



*Dynamic Chiropractic* – February 27, 1995, Vol. 13, Issue 05

## **Fibromyalgia Syndrome Arising de novo from Trauma**

By Arthur Croft, DC, MS, MPH, FACO

Fibromyalgia syndrome (FS) is a chronic disorder involving not regions but the entire body, both above and below the waist and on both sides. It is much more prevalent in females and has generally not been associated with trauma. It has, however, been associated with more than 46 different infectious, metabolic, neurologic, and neoplastic diseases.<sup>1</sup> In such cases it is referred to as "secondary fibromyalgia syndrome." When free-standing it is usually referred to as "primary fibromyalgia syndrome" or simply "fibromyalgia."

Patients with FS have a number of complaints in common and these are listed in Table 1, which is from Wolfe<sup>2</sup> and is compiled from the American College of Rheumatology (ACR) 1990 criteria study.<sup>3</sup>

**Table 1: Characteristics of FS**

<b>Symptom</b>	<b>Percent of patients</b>
1. Widespread pain	97.6
2. Tenderness (11 of 18 tender points)	90.1
3. Fatigue	81.4
4. Morning stiffness (>15 min.)	77.0
5. Sleep disturbance	74.6
6. Paresthesias	62.8
7. Headache	52.8
8. Anxiety	47.8
9. Dysmenorrheic history	40.6
10. Sicca symptoms	35.8
11. Prior depression	31.5
12. Irritable bowel syndrome	29.6
13. Urinary urgency	26.3
14. Raynaud's phenomenon	16.7

The ACR also established 18 "tender points" which are commonly found in these patients. To qualify as having FS a patient must have the following:

1. widespread pain (above and below the waist and on both sides of the body) for at least three months duration;
2. axial spine pain (cervical, chest, thoracic or lumbar);
3. at least 11 of 18 "tender points";
4. at least three of the following:

- a) fatigue*
- b) sleep disturbance*
- c) anxiety*
- d) irritable bowel syndrome*
- e) headache*
- f) paresthesiae*

It is, of course, difficult sometimes to decide just what it is that constitutes fatigue. And what level of anxiety is important? How often should headaches occur? Does one per week qualify? The vague and nebulous nature of these criteria has raised a fair amount of criticism from some authorities. On the other hand, there is a strong association between poor sleep and increased symptom expression, and Romano<sup>4</sup> has recently correlated irritable bowel syndrome with FS rather definitively.

Several authors have noted that patients with FS tend to be rather asthenic. Mengshoel et al.,<sup>5</sup> found FS patients to fatigue faster than controls. Bennet<sup>6</sup> feels that the weight of evidence supports the theory of the muscle as the end organ responsible for FS. Unfit muscles are more likely to be injured. He also speculates on the role of sleep disturbance suggesting that growth hormone is decreased and this may also delay the healing process. With regard to the sleep disturbance, a number of investigators have suggested the possibility of a neurohormonal mechanism<sup>7</sup> which might be responsible. Years ago, physicians began to notice that patients with FS complained of poor sleep. Investigation of this uncovered the fact that most of these patients have alpha wave intrusion into their stage IV delta wave sleep.<sup>8</sup> In essence, their quality sleep is interrupted by a very shallow and non-restorative sleep. They typically awaken and feel unrested. It was also noted by using a visual analog pain scale that their pain was inversely proportional to their perceived quality of sleep (i.e., they had more pain on the days following nights of poor quality sleep).

Serotonin is known to modulate pain and play an important role in the regulation of deep sleep. If serotonin is deficient in FS patients, it would explain these sleep disturbances and possibly also their increased sensitivity to pain.<sup>7</sup> In a recent small study, serotonin was found to be significantly lower in FS patients.<sup>9</sup> This may be due in part to excessive platelet re-uptake.<sup>7</sup>

Going on this information, another group theorized that by giving supplemental tryptophan, a precursor of serotonin, they might improve sleep and decrease the pain.<sup>10</sup> In the end they did measure an improvement

in sleep but also an increase in pain. However, this study was repeated by Caruso et al.,<sup>11</sup> who showed improvement in all clinical parameters with the administration of 5-hydroxytryptophan. One wonders if the effects will last. Experience with migraine headaches has shown that tryptophan is effective for only 4-6 weeks.

Another important finding is that substance P is a key player in FS syndrome. It normally has a dampening effect on sensory nerve discharge in the presence of high concentration of serotonin. When serotonin is depleted, it does not exert this control. Substance P has been found to be elevated in the CNS of FS patients.<sup>12</sup> There is also evidence that hyperparathyroidism<sup>13</sup> and hypothyroidism may play significant parts in FS.<sup>14,15</sup> In these cases, however, FS would be described as secondary FS.

Recently, several authors have begun to cite trauma as an etiological factor in the development of fibromyalgia<sup>16-18</sup>. In reality, what we see clinically ranges from the de novo appearance of the disorder to traumatic aggravation of pre-existing fibromyalgia. In the latter case, patients who may have been previously symptomatic yet functional may become dysfunctional following their injury. It is also likely that complex interrelationships exist between endocrine, neurological, and musculoskeletal systems in such cases that virtually defy precise understanding from a pathophysiologic standpoint. It is also likely that patients predisposed to developing fibromyalgia may, after an injury, suffer not only from FS but from myofascitis, thoracic outlet syndrome, or other conditions as well. Further research is needed to investigate the possibility that some cases of FS arise directly from trauma. I have recently reported four cases in which this appears to have occurred.<sup>19</sup>

### *References*

1. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed (DSM-III). American Psychiatric Association, Washington, D.C., 1980.
2. Wolfe F. Diagnosis of fibromyalgia. *J Musculoskel Med* 53-69, 1990.
3. Wolfe F, Smythe HA, Yarnus MB et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the multicenter committee. *Arthritis Rheum* 33: 160-172, 1990.

4. Romano TJ. Concurrent fibromyalgia and irritable bowel syndrome. *Pain Pract* 3(1): 4-7, 1991.
5. Mengskoel AM, Ferre A, Komhnaes HB. Muscle strength and aerobic capacity in primary fibromyalgia. *Clin Exper Rheum* 8: 475-479, 1990.
6. Bennet RM. Muscle physiology and cold reactivity in fibromyalgia syndrome. *Rheum Dis Clin N Amer* 15(1): 135-147, 1989.
7. Russell IJ. Neurohormonal aspects of fibromyalgia syndrome. *Rheum Dis Clin N Amer* 15(1): 149-167, 1989.
8. Moldofsky H. Rheumatic pain modulation syndrome. The interrelationship between sleep, central nervous system serotonin, and pain. In: Critchley M, Friedman M, Gorini S, et al., eds. *Advances in Neurology*, vol 33. New York: Raven Press, 1982, pp 51-57.
9. Russell IJ, Bowden CL, Michalek J, et al. Imipramine receptor density on platelets of patients with fibrositis syndrome: correlation with disease severity and response to therapy. *Arthritis Rheum* 30: 563, 1987.
10. Moldofsky H, Warsh JJ. Plasma tryptophan and musculoskeletal pain in non-articular rheumatism ("fibrositis syndrome"). *Pain* 5: 65-71, 1978.

11. Caruso I, Puttini PS, Cazzola M, Azzolini V. Double-blind study of 5-hydroxytryptophan vs. placebo in the treatment of primary fibromyalgia syndrome. *J Intl Med Res* 18: 201-209, 1990/
12. Vaeroy H, Helle R, Forre O, et al. Elevated CSF levels of substance P and high incidence of Raynaud's phenomenon in patients with fibromyalgia: new features for diagnosis. *Pain* 32: 21-26, 1988.
13. Beetham WP, Jr. Diagnosis and management of fibrositis syndrome and psychogenic rheumatism. *Med Clin N Amer* 63: 433-439, 1979.
14. Wilke WS, Sheeler LR, Makarowki WS. Hypothyroidism presenting symptoms of fibrositis. *J Rheumatol* 8: 626-631, 1987.
15. Forslind K, Frediksson E, Nived O. Does primary fibromyalgia exist? *Br J Rheum* 29: 368-370, 1990.
16. Moldofsky H, Wong MTH, Lue FA. Litigation, sleep, symptoms, and disabilities in post-accident pain (fibromyalgia). *Journal of Rheumatology* 20(11): 1935-1940, 1993.
17. Zimmermann M. Pathophysiological mechanism of fibromyalgia. *Clinical Journal of Pain* 7(S1): S8-S15, 1991.
18. Culclasure TF, Enzenauer RJ, West SG. Post-traumatic stress disorder presenting as fibromyalgia. *American Journal of Medicine* 94(5): 548-549, 1993.

19. Croft AC. Post-traumatic fibromyalgia -- case reports. Submitted.

*Arthur Croft, DC, MS, FACO*

*San Diego, California*

---

Click [here](#) for more information about Arthur Croft, DC, MS, MPH, FACO.



Page printed from:

[http://www.chiroweb.com/mpacms/dc/article.php?id=40097&no\\_paginate=true&p\\_friendly=true&no\\_b=true](http://www.chiroweb.com/mpacms/dc/article.php?id=40097&no_paginate=true&p_friendly=true&no_b=true)