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Case Report

Neglected bilateral rupture of the patellar tendon: A case report

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ABSTRACT

Simultaneous bilateral rupture of the patellar tendon (PT) is extremely rare and is generally associated to some chronic diseases. When the rupture becomes chronic, it is more difficult to repair than as it remained untreated.

The diagnosis, which is clinical, is often delayed, guided by standard radiography and confirmed by ultrasound or MRI.

The management of a bilateral neglected, chronic patellar tendon rupture must address some serious difficulties: the proximally retracted patella, the reconstruction of the patellar tendon, finally, the temporary protection of this repair.

We report a case of neglected bilateral rupture of the patellar tendon in a chronic hemodialysis patient, treated with a plastic surgery of the ipsilateral quadriceps tendon.

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1. Introduction

Bilateral rupture of the patellar tendon (PT) is an extremely rare affection; these ruptures appear especially in patients with systemic disease or predisposing conditions. Most patients who sustain a spontaneous patellar tendon rupture have risk factors for tendinopathy including rheumatoid arthritis, chronic renal failure systemic, lupus erythematosus (SLE), rheumatoid arthritis, hyperparathyroidism, hereditary disorders of the connective tissue (like Ehlers-Danlos

syndrome) or long term medication, such as corticosteroids or fluoroquinolones.^{1,2} We report a case of a neglected bilateral rupture of the patellar tendon in a chronic hemodialysis patient.

2. Case report

It is about 40-years-old patient who has been undergoing haemodialysis for 12 years for an idiopathic chronic renal failure, the patient reports having dropped its height with a

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Fig. 1 – Clinical aspect of bilateral patellar tendon rupture.

reception on both knees flexed, following a sensation of instability of the right knee without notion of trauma or stumbling. He was presented to the emergency department in another hospital with knee pain and total functional impairment of both knees; the diagnosis of mild trauma was retained. Remaining bedridden, he consulted, three months later, in our department. The clinical examination revealed swollen knees with a sub-patellar defect (Fig. 1) and a lack of active bilateral knee extension. X-rays on both knees showed a

Fig. 2 – Lateral X-ray of the knee showing a patella alta.

patella alta with a Caton-Deschamps Index >1.2 (Fig. 2). The diagnosis of a neglected bilateral rupture of the patellar tendon was retained. In our case we did not find that it is necessary to do other complementary examinations such as ultrasound or MRI given their cost and therapeutic delay they cause. However Blood chemistry results were as follows: phosphate was 46 mg/l, serum calcium corrected by albumin was 92 mg/L, parathyroid hormone (PTH) level was 620 pg/mL and blood pH was 7.3.

Surgical treatment was identical for both knees using an anterior approach. The rupture of the PT was located in the middle third at the right (Fig. 3a), and in the proximal area on the left (Fig. 3b). The ends of the ruptured patellar tendons were frayed, and tendons seemed to be fragile. The reconstruction of the PT was performed by using a graft, extracted from the middle third of the ipsilateral quadriceps tendon (QT), with 10 cm of length and 15 mm of width, pedicled on the patella and returned to 180° (Fig. 4). The graft is passed through a tibial tunnel with an inlet orifice at the insertion of the native PT and an outlet orifice at 15 mm below. A wire strapping making a frame between the patella and the tibial tuberosity can adjust the height of the patella under fluoroscopic control with obtaining a Caton-Deschamps Index around 1. Proximally, the graft banks are sutured to the adjacent soft tissues by a non-absorbable wire, acting as brakes. The tight graft is sutured to itself. It is attached to the patellar ailerons and the stumps of native tendon (Fig. 5).

Removable splints have been prescribed for 6 weeks; the passive rehabilitation was undertaken since second day in a range of mobility of $0/60$ by arthromotor. One month after, the patient started doing partial weight-bearing with crutches and at the third month, he underwent an active rehabilitation based mainly on stretching quadriceps, the gradual increase of its resistance in eccentric and the change in execution speed movement. At the sixth month, the strapping wire was removed and after 7 months, the patient regained normal knee mobility ($0^\circ/120^\circ$) and resumed daily life activities.

3. Discussion

Traumatic ruptures of the PT are the result of significant pressure on the extensor mechanism in young athletes.³ However, its bilateral rupture is extremely uncommon² and occurs after low-energy trauma or without trauma.

The extensor mechanism consists of the quadriceps tendon, the patella, the patellar tendon, and the insertion of the patella on the tibial tubercle. The muscle moment arm of the extensor mechanism is increased by the patella. Patellar tendon rupture results from contraction of the quadriceps in a flexed knee. Opposite contractile forces create a superior moment arm across the quadriceps and an inferior moment arm pointing towards the tibial tubercle, strong enough to tear a normal patellar tendon if the force is 17.5 times the body weight.²

Usually the spontaneous bilateral patellar tendon rupture has been associated with systemic diseases such as SLE, rheumatoid arthritis, chronic renal failure and prolonged corticosteroid fluoroquinolones treatments.³ In addition to ageing, these conditions lead to the degeneration of collagen

Fig. 3 – Surgical view of patellar tendon ruptures: a) right knee, and b) left knee.

fibrils and the loss of tendon vascularisation.⁴ more specifically In CKD patients on long term hemodialysis; hyperparathyroidism (by causing dystrophic calcification-weakening tendon and subperiosteal bone resorption-weakening osteotendinous junction), acidosis (disrupting structure of protein-polysaccharide complex which is responsible for collagen maturation) and beta 2 amyloid deposition may be responsible for tendinopathy and rupture may occur with little force.^{2,5} Treatment of underlying hyperparathyroidism (calcium and vitamin D analogs-will address metabolic bone disease as well; sometimes a total parathyroidectomy with autotransplantation

of a parathyroid gland to avoid hypoparathyroidism) may prevent tendon ruptures.⁵

The scarcity of bilateral ruptures as well as the symmetry of signs found on clinical examination may obscure the diagnosis.⁶ Functional impairment of knees with defective active extension direct the diagnosis which will be confirmed by a profile X-ray of the knees showing patella alta.⁷ Ultrasound as well as MRI can be useful to confirm the diagnosis. MRI may also help to exclude other ligamentous or meniscal injuries especially in sports injury cases.^{8,9}

The treatment of neglected ruptures of PT is surgical. However, the surgical modalities must be adapted to the anatomopathologic aspect of tendinous structure. Infrequently, direct or transosseous sutures, protected by tibiopatellar cerclage wiring, are possible. After six weeks, repairing neglected forms becomes difficult due to the retraction of the quadriceps and the ascension of the patella.¹⁰ To fix this retraction problem, some authors propose the establishment of a percutaneous trans-patellar traction or an external fixator, or even an incision of the rectus femoris proximal tendon using Hueter approach, in order to gradually lower the patella before repairing the PT.^{7,11} These techniques should be reserved for ancient forms.

As is the case of our patient, the quality of the remaining patellar tendon requires reconstruction. This may involve the use of semitendinosus alone¹² or combined with gracilis tendon,¹³ fascia lata,⁶ or semitendinosus associated with a quadriceps flap.¹⁴ A PDS® band can also be used so as to strengthen the tendinous reconstruction.¹⁴ As the quality of the remaining tendon stump is insufficient, the use of contralateral autograft is the technique of choice for H. Dejour. Besides its iatrogenicity, this technique cannot be adopted in case of bilateral lesions.¹⁵

The reconstruction of PT by allograft using Achilles tendon or PT has been reported but the risk of infection restricts its use.¹⁶

Fig. 4 – The graft from the middle third of the ipsilateral quadriceps tendon.

Fig. 5 – Final position of the graft protected by cerclage wire: a) scheme face, b) profile scheme, c) surgical view.

To the best of our knowledge, only one author has used an ipsilateral quadriceps tendon graft that is sutured termino-terminally to the distal end of the native tendon.¹⁷ In our technique, the graft is passed through a tibial bone tunnel and sutured to itself. We believe that this device improves the immediate and secondary mechanical strength of the graft, thereby allowing an early rehabilitation of the patient. In the long run, this method will reduce the risk of re-rupture, especially in case of fatigue failure of the cerclage wire.

Postoperative immobilization is advocated by most of the authors for three to six weeks, with passive motion exercises depending on the stability and durability of the repair. Active rehabilitation begins at the sixth week.^{7,8} However, in the ancient forms, amyotrophy of quadriceps may persist and hinder the movement of the knee. In this case, the permanent wearing of a knee extension brace becomes necessary.¹⁸

4. Conclusion

The bilateral chronic rupture of PT is extremely rare. Emergency physicians as in the present case frequently miss this diagnosis. High index of suspicion, proper physical examination and ultrasonography in the emergency room may help to prevent missing such cases. Early detection and treatment may improve prognosis. Reconstruction to QT is a reliable and little iatrogenic technique.

Conflict of interest

All authors have none to declare.

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