Name and Classification of Soft-Tissue Rheumatism

José Álvarez-Nemegyei[a] and Juan J. Canoso[b]

[a]Unidad de Investigación Médica, UMAE # 25, Instituto Mexicano del Seguro Social, Mérida, Yucatán, México
[b]American British Cowdray Medical Center, México DF, México

The term soft-tissue rheumatism is one of the myriad designations of a group of entities that are characterized by regional pain in the extremities and are usually due to the abuse on tendons, enthesis, and bursae.[1] Clinical and ethiopathogenic similarities have led to the inclusion of some entrapment neuropathies, such as carpal tunnel syndrome, into this group, as well as certain vascular processes, for example thoracic outlet syndrome and chronic compartmental syndromes. The usual therapeutic sequence in soft-tissue rheumatism initially consists of the suppression or modification of the causal factors, the use of anti-inflammatory medication and physiotherapy, and in cases that are resistant to therapy, the local injection of glucocorticoids or even surgery.[2-4]

The epidemiologic impact and the socioeconomic consequences of soft-tissue rheumatism are significant. It is estimated that its prevalence varies between 3% and 15%,[5-8] according to the case-definition and the population in which the survey was carried out, surpassing that for rheumatoid arthritis and systemic lupus erythematosus. Paradoxically, in contrast to those diseases, resources assigned to research and diffusion of knowledge on soft-tissue rheumatism are meager or inexistent.[1] The medical community's lack of interest in these illnesses undermines the quality in the attention that patients receive and are translated into a heavier burden, in many ways avoidable, of suffering, handicap and personal and collective social, and economical losses. An important consequence of the lack of interest in these syndromes is the variety in the collective names used to name them: soft-tissue rheumatism, soft-parts rheumatism, extra-articular rheumatism, regional rheumatism, regional pain syndromes, tendonitis-bursitis syndromes, regional syndromes, and periarthritis, among others. All of them present with conceptual or factual errors that impact not only on their ethiopathogenic conception, but also on the design of preventive measures and their therapeutic approach.[1] A flagrant mistake is the use of the "itis" suffix, which indicates inflammation, in the designations of "tendonitis-bursitis syndromes" and "periarthritis", when hystopathologic evidence in diverse hand, wrist, elbow, shoulder, and foot syndromes[9-13] does not reveal inflammation but an angiofybroblastic tendinosis. This process is repeated with small variations in the different presentations, characterized by vascular hyperplasia, collagen fiber disorganization, an increase in the intercellular substance, microfybroblastic hyperplasia, and fibrocartilage metaplasia.[13,14] An obligatory conclusion derived from the absence of inflammation should be a critical review of the use of systemic or local anti-inflammatory drugs in these syndromes.[14] Apart from this, alternative hypothesis should be considered, such as the tendency toward a spontaneous resolution with the passage of time, such as is the case in lateral "epycondylitis,"[15] to explain the apparent efficacy of the current treatment algorithm, whose interventions are primarily anti-inflammatory. This aberration in the generic designations is also applied to individual entities such as "stenosing tenosynovitis," "de Quervains tenosynovitis," "lateral epycondilitis," and "plantar fasciitis." In the first, the cause of pain and tendinous entrapment is a predominantly fibrocartilaginous angiofybroblastic lesion of the flexor pulley of A1.[11,16,17] In the second, an angiofybroblastic lesion of the retinaculum above the first extensor compartment of the wrist leads to the manifestations.[10,16] On the other hand, "lateral epycondilitis" is originated by an angiofybroblastic lesion of the extensor tendons of the wrist, near their proximal insertion in the lateral epycondile[9]; while most of the cases of "plantar fascitis" are explained by a lesion of an angiofybroblastic nature that affect the insertion portion of the plantar fascia.[12] A correct denomination of these syndromes would imply the substitution of "itis" for "pathy" or "osis": tendinosis or stenosing digital tendinopathy, tendinosis or de Quervains' tendinopathy, epicondylosis, and plantar fasciopathy (fasciosis seems cacophonic). Equally erroneous are the designations of trochanteric bursitis and anserine bursitis. In clinically well defined syndromes of "trochanteric bursitis" studied through magnetic resonance imaging, the most frequently isolated anomaly is a tendinopathy, accompanied or not by an anatomical interruption of the gluteus medius muscle near its insertion to the larger trochanter. In contrast, the presence of an effusion in any of the perytrochanteric bursae is an infrequent finding in these patients.[18,19] In "anserine bursitis," maybe the most common cause of pain in the

Correspondence: Dr. J. Álvarez Nemegyei.
Calle 43# 340x 26y 26-A. Fracc. Monte Albán. 97114 Mérida.
Yucatán. México.
E-mail: nemegyei@yahoo.com.mx

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medial region of the knee, ecographic evidence indicates that not only is the anserine bursa not affected, but there are no other alterations in the bursa-anserine tendon complex that explain the syndrome.[20,21]

In conclusion, not enough research into soft tissue rheumatism has been done, and the knowledge generated by the scarce contributions from research has not been appropriately spread. Therefore the erroneous denominations used to define this entity, both collectively and individually mustn't surprise us. As an unfortunate consequence, the confidence put in these denominations has perpetuated a possibly erroneous therapeutic paradigm. The scientific community dedicated to the study of "soft-tissue rheumatism" has before them the unpostponable challenge of reviewing discussing and unifying the collective and individual denominations of these entities. The beneficiaries will be our patients and society in general. The task will be arduous, because it is a terrain that is shared by rheumatologists, physical therapists, orthopedic surgeons, and neurologists. Let the leadership be ours.

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