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Bilateral spontaneous rupture of Achilles tendons: A case report

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ABSTRACT

Spontaneous bilateral rupture of Achilles tendon is rare. Rupture of the Achilles tendon has been described in patients on oral corticosteroid therapy. The sudden dorsiflexion of the plantar-flexed foot is the usual mechanism of injury. Spontaneous bilateral rupture is common in the degenerated tendon, which is often seen in patients with long-term corticosteroid therapy. This case is unusual because the patient has never taken steroids. We discuss the mechanism of injury and other probable causes.

Key words: *Achilles tendon; rupture, spontaneous; tendon injuries*

INTRODUCTION

The rupture of the Achilles tendon does not occur commonly, although it is the most frequently ruptured lower limb tendon and accounts for approximately 20% of all large tendon injuries.[1] Simultaneous spontaneous bilateral rupture of Achilles tendon is even more rare.[2] Patients with chronic diseases like systemic lupus erythematosus and rheumatoid arthritis who are treated with corticosteroids; and healthy individuals or athletes who are active in physical activities seem to be more prone to have Achilles tendon rupture.[1] Habusta[1] reported that the incidence of Achilles tendon rupture was around 0.02% in the western population. Less than 1% of them had bilateral simultaneous rupture.

Many of these cases have not been reported in the English medical literature. Our case is unusual because

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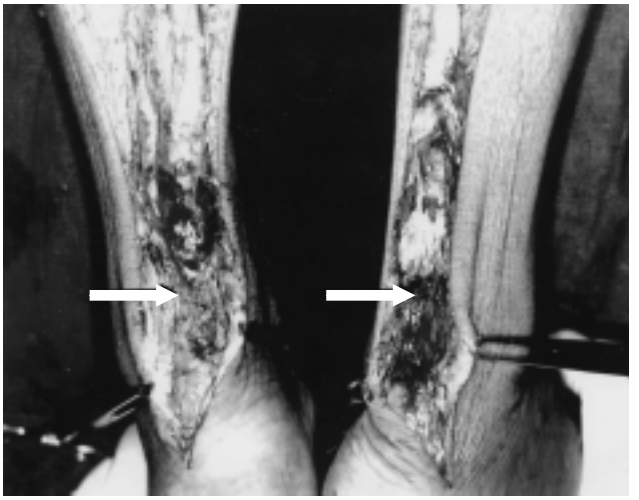

Figure 1 Bilateral rupture of Achilles tendon showing gap between 2 cut ends (arrows).

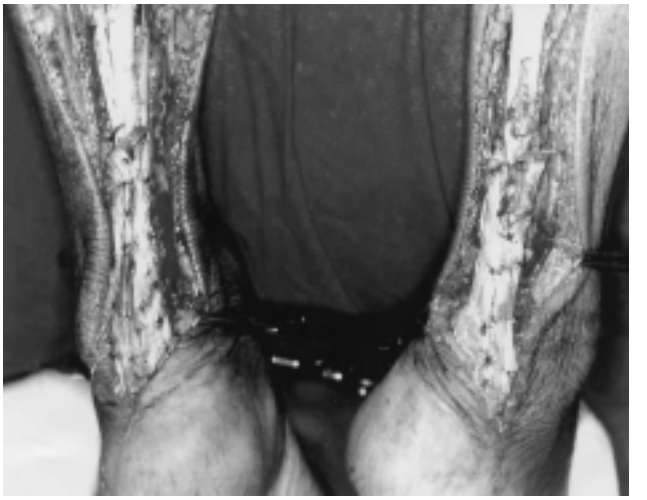

Figure 2 Repaired Achilles tendon.

the spontaneous bilateral rupture of Achilles tendon occurred in a woman who had never taken steroids.

CASE REPORT

A 38-year-old woman who was diagnosed to have hypothyroidism had been treated with thyroxine for 6 months before presentation. She presented to the orthopaedic out-patient department of Kasturba Medical College Hospital in March 2002 with pain in the retrocalcaneal region of both ankles. She was unable to plantar flex both feet. She had experienced a sudden snap and pain at the back of the left ankle while walking on plane ground at home. She had wrapped her foot with a cold pack and rested the ankle. A day later, she experienced a similar snap and pain in the right ankle in an attempt to walk. Physical examination revealed palpable gap and tenderness approximately 4 cm above the insertion of both Achilles tendons. Thompson's test was positive on both sides. Biochemical investigation showed marked increase in serum cholesterol level. Her thyroid function tests were within normal limits and she did not have any other associated diseases. Exploratory examination confirmed the physical findings of rupture of the tendons (Fig. 1). Primary surgical repair of both tendons and reinforcement with fascial graft from gastrocnemius aponeurosis was performed (Fig. 2). The patient's legs were placed in a bilateral long-leg cast with both feet in minimal plantar flexion for 6 weeks. Six weeks postoperatively, she regained a full range of motion in both ankles and her muscle strength gradually improved. Specimens of the ruptured Achilles tendon showed degenerative changes. Nine months postoperatively, the patient was able to walk without any difficulty or pain.

DISCUSSION

Bilateral simultaneous and spontaneous rupture of the Achilles tendon has been reported to occur as a complication of long-term corticosteroid therapy. Raunest et al.[3] reported that patients with hypercholesterolaemia had a higher risk of rupture. Rupture of the Achilles tendon was seen more often in a younger age-group with a mean age of 38 years.[4] Less than 20 cases of bilateral simultaneous Achilles tendon rupture have been reported and the majority of them were related to corticosteroid use.[1] The mechanism of injury of this case was probably because of sudden dorsiflexion of the plantar-flexed foot. This happened while the plantar-flexed foot gaining momentum against the ground to push off the foot from the floor, which resulted in forceful contraction of the gastrocsoleus group of muscles. The combination of eccentric loads from the contracted gastrocsoleus and ground force initiated the rupture at the weakest point of the Achilles tendon. Habusta[1] reported that such spontaneous ruptures were common in degenerated tendons. Another contributing factor for bilateral rupture of Achilles tendon is chronic weakening of the collagen structure of the tendon. This condition is usually

seen in patients with long-term corticosteroid therapy[2,5,6] and hypercholesterolaemia.[3]

Several mechanisms have been postulated for rupture of Achilles tendon in hypothyroid patients. Hypothyroidism causes decreased synthesis and degeneration of collagen. It also inhibits epimerase, which results in reduction of chondroitin sulphate and elevation of hyaluronic acid, hence weakening the matrix. Transient hypercalcaemia resulting from hypothyroidism causes calcification of tendon and small vessels. This decreases the vascularity of the tendon and further reduces the tendon strength. Extra intracellular accumulation of fat caused by hypercholesterolaemia increases the size of the cells. Lipomatous infiltration in the tendon causes fibrillation of collagen, produces cleavage of planes, alters the vascularity pattern, and finally reduces tendon strength.

Histopathological results of the specimens of this case showed degenerative changes in the Achilles tendon. It was possible that the pain several months before the final tendon rupture might be due to repeated minor trauma, leading to minute tears in the degenerated tendon, which was expected to heal in healthy individuals. However, hypercholesterolaemia and the earlier hypothyroid condition probably contributed to the delayed healing of the degenerated tendon and led to the final rupture.

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