

Inflammatory Fibromyalgia: A Diagnostic Dilemma

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ABSTRACT

Fibromyalgia in many ways remains a challenge for the medical profession. Patients are forced to give up work and become increasingly isolated, misunderstood and frustrated. The etiology and mechanisms of fibromyalgia are not well understood. One of the features distinguishing fibromyalgia from inflammatory conditions is the absence of elevated inflammatory markers. More recently, reports of inflammatory fibromyalgia are increasingly reported where patients have negative rheumatic serology but elevated inflammatory markers. They present a diagnostic dilemma and may receive unnecessary treatment. We aim to highlight association of inflammation and fibromyalgia and emphasize the importance of treating the patient and not focusing exclusively on abnormal laboratory results.

Keywords: Case report, Creatine phosphokinase, Disease-modifying antirheumatic drugs, Inflammatory fibromyalgia, Polymyositis/Dermatomyositis.

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INTRODUCTION

Fibromyalgia is not a true rheumatological condition, although it can coexist with many chronic pain conditions like rheumatoid arthritis (RA), lupus, and osteoarthritis. Prevalence¹ is approximately 2–8% of the population worldwide, and it is second only to arthritis. The female-to-male proportion is 6:1, making it more common in women. Presently, fibromyalgia is a fairly well-understood disease, and it is also termed centralized pain syndrome.² Diffused hyperalgesia and/or allodynia is an indicator of fibromyalgia, suggesting that these patients struggle with pain or sensory processing and not to any abnormality confined to the region where the patient is currently experiencing pain. They also show an elevated level of biomarkers associated with pain, including glutamate and substance P.

Herein we describe a case of a 38-year-old female patient experiencing diffuse musculoskeletal pain and elevated erythrocyte sedimentation rate (ESR) who was misdiagnosed with RA and did not respond to disease-modifying antirheumatic drugs (DMARDs). She had a picture of inflammatory arthritis with unrevealing rheumatic serology. Ultimately, the patient was diagnosed with fibromyalgia.

CASE DESCRIPTION

A 38-year-old female presented to the pain clinic with easy fatigue, weakness of legs, and bilateral heel pain for the past 6 months. Heel pain adversely affected her life, prevented her from sleeping, and had a negative effect on her emotions. Her 10-point visual analog scale (VAS) was 8/10 with activity and 3/10 at rest.

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Past history showed that the patient was being treated with high-dose disease-modifying drugs and nonsteroidal agents (indomethacin). There was no resolution of symptoms with these medications, instead became anorexic and lost weight. She underwent extensive autoimmune workup, which was negative for RA factor, anti-citrullinated peptide antibodies, antinuclear antibody, complete blood count, and C-reactive protein (CRP), except for elevated ESR (46).

Clinical examination revealed hypotension and hypoglycemia. There were no constitutional symptoms of rash, fever, morning stiffness, or bowel symptoms. Musculoskeletal examination showed hyperalgesia in calves and heel pain with no edema/synovitis. Her pain was less clearly localized to joints, but she had diffuse pain in her legs and heel with emotional problems and insomnia.

Due to protracted history and elevated inflammatory markers, we decided to further evaluate the patient.

The differential diagnosis now was:

- Fibromyalgia.
- Polymyositis (PM).
- Restless leg syndrome.
- Myofascial syndrome.

CLINICAL COURSE

The patient was tapered off immunosuppression. Repeat blood work was done to exclude PM. Creatine phosphokinase was 33 (within the normal range). Her bone densities were normal. The ultrasonographic examination of the foot showed mild plantar fasciitis in the left foot with no fat pad atrophy. The right foot was grossly normal.

We started her on neuropathic drugs (pregabalin), a short course of steroids. On the subsequent visit, after a 2-week follow-up, she had a 50% improvement in symptoms. Pain scores in the leg were almost 2/10 with activity, but her heel pain still persisted after standing for prolonged periods. Repeat blood work was done, which showed consistent elevation of ESR with CRP normal.

Marked improvement of symptoms with anticonvulsants and opioids goes in favor of centralized pain syndromes.

In spite of having raised inflammatory markers and unrevealing rheumatic serology, this patient was diagnosed as a case of fibromyalgia. Fibromyalgia management principles were discussed as it is a remitting, relapsing disease. Dietary and physical exercise was discussed.

DISCUSSION

Fibromyalgia, a form of chronic widespread pain disease, has a prevalence reported to be 2–8% of the population. This is not a true rheumatologic condition, although it can coexist with lupus. Fibromyalgia is extremely common with Female:Male of 6:1. Secondary fibromyalgia occurs in 10–30% of autoimmune diseases, although pain often waxes and wanes.³ It is often accompanied by fatigue, deep muscle pain, and stiffness, sensitivity to touch, cognitive disturbance, and multiple somatic symptoms. Although it can occur at any age but it is seen to be more common in middle-aged females.

There are a whole lot of factors that can cause these generalized body-wide symptoms; the most famous ones are psychological and environmental. It can also run in families having genetic relationships as well. Several studies have shown relationships between its development and dysfunctional genes controlling neurotransmitters, especially serotonin, dopamine, and norepinephrine.

Getting a diagnosis of fibromyalgia is not always easy. There is a lot of controversy surrounding fibromyalgia, whether it should be kept as a possibility or not. In 2016, fibromyalgia was declared not to be a disease of exclusion. So today, it is looked at in the context of biological, psychological, and social factors combined. A criterion based on tender spot evaluation was used in the past as diagnostic criteria—11 out of 16 tender points were considered positive for fibromyalgia, but this

criterion was disreputed as the patient felt pain everywhere. Nearly three-fourths quadrant of the body is in pain. Presently, widespread pain index (WPI) with scores ranging from 0 to 19 and symptom severity (SS) score with scores ranging from 0 to 12 are used with total scores combined ranging from 0 to 31.

The 2010 ACR diagnostic criteria for fibromyalgia:

- Widespread pain index (WPI) >7 and symptoms severity scale score >5.
- Symptoms have been present at a similar level for at least 3 months.
- The patient does not have a disorder that would otherwise explain the pain.

The SS scale score is the sum of the severity of the three symptoms, including fatigue, waking unrefreshed, and cognitive symptoms.

Fibromyalgia is now a fairly well-understood disease where the central nervous system takes inputs from peripheral regions—arms, shoulders, abdomen, and legs. The patient's brain processes pain differently from a normal person's. Like if we take an magnetic resonance imaging of a fibromyalgia patient and press his thumb under the scanner—the pro pain areas of the brain will light up like a Christmas tree. The brain actually amplifies pain. Higher scores mean more regions are getting activated. Fibromyalgia is also categorized under centralized pain syndromes.

There are many overlapping pain conditions like-inflammatory bowel disease, migraine, low back pain, and endometriosis which can present with hyperalgesia and are coexistent with fibromyalgia as well.

There is no specific test that is diagnostic of fibromyalgia. It is a disease of exclusion. One of the features distinguishing fibromyalgia from inflammatory conditions is the absence of elevated inflammatory markers. More recently, inflammatory fibromyalgia has also been described. These patients present a diagnostic dilemma and may receive unnecessary treatment. We aim to highlight the association between inflammation and fibromyalgia and emphasize the significance of treating the patient and not focusing exclusively on abnormal lab results.

Fibromyalgia should be recognized as a complex condition that requires a multidisciplinary approach⁴ with a combination of nonpharmacological and pharmacological treatment modalities tailored according to pain intensity, function, and associated features such as depression, fatigue, and sleep disturbance in discussion with the patient.

Motivation is certainly the key to treating these patients; they should improve their exercise and activity tolerance. Behavioral therapy can also be tried, such as cognitive restructuring and psychotherapy.

Few pharmacological therapies⁵ have been approved by the Food and Drug Administration (FDA) for the treatment of fibromyalgia.

- Pregabalin (first to get approval by FDA).
- Duloxetine.
- Milnacipram.

These drugs have also been recommended by European League Against Rheumatism for fibromyalgia with strong evidence (class 1A). An ideal combination would be one which addresses multiple symptoms at the same time with minimum side effects and drug interactions.

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