



Images in clinical rheumatology

Calcic tenosynovitis in a patient with undifferentiated connective tissue disease

Tenosinovitis cálcica en paciente con enfermedad indiferenciada del tejido conectivo

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We present the case of a 50 year old woman who was diagnosed with undifferentiated connective tissue disease (UCTD), based on severe Raynaud's phenomenon with ischemic ulcers, extensive calcinosis, sclerodactilia, polyarthritis, glomerulonephritis, polyserositis, chronic hypertransaminasemia and had undergone the bilateral suprachondyleal amputation with severe acute ischemia (associated to antiphospholipid syndrome and structural vasculopathy due to the disease). She was positive to anti-MI2 and anti-Ro52, AMA with a titer of 1:640, with negative ANA, anti-DNA, anti-U1RNP, anti-SCL70, anti-LKM and ANCA. The patient was clinically stable under treatment with steroids, antimalarials, cumarin and cyclic intravenous iloprost.

The patient had complained of chronic, painless swelling on both sides of the wrists, with little functional limitation, clinically compatible with flexor and extensor tenosynovitis. The ecography (Logic 9 series, General Electric) showed distension of the common extensor tendon sheaths of the fingers, common flexor tendons and first finger flexor (Figure), with an associated *power*-Doppler signal, with multiple hyperechogenic images on the sheaths and subcutaneous tissue, with posterior sonic shadow, compatible with hydroxiapatite calcium deposits (in the context of the patients' calcinosis). Hand x rays also showed multiple calcifications on soft tissue of the wrist and fingers, without being able to determine the localization of the deposits. A guided infiltration with triamcinolone was performed with an excellent clinical response.

Discussion

Dystrophic calcification of soft tissue, or calcinosis, is a frequent finding in connective tissue disease, especially systemic sclerosis (25% of patients), dermatomyositis (more prevalent in juvenile forms, affecting between 30% and 70% of patients) and systemic lupus

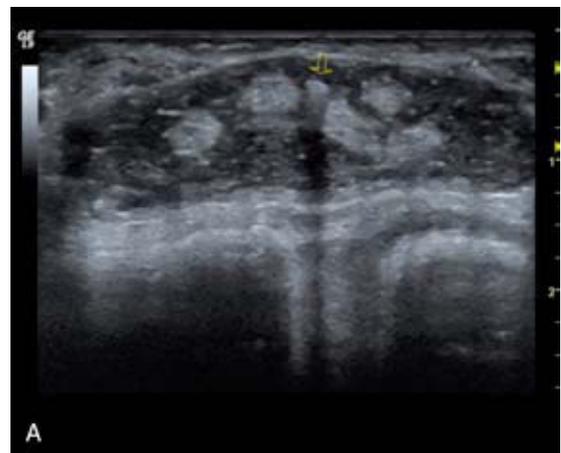


Figure 1. Transverse (B) and longitudinal (B) projection image of the extensor compartment 4, corresponding to the common extensor of the fingers. Longitudinal image corresponding to the first finger long extensor (C) on grayscale.

On transverse images the crossbeam software has been nullified, sending ultrasound waves in an oblique manner, minimizing the ecographic artifact known as posterior shadow, because this is useful in evaluating zones of calcinosis. Great distension of the tendon sheaths is observed, with the presence of hyperechogenic regions, with multiple sizes, with a posterior acoustic shadow (the most evident images are pointed by arrows) corresponding to deposits of hydroxiapatite. tecd indicates common extensor finger tendon; tfpd, first finger long extensor flexor; r, radius.

erythematosus (17% of patients).¹ The prevalence of UCTD is not well established, but may be similar.² Its pathogenesis is unknown, but has been related to a focal ischemic phenomenon, probably due to

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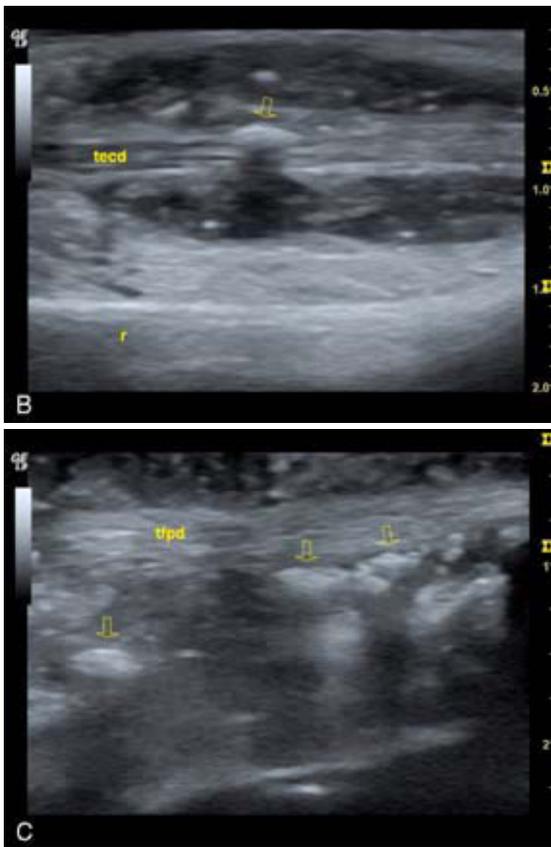


Figure 1. (Continuation)

alterations of microvascularization. It may also possibly contribute to the inflammatory process due to an elevated tissue presence of macrophages and proinflammatory cytokines (interleukin-1, interleukin-6, tumor necrosis factor).³

Calcinosis generally appears as plaques and subcutaneous nodules, variable in size, localized on pressure zones and extensor surfaces of the limbs. They are generally asymptomatic and can, on occasion, ulcerate or infect. They may also be accompanied by local inflammatory processes, although without a clear pathogenic relationship, because these are also manifestations of the underlying connective tissue disease (which in our patient may be a particular characteristic of her tenosynovitis). In a literature search we did not find any trials that critically evaluate the role of echography in calcinosis-associated tenosynovitis, nor those related with connective tissue disease or other clinical contexts. There are descriptions of tendon sheath distension, with an associated power Doppler signal, and the presence of hyperechogenic areas in the interior of the sheath, which project a posterior sonic shadow and correspond to calcium deposits.⁴

There is no effective pharmacologic treatment for the prevention or reduction of calcinosis.¹ Data regarding the use of warfarin, colchicine or calcium channel antagonists are limited and originate from uncontrolled trials. Large lesions, or those that are greatly symptomatic may benefit from surgery or, as in our case, from local steroid infiltration.

Conflict of interest

The authors have no conflict of interest.

References

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