Dear Sir,

Spontaneous tendon rupture is a rare but well-known complication of renal or systemic diseases due to an accelerated degeneration of collagen. Presented here is a renal transplantation (RT) patient who experienced nontraumatic bilateral rupture of both Achilles tendons at the time of an abrupt dehydration.

A 61-year-old man suffering from IgA nephropathy and treated with continuous ambulatory peritoneal dialysis since March 1990 was admitted to hospital on October 12, 1991 for an RT. Laboratory tests performed before RT ruled out hyperparathyroidism. Serum calcium was 2.1 mmol/l, while serum phosphorus was elevated to 1.8 mmol/l. There was no increase in serum alkaline phosphatase (120 mU/ml, normal value 70-210) or in parathyroid hormone (intact PTH) as measured by immunoradiological assay (60 pg/ml, normal value 15-65). The baseline immunosuppressive regimen consisted of antilymphocyte globulin (ALG) for 13 days later replaced by cyclosporine (CsA) with a loading dose of 6 mg/kg/day. ALG was combined with intravenous methylprednisolone (40 mg per day) and CsA with oral prednisone at the initial dose of 1 mg/kg/day tapering off to 0.3 mg/kg/day. In the posttransplantation period, immediate oliguric acute tubular necrosis required 3 hemodialysis sessions and peritoneal dialysis was again started on the 10th postoperative day. On day 13, polyuria developed and the patient lost 3 kg in 24 h. At this time, the patient complained of bilateral pain and stiffness in the mid-calf when walking. Physical examination revealed bilateral diffuse slight swelling in the area of the Achilles tendon. There was no abnormality on dorsiflexion. Despite appropriate rehydration, the patient continued to lose weight because of persistent polyuria (more than 4 liters a day). Sodium was 144 mmol/l, total serum proteins were 54 g/l and hematocrit 28%. Serum creatinine regularly decreased to 389 µmol/l at the 19th day and blood urea nitrogen was still elevated to 33.3 mmol/l. On the 20th day, an elevation in fasting blood glucose to 13.8 mmol/l not requiring any treatment was noted. The patient had no past history of diabetes. On the 25th day, total weight loss was 8 kg. The patient suddenly experienced inability to walk and he could...
not stand on his toes. No violent muscle contraction preceded the gait disorder. A hematoma was visible in the area of the Achilles tendons and a distinct gap could be felt suggesting the diagnosis of total rupture of both Achilles tendons. Tendon discontinuity was seen on magnetic resonance imaging. Laboratory data were as follows: serum creatinine 194 \( \mu \text{mol/l} \), blood urea nitrogen 15 \( \text{mmol/l} \), fasting blood glucose 6.6 \( \text{mmol/l} \), serum calcium 2.1 \( \text{mmol/l} \), phosphorus 1.14 \( \text{mmol/l} \), PTH 65 pg/ml, and alkaline phosphatase 82 Ul/ml. Treatment was orthopedic consisting of bilateral immobilization first with boot cast in equinus position for 4 weeks followed by a boot cast for walking for 4 weeks. Six months later, the outcome was characterized by a persistent gap in both tendons, instability and a limping gait.

Sporadic cases of tendon rupture have been reported in successful renal transplantation patients (Achilles or quadriceps tendons) [1-3], with 1 case of multiple spontaneous rupture of the tendon not having any clear explanation [4]. Bilateral tendon rupture suggests some generalized condition predisposing to weakness of the tendon [5,6]. This is the case in systemic disease such as systemic lupus erythematosus [7], inflammatory reaction, diabetic arteriosclerosis [8] and chronic renal failure [5, 6]. In hemodialyzed patients,

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tendon rupture is related to the duration of dialysis and to the severity of secondary hyperparathyroidism [5, 9]. One third of the patients who presented with spontaneous tendon rupture had experienced previous symptoms such as tenderness, stiffness, pain or discomfort for a variable period before rupture occurred [6]. Thus, rupture occurs at a very late stage of the degenerative process, when the tendon is weak enough to be prone to rupture [6]. In the case reported here, previous symptoms preceded total bilateral rupture by 12 days. Such symptoms can be attributed to partial rupture which became total when the patient engaged in a normal physical activity. Histopathological studies of spontaneous ruptured tendons show obvious preexisting structural changes including hypoxic degenerative tendinopathy, mucoid degeneration and calcifying tendinopathy [6]. It has also been suggested that blood flow decreases in spontaneous ruptured tendons leading to persistent hypoxia [6]. Thus, it can be postulated that in our patient, dehydration resulted in a decrease in arterial blood flow in the tendons with hypoxia and impaired metabolic activity. Such vascular changes in a previously weakened tendon may have lead to the rupture. Age also can play a part in degenerative changes of collagen [8, 6]. Steroids may also alter collagen structure but they cannot be regarded as the sole factor because tendon ruptures have not often been observed in patients receiving long-term steroid therapy for systemic disease [8]. Finally, several conditions could have been implicated in combination in this related case of bilateral simultaneous rupture of the Achilles tendons, i.e., natural degenerative changes of old age, lesions due to past chronic renal failure and to steroid treatment. We wish to stress the role played by abrupt dehydration which may have caused acute changes resulting in the rupture of both tendons. The presence of previous symptoms emphasizes the need to consider the diagnosis in a patient at risk presenting a gait disorder in an effort to prevent total rupture by immobilisation during the stage.

References


