Exuberant plantar calcifications after corticosteroid injections

Raposo A[1], Santos R[2], Matias C[2]

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INTRODUCTION

Plantar fasciitis is a common cause of heel pain, usually a self-limiting condition. The aetiology of plantar fasciopathy is likely to be multifactorial, several risk factors have been reported (anatomic, biomechanical and environmental), but also inflammatory rheumatic diseases and neurologic conditions must be considered[1].

The studies showed that corticosteroid injections (CI) result in improvement of plantar fasciitis, reducing the heel pain and plantar fascia thickness[2]. Plantar calcifications are a rare secondary consequence after CI while plantar fascia rupture and the heel fat pad atrophy are the most feared complications, generally associated with multiple injections[2]. Plantar fascia rupture ranged from 2.4% to 6.7% in two retrospective studies, with an average of 2.67 *vs 2.1* CI in the sample[2].

CASE REPORT

A 54-year-old woman was referred to the Physical Medicine and Rehabilitation department for plantar fasciitis, with 15 years of duration, refractory to physical therapy, oral anti-inflammatory treatment and CI.

A previous plain X-ray (Figure 1-A), four years before, revealed a heel spur. She had been submitted to six CI with betamethasone (5) and methylprednisolone acetate (1) suspensions, with poor clinical response.

During the last two years the patient complained of increasing heel pain while walking and with prolonged weight bearing. A new X-ray (Figure 1-B) of her foot showed a calcification near the plantar fascia insertion and the computed-tomography (Figure 2) an exuberant conglomerate of calcifications in the medial aspect of plantar fat pad associated with subcutaneous oede-

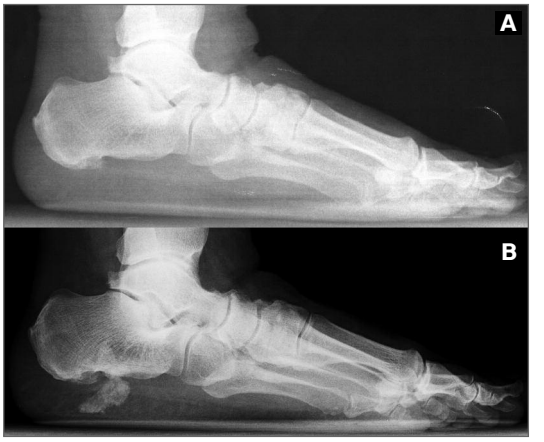

FIGURE 1. X-Ray image of A) 2009; and B) 2015, with a new area of calcification near the plantar fascia insertion

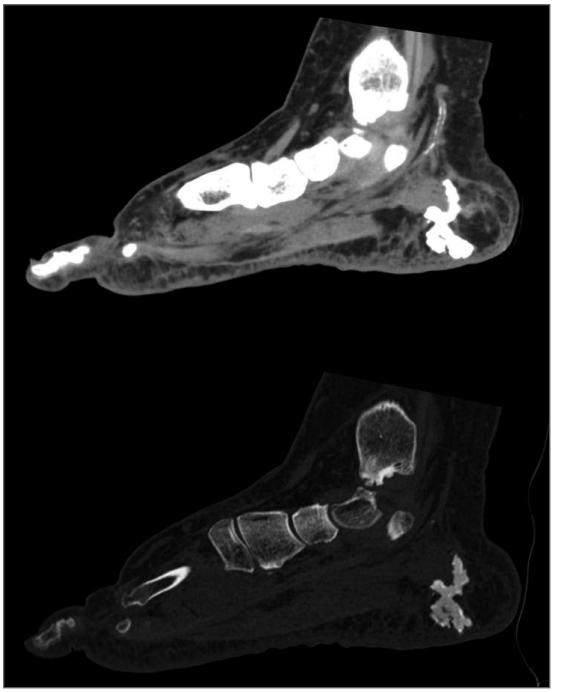

FIGURE 2. Computed-tomography image with an exuberant conglomerate of calcifications in the medial aspect of plantar fat pad associated with subcutaneous oedema

1. Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima;
2. Serviço de Medicina Física e de Reabilitação, Hospital de Braga

ma suggesting steatonecrosis. The patient was also refractory to posterior extracorporeal shockwave therapy.

DISCUSSION

Very few case-reports published plantar calcifications following CI[3-5]. It was suggested that the accumulation of insoluble steroid acts as a foreign body and induces a chronic inflammatory process with subsequent calcification[3].

Incorrect injection of corticosteroids can also induce necrosis and atrophy of the plantar fat pad, associated with increased morbility[4].

Although CI in plantar fasciitis is generally associated with low serious complications, the repetitive procedure and the slightly soluble, long acting corticosteroid used in this case (betamethasone), could be implicated as the cause for the formation of these exuberant calcifications.

CORRESPONDENCE TO
Ana Raposo
Serviço de Reumatologia
Unidade Local de Saúde do Alto Minho,
Ponte de Lima, Portugal
E-mail: anaeraposo@gmail.com

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