MULTIPLE, SPONTANEOUS TENDON RUPTURES WITH ASSOCIATED SPONDYLOLISTHESIS AND OSTEOARTHRITIS: A CASE SERIES

Jason Seewoodhar and Roger Wolman

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INTRODUCTION

Multiple spontaneous tendon ruptures are rare. The aetiology of these conditions is largely unknown and treatment is difficult. Trauma and chronic medical conditions, such as renal failure, diabetes mellitus, hyperparathyroidism, systemic steroid therapy, fluoroquinolone usage, and more recently aromatase inhibitors, e.g. anastrazole, have been associated with multiple spontaneous tendinopathies.(1,2)

We present three cases of multiple spontaneous tendon ruptures. The aetiology of multiple tendinopathies and whether the clinical presentation of spontaneous tendon ruptures should lead to screening for associated medical conditions and other susceptible tendon sites is discussed. The association between multiplespontaneous tendinopathies and other rheumatological conditions such as degenerative spondylolisthesis and predisposition towards secondary osteoarthritis is critically considered. Furthermore, the level of functional disability and difficulties in the management of multiple spontaneous tendon ruptures is discussed.

1 Dr Jason Seewoodhary, Department of Diabetes and Endocrinology, Swansea School of Medicine, University of Wales, Swansea, UK, seewoodharyj@hotmail.com
Case 1

A 74-year old lady presented with multiple tendon ruptures. Aged 72 she spontaneously ruptured her right Achilles tendon and six months later she ruptured the left. Aged 73 she ruptured her right subscapularis and biceps tendons following a fall. In view of her age the functional needs for her shoulder were relatively modest and this was, therefore, treated with a cortisone injection and a course of physiotherapy. She also suffers from unilateral chronic grade 2 tibialis posterior tendon insufficiency.

The multiple tendon ruptures caused marked functional disability. She had difficulty using her dominant right upper limb for personal care, driving, and writing, which led to difficulties fulfilling activities of daily living. As a result of the lower limb tendinopathies she could only walk short distances.

She previously had two spinal fusions at L4/5 and L5/S1 for a degenerative spondylolisthesis. Furthermore, she has had bilateral patellar dislocations with stretching of the medial retinacula. She also has severe widespread osteoarthritis.

She has no medical risk factors for tendon rupture.

Case 2

A 67-year old lady presented with multiple tendon ruptures. Aged 54 she spontaneously ruptured her left Achilles tendon, which was surgically repaired. Aged 60 she complained of shoulder pain and an MRI scan revealed a tear to the left supraspinatus tendon. There was no preceding trauma to the left shoulder. Later that year she suffered a full thickness tear to the right supraspinatus tendon. Aged 63 she ruptured her right tibialis anterior tendon and soon after she sustained a partial rupture to the right Achilles tendon. All the tendon ruptures were treated conservatively with physiotherapy.

Her multiple tendon ruptures have caused a combination of ongoing bilateral shoulder and foot pain. She has difficulty walking and climbing stairs due to a right sided foot drop and she struggles with personal hygiene and dressing secondary to a reduced ability to abduct her arms. This has significantly compromised her quality of life.

She has a degenerative spondylolisthesis at L2/L3 and has widespread osteoarthritis.

She has risk factors for tendinopathies. She is an insulin dependent diabetic, suffers from hypertension and hypercholesterolaemia, which are being treated with a thiazide diuretic and a statin respectively. She developed hyperuricaemia secondary to the thiazide diuretic, which may have contributed to her musculoskeletal problems.

She has no history of chronic renal failure, hyperparathyroidism, systemic steroid treatment or fluoroquinolone usage.
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Case 3

A 68-year old lady sustained multiple tendon ruptures over a three year period. Aged 65 she complained of left shoulder pain and an ultrasound scan showed impingement tendonitis with a small, early, full thickness tear of the supraspinatus tendon. Aged 66 she suffered a fall and injured her left shoulder, which aggravated the tear. Aged 67 she complained of progressively worsening right shoulder and arm pain, which she sustained after a fall. Radiographs of the right shoulder showed upward migration of the humeral head suggestive of a rotator cuff tear and an ultrasound scan confirmed full thickness tears of the right supra- and infraspinatus tendons. Furthermore, she has a longstanding history of pain in the right ankle, foot, shin and calf as a result of tibialis posterior tendon dysfunction, which has been treated conservatively with an orthotic prosthesis.

Her multiple tendon ruptures have caused significant functional impairment and pain, which has markedly affected her quality of life. This was treated conservatively with physiotherapy focusing on general conditioning exercises and a long term stabilisation home exercise plan.

She has a grade 1 spondylolisthesis at L4/5 and at L5/S1. MRI scanning also revealed marked canal stenosis at L5/S1. She suffers from widespread osteoarthritis particularly in the knees, shoulders and lumbar spine, which have been treated with multiple intra-articular steroid and hyaluronic acid injections separately.

She has no medical risk factors for tendon rupture.

DISCUSSION

The pathogenesis of tendinopathy in the above three patients remains difficult to study because tendon biopsies were not obtained before the tendons ruptured. The term ‘spontaneous tendon rupture’ describes ruptures that occur without any preceding clinical symptoms, but because tendons can withstand very high tensile loads these conditions are rarely truly spontaneous and are associated with at least some degree of matrix degeneration.(3) Studies of degenerate supraspinatus tendons found a significant decrease in the total collagen content, and an increased proportion of type III collagen relative to type I collagen.(4) Post-translational modification of the collagen network was also different compared with age-matched normal tendons with a significant increase in hydroxylsine and the mature hydroxylsylpyridinoline and lysylpyridinoline collagen cross-links.(5) Such a genetic aetiology may lead to the development of novel molecular therapeutics aimed at restructuring degenerate tendons.

Spontaneous tendon ruptures are reportedly associated with systemic diseases, such as chronic renal failure, diabetes mellitus and hyperparathyroidism.(1) There are currently no guidelines on whether the presentation of multiple spontaneous tendinopathies should lead to screening.
for these associated medical conditions. It is our opinion that screening for associated medical conditions should be part of the routine management of patients presenting with multiple tendinopathies.

All three patients had suffered a significant disability as a result of their multiple spontaneous tendinopathies. Studies have found that long-term adverse affects of tendon ruptures coupled with the difficulties in managing them include abnormal gait, chronic pain, and inability to function normally, resulting in loss of occupation and/or depression.(6) These patients are a challenge to manage and we would agree with Palmer, who suggested patients need a thorough assessment, early recognition and comprehensive management to enhance functional outcomes and reduce disability.(6)

There is currently no evidence on whether spontaneous tendon rupture at one anatomical site should lead to screening of other tendon sites. It may be that by routinely screening other sites of potential tendon rupture would lead to earlier diagnosis and thereby reduce future disability. Further research into this concept is required.

All three patients with multiple tendinopathies had radiological evidence of spondylolisthesis. This may be due to a chance association as there is currently no evidence that multiple tendinopathies are associated with spondylolisthesis.

However, it is possible that there is a unifying genetic aetiology. It is known that tendons which spontaneously rupture have a collagen content that is structurally weaker than normal with an increased proportion of type III to type I collagen and glycosaminoglycan accumulation.(4) Unfortunately, there are no equivalent human studies investigating the collagen content of ligaments that have ruptured after minimal trauma. However, studies in dogs have shown that ligaments are 90% type I collagen although the extracellular matrix content of ruptured ligaments has significantly higher amounts of immature cross-links, total and sulphated glycosaminoglycans and water content, compared with that of intact ligaments.(7)

Whether the weakened connective tissue content and molecular changes of the multiple ruptured tendons is also found in paravertebral and spinal ligaments, which could then predispose onto a spondylolisthesis requires further research.

There is some evidence that rupture of the tibialis posterior tendon is associated with secondary osteoarthritis of the subtalar and ankle joints.(8) Furthermore, it is also well established that rotator cuff arthropathy predisposes to secondary osteoarthritis of the shoulder joint, so called cuff arthropathy.(9) All three of our patients with multiple tendinopathies had osteoarthritis. Whether tendon ruptures predispose to secondary osteoarthritis or whether osteoarthritis is an independent risk factor for spontaneous tendon ruptures at nearby or distant sites warrants further research.

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REFERENCES