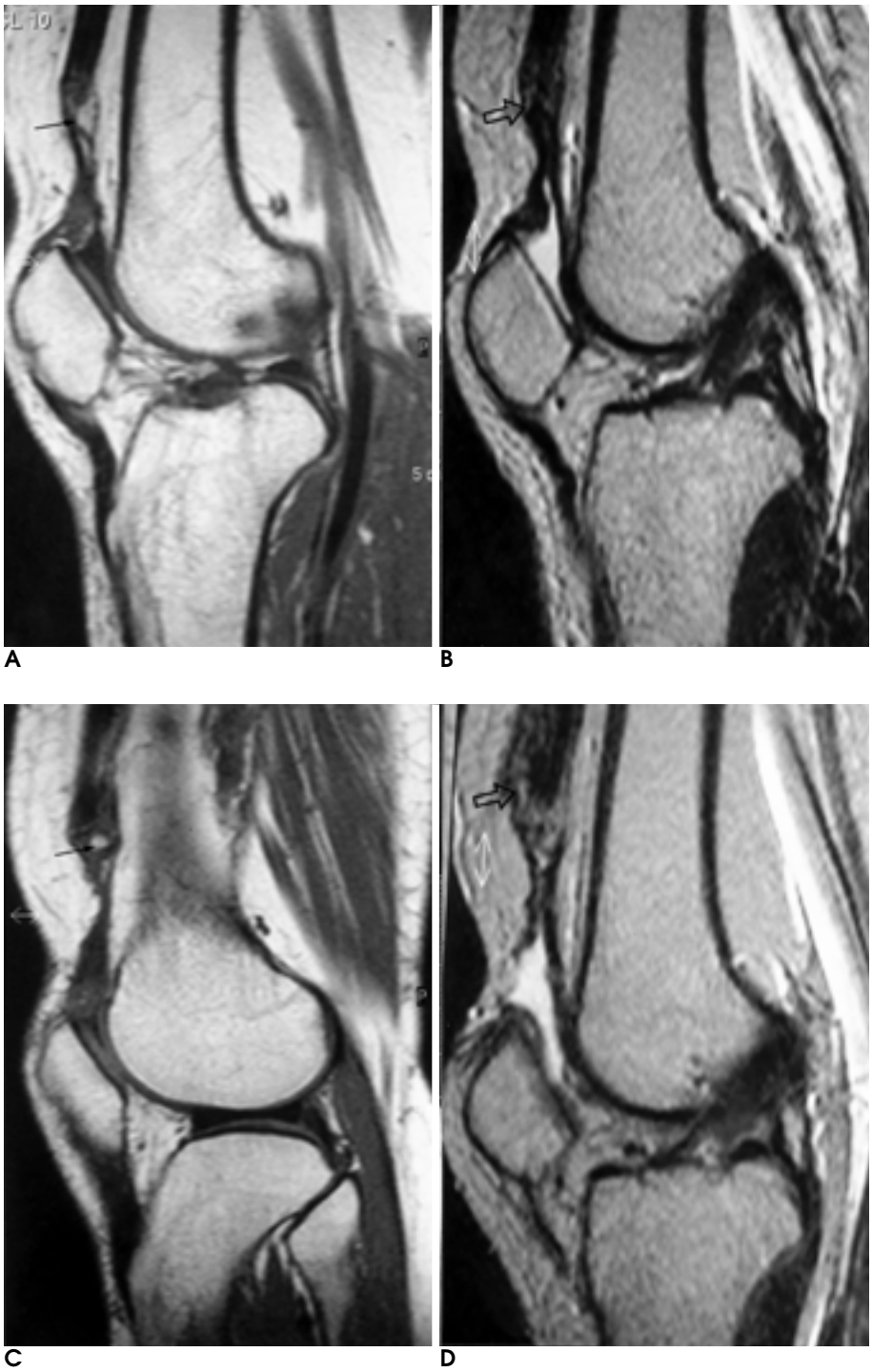


Fig. 3. Sagittal T1- and T2-weighted MR images of the right (**A, B**) and left (**C, D**) knees reveal disruption of the quadriceps tendons from the superior pole of the patella and proximal retraction of the torn quadriceps tendon (open arrows) including focal ossified fragments (arrows). Suprapatellar fibrous tissue and hemorrhage are also noted.

ed in the pathogenesis (3, 5, 6). It has been suggested that a chronic acidotic state leads to elastin deposition in the tendons, predisposing them to spontaneous rupture. A reduced turnover of collagen, with replacement by elastic tissue, has been postulated as a cause of weakening of the tendon (5, 7). In contrast to the ruptures seen in elderly or obese patients with fatty degeneration, tendon ruptures in dialysis patients tend to occur at a younger age, usually under 40 years (5).

Renal failure as an underlying cause of bilateral quadriceps tendon rupture is directly related to the duration of renal failure and the period of dialysis (3, 5). In this 36-year-old-female, chronic renal insufficiency and secondary hyperparathyroidism were precursors and causative conditions for the attenuation that precipitated spontaneous rupture. The osteotendinous junction is thought to be weakened by bone resorption secondary to hyperparathyroidism, and it may be for this reason that it is the most frequently involved site (3). Primary or secondary hyperparathyroidism has contributed to quadriceps tendon rupture by causing dystrophic calcification and subperiosteal bone resorption, which weakens the osteotendinous junction. The result of this may be repeated minor avulsion fracture of the bone cortex, ultimately leading to scarring and weakening of the tendon attachment site, and total rupture (7, 8). Thus, the most important causes of tendon rupture appear to be primary tendon disease and bone erosion at the site of tendon insertion. In our case, avulsion fractures of the patellar cortex were not present, but there were focal subperiosteal erosions in both patellae and ossified fragments with combined dystrophic calcification in the retracted quadriceps tendons.

To prevent residual deformity and functional loss, early diagnosis and repair are necessary. The clinical findings are not always clear, and after hematoma formation, the tendon defect may not be readily palpable (9). Plain radiography is useful for demonstrating indirect signs of tendon rupture, such as poorly defined suprapatellar swelling and forward tilting of the patella. Small fragments of avulsed bone or dystrophic calcification may be observed in the suprapatellar region. The patella is often, but not always, low lying (1). Sonography can demonstrate effusion and a ruptured tendon, though tendon separation and proximal retraction are most clearly defined by MRI. In summary, a case of simultaneous, spontaneous, bilateral rupture of the quadriceps tendon in secondary hyperparathyroidism is presented. Although the diagnosis is clinical, MRI can accurately determine the site and extent of tendon rupture, findings which can be very helpful for confirming the clinical diagnosis and for preoperative planning.

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J Korean Radiol Soc 2001;45:507 - 511

대한방사선의학회지 2001;45:507 - 511

이차 부갑상선기능항진증 환자에서 대퇴사두건 양측 동시 파열: 1예 보고[1]

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특별한 외상의 병력없이 양측 대퇴사두건이 동시 파열되는 경우는 매우 드물며, 만성 신장질환이나 이차 부갑상선기능항진증과 같은 만성 대사질환에서 발생할 수 있다. 저자들은 만성 신부전에 의한 이차 부갑상선기능항진증 환자에서 대퇴사두건이 양측 동시 파열된 1예를 경험하였기에 자기공명영상과 단순 촬영 소견을 문헌고찰과 함께 보고하고자 한다.