Clinical case

Spontaneous rupture of Achilles tendon and Cushing’s disease. Case report

Maladie de Cushing et rupture spontanée du tendon d’Achille.
À propos d’un cas

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Abstract

Spontaneous rupture of Achilles tendon is observed in patients with tumors, metabolic disorders, infections and systemic diseases. Fluoroquinolones may cause tendinopathies. In addition, spontaneous rupture uncommonly occurs in Cushing’s syndrome. We report a case of Cushing’s disease revealed by recurrent rupture of Achilles tendon. Tendon tears are described in patients receiving long-term steroid treatment, probably via a mechanism similar to that one occurring in Cushing’s syndrome. Although exceptional, this diagnosis should be discussed in patients with spontaneous rupture of Achilles tendon.

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Résumé

Certaines pathologies tumorales, métaboliques, inflammatoires et infectieuses s’accompagnent exceptionnellement de rupture spontanée du tendon d’Achille. Des médicaments tels que les fluoroquinolones peuvent être à l’origine de tendinopathie. À cette liste, il faut ajouter, dans de très rares cas, le syndrome de Cushing. Nous rapportons le cas d’un patient présentant une maladie de Cushing révélée par une rupture récidivante spontanée du tendon d’Achille. Les ruptures tendineuses sont décrites chez les patients traités par des corticoïdes au long cours, c’est probablement par un mécanisme physiopathologique proche que le syndrome de Cushing donne, dans de très rares cas, des lésions achilléennes. Bien qu’exceptionnel, il faut donc savoir évoquer ce diagnostic devant une rupture spontanée du tendon d’Achille.

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Mots clés : Maladie de Cushing ; Rupture spontanée du tendon d’Achille ; Effet de l’hypercorticisme sur les tendons

1. Introduction

Nontraumatic rupture of Achilles tendon is uncommon. It is reported in patients with tumors, infections (tuberculosis, syphilis, ...) [1], metabolic disorders (gout [2], primary and secondary hyperparathyroidism, type II hyperlipoproteinaemia [3]) and systemic diseases (rheumatoid arthritis [4], systemic lupus erythematosus [5], granulomatous vasculitis [6]). It is sometimes associated with fluoroquinolone therapy.

In addition, spontaneous rupture of Achilles tendon is uncommonly described in subjects with steroid excess from an exogenous or exceptionally endogenous source. In the literature, spontaneous tears of Achilles tendon have been reported in only four patients with Cushing’s syndrome [7–9]. This association is so unusual that the hypothesis of a coincidence may not be excluded. We report the first case of Cushing’s disease revealed by recurrent rupture of Achilles tendon.

2. Case report

A 44-year-old man with a medical history of hypertension treated by nicardipine (100 mg daily) and diabetes treated by glibenclamide (15 mg daily) not complicated by arteritis, developed pain in the left heel without previous trauma or quinolone therapy. Spontaneous rupture of the left Achilles tendon was diagnosed and treated by tendon suture.

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Five months later, the patient presented a new episode of pain in the same heel due to an elongation of Achilles tendon without rupture treated by immobilisation with a plaster cast for five weeks.

A few weeks later, the patient complained of low-back pain. A routine X-ray detected vertebral compression fractures L3/L4 and dual energy X-ray absorptiometry confirmed the diagnosis of osteoporosis (femoral neck T-score: −2.55 S.D., lumbar spine T-score: −1.43 S.D.).

The patient was hospitalized for suspicion of Cushing’s syndrome. Typical cushingoid features were then noticed at examination (hypertension, skin thinning, proximal muscle weakness, moon face).

Laboratory studies showed a calcium level of 2.43 mmol/l (normal range, 2.2–2.7 mmol/l), a phosphate level of 0.75 mmol/l (normal range, 0.75–1.2 mmol/l), a 25OHD concentration of 12.9 ng/ml (normal range, 10–68 ng/ml). Blood glucose was elevated at 160 mg/dl (reference range 70–100 mg/dl) and glycosylated haemoglobin A1c was 6.1%.

Endocrinology investigations revealed an elevation of serum cortisol 8 h at 738 nmol/l (reference range, 138–690 nmol/l), and of serum cortisol midnight at 836 nmol/l. The 24 h urine free cortisol excretion was 2017 nmol/24 h (reference range, 80–280 nmol/24 h). The baseline plasma ACTH value was in normal range (<46 pg/ml). The serum cortisol after 48 h low dose dexamethasone suppression test was 440 nmol/l. The CRH stimulation test revealed an increase in cortisol level at 1521 nmol/l and ACTH at 174 pg/ml. These results confirmed Cushing’s syndrome due to a pituitary adenoma.

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MRI detected a low signal intensity lesion (5 mm) with less enhancement in the right half of the pituitary gland which confirmed a pituitary microadenoma.

Treatment by transphenoidal pituitary adenomectomy was successful. Postoperative basal levels of plasma ACTH and serum cortisol were 5 pg/ml and 29 nmol/l respectively. Treatment by hydrocortisone (30 mg daily) was introduced.

Five years later, the patient remains free of any tendon disorder.

3. Discussion

Spontaneous rupture of Achilles tendon in Cushing’s syndrome occurs in middle-aged adults, in the fourth decade. Two out of the four patients previously described were women [8,9]; two of them had a bilateral rupture [7,8]. Only our patient presented a recurrent rupture.

Spontaneous rupture of Achilles tendon is described in patients receiving long-term steroid treatment [10]. Indeed during glucocorticoid excess, changes in the connective tissue can be observed.

Glucocorticoids alter the quantity and the chemistry of hyaluronic acid, an extremely active component of the ground substance, produced by the mast cells. As a consequence, fibroblast proliferation and maturation are decreased, and less collagen is produced [11].

However, the effects of glucocorticoids on the proliferation of human fibroblasts are controversial according to more recent data. Indeed, a Greek study (2005) [12] reports that in vivo, long-term exposure to high level of cortisol in Cushing’s syndrome leads to an increased proliferative ability of skin fibroblasts. On the other hand, excess endogenous glucocorticoid induces alteration of several parameters of extracellular matrix homeostasis, such as a decrease in collagen synthesis and an altered expression of matrix metalloproteinase and their inhibitors, finally leading to a reduction of the collagen mass.

Yet, no study has investigated the specific effects in tendon tissue of glucocorticoid excess.

Finally, a case of rupture ossified Achilles tendon has been described in a diabetic woman [13]. In the present case, hyperglycemia most likely had a direct effect on the tendon tissue, leading to ossification and rupture.

4. Conclusion

The incidence of Cushing’s syndrome is estimated at one to ten cases per million. Clinical features are nonspecific. A missed diagnosis may be associated with significant morbidity and morbidity. Although this clinical presentation is exceptional, Cushing’s syndrome should be discussed in patients with spontaneous rupture of Achilles tendon.

References