CASE REPORTS

PULMONARY EMBOLISM WITH BILATERAL PULMONARY INFARCTION AFTER SURGICAL CORRECTION OF ACHILLES TENDON - A RARE COMPLICATION

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Abstract

Introduction: *Pulmonary embolism (PE) is a potentially fatal disorder that occurs as a result of a thrombus formed in the deep venous system that detaches and obstructs the pulmonary artery or one of its branches. Herein we report a rare, bilateral PE after surgical correction of the Achilles tendon.*

Objectives: *To report a bilateral PE after calcaneus tendon repair, and to review the literature on this rare condition.*

Materials and Methods: A search of the literature was carried out in electronic databases and a review of medical records.

Conclusion: *PTE, although rare, is a serious and potentially fatal complication, requiring adequate and early treatment. Pharmacological prophylaxis in these situations is still controversial in the medical literature; however, there is consensus for the use of intermittent pneumatic compression in the postoperative period.*

Keywords: *Calcaneal tendon; Pulmonary Embolism; Computed Tomography Angiography; X-Ray Computed Tomography*

INTRODUCTION

The calcaneal tendon is the thickest and strongest tendon in the human body and can withstand forces up to 12 times the body weight.1 Rupture of the calcaneal tendon occurs mainly from 30 to 50 years of life, occurring as accidental trauma and athletic activities, like throwing and jumping.[1,2]

Its injury mechanism often involves a sudden and powerful contraction of the gastrocnemius / soleus muscles with a flexed foot.[2] Spontaneous lesions of the calcaneal tendon are very unusual, representing less than 1% - Important risk factors are:1 medication with anabolic steroids, corticosteroids and fluoroquinolones, rheumatoid arthritis, systemic lupus erythematosus, gout, hyperthyroidism, limb Ischemia and previous rupture of the calcaneus tendon.

Amongst calcaneal tendon rupture complications, venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), may oc-

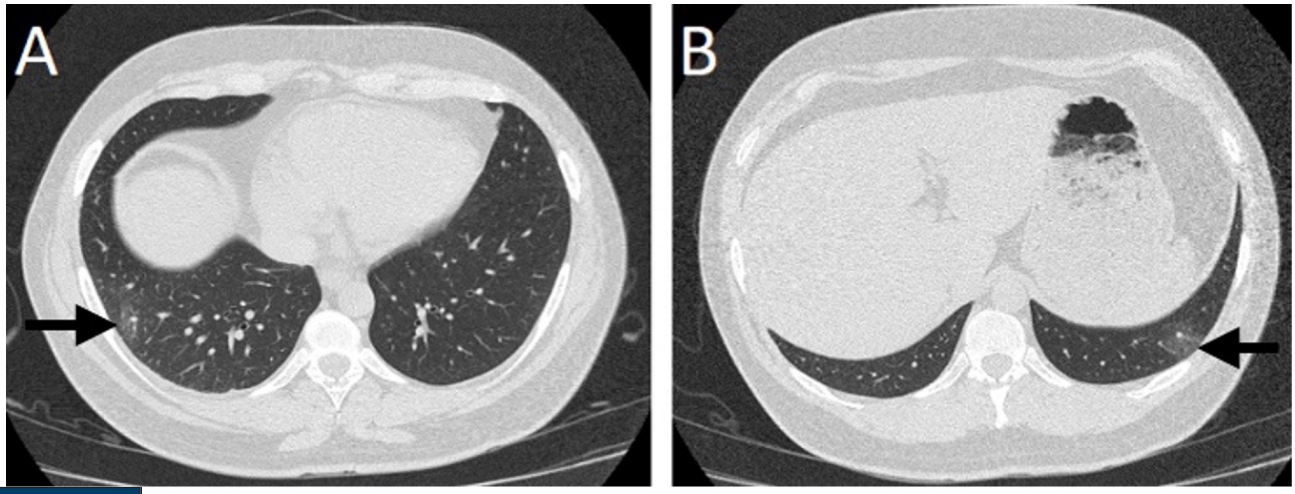

Figure 1 *Computed tomography of the chest without contrast, showing ground-glass opacities in the right lung base in A and the left lung base in B (black arrows).*

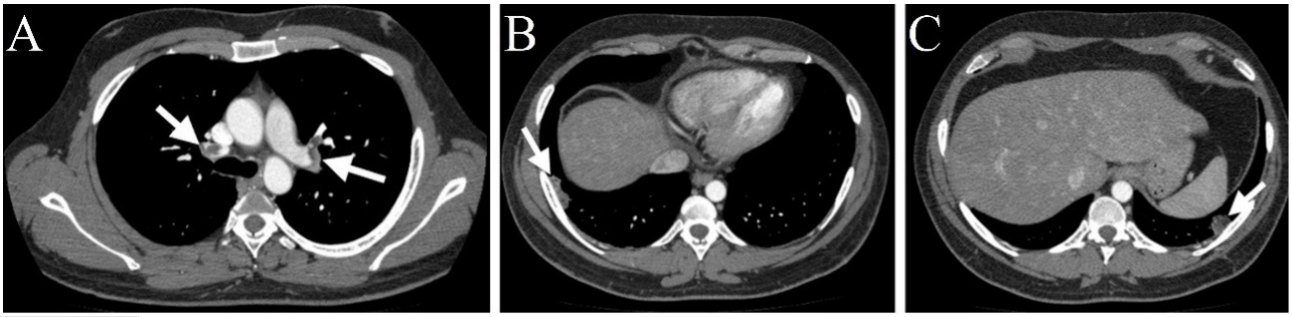

Figure 2 *In A, chest computed angiotomography scan showing filling defects in the main pulmonary arteries, inferring massive pulmonary thromboembolism (white arrows). The chest computed angiotomography scan showing wedge-shaped opacities on the inferior periphery of lungs (Hampton sign)–in the right in 2B and in the left in 2C (white arrows).*

cur due to immobilization of the injured limb.[2] Calder et al performed a meta-analysis investigating the incidence of VTE in foot and ankle surgery and found a VTE incidence of 0.6% in the clinical evaluation group, versus 12,2% in the group diagnosed with radiological aid.[2,3] However, VTE incidence in calcaneal tendon rupture was 7% in the clinical evaluation group, versus 35.3% in the group with imaging exams.[2,3]

Herein we report a case of PE with bilateral pulmonary infarction after surgical repair of the calcaneal tendon.Informed consent was obtained from the patient.

CASE REPORT

A male patient of 35 years old, in the 10thpostoperative day of surgery for correction of a ruptured calcaneal tendon (and no other pathological history), presented to the emergency department (ED) with respiratory distress, characterized by sudden, progressive, dyspnea, associated with stabbing chest pain, without irradiation. During this period, he reported peripheral cyanosis, hematemesis, and progressive dyspnea, being initially treated as COVID-19 at another ED, having received azithromycin and vitamin D; his computed tomography presented two pulmonary opacities in inferior lobes (Figure 1).Latter, real-time PCR for SARS-Cov-2 was negative.

After 2 days, the patient deteriorated, and had a syncope associated with central and peripheral cyanosis when performing minimal effort; he presented to our ED, being admitted to the ward for 2 days. A chest computed angiotomography(CCAT) was performed, which detected a bilateral (Figure 2A) with peripheral,wedge-shaped,

opacities, suggesting pulmonary infarction (Hampton sign) - Figures 2B and C. Patient was, then, transferred to intensive care unit (ICU), and started Enoxaparine at a 1mg/Kg BID for 2 days, later modified to Rivaroxaban 15mg BID; a venous Doppler ultrasound of the lower limbs revealed thrombosis of the superficial femoral vein and the supra-articular segment of the popliteal vein. He was discharged after 7 days as an outpatient follow-up.

After 3 days of hospital discharge, the patient reported hemoptysis of small quantity and stabbing pain in the left hypochondrium. As a pulmonology outpatient, he performed an abdominal and thoracic angiotomography, that revealed normal abdominal circulation, bibasal opacities, with filling flaws characterizing massive PTE (increased comparing to previous).

The patient had no hospitalization criteria, and so remained in home isolation; clinical picture improved and, so, anticoagulation with Rivaroxaban was maintained, with dose modification, after 21 days, to 20mg for 6 months.

DISCUSSION

In literature, few case reports are documenting DVT and PE after calcaneal tendon rupture.4 DVT incidence diagnosed by Doppler ultrasonography reaches 34% in the first two months of immobilization[2]; PTE is rare and occurs in about 1.5-3.0% of such cases.2,4,5 The incidence of DVT in the non-surgical treatment of Achilles tendon rupture varies from 1.2% to 4%.1However, Patel et al carried out a retrospective study, evaluating 1172 symptomatic patients after calcaneal tendonrupture for the incidence of PTE and DVT, revealing an incidence of 0.34 % and 0.43 %, respectively.[6]

Several factors contribute to thedevelopment of DVT/PTE with lower limb cast immobilization[5]; this includes trauma, prolonged immobilization, use of a tourniquet, and surgery[5,6]. However, some authors disagree with surgery as a risk factor, claiming that DVT/PTE can occur before the procedure, and be asymptomatic.[5] PTE or DVT may be a clinical manifestation of anundiagnosed prothrombotic disease[4,8]; therefore, all patients who develop DVT/PTE must undergo a detailed investigation of underlying conditions.[4]

CCAT is the gold standard for the diagnosis of PTE, due to its excellent accuracy, wide availability, good spatial resolution, and multi-planar reconstruction capability – main findings are filling defects within the pulmonary arteries, often surrounded by a contrast.7 Magnetic resonance imaging (MRI) is an alternative when there is a contraindication to the use of iodinated contrast.[7]

Complications of acute PE includeright ventricular dysfunction and pulmonary infarction (PI).[7] PI can be identified on the calcaneal tendon, MRI, or chest X-ray, as a wedge-shaped opacity at the lungperiphery (Hampton sign, as shown in the case), often with central ground glass (inverted halo sign).[7] PI occurs in 10 - 15% of patients with acute PTE, especially in patients with left heart failure, which decreases collateral blood supply through the bronchial arteries.8PTE mortality ranges from 10 to 30%.[7]

When DVT is suspected, it is recommended that treatment of community anticoagulation with the use of low molecular weight (or fractionated) heparin combined with oral warfarin therapy.1 Low molecular weight heparin once daily for 5 to 7 days is commonly used as initial therapy.[1] Systemic thrombolytic therapy is supported by the main guidelines as a first-line treatment for PTE.[8] However, such therapy is accompanied by a significant risk of 2% intracranial hemorrhage and severe general bleeding of up to 20%.[7]

Surgical treatment of calcaneal tendon can be performed open or percutaneous fashion, and non-surgical treatments include cast immobilization and functional brace.[2] Surgical treatment is a relatively simple procedure, but complications such as wound infection, delayed cicatrization, and adhesion, may occur; re-rupture can occur as a complication of non-surgical approaches.[2]

A meta-analysis by Calder and cols suggested that chemical prophylaxis with enoxaparin was not beneficial after surgery foot and ankle surgery;3 however, the authors noted that intermittent pneumatic compression may be useful for thromboprophylaxis incalcaneal tendonrupture.3 Testroote and cols performed a meta-analysis revealing a 55% lower risk of DVT for low molecular weight heparin versus placebo; however, no significant difference was found for the incidence of PTE.[9]

Domeij-Arverud et al evaluated the use of intermittent pneumaticcompression in the lower limb postoperative period and found that an incidence of DVT of 21%, versus 37% in the control group.[10] Muscular pumping action of the feet, calves, and thighs, perform most of the venous return from lower extremities.[2] In patients with calcaneal tendonrupture, loss of function of the calf muscle pump due to orthosis with plantar flexion of the ankle reducesvenous blood return flow; thus, due to changes in the Virchow triad, DVT may arise.[2]

CONCLUSION

PTE and DVT are complications of calcaneal tendon rupture. PTE, although rare, is a serious and potentially fatal complication, requiring adequate and early treatment. Pharmacological prophylaxis is still controversial in the medical literature, but there is consensus for the use of intermittent pneumatic compression in the postoperative period.

Triangular, wedge-shaped, lung opacities, associated with sudden dyspnea, chest pain, should always rise the hypothesis of PTE, especially in patients with predisposing factors, even with normal clinical parameters.

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