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Short Communication

Fibromyalgia, Generalized Pain Stage of the Fibromyofascial Syndrome

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Fibromyalgia, as defined and characterized by the American College of Rheumatologists (ACR), is a chronic neuromusculoskeletal disease, of a cause unknown until now, which has existed since a patient has more than 11 "tender points". Although in recent years, some other semiological variables have been added to refine its diagnosis, and it has related to varied emotional, psychiatric, and neurological pathology. Its treatment is only symptomatic and, even so, of weak and short therapeutic response. The most recent ACR medical recommendation for this disease is: "teach these patients to live with pain".

There are no references to patients who have 1 to 11 "tender points". They do not exist for current medicine, especially if they are people of productive working age and even less if they belong to a health insurance system. These people are victims of pain, increasingly intense, persistent, and widespread. That has no known cause and therefore has no curative treatment. Moreover, many times, they are treated as "simulators" by their health insurance systems, even if they have more than 11 tender points.

Another disease, myofascial syndrome, is characterized by being localized, of a recurrent, non-chronic course, characterized by presenting "trigger points" shares similar features, such as unknown and poor cause or no response to analgesics. It would correspond to the initial stages of Fibromiofascial Syndrome.

However, the problem that afflicts a large number of people who have these diagnoses is that their pain persists and worsens over time since his treatment has been only symptomatic.

Given this reality, a clinical study was conducted to find the cause of this disease, its characterization, and natural history. Aetiological hypothesis and treatment have established according to this hypothesis. Also, is evaluated the response to this treatment, which in the study was 100% effective in each participant. In the Journal of the Spanish Pain Society in 2017 this work was published. Its link is: https://www.resed.es/caracterizacion-del-dolor-fibromiofascial-hipotesis-etiologica-aplicacion-y-efecto-del-treatment142

Before and during the aforementioned clinical study, the clinical semiological characterization of the disease and its natural history was constructed, from before the presence of 1 to 11 tender points.

The natural history of the disease takes us to its earliest stages, expressed as fleeting episodes of paraesthesia, located in different anatomical musculoskeletal segments and, much less frequently, visceral. These episodes are repeated more frequently and for more extended periods, until they become persistent, in some cases. The progressive evolution of the disease expressed as fleeting episodes of

indefinable pain in different body segments, especially limbs. Such pains appear and cease without attributable cause, in more frequent and intense episodes and progressively more extended periods and migratory evolution. They are evolving until becoming generalized and progressively more intense and significantly affecting the functionality of the musculoskeletal system. Then, we sought to explain the physio-histo-pathological cause of fibromyofascial, non-inflammatory pain.

After the investigative stage of the clinical study that supports this report, a differential semiological-clinical study it has made between chronic diseases of the musculoskeletal system with known cause. Of which, it could conclude that the disease with semiological-clinical characteristics more similar to the syndrome studied is gout. Although such disease differs in its advanced evolution and in that it is a chronic process with acute highly inflammatory episodes. Unlike our case, in which its distinctive component is pain, which eventually accompanies edema. This syndrome understudy is never inflammatory.

According to the review of the literature currently available, we can theoretically affirm that the pain would have caused by the progressive impregnation of monosodium urate crystals in the extracellular matrix [1]. Especially in its basal laminae or fascia [2] corresponding to the neuromusculoskeletal apparatus and in the loose connective tissue, in a high amount that exceeds the phagocytic capacity of its migratory resident cells, as mast cells, macrophages, plasma cells, lymphocytes, and granulocytes, which would trigger the so-called monocyte-mediated pain cascade by neutrophils, and their cytokines [3-5]. Then, such excessive impregnation of crystals would yield due to their phagocytosis and subsequent cellular apoptosis, mediated by caspases, produced mainly by macrophage.

The relationship between the intensity or density of impregnation of crystals in the connective tissue and the phagocytic capacity of its mobile resident cells is what would give this disease its natural evolution, initially focal and self-limited, then recurrent and finally migratory, progressive and generalized.

The density of the impregnation of monosodium urate (insoluble acid salt) in the connective tissue is independent of the plasma levels of uric acid in each patient. Since the pain of patients treated with uricosuric or hypouricemic agents, decreases or ceases, regardless of plasma uric acid levels - the one that remains on average or slightly low ranges even after one year of treatment.

Painful edema of the legs or hands, which participates in some cases as part of the syndrome or as a single sign, could be caused by the stimulation of monosodium urate on mast cells of the tegumentary and lax conjunctive fascia, releasing autacoids, especially kallikrein-kinins, which have an intense local vasoactive effect.

The trigger points [6,7] or tend points observed in some patients, should be due to the accumulation of these irritating crystals in some neuromuscular tissue foci that, as we know, are tissues formed by very excitable cells. Causing, such persistent stimulus, the cytoelectroionic blockade of these, which could be explained as a blockage in the refractory period of the cell membrane [8].

With this theoretical basis, we selected people who had the diagnosis of fibromyalgia and had been treated, without success, with the therapeutic indications given by the American College of Rheumatologists, for a variable number of years.

Classic antigout drugs: Colchicine for the initial phase or acute episodes, and allopurinol as a permanent treatment in usual doses have determined as appropriate treatment.

During the study, the syndrome could be characterized in its signs, symptoms, and natural history. With that, we were able to investigate patients with a primary diagnosis of Fibromiofascial Syndrome, whom we included in the study.

The effect of this treatment was 100% absence of initial pain in all participants of a sample of 49 people, in a variable time from 2 weeks to 1 year, with a median of 2 months. 25% of the people in the sample had tolerable adverse reactions.

I continued treating patients after the aforementioned study in the general population of my city, with semiology and anamnesis of fibromyofascial syndrome, with and without previous diagnosis of fibromyalgia, and have reproduced its good results.

Conclusion

Today, fibromyalgia and myofascial syndrome patients have a specific therapeutic alternative that has been shown to really cure their disease and restore their healthy and normal life.

Based on the hypotheses and experiences reported here, researchers can now investigate the intimate, cytochemical, and genetic processes that produce this painful disease. Also, they can produce more effective medications and non-invasive biomedical procedures that allow measuring the density of monosodium urate in the human connective tissue.

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