

A case of bilateral Achilles tendon spontaneous rupture in a hemodialysis patient: Who is the real culprit? Acidosis? Or hyperparathyroidism?

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ABSTRACT

Patients who are on regular hemodialysis and who have advanced renal failure but have not yet started on renal replacement therapy have a high risk of tendon rupture, although this is a rare situation. We reported a case of 55-year-old male patient who had been on regular hemodialysis for the past 10 years. He presented with a sudden onset of pain and swelling in the left posterior ankle while climbing stairs. Ultrasonography revealed a complete disruption of the Achilles tendon, which was surgically repaired. His medical history was remarkable for the right Achilles tendon rupture a year ago. Investigations revealed tertiary hyperparathyroidism with chronic metabolic acidosis and high β -2 macroglobulin. With surgical repair, physiotherapy, and parathyroidectomy, the patient regained full active mobility. This case emphasized the importance of high parathyroid hormone level, metabolic acidosis, and high β -2 macroglobulin in the pathogenesis of tendons ruptures.

Key words: Achilles tendon, Hemodialysis, High β -2 macroglobulin, Hyperparathyroidism, Rupture

The Achilles tendon is the largest and most resistant tendon of the human body. The spontaneous Achilles tendon rupture (ATR) is a rare but well-known complication of systemic diseases, such as systemic lupus erythematosus, inflammatory reaction, diabetic arteriosclerosis, and chronic renal failure due to an accelerated degeneration of collagen. ATR may occur on one or both sides. The bilateral tendon rupture is rare, accounting for less than 1% of all ATRs [1]. An association between the kidney disease and spontaneous ATR has been documented in many case reports [2-8]. The spontaneous ATR in hemodialysis patients is a rare complication. The majority of these patients have additional predisposing factors, such as secondary hyperparathyroidism (HPTH), diabetes mellitus, obesity, rheumatoid arthritis, gout, statins, steroids, fluoroquinolones, and the presence of B and/or C hepatitis virus [9].

In chronic kidney disease patients, the most common cause of tendon rupture is secondary HPTH, which is present in most of these patients. Other important factors are chronic metabolic acidosis and high levels of β 2-microglobulin in dialysis patients, as indicated in several recent reports. We present here a case of spontaneous ATR in a patient undergoing long-term hemodialysis with multiple risk factors predisposing him to this condition. The case report aims to raise awareness among physicians about this unusual complication in patients on hemodialysis.

CASE REPORT

A 55-year-old male patient, a case of end-stage renal disease, had been undergoing hemodialysis, secondary to hypertensive nephropathy, 3 times a week since 2007. He was admitted to the emergency department as he presented with a sudden onset of the left heel pain while he was climbing the stairs. His medical history was remarkable for hepatitis B and C for which he received treatment. Furthermore, he had a history of partial rupture of the right Achilles tendon, and it was treated using a right leg cast. In the last year, he developed tertiary HPTH which was evidenced by the excessive secretion of parathyroid hormone (PTH) at a level of 1900 pg/ml, resistance to medical treatment, and detection of parathyroid adenoma by the parathyroid scan. There was no history of using corticosteroids or fluoroquinolones.

On examination, a gap was discovered at the site of the Achilles tendon, and there was swelling around the left posterior ankle with tenderness on palpation above the insertion of the tendon. Ultrasonography revealed complete disruption of the Achilles tendon, which was surgically repaired, and a short leg cast was applied. He received follow-up treatment in the orthopedic clinic, and the cast was removed after 4 weeks. With regular physiotherapy sessions, he completely regained his normal ankle function.

Investigations showed that the patient had persistent HPTH despite using calcium supplements, phosphate binders,

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Vitamin D analogs, and calcimimetic agents. Corrected calcium was 2.34 mmol/L. In addition, the patient's arterial blood gas analysis showed persistently low pH (7.2) and the bicarbonate level of 16 mmol/L despite dialysis with high bicarbonate dialysate (40 meq/L). Serum β -2 macroglobulin was 29 mg/L (normally <2 mg/L), and the thyroid function test was within normal limits.

The patient was referred to a surgeon for parathyroidectomy. After the early post-parathyroidectomy period, Vitamin D was given to prevent hypocalcemia, and sodium bicarbonate tablets were given to correct refractory acidosis. At the 6-month follow-up, his PTH and bicarbonate levels returned to normal.

DISCUSSION

In patients with chronic renal failure, the tendon rupture occurred at a younger age as compared to patients with other etiologies [5]. The average age of patients from one series was 52 years [9]. The most frequently affected tendon was the quadriceps tendon followed by the patellar tendon and the Achilles tendon [4]. Furthermore, a more rare case of the rupture of the pectoralis major muscle was reported in a hemodialysis patient [10]. Cases of ATR in healthy patients without identified risk factors have been reported as well [11,12].

The causal relation between chronic dialysis and the spontaneous tendon rupture is not well understood. Bilateral ruptures are very rare and strongly associated with systemic illnesses, such as HPTH, renal failure, rheumatoid arthritis, gout, obesity, systemic lupus erythematosus, and diabetes mellitus. Regarding the pathogenesis of ATR, our case highlights three main issues that deserve special attention.

First, our patient had HPTH, which is in line with many reports in the literature [2-7]. HPTH in renal failure is the result of retention of phosphate due to decrease in glomerular filtration rate and decrease in the active form of Vitamin D with consequent hypocalcemia resulting in parathyroid gland stimulation. Tertiary HPTH is a state of excessive secretion of PTH after a long period of secondary HPTH and dialysis. Restoration of serum calcium and phosphate is not without increased bone resorption due to high PTH. Relatively minor trauma can cause the spontaneous rupture of tendon at tendon-bone junction [13].

Second, our patient showed persistent metabolic acidosis, refractory to hemodialysis, and oral bicarbonate. The role of chronic acidosis in the pathogenesis of ATR is not well understood. It has been suggested that metabolic acidosis may lead to tendon degeneration due to an alteration in the structure of the protein-polysaccharide complex responsible for collagen maturation. While other authors suggested that increased subperiosteal resorption together with connective tissue elastosis due to chronic acidosis may weaken the osteotendinous junction and cause spontaneous tendon rupture [14-16].

Third, the patient's serum β -2 macroglobulin was high. Renal failure with protracted hemodialysis can cause several complications that are linked to the dialytic process, such as dialysis-related amyloidosis (DRA) [17]. In this particular type of

amyloidosis, there is an abnormal production of β -2-microglobulin (β -2-m). In healthy people, β -2-m is metabolized by the kidneys, whereas in uremic patients, especially those who underwent dialytic treatment with insufficiently permeable filters, it accumulates in blood with rates 30–40 times greater than the normal values. In the DRA, the unexcretable plasma β -2-m tends to collect in particular areas, such as joints, bones, and tendon structures.

Diagnosis of ruptured tendon in a hemodialysis patient starts with clinical examination. There is tenderness at the injury site with limited mobility of the affected limb and a tendon gap. Ultrasonography reveals the injury with good sensitivity (96–100%) and specificity (83–100%) [18]. MRI may be helpful when the diagnosis remains unclear, especially in the quadriceps tendon rupture [19]. Early surgical repair and treatment of ruptured tendons with post-operative physiotherapy and controlling of predisposing factors result in better outcomes than delayed treatment [18]. Patients need prolonged physical therapy after surgical treatment. Significant problems with wound healing occur in renal transplant recipients who receive immunosuppressive therapy.

CONCLUSION

Based on the lack of history of the use of corticosteroids or fluoroquinolones, our case highlights the importance of HPTH and acidosis in the pathogenesis of ATR. The spontaneous rupture of the Achilles tendon is rare but should be considered in the case of any individual who undergoes long-term hemodialysis and presents with acute heel pain.

AUTHORS' CONTRIBUTION

Khan S contributed to writing the manuscript and reviewing the literature. Othman M contributed to developing the work idea composing and revising the manuscript. Elesnawy MA contributed to composing and revising the manuscript. All authors read the manuscript and agree to its publication.

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