

Introduction and historical review of the fibromyalgia syndrome

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Chapter 1

Introduction

The fibromyalgia syndrome is a form of nonarticular rheumatism characterized by chronic and diffuse musculoskeletal aching and stiffness accompanied by exaggerated tenderness at specific anatomical sites, known as tender points (1). The symptoms are modulated by certain factors e.g., weather, physical activity, physical or mental stress, and sleep quality. Other symptoms are fatigue and a disturbed sleep, not seldom accompanied by headache and symptoms of irritable bowel syndrome.

Since some or many of these findings may be secondary to various underlying disorders such as rheumatoid arthritis (RA), trauma, significant osteoarthritis (OA), infection and hypothyroidism, the term primary fibromyalgia was employed to describe a distinct entity. The condition was called primary when an underlying related condition was absent.

Between 1970 and 1990 several sets of criteria for the diagnosis of fibromyalgia have been proposed (Yunus, Campbell, Smythe) (1,2,3), summarized in table 1. Wolfe (4,5,6) attempted to summarize these movements through the years and put forward his own ideas and findings concerning this patient group. Bengtsson et al. (7) already did the same in describing the clinical and laboratory findings of her patient group. They also compared this group with a rheumatoid arthritis group on the different items. Their fibromyalgia group expressed a more intense feeling of illness than did the rheumatoid arthritis patients, although on objective measures one would expect the opposite.

The Multicenter Criteria Committee in defining the American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia (8) proposed to abolish the distinction between primary and secondary-concomitant fibromyalgia at the level of diagnosis, because the two were essentially indistinguishable with the study variables used, and the proposed criteria worked equally well in both groups. These ACR 1990 criteria are now worldwide acknowledged and comprise 1) widespread pain in combination with 2) tenderness at 11 or more of 18 specific tender point sites (table 1). It should be noticed that these criteria are designed for classification criteria and not meant for diagnostic purposes.

Table 1
 CRITERIA FOR DIAGNOSIS OF PRIMARY FIBROMYALGIA SYNDROME
 (YUNUS, 1981)

1. Obligatory criteria	A	Presence of generalized aches and pains or prominent stiffness, involving 3 or more anatomic sites, for at least 3 months
	B	Absence of secondary causes, e.g., traumatic (due to repetitive or more direct trauma), other rheumatic (including degenerative), infective, endocrine or malignant, with normal laboratory tests (CBC, ESR, rheumatoid factor, ANA, muscle enzymes) and röntgenograms
2. Major Criteria		Presence of at least five typical and consistent tender points.
3. Minor Criteria	A	Modulation of symptoms by physical activity
	B	Modulation of symptoms by weather factors
	C	Aggravation of symptoms by anxiety or stress
	D	Poor sleep
	E	General fatigue or tiredness
	F	Anxiety
	G	Chronic headache
	H	Irritable bowel syndrome
	I	Subjective swelling
	J	Numbness

All primary fibromyalgia patients must satisfy the 2 obligatory criteria, as well as either the major criterion plus at least 3 minor criteria. If the patient has only 3 or 4 tender points, then 5 minor criteria are suggested.

CRITERIA OF CAMPBELL (1983)

1. A questionnaire to define possible fibromyalgia.

Items on this questionnaire:

- 1 Exercise makes me feel better.
- 2 I sleep well at night.
- 3 I feel well rested when I get up in the morning.
- 4 I wake up frequently at night.
- 5 I tire easily.
- 6 I am too tired during the day to do what I want to do.
- 7 I have pain in neck and shoulders.
- 8 I am stiff in the morning.
- 9 I have pain in my muscles and joints.
- 10 I ache in the morning.
- 11 Pain wakes me up at night.
- 12 Heat (such as heating pads) helps my pain.
- 13 My pain is affected by weather.
- 14 I have more pain when I am emotionally upset.
- 15 My pain is worsened by noise.

Patients are asked to answer these questions on a 4-point scale: Never, Sometimes, Often, and Almost Always. The diagnosis of possible fibromyalgia required: 1) Questions 7 or 9: Often or Almost Always + 2) Questions 8 or 10: Often or Almost Always + 3) Question 3: Never or Sometimes + 4) Questions 1, 12-15 (any 2): Often or Almost Always.

2. Objective tenderness at dolorimeter pressures of less than 4 kg/1.54 cm² in at least 12 of 17 tender point localisations.

CRITERIA OF SMYTHE (1980)

- 1. Widespread aching of more than 3 months' duration.
- 2. Local tenderness at 12 of 14 specified sites.
- 3. Skin roll tenderness over the scapular region.
- 4. Disturbed sleep, with morning fatigue and stiffness.
- 5. Normal laboratory findings.

CRITERIA OF WOLFE ET AL. (1990), ACR CRITERIA

1. History of widespread pain.
Pain is considered widespread when all of the following are present: pain in the left side of the body, pain in the right side of the body, pain above the waist and pain below the waist. In addition axial skeletal pain must be present.

2. Pain in 11 of 18 tender point sites on digital palpation.
These 18 sites (9 bilateral) are:
 - Occiput, bilateral, at the suboccipital muscle insertions.

 - Low cervical, bilateral, at the anterior aspects of the intertransverse spaces at C5-C7.

 - Trapezius, bilateral, at the midpoint of the upper border.

 - Supraspinatus, bilateral, at origins above the scapula spine near the medial border.

 - Second rib, bilateral, at the second costochondral junctions, just lateral to the junctions on upper surfaces.

 - Lateral epicondyle, bilateral, 2 cm distal to the epicondyles.

 - Gluteal, bilateral, in upper outer quadrants of buttocks in anterior fold muscle.

 - Greater trochanter, bilateral, posterior to the trochanteric prominence.

 - Knee, bilateral, at the medial fat pad proximal to the joint line.

Historical review of previous designations

Various forms of nonarticular rheumatism under a variety of names have been described, and particularly in the last century.

Gowers in 1904 (9), in "A Lecture on Lumbago: It's Lessons And Analogues", was the first to use the general term "fibrositis" to indicate the then generally held belief that these varied clinical conditions were the result of a proliferation or inflammation of subcutaneous and muscular fibrous tissue. Gowers based the term "fibrositis" on the marked tenderness he had found to be associated with regional pain syndromes. He did not describe diffuse body pain, only asymmetrical regional pain syndromes. He gave great importance to the distinction between spontaneous pain and sensitiveness. The sensitive sites were asymptomatic at rest and produced distress only when punched, or when muscle action caused tension in hypersensitive tendinous structures connecting tissue to bone (10). Gowers also described the concepts of pain amplification, posttraumatic syndromes, lack of inflammatory products, failure of salicylates, help by distraction and gentle manipulation, counterirritation and cocaine injections. He even mentioned sleep disturbance and exhaustion as consequences of pain. The term "fibrositis" has persisted in the literature despite the failure of several authors to show reproducible and consistent changes in structure of these connective tissues. Simons gave an extended historical review on muscle pain syndromes (11,12).

Depending upon which characteristics the authors wished to emphasize, many names have been applied to these painful clinical conditions: fibrositis, fibrositis syndrome, interstitial myofibrositis, Muskelschwiele (German for muscle callus or welt), myogelosen (muscle gelling or myogeloses), Muskelhärten (muscle hardening), muscular rheumatism, non-articular rheumatism, or Weichteilrheumatismus (soft-tissue rheumatism), myofascial (pain) syndrome, myofascitis, or trigger points, and myalgia or myalgic spots. The most extensive literature was published in Germany in the 19th century. After the turn of the century the original fibrositis literature appeared in Great Britain. Approaching mid-century a distinctive American contribution appeared.

Fibrositis is the one term most used in all English language literature to identify this painful condition of muscles. From the articles by Simons we can learn that the thoughts and beliefs on pathogenesis evolved from an interstitial myositis, together with a similar inflammatory process in the connective tissue of adjacent fat, fasciae, and especially nerves, to inflammation of fibrous tissue of the muscle. Bacterial infection was presumed to cause the connective tissue hyperplasia.

As mentioned before Gowers introduced in 1904 the term "fibrositis", in a paper on lumbago that included muscular fibrositis of the arm. He characterized muscular rheumatism in general (and lumbago specifically) as an inflammation of fibrous tissue of the muscle. He hypothesized that the inflammation in lumbago originated behind the sacrum and spread to involve the fibrous sheath of the sciatic nerve to produce the pain distribution. He did not consider it an ordinary inflammation because of the absence of regular "inflammatory products". However the term "fibrositis" had been established for many years to come. Gowers deliberately chose not to use the word "myalgia" for the affection he wanted to describe, because its analogy with "neuralgia" suggested unexcited and spontaneous pain. Aggravating or precipitating factors included exposure to cold, and acute and chronic muscular overstrain. Gowers did not mention any palpable findings, but attributed the pain to hypersensitivity of the muscle spindle.

Supposed clinical pathological correlations

Simons mentioned the description of patchy, inflammatory changes in "white fibrous tissue" biopsies from areas of nodular soft tissue in patients complaining of "chronic rheumatism" by one of the earlier investigators. As a specific diagnosis "fibrositis" was often argued. It became increasingly apparent to multiple observers that the finding of soft tissue nodules depended on the desire of the examiner to appreciate their presence. Such nodules were often absent in patients with chronic rheumatic complaints, and similar subtle soft tissue nodules were frequently present in normal or symptom-free individuals.

Early in the twentieth century chronic articular rheumatism (rheumatoid arthritis) was

renamed as articular fibrositis, in distinction to myo-fibrositis (muscular rheumatism), and neuro-fibrositis or fibrositis of the nerve sheaths associated with pain in the distribution of the nerve. Myo-fibrositis was then defined as an "acute or chronic inflammatory change in the interstitial fibrous tissues of a striated or voluntary muscle, the parenchymatous elements of which are only secondarily implicated". This was considered the muscular manifestation of a process that applied equally to the connective tissue of fat, fascia and neural sheaths. Bacterial infection, familial predisposition, occupation (increased incidence with more physical labour), exposure to drafts or chilling and exercise beyond tolerance were considered significant predisposing factors.

A few years later, again according to Simons, an American publication emerged where the symptoms of muscular pain, stiffness and exhaustion were associated only occasionally with palpable nodules. Laboratory blood findings were normal. Muscle biopsies showed fibroblastic activity and mild degenerative changes in chronic cases. Other investigators produced pain by injecting hypertonic saline into deep structures of themselves and other volunteers and observed that the experienced pain was referred, nearly always distally, to deep tissues in a vaguely demarcated distribution determined chiefly by the segmental nerve supply. The referred pain might also be accompanied by referred tenderness and by muscle spasm. They noted that segmental reference of pain was characteristic of deep pain origin and not seen in cutaneous pain, and that deep somatic structures gave rise to segmental referred pain. They also noted that the referred sensations might have qualities of numbness and tingling as well as aching. These findings can be related to the concept of tender points. During the years more papers with different biopsy findings were written, possibly as a result of the frustration of no confirmatory biopsies in the studies earlier performed. Simons concluded that these either obtained negative results or found other diagnosable conditions. The doubts as to the anatomical validity of the fibrositis concept, raised by these papers, were even more intensified, when it was found that the pressure sensitive ("trigger points") palpable nodules associated with low back pain symptoms, were located within the distribution of pink fat. Postmortem studies showed a common pattern of fat herniation and pedunculation through adjacent fascial planes.

At the same time substantial evidence was presented for a neurogenic mechanism in at least some cases. A controlled EMG-study on patients with proven herniated disease showed spontaneous isolated motor unit action potentials firing at 8-12 per second in tender muscle, but not in adjacent non-tender muscle. Pressure on the tender muscle markedly increased EMG-activity, but pressure on adjacent non-tender muscle had no effect. These findings meant that nerve root pressure could induce a condition of hyperirritability of the muscle that involved spinal anterior horn cells and gave the appearance of typical fibrositic lesions. Some palpable nodules were attributed to muscle contraction (spasm), which contributed to the pain but did not fully account for it. The sustained contraction of part of a muscle was supposed to lead to increased

irritability and pain sensitivity and eventually to pathological changes.

In his journey through the years Simons mentions a publication of just after World War II where a very clear and detailed presentation of typical findings in fibrositis is presented, e.g. myalgic spots, in specific muscle throughout the body. The most common associations included headache, shoulder pain, forearm and hand pain, pleurodynia, sciatic pain and a painful knee. Four features were considered evidence of reflex activity originating from the myalgic spot itself: widespread referred pain, deep hyperalgesia in that same area, edema of involved tissues, and stiffness or wasting of muscles. Reflex effects were therefore implicated in both the origin and clinical expression of the myalgic lesions of fibrositis.

Still, there was no agreement on which pathophysiological findings really were related to the fibrositis syndrome. The concept that the palpable findings and the pain were caused by muscle spasm was not in concordance with the finding in one patient that severe fibrositic pain induced by sustained activity was quite independent of the rate of motor unit-activity.

In the review of Simons a description of the fibrositis syndrome emerged in the late sixties, and included four essential features: exquisite point tenderness of the muscle, a palpable "rope" in the muscle, increased dermographia, and reduction of pain by ethyl chloride spray. At this time, after studies with EMG, it was concluded that the "rope" was not due to muscle contraction, e.g. spasm, but must have an other cause, like localized edema.

Fibromyalgia: a clinical entity?

Actually Smythe was one of the first of the new generation who looked closer in to the concept of fibrositis or fibromyalgia. Another group, under leadership of Simons and Travell, was still most interested in the myofascial pain syndrome. These two conditions are very hard to discriminate, and looking at the published papers, in particular those before 1970, it is often very hard to tell which condition is under consideration. In 1989 there was a first serious effort to bring more clearness with regard to those two overlapping syndromes, by means of an international symposium on myofascial pain and fibromyalgia in Minneapolis, USA. Here a lot of questions were raised, and only a few answers were obtained. At the second world congress, three years later in 1992 in Copenhagen, Denmark, an official document was made up at the end of the congress and is known as "The Copenhagen Declaration" on fibromyalgia. In this document the results of a consensus conference held by several international experts on fibromyalgia are summarized. During this consensus conference several questions regarding the different aspects of the fibromyalgia syndrome are addressed.

Myofascial pain syndrome is characterized by trigger points in muscle. A trigger point is defined (by Travell) as circumscribed tenderness, a localized twitch or fasciculation on stimulation by pressing or pinching that portion of the muscle which contains the trigger area, and referred pain produced by pressure on the trigger point (see chapter 3).

From 1970 onwards there has been an increasing interest in the concept of fibromyalgia, concluding in numerous papers on different topics and implications of the fibromyalgia syndrome. The reason we prefer to speak of fibromyalgia, and not fibrositis, is the fact that there has never been substantial proof of an infection or inflammation of fibrous tissue. Therefore the term fibrositis is confusing and not justified.

A consensus about the existence of the fibromyalgia syndrome has not been reached, and there is still lot of discussion on this topic known, but it is increasingly accepted that this syndrome is a definable clinical entity, of sufficient uniformity to be diagnosable by clinical criteria. The discussion is more and more focused on the possible peripheral or more central origin of the pain. It seems the central theory gains the most favourable support (see chapter 2).

However, most physicians do agree they see these patients in their practices with these complaints, as fit in with our diagnosis fibromyalgia. The problem of this group of patients is to find an adequate treatment.

During the last years a number of articles (editorials, reviews) have been published in leading journals in rheumatology or general internal medicine or in pain management. An example of this is an editorial written by Mufson and Regestein (13). They favour the concept of a generalized pain-modulation disorder. They distinguish a primary fibromyalgia syndrome, a fibromyalgia syndrome with comorbid psychiatric disorder

and thirdly a psychiatric disorder with fibromyalgia-like symptoms. The background of these authors is psychiatry. Regarding treatment possibilities the authors state that a firm therapeutic alliance is essential. The patient should be made an active participant in the treatment plan. A model of rehabilitation in which active rehabilitation with patient participation and physical exercise is pursued, rather than a passive curative approach, will help the patient to become a partner in treatment and this will linger the frustration and anger that often arises when the patient expects the doctor, or other health professionals, to render immediate cure. We can certainly support this point of view.

In a review article by Cohen and Quintner (14) the fibromyalgia syndrome is addressed as a problem of tautology. They criticize the concept in which the diagnostic criteria convey no pathophysiological insight and where these criteria have been, in the authors' opinion, validated via a circular argument in which the evidence on which the construct (concept) is based is taken as proof of its veracity. The authors suggest an alternative approach to the clinical presentation of the fibromyalgia syndrome, namely via secondary hyperalgesia. Cohen published another article on this last subject together with Arroyo (15).

Inappropriate stress coping

Another review was written by Lorentzen (16), from the department of rheumatology of the university of Copenhagen, Denmark. The title is stimulating to the readers "Fibromyalgia : a clinical challenge". Point of view of this author is that fibromyalgia is not a disease entity, but the symptoms often reported by fibromyalgia patients supposedly reflect difficulties in coping with various types of environmental stress. Lorentzen sees this as the key in the process and therefore that identification of these environmental factors and subsequently early intervention should have high priority. The experienced stress may lead to sleep disturbances, fatigue and a low level of physical activity and fitness. This again may lead to muscle pain and tenderness. The syndrome becomes chronic because of the vicious circle one ends up in. Lorentzen makes some critical remarks on the concept of fibromyalgia. Fibromyalgia syndrome contains an unusually large and heterogenous number of symptoms and this makes it very difficult to assume a common pathogenetic factor. Also the high percentage of women overall and especially between 40 and 50 years old in the western countries finds Lorentzen remarkable. The examination of tender points is also disputable, when one exerts too much pressure on palpation everyone can become a fibromyalgia patient. The nature of the heterogeneous symptoms in fibromyalgia could indicate a psychosomatic component. Lorentzen makes a comparison between fibromyalgia syndrome and the major epidemic of localized fibromyalgia in Australia in 1980. The epidemic was ended when a jury rejected compensation for a patient (see chapter 8). It is very difficult to assess the severity of the syndrome, and also the estimation of the

degree of work disability. Furthermore he states that every effort should be made to counteract the patients' disability and working incapacity.

A non-disease?

A critical sound was earlier made by Hadler (17). The main topics of his critics are that the name fibrositis implies certain pathophysiologic insights which are not valid and proven. Following this, the patients may get the believe that they have this illness with the suggested patho-anatomic derangement and this, Hadler believes, is in many cases counterproductive towards relieve of symptoms and recovery. Hadler said that labelling of a non-disease can cause patients to perceive themselves as ill, and he illustrates this with a number of references. However he acknowledges the existence of patients with these fibromyalgic symptoms, and apart from calling them a "name", he manages these patients in the same way as the other colleagues do, with attention to physical fitness, repeated reassurance as to the absence of progressive damage, interventions to decrease psychologic stresses when possible.

A very intriguing title emerged in an article in 1990, written by three Swedish authors of the department of Rheumatology of the university hospital of Lund (18): "Does primary fibromyalgia exist?" The authors re-examined 21 of 25 consecutive patients that were diagnosed with fibromyalgia, during a five-year period in a tertiary care day-ward for pain syndromes. Fifteen of these patients fulfilled criteria for fibromyalgia, but all patients had either psychiatric disturbance or thyroid dysfunction. Four patients that were not seen for follow up developed other diseases, two neurological disease, one rheumatoid arthritis and one hypothyroidism. Six patients were not classified as fibromyalgia on follow-up, but also had other diagnoses. The authors conclude from their results that none of the 25 patients, earlier on diagnosed as (primary) fibromyalgia, had at follow-up primary fibromyalgia. They hypothesize that it could be possible that a day ward specializing in pain syndromes does not have a single case of primary fibromyalgia during a 5-year period, and also they suggest that an underlying disease could be neglected by accepting primary fibromyalgia as a separate entity. In their study most patients were occupied as manual workers, mostly cleaners, and this could indicate that occupational load could be important for the development of symptoms. Actually none of the patients had returned to work. This article brings forward other outcomes and conclusions than most other studies. What can we make of it? The distinction between primary and secondary fibromyalgia is no longer made, although any treatable concomitant disease should be treated effectively. The patient group described in the article is probably a very selected group, on a tertiary care day-ward, where a great deal of psychiatric symptoms, e.g. diseases, could be expected.

Chronic pain syndrome

In 1988 appeared a review article by McCain and Scudds (19). The authors presented a classification model of different chronic musculoskeletal pain syndromes and they hoped this would be a starting point for epidemiological studies outlining the similarities and differences between these clinically observable and different musculoskeletal syndromes. Although the authors tried to identify homogeneous patient's groups, they also stated that there are patients that might fit more than one of the descriptions of the different disorders. Visualizing this overlapping they designed the Venn diagram.

In 1991 a review article appeared, divided in two parts, which aimed at an understanding of the fibromyalgia syndrome. First part concerned medical and pathophysiological aspects and the second part concerned more the psychological and phenomenological aspects of the fibromyalgia syndrome (20,21).

Still earlier, in 1986, Bennett (22) published an editorial "Fibrositis: Evolution of an enigma". He pointed out that there was at that time a widespread acceptance of fibrositis by North American physicians, and that it became recognized as being one of the most common rheumatic complaints with a clinical prevalence between 6 and 20%. Furthermore he emphasized the importance of the differentiation between general fibrositis (fibromyalgia) and local fibrositis (myofascial pain). He commented on several reports on possible pathogenetic mechanisms published earlier. At that time Bennett saw a crucial role in the stage 4 sleep disturbance and he created an etiological paradigm for fibrositis (fibromyalgia). He hypothesized that many initiating factors, like joint pain or trauma or even the use of caffeine and alcohol, could lead to a final common pathway leading to the induction of a stage 4 sleep anomaly. Fibromyalgia is a syndrome, rather than a disease entity, but it can be recognised as a clinical entity.

Not only in the English language papers on fibromyalgia are found. Houvenagel wrote an editorial (23) giving an overview concerning different aspects and theories of fibromyalgia. In his references he mentioned the well-known Anglo-American publications next to a few French publications. The name used in earlier years in the french areas is "Le syndrome polyalgique idiopathique diffus". In the late eighties the name fibromyalgia is also emerging in the French publications.

To emphasize the current interest in the fibromyalgia syndrome one could find several journal issues which are totally filled with articles on fibromyalgia. Giving fibromyalgia and its consequences in daily life a place is extremely difficult. Following the WHO classification from disease to impairment to disability and finally handicap is one line that is very difficult to follow in fibromyalgia. In the real sense of the word there is no disease, but there is pain (impairment) which leads to disabilities and many fibromyalgia patients surely feel handicapped. The sequence of impairment, disability and handicap is well known in rehabilitation medicine and treatment programs try to interfere as early as possible in the process of this triad. To

measure disability in fibromyalgia, however, is a very difficult task (see also chapter 8).

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