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Challenging Rehabilitation After Patellar Tendon Rupture in a Patient with Werner Syndrome

Werner Sendromlu Bir Olguda Patellar Tendon Rüptürü Sonrası Zorlu Rehabilitasyon Süreci

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ABSTRACT Werner syndrome (WS) is a rare autosomal recessive disorder characterized by features of premature aging and various musculoskeletal complications, including tendon calcifications and muscle wasting. These changes may predispose patients to spontaneous tendon ruptures. We present the case of a 48-year-old man with WS who experienced bilateral spontaneous patellar tendon ruptures 5 years apart. After surgical repair of the second rupture, rehabilitation was initiated 2 months postoperatively due to limited knee motion. The program was complicated by early synovitis, requiring temporary cessation of exercises and corticosteroid treatment. At the end of physical therapy, the patient's knee flexion improved only to 60-65 degrees, and crutches were still needed at discharge. This case underscores the difficulty of tendon healing and rehabilitation in WS patients, emphasizing the importance of early intervention and tailored rehabilitation protocols to optimize functional outcomes.

Keywords: Werner syndrome; patellar tendon rupture; rehabilitation

ÖZET Werner sendromu (WS), erken yaşlanma belirtileri ve tendon kalsifikasyonları ve kas kaybı gibi çeşitli kas-iskelet sistemi komplikasyonları ile karakterize nadir bir otozomal resesif hastalıktır. Bu değişiklikler hastaları spontane tendon rüptürlerine yatkın hâle getirebilir. Bu yazıda, 5 yıl arayla bilateral spontane patellar tendon rüptürü gelişen 48 yaşındaki bir WS hastası sunulmuştur. İkinci operasyondan 2 ay sonra hareket kısıtlılığı nedeniyle rehabilitasyon programına alındı. Rehabilitasyon süreci, erken sinovit nedeniyle komplike hâle geldi ve egzersizlerin geçici olarak kesilmesini gerektirdi. Fizik tedavinin sonunda diz fleksiyonu yalnızca 60-65 dereceye kadar arttı ve hasta taburcu olurken hala koltuk değneklerini kullanıyordu. Bu vaka, WS hastalarında tendon iyileşmesi ve rehabilitasyonunun zorluğunu vurgulayarak, fonksiyonel sonuçları optimize etmek için erken ve bireyselleştirilmiş tedavi protokollerinin önemini vurgulamaktadır.

Anahtar Kelimeler: Werner sendromu; patellar tendon rüptürü; rehabilitasyon

Werner syndrome (WS) is a rare autosomal recessive disorder characterized by accelerated aging features.[1] The estimated prevalence of WS is approximately 1:100.000 in the Japanese population and ranges between 1:1.000.000 and 1:10.000.000 in populations outside Japan.[2] Patients are typically asymptomatic until adolescence. Common clinical features include graying hair, alopecia, a characteristic "bird-like" facial appearance, scleroderma-like skin changes (including skin atrophy and refractory ulcers), bilateral cataracts, Type 2 diabetes mellitus, and hypogonadism.[2] WS is also associated with various musculoskeletal manifestations; insufficiency fractures, nonunion, tendinitis, soft-tissue tumors, os-

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teoporosis, chronic osteomyelitis, soft-tissue calcifications, muscular atrophy, and foot deformities.[3-5]

Patellar tendon rupture (PTR) is a complete tear of the tendon that connects the inferior pole of the patella to the tibial tubercle. It is often associated with underlying tendinosis or tendon weakening. Surgical repair is necessary for complete ruptures, regardless of the patient's age or activity level.[6] Rehabilitation is essential for restoring joint function and muscle strength, and a good range of motion (ROM) is typically expected within 12-16 weeks after repair.[7]

This article discusses the rehabilitation outcome following a spontaneous PTR in a patient with WS.

CASE REPORT

A 48-year-old male had been under follow-up for scleroderma due to skin lesions. Genetic analysis later confirmed the diagnosis of WS. Five years earlier, he experienced a sudden rupture of the left patellar tendon during knee flexion, which was surgically repaired. Five years after the first surgery, while sitting on his knees, he felt a popping sensation in his right knee followed by sudden pain and swelling. He was evaluated by the orthopedics department and underwent surgery for a right PTR. The patellar tendon was sutured and reattached to the superior patella, and the retinaculum and medial patellofemoral ligament were also repaired. A long leg cast was applied.

One month later, the cast was replaced with a splint, and an adjustable-angle knee brace was prescribed. The patient was referred to our outpatient clinic 2 months after the operation because of significant limitation in knee movement. On initial examination, knee flexion was limited to 15 degrees. There was no warmth or swelling, but the joint movement was painful. Pain intensity was rated as 5/10 on the visual analog scale (VAS).

The primary goal of rehabilitation was to normalize ROM and restore functional mobility. Supervised physiotherapy sessions were held 5 times per week. Each physiotherapy session consisted of 20 minutes of hot pack application, 10 minutes of ultrasound therapy, 20 minutes of transcutaneous electrical nerve stimulation (TENS), and 30 minutes of supervised exercises including isometric quadriceps contractions (3 sets of 10 repetitions), passive-assisted ROM exercises (3 sets of 10 repetitions within tolerance), and progressive resistive training as tolerated (starting at 2 sets of 10 repetitions with gradual increase depending on pain and muscle strength). On the 5th day of therapy, the patient reported increased knee pain and swelling. Examination revealed increased warmth and mild joint effusion. Ultrasonography showed minimal suprapatellar fluid. Heat therapy was discontinued and replaced with cold therapy 3 times daily. The stretching exercises were paused, and non-steroidal anti-inflammatory drugs (NSAIDs) were initiated for pain management. Due to persistent swelling and slightly elevated sedimentation and C-reactive protein levels, septic arthritis and crystalline arthropathy were considered in the differential diagnosis; however, clinical signs and laboratory parameters (normal leukocyte count, absence of fever, and negative joint aspiration findings) were not supportive. Therefore, low-dose methylprednisolone (8 mg) was added to the treatment. The patient's symptoms improved significantly after steroid initiation, and follow-up ultrasonography showed resolution of the suprapatellar fluid.

Rehabilitation was continued with TENS for analgesia and stretching/strengthening exercises within the pain threshold. Methylprednisolone was tapered and discontinued over 4 weeks.

After 30 sessions of rehabilitation, knee flexion improved to 60-65 degrees. Pain intensity was rated 5/10 on the VAS. At discharge, the patient was advised to use crutches and follow a home exercise program.

At the 6-month follow-up, the patient reported modest functional improvement with knee flexion reaching 70°, but continued to use crutches intermittently. No recurrence of effusion was noted.

Written informed consent was obtained from the patient.

DISCUSSION

Musculoskeletal findings commonly seen in WS include muscle wasting, calcific deposits in tendons and ligaments (especially in the knees, ankles, and hands), osteoporosis, insufficiency fractures, and foot deformities.[4] In our case, calcific deposits were noted

in the patellar and Achilles tendons, as well as osteoporosis, toe flexion contractures, and lower limb muscle wasting (Figure 1).

PTR is a relatively rare injury, typically occurring in individuals under the age of 40 years, usually due to trauma or advanced tendinopathy.[6] Certain systemic conditions can predispose individuals to PTR, including rheumatoid arthritis, chronic renal failure, prolonged corticosteroid use, diabetes, hyperparathyroidism, and connective tissue disorders.[6]

At the molecular level, WS is caused by mutations in the Werner syndrome helicase (WRN) gene, which encodes a RecQ helicase involved in DNA replication, repair, and telomere maintenance. Loss of WRN function leads to genomic instability, impaired cellular senescence regulation, and defective tissue regeneration. These mechanisms are thought to contribute to tendon degeneration and impaired healing capacity. In particular, reduced fibroblast proliferation and abnormal extracellular matrix turnover may underlie the increased vulnerability to tendinopathy and spontaneous tendon ruptures observed in WS patients.[1] This molecular background helps explain the chronic, poorly healing nature of tendon injuries in WS and supports the need for modified rehabilitation approaches in this population.

To our knowledge, there are no previous reports in the literature of spontaneous PTR in patients with WS. In our case, bilateral spontaneous ruptures occurred after age 40, without clear trauma. Although we did not observe the pre-rupture period, we believe the ruptures were due to chronic tendon degeneration and tendinopathy, supported by imaging evidence of patellar tendon calcification on both X-ray and magnetic resonance imaging (Figure 2, Figure 3).

Soft tissue calcification, especially tendon calcification, is one of the diagnostic criteria for WS.[8] Calcific tendinopathy worsens clinical symptoms,

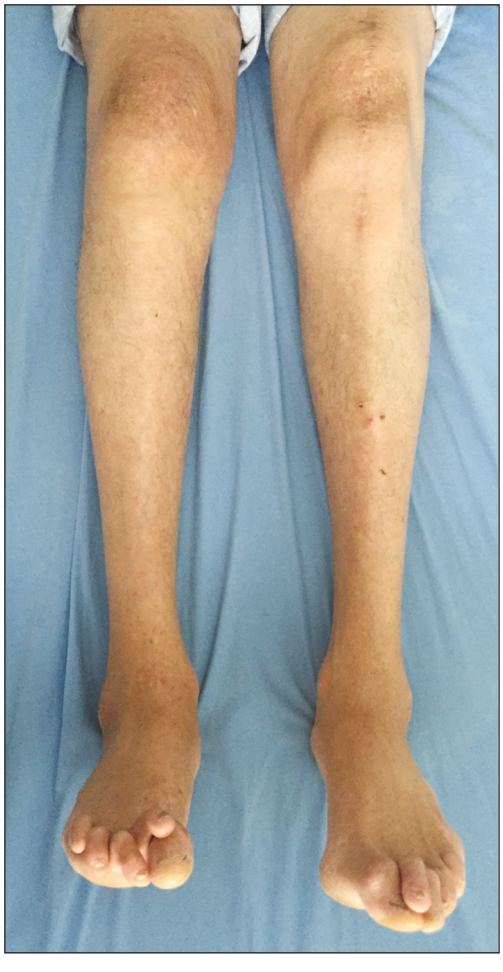

FIGURE 1: Feet deformities (toe flexion contractions) and muscle wasting in the lower limbs

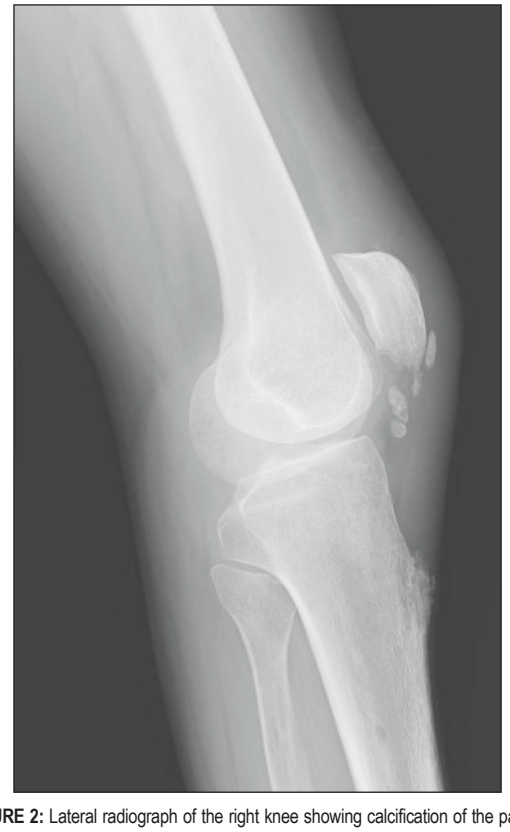

FIGURE 2: Lateral radiograph of the right knee showing calcification of the patellar tendon

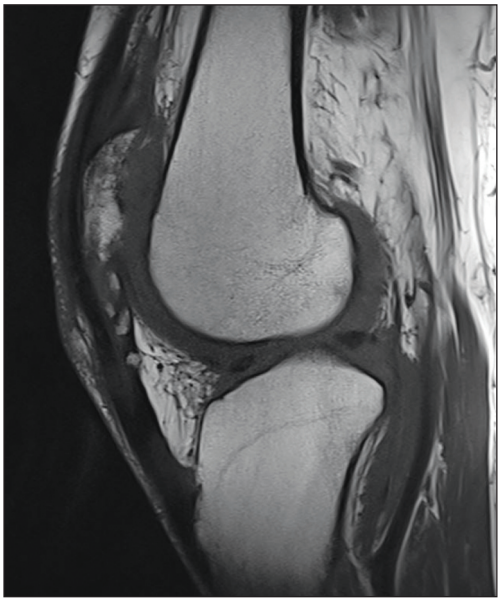

FIGURE 3: Magnetic resonance imaging of right knee showing calcification of the patellar tendon

increases rupture risk, prolongs recovery, and raises the likelihood of complications following surgery.[9] Additionally, our patient had Type 2 diabetes mellitus, another known risk factor for tendon rupture.[9]

Tendons affected by prior tendinopathy have reduced adaptability to mechanical loads. The chronic degenerative nature of PTR in these cases necessitates a more prolonged and cautious rehabilitation protocol.[10] Early rehabilitation following PTR repair should begin within the first week post-op, limiting motion from 0-45 degrees, allowing only active flexion and passive extension. After 2 weeks, ROM can be increased by 15 degrees per week until full ROM is achieved. Full weight-bearing is allowed for 6 weeks with the knee locked in extension and with crutches. Active extension exercises begin thereafter, and by 12 weeks, crutches are typically discontinued as quadriceps strength improves.[6]

In our case, the outcome was suboptimal even 4 months post-surgery. Full ROM was not achieved, and the patient continued to require crutches. Several factors may have contributed to this: 1st the patient was referred to rehabilitation 2 months post-surgery, which is considered delayed. Earlier rehabilitation may have led to better outcomes. 2nd Synovitis developed early in the rehabilitation process, necessitating steroid and NSAIDs use and a pause in exercises. 3rd Patellar tendon calcification due to WS likely caused chronic tendon weakness, impairing healing. Prior research by Walton et al. also indicated poor soft tissue healing in WS patients.[4] 4th Foot deformities and wounds due to WS further impaired mobility and rehabilitation progress.

Patients with WS are prone to musculoskeletal complications due to chronic tissue degeneration and calcification, which can compromise both surgical outcomes and rehabilitation progress. Spontaneous tendon ruptures although rare, may occur and require individualized management strategies. This case highlights the need for early, tailored, and closely monitored rehabilitation protocols in WS patients following tendon repair. Multidisciplinary coordination, early intervention, and awareness of the syndrome's unique challenges are essential to optimize functional recovery in these patients.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

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