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Observations about subcalcaneal adventitial bursitis (heel fat pad inflammatory lesion) in rheumatoid arthritis. Comment on the article of Suzuki and Shirai

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Dear Editor,
We read with great interest the clinical report of Suzuki and Shirai recently appeared in this journal (1). The authors accurately described two patients affected by undifferentiated palindromic pain in hands and foots, in whom a definite diagnosis of seropositive rheumatoid arthritis (RA) was made after an ultrasound (US) examination demonstrating subcalcaneal bursitis with synovial proliferation.
Scanty works described this peculiar type of subcalcaneal lesion, and some aspects of its pathogenesis and prevalence needs to be clarified. Previous reports from our group on this topic reported a frequency of 6.6% in RA (2). In a recent series of consecutive patients with definite RA who underwent US examination in our Institute between September 2019 and February 2020, we have diagnosed a subcalcaneal inflammatory lesion in 8%. Moreover, some observations have to be raised.

DEFINITION AND NOMENCLATURE

The authors defined the lesion *subcalcaneal bursitis with synovial proliferation*. In our previous papers we used the term *heel fat pad inflammatory-edematous lesion* to underline the peculiar hypervascularity and not homogeneous swelling of heel fat pad (3). Other authors described this lesion as *subcalcaneal panniculitis* (4), or *adventitial (adventitious) bursitis* (5, 6).

Nevertheless, to date, a permanent native synovial space (synovial bursa) in the subcalcaneal region has not been demonstrated. On the other hand, there are hystopatologic demonstrations that adventitial bursae can develop in adulthood in sites exposed to high pressure and friction (*i.e.,* pre-patellar bursitis, submetatarsal bursitis). In these conditions, a subcutaneous adventitious bursa develops as a coalescence of pre-existing small spaces in loose connective tissue. The walls progressively become differentiated from the adjacent connective tissue and a well-defined fluid-filled cavity is formed, surrounded by columnar cells, when friction forces are mantained. Unlike permanent bursae, they lack a mesothelial lining and synovia (5, 6). We suppose that a biomechanical overload could have triggered the bursal formation also in the two patients described, as they developed heel pain at the same time with forefoot pain. These considerations lead to choose the term *subcalcaneal adventitial bursitis* as the best definition for these lesions.

CAN SUBCALCANEAL ADVENTITIAL BURSITIS BE CONSIDERED AN EQUIVALENT FOR SYNOVITIS?

The authors stated that the subcalcaneal inflammatory lesion was the main synovitis that permitted to define a RA diagnosis and to start an appropriate treatment (1), but presently there are no hystopathologic

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demonstrations that these lesions could have an inflammatory infiltrate, like in rheumatoid synovitis. The intra- and perilesional hypervascularisation demonstrated with Power Doppler US is not sufficient to define it as synovitis. Moreover, in gray-scale US the echo-texture of the subcalcaneal lesion is similar to the normal heel fat pad, except for the higher content of hypoechoic areas (probably expression of oedema and inflammatory neovessels). On the other hand the good responsiveness of these lesions to biological therapies, rather than to soft plantar orthoses, indicates that immunologic mechanisms could be also implicated in maintaining inflammatory neovascularisation and related pain (1, 3).

IS SUBCALCANEAL ADVENTITIAL BURSITIS SPECIFIC FOR RHEUMATOID ARTHRITIS?

Although we cannot consider these lesions as an equivalent for synovitis (for their structure and pathogenesis), their higher prevalence in RA is undeniable (2-5) and, according to the literature, they are reported only in association with RA. In our case series we have rarely diagnosed subcalcaneal adventitial bursitis also in patients with systemic lupus erythematous (SLE), another immune-mediated condition (3). This observation could open new hypothesis on pathogenesis of fat inflammation and degeneration in RA and other immune-mediated conditions, as some previous reports described intrinsic abnormalities of composition and of viscoelastic biomechanical properties of fat tissues (body fat, knee and heel fat) in RA (7, 8).

In conclusion, the interesting clinical cases described by Suzuki and Shirai have contributed to highlight a cause of subcalcaneal pain in the early phases of RA, and to confirm the clinical relevance of US examination in differential diagnosis of hindfoot pain in RA.

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