The Role of Tendinitis in Fibromyalgia Syndrome

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Fibromyalgia Syndrome (FS) is a common disease characterized by diffuse, widespread pain and multiple tender points. The syndrome has been subclassified as primary (PFS) and secondary (SFS) fibromyalgia. The aim of this study was to evaluate the role of common tendinitis (rotator cuff tendinitis, bicipital tendinitis, lateral epicondylitis, DeQuervain's tendinitis and pes anserinus tendinitis) in FS. Twenty female patients with PFS, 20 with SFS and 20 female controls, matched by age and body mass index, participated in the study. Existence of common tendinitis was evaluated with specific examination methods. Right and left rotator cuff tendinitis, pes anserinus tendinitis and left lateral epicondylitis were significantly more common in patients with PFS and SFS than in control subjects. As a result, considering the central hyperexcitability present in the fibromyalgia patients, concomitant pathologies such as tendinitis which lead to shoulder, arm, and leg pain must be evaluated. Follow up and therapy for the disease must be planned according to these factors which are not only probable symptoms of FS, but also leading causes for the occurrence and continuity of the pain in this disease.

Key Words: Fibromyalgia, tendinitis

INTRODUCTION

Fibromyalgia Syndrome (FS) is a common disease characterised by widespread musculoskeletal pain and tenderness on palpation of specific tendinomusculoskeletal sites, called "tender points". FS has been subclassified as primary (PFS) and secondary (SFS) fibromyalgia. Patients with PFS have diffuse, widespread pain and multiple tender points in the absence of underlying, causative, or significant concomitant condition. When these conditions are present FS is classified as SFS. Studies have shown that the clinical characteristics of FS in SFS patients are not significantly different from those of PFS patients. The most common characteristics of the syndrome are non-restorative sleep, tension-type headache, subjective soft tissue swelling, morning stiffness and paresthesias. Peripheral mechanism has been postulated as a possible pathophysiological theory for FS. This theory focused on localised ischemia due to disturbed microcirculation that causes muscle pain. To explain widespread pain at rest, a characteristic of FS, this theory invokes disturbed pain modulation in the central nervous system (CNS). The aim of this study was to evaluate the role of common tendinitis as a peripheral factor in primary and secondary FS. We also assessed the most common characteristics of fibromyalgia in our patients.

MATERIALS AND METHODS

Twenty female patients with PFS and 20 with SFS (due to type-2 diabetes mellitus (DM)) who had been referred to our outpatient clinic, along with 20 female controls matched by age and body mass index (BMI), participated in the study. None of the subjects had any previous history of trauma at tendon sites and all of them had right-hand predominance. All patients fulfilled the classification criteria for fibromyalgia proposed by the American College of Rheumatology (ACR). Initially, the demographic features of the patients were noted, along with clinical characteristics of FS such as non-restorative sleep, tension-type head-
ache, morning stiffness, subjective soft tissue swelling and paresthesias. Then all patients underwent detailed locomotor and systemic examination.

Tender point (TP) and control point (CP) examinations were performed with a Fischer's tissue compliancemeter which may be used as a pressure pain algometer.\(^5\)\(^\text{,}\)\(^7\) Eighteen TP and 4 CP (mid forearm and thumbnail-bilateral)\(^9\) were evaluated. The compliancemeter was applied to these specific points and the amount of pressure causing pain (pain pressure threshold - PPT) was recorded in kg/cm\(^2\). Points that were painful with less than 4 kg/cm\(^2\) pressure were accepted as tender points. The severity of FS was assessed with total myalgic score (TMS) and control point score (CPS). The sum of the PPTs of the 22 points (18TP and 4CP) was calculated as TMS (kg/cm\(^2\)) and the sum of the PPTs of the control points was recorded as CPS (kg/cm\(^2\))\(^6\)\(^,\)\(^7\).

Rotator cuff tendinitis (RCT), lateral epicondylitis (LE), bicipital tendinitis (BT), De Quervain's tendinitis (DQT), and pes anserinus tendinitis (PAT) were evaluated with specific clinical tests.\(^8\)\(^,\)\(^9\)

SPSS 10.0 for Windows was used for statistical analysis. Chi-square and one-way ANOVA tests were selected for analysis and post hoc analysis was performed with Bonferroni test. \(p\) values less than 0.05 were accepted as significant.

RESULTS

The mean age of the 20 females with PFS was 51.25 ± 8.82 years (range 34 - 70), of the 20 females with SFS was 55.65 ± 11.06 years (range 36 - 78) and of the 20 female controls was 51.40 ± 7.68 years (range 40 - 65). Demographic features of fibromyalgia and control groups, and mean disease durations are given in Table 1. There was no statistically significant difference between the 3 groups with respect to mean age, height, weight, BMI and duration of the disease (\(p>0.05\)). The mean DM duration was 9.8 ± 5.27 years and mean fasting blood glucose level was 193.5 ± 63.42 mg/dl in the SFS group.

There were no differences in any of the clinical parameters between the two FS groups (\(p>0.05\)). Non-restorative sleep, subjective joint swelling, morning stiffness and paresthesias were found more in the PFS and SFS groups than in the controls (\(p<0.001\)). However the incidence of tension-type headache was equal for all three groups (\(p>0.05\)). Right and left RCT, PAT and left LE were significantly more in the patients with PFS and SFS than in the control subjects (\(p\) values shown in Table 2). No statistical differences among the three groups were found for right and left DQT, BT and right LE (\(p>0.05\)). Number of tender points was higher (\(p<0.001, p<0.01\), and TMS and CPS were lower (\(p<0.001\)), in the PFS and SFS groups than in the control subjects. These clinical features and statistical analyses are shown in Table 2.

DISCUSSION

Pathophysiologic theories of FS can be divided into three groups based on the following proposed mechanisms. (1) Primarily central: This theory is based on the comorbidity of fibromyalgia with major depression, migraine, irritable bowel syndrome, chronic fatigue syndrome, panic disorders and the alpha electroencephalographic sleep anomaly. (2) Combination of central and peripheral mechanisms based on "central neuro-hormonal dysfunction". Decreased serotonin level, which may be triggered by non-specific stress-

| Table 1. Demographic Features and Mean Disease Durations of FS and Control Groups |
|-------------------------------|----------------|----------------|----------------|-----------------|----------------|
|                              | Age (year)    | Height (m)    | Weight (kg)   | BMI (kg/m\(^2\)) | Duration of FS (year) |
| PFS                           | 51.25 ± 8.82  | 1.60 ± 0.06   | 72.05 ± 10.71 | 28.09 ± 4.97    | 4.00 ± 2.73      |
| SFS (Type-2 DM)               | 55.65 ± 11.06 | 1.56 ± 0.07   | 73.55 ± 10.87 | 30.12 ± 4.43    | 4.45 ± 2.37      |
| Control                       | 51.40 ± 7.68  | 1.58 ± 0.05   | 75.50 ± 10.69 | 30.00 ± 4.39    | > 0.05           |
| \(p\)                         | > 0.05        | > 0.05        | > 0.05        | > 0.05          | > 0.05           |

FS, Fibromyalgia; PFS, Primary Fibromyalgia; SFS, Secondary Fibromyalgia; DM, Diabetes Mellitus; BMI, Body Mass Index.

from trauma, viral infection or mental stress, is one of the possible mechanisms. (3) Primarily peripheral: This theory focuses on localised ischaemia due to disturbed microcirculation that causes muscle pain. To explain the widespread pain at rest that is characteristic of FS, this theory invokes disturbed pain modulation in the CNS.5

One of the ACR criterion for the diagnosis of FS is the existence of “sensitive points”. While former data indicated pain only in these described points, recent studies have shown an increase in sensitivity throughout the body.10 It is stated in these recent studies that a central hyperexcitability exists in FS patients and that as a consequence the afferent input originated from the periphery is amplified and continued by the CNS.11-13 Sorensen, et al. measured the PPT in twelve patients with FS and twelve control subjects who did not have spontaneous pain in the anterior tibial (AT) muscle. They applied single and continuous electrical stimulation to the skin and assessed the PPT of the right AT muscle. They also augmented the pain in the left AT muscle by injecting sterile hypertonic saline, recorded the intensity and time of the saline-induced pain continuously via electronic visual analog scale and mapped the expansion of the pain. They concluded that the pain threshold in the FS patients was lower than in the controls. Both groups had been found to have the same thresholds for electrical stimulation through the skin. Lowering of pain threshold after consequent intramuscular injections had been found to be more profound in patients with FS and stated to be due to the dominance of temporal nociceptive summation in this group. They pointed out that the augmented pain resulting from the injection of hypertonic saline lasted longer and spread more. This was accepted to be a sign of central sensitization. They also stated that almost all of the patients had spontaneous pain in the shoulder.
area and that this was an indication of erroneous microcirculation and/or a defect in the energy metabolism. These alterations are not specific to FS, but they may be triggering factors of pain, especially in the existence of central hyperexcitability.  

Tendons such as the rotator cuff at the shoulder and the biceps include “critical zones” because of their vascular structures. Thus, microcirculatory defects and altered energy metabolisms occur in these regions in cases of tendinitis. In our study, we observed a high ratio of bilateral RCT, left LE and bilateral PAT in both primary and secondary FS patients. As mentioned before, these kinds of tendinitis can be the factors for initiation and maintenance of pain in primary and secondary fibromyalgia because of the central hyperexcitability found in this disease. Tendinitis also can be a symptom of fibromyalgia because of its coexistence with FS and its occurrence in both primary and secondary FS. For this reason, tendinitis requires careful assessment in the follow up and treatment of FS patients.

Another important finding in our study was that no patient or control subject had a previous history of trauma at tendon sites. In addition, although all of them had right-hand predominance, left LE was significantly more common in our fibromyalgia patients, whereas right LE was not different from the control subjects. Based on these findings regarding the role of tendinitis in FS we concluded that tendinitis is a symptom of fibromyalgia and seems to be a part of the disease nature, but not a concomitant condition.

Multiple theories about the pathogenesis of this disease dictate various therapy regimens. As the identification of the exact pathogenic factors is difficult, current therapeutic approaches seem to be inadequate. After four years of follow up, Ledingham, et al. pointed out a poor prognosis characterised by a high degree of functional impairment and persistence of symptoms. In conclusion, considering the central hyperexcitability present in the patients and the similar properties of tendinitis and fibromyalgia, follow up and therapy for the disease must be scheduled according to these factors which are not only probable symptoms of FS, but also leading causes for the occurrence and continuity of the pain in this disease.

REFERENCES


