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A GENERAL PRACTICE APPROACH TO MANAGEMENT OF CHRONIC WIDESPREAD MUSCULOSKELETAL PAIN AND FIBROMYALGIA

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- **Fibromyalgia and chronic widespread pain are common disorders in primary care that are often a source of frustration for both the patient and the physician**
- **There is increasing evidence that the pain in fibromyalgia and chronic widespread pain are caused by peripheral factors interacting with an altered central nervous system (CNS) processing**
- **The general practitioner (GP) should adopt a strategic approach to management of patients with chronic widespread pain and fibromyalgia based on current knowledge of pathogenesis**

INTRODUCTION

General practitioners (GPs) frequently see patients with musculoskeletal pain. Acute and localised pain can often be related to a specific cause, but this is rarely the case when pain is more long-standing and widespread. Instead of being a signal of tissue damage, pain has then in itself become the disorder. This is also supported in the widely used and accepted definition of pain proposed by the International Association for the Study of Pain (see p.125 this volume).

The situation is often a source of frustration for both the affected individual and the physician. One reason for this may be the old dualistic distinction between body and soul that still has implications in modern medicine. Even today, patients suffering from pain without an obvious organic cause are often considered as having a psychological cause for their pain. Recent research in pain physiology and stress has revealed how mechanisms connecting 'body' and 'soul' make the system react as a united whole. This knowledge should be put into practice in a structured treatment and rehabilitation programme. This article focuses on the GP's management of patients with established chronic widespread pain and fibromyalgia or at risk of developing the disorders; it also gives a short background to definitions, epidemiology and pathogenesis.

CHRONIC WIDESPREAD PAIN AND FIBROMYALGIA

Fibromyalgia (FM) was introduced as a diagnostic concept by Yunus et al in 1981,¹ and gained an official status in 1990, when the American College of Rheumatology (ACR) published their still accepted criteria.² The diagnosis of FM is based on a history of chronic widespread pain (CWP) and the presence of at least 11 out of 18 specified 'tender points' (Table 1).

Since 1990 several researchers, including some of the authors behind the ACR 1990 criteria, have stated that FM should be regarded not as a distinct entity^{3,4} but rather as one end of a continuous spectrum.⁵

TABLE 1. The American College of Rheumatology (ACR) 1990 criteria for the classification of fibromyalgia.²

1. History of widespread pain

Definition: 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 (cervical spine or anterior chest or thoracic spine or low back) must be present. In this definition, shoulder and buttock pain is considered as pain for each involved side. 'Low back' pain is considered lower segment pain.

2. Pain in 11 of 18 tender point sites on digital palpation

Definition: Pain, on digital palpation, must be present in at least 11 of the following 18 tender point sites:

- *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 the origins, above the scapulae 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 outer quadrants of buttocks in anterior fold of muscle
- *Greater trochanter:* bilateral, posterior to the trochanteric prominence
- *Knee:* bilateral, at the medial fat pad proximal to the joint line.

Digital palpation should be performed with an approximate force of 4 kg. For a tender point to be considered 'positive' the subject must state that the palpation was painful. 'Tender' is not to be considered 'painful'.

For classification purposes, patients will be said to have fibromyalgia if both criteria are satisfied. Widespread pain must have been present for at least 3 months. The presence of a second clinical disorder does not exclude the diagnosis of fibromyalgia.

According to the ACR criteria pain is considered chronic and widespread when present for at least 3 months in both the left and right side of the body, both above and below the waist, and in the axial skeleton. MacFarlane et al have proposed a more stringent 'Manchester' definition of widespread pain.⁶ The main difference in this definition is that pain must be present in *two* separate sections of a body quadrant in order to be classified as positive. It has been proposed that this definition of chronic widespread pain will more precisely identify individuals with truly widespread pain and stronger associations to adverse psychosocial factors.⁷ The fact that CWP is also associated with other somatic symptoms has been used to support a hypothesis that CWP is one feature of somatisation.

Somatisation: a process whereby a mental event is expressed in a body disorder or physical symptom

The prevalence of CWP in the population has been shown to be just above 10% when the ACR 1990 definition has been used⁸⁻¹⁰ and around 5% with the more stringent 'Manchester' definition.⁷ The prevalence has been reported to be about twice as high for women as for men. The prevalence of FM has been shown to vary from 0.7% to 4.8% in comparable studies using the ACR 1990 criteria. The predominance of women is strong, with a female to male ratio between 3:1 and 7:1.¹⁰⁻¹²

A THEORY ON PATHOGENESIS IN CHRONIC WIDESPREAD PAIN AND FIBROMYALGIA

The pathogenesis of FM has long been regarded as an enigma but there is now increasing evidence that peripheral factors interact with an altered central nervous system (CNS) processing of nociceptive stimuli.¹³ Table 2 summarises some of the suggested pathogenetic mechanisms that are especially important for the treatment strategy proposed in this article.

As the main complaint in FM is pain in muscles and soft tissues, considerable research to find pathological changes in the muscles has been carried out. Although no specific changes in peripheral tissues have been found, it is assumed that different types of peripheral 'pain generators' could be present in patients with CWP and FM.

The nociceptors in the peripheral tissues are normally silent in the absence of a noxious stimulus but may be sensitised (*peripheral sensitisation*), partly by influence of prostaglandin that is released at the site of tissue injury or inflammation. A sensitised nociceptor gives an enhanced response to noxious stimuli (*hyperalgesia*) and may also respond to non-noxious stimuli such as normal touch (*allodynia*). It may also begin to discharge spontaneously. Impulses from the nociceptors are conducted via the primary afferent neurons (A- δ and C-fibres) to synapses in the spinal cord's dorsal horn.

TABLE 2. Pathogenetic mechanisms in chronic widespread pain and fibromyalgia that are especially important for the proposed treatment strategy.

1. Peripheral pain generator

- traumatic tissue damage
- osteoarthritis
- inflammatory rheumatic diseases
- muscular tension
- disc herniation
- endometriosis
- migraine

2. Peripheral sensitisation

- more responsive nociceptors

3. Central sensitisation and disinhibition

- facilitated transmission of pain signals

4. Cognitive processes

- thoughts and memories

5. Emotional processes

- anxiety and depression

6. Stress and psychosocial situation

7. Behaviour

- avoidance and immobilisation
- withdrawal and escape
- muscle contraction

The signal is further conducted via the spinothalamic tract to the thalamus and finally to the cerebral cortex, where the pain is perceived.

The transmission in the dorsal horn synapses is under influence from descending signals from the brain and brain stem (*central inhibition*), mediated

partially by endorphins and serotonin, and inhibition from neurons (A-β) conducting normal sensitivity from the periphery (*gate-control*) (Figure 1). The transmission in the dorsal horn synapses can be facilitated by reduced central inhibition and a change in the sensitivity of the post-synaptic neuron (*central sensitisation*) mediated mainly by N-methyl-D-aspartate (NMDA) receptors.¹⁴ The central sensitisation and a diminished central inhibition can, together with a peripheral sensitisation, contribute to a problematic pain situation, where the peripheral noxious stimulus may be small or no longer existent. Allodynia and hyperalgesia are thus key features in FM and are expressed in the tender points.

Patients with FM, low back pain and chronic fatigue syndrome have been shown to have perturbation in the hypothalamus–pituitary–adrenal (HPA) axis. This is believed to be an effect of long-standing stress. Although chronic pain is a factor of stress that may lead to the noticed disturbances, the disturbances in the neuro-hormonal systems also interact with pain perception and seem to add to the vulnerability for developing chronic musculoskeletal pain. Furthermore the neuro-hormonal disturbances may contribute to other symptoms reported in association with CWP and FM.¹³

The perception of pain cannot be described only in anatomical and physiological terms of sensory input and transmission. Psychological factors must also be

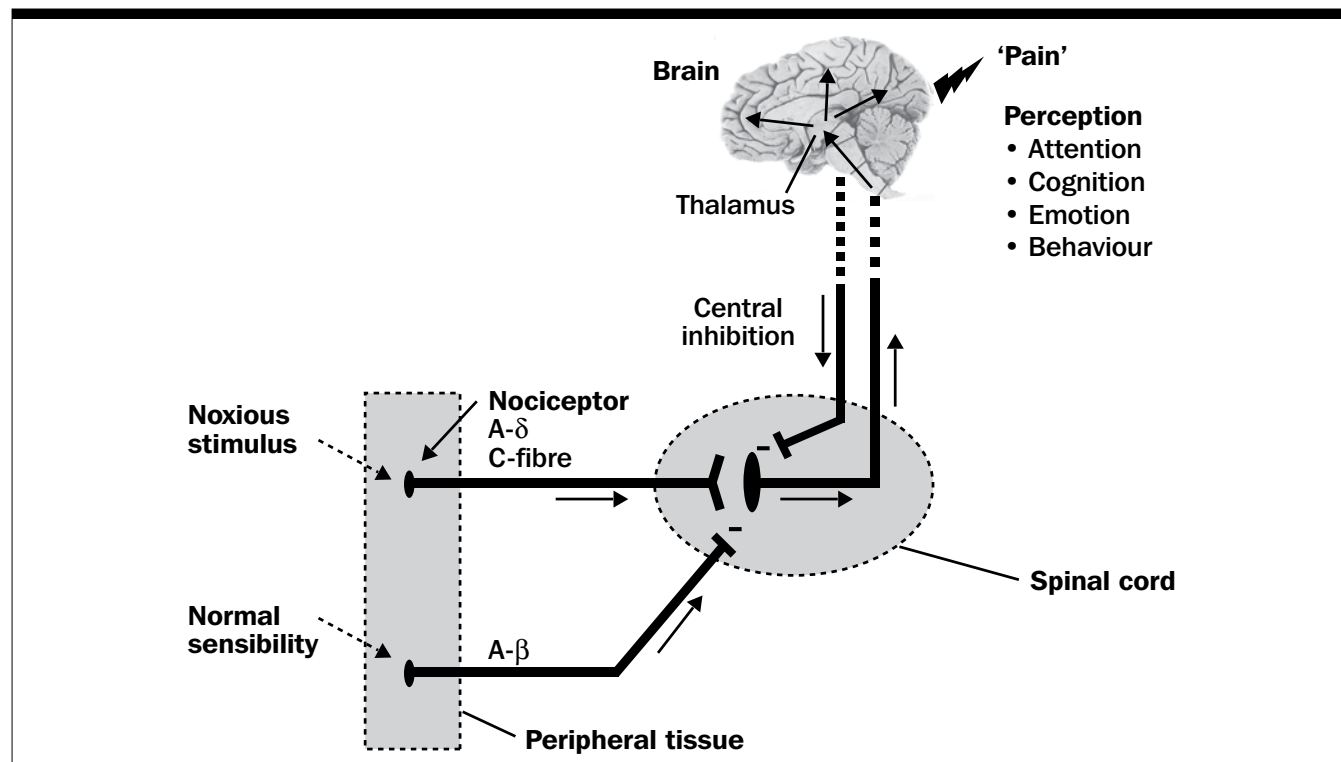


FIGURE 1. The perception of nociceptive pain not only involves the sensation transmitted and regulated by peripheral and central neurons, but also is affected by higher brain functions.

considered, such as attention, cognition (thoughts and memories), emotions (fear, anxiety, stress and depression), and response in behaviour (avoidance, withdrawal, escape and muscle contraction). This is especially the case when pain becomes long-standing and widespread.

WHO IS AT RISK?

The strongest risk factor for developing CWP is the presence of a long-standing regional pain. The development of a more widespread pain is not a dead end. Individuals with CWP move within the pain spectrum continuum and in longitudinal studies have reported less or no pain at follow-up.¹⁵ The prognosis is worse for those with more widespread pain and higher tender point counts.

Development and persistence of CWP is associated with several sociodemographic, psychosocial and lifestyle risk factors. These factors include: female sex, older age, lower educational level, lower socio-economic class, having a family history of chronic pain, not having social support, and smoking. Furthermore, features of somatisation,¹⁶ self-reported poor health status and sleep disturbances have been shown to predict the development of CWP.

A PRACTICAL APPROACH TO THE PAIN PATIENT

Table 3 summarises a practical approach for assessment and treatment of patients with musculoskeletal pain from a GP's point of view. The first three points are applicable to all patients with pain; the remainder focus on management of patients with potentially chronic widespread pain. The recommendations are further developed and discussed below. It is usually not possible to implement all these steps at the first visit and it is also important to individualise management depending on the patient's problem.

1. Believe in the patient's experience and description of pain

Often when pain is reported no tissue damage is found and the search for a specific cause may be in vain. Bearing in mind the subjective nature of pain, it is important to believe in the patient's experience.

2. Do a thorough clinical examination at the first visit

This in itself is part of the treatment as it helps to create empathy and so gives the patient confidence. The clinical examination, together with the case history, forms the foundation for all other measures and management.

TABLE 3. A practical approach for assessment and treatment of patients with musculoskeletal pain from a GP's point of view.

1. Believe in the patient's experience and description of pain
2. Do a thorough clinical examination at the first visit
3. Treat acute pain adequately
4. Search for peripheral pain generators
5. Pay attention to cognitive factors
6. Pay attention to emotional factors
7. Pay attention to stress, sleep problems and psychosocial factors
8. Pay attention to central sensitisation and central disinhibition
9. Encourage physical exercise
10. Massage therapy, acupuncture and transcutaneous electrical nerve stimulation (TENS) could be adjuvant
11. Consider cognitive behavioural therapy
12. Educate and motivate the patient
13. Get yourself a multidisciplinary team or create one in your Primary Care Trust

- Is the pain localised or widespread? Widespread pain is a prerequisite for fibromyalgia. A pain drawing could be used in the assessment. Widespread pain indicates that factors other than strictly biomedical ones must be taken into account.
- Tender point examination to show if the patient has disturbed pain processing with hyperalgesia or allodynia. A tender point count of less than 11 out of the 18 specified for fibromyalgia does not exclude the possibility of the patient having a disturbance in the nociceptive system. It is wise to test for tenderness on other possible tender point sites. Note that a general tenderness does not exclude fibromyalgia, but instead strengthens the suspicion. The term 'tender point' could be misleading: according to the ACR criteria for fibromyalgia, palpation should be 'painful' and not merely 'tender' in order to be classed as a positive result.
- Swollen or painful joints could indicate the presence of osteoarthritis or an inflammatory rheumatic disease.
- Muscular tension is common as a result of pain, but could also be a source of pain. There is an obvious risk of a vicious circle developing.
- It must be remembered that CWP and FM do not exclude other disorders. Pay special attention to the possibility of malignancies and endocrine disorders. Both the patient's history and the clinical examination are crucial for decisions on further assessment and referrals.

3. Treat acute pain adequately

Acute and localised pain should be adequately treated with analgesics following the World Health Organization (WHO) Analgesic Ladder (see p.127 this volume). Adequate treatment of acute pain also includes realistic and confident reassurance of what the patient can expect during the healing process. The patient should be advised and encouraged to continue or return to normal activities as soon as possible as this has been found to be very effective in reducing chronic problems in acute back pain and after neck injuries.¹⁷ Diagnostic procedures should be kept to a minimum as they may introduce fear and negative expectations.

4. Search for peripheral pain generators

In widespread pain situations peripheral pain generators should be identified and treated. The pain generator could, for example, be a joint with rheumatic arthritis or osteoarthritis. It has also been hypothesised that muscle tension in a static work situation could lead to a local 'energy crisis' and peripheral sensitisation of the nociceptors in that area. A referral to an occupational therapist or physiotherapist would be justified and helpful if an adverse ergonomic work situation is suspected. Pain generators outside the musculoskeletal system, such as endometriosis and migraine, could also contribute to the nociceptive load.

5. Pay attention to cognitive factors

Thoughts and memories could influence pain perception and behaviour. Cognitive factors are believed to modulate pain transmission in the nociceptive pathways. It could be of value to ask the patient why s/he has chosen to seek help, and why now? Fears and earlier experiences should be asked about and discussed. Vicious circles in thought and behaviour can often be identified and explained to the patient.

6. Pay attention to emotional factors

Depressive disorders are common among patients with chronic pain. It could be the result of having a painful disorder that has been misinterpreted and where the search for help has been in vain. A depressive disorder may also precede the pain and contribute to the patient's vulnerability. The depressive disorder must not be ignored and should be treated together with the pain symptoms. Selective serotonin reuptake inhibitors (SSRIs) may be the drugs of choice as some studies have shown them to be effective treatment for other concomitant symptoms.

7. Pay attention to stress and psychosocial factors

A chronic stress situation contributes to an exaggerated transmission and perception of pain, and also to symptoms such as fatigue, sleep problems, irritable bowel syndrome and cognitive dysfunction. Treatment and rehabilitation will not succeed unless the correct psychosocial conditions are present for a full recovery and return to work. Sleep problems need to be assessed and properly treated.

8. Pay attention to central sensitisation and central disinhibition

Central sensitisation should be suspected when there is a history of widespread pain and the clinical examination reveals hyperalgesia and allodynia.

- Peripherally working analgesics are often of little help in this situation and most opioids do not affect the sensitisation process, although the pain diminishes during treatment. Patients with FM are often drug sensitive and have frequent problems with adverse drug effects. Tramadol is one drug to try. It has been reported that tramadol in combination with dextromethorphan could have an NMDA-receptor antagonist effect.
- Antidepressants may be helpful, especially tricyclic antidepressants: in particular, amitriptyline has been proven to be effective when given in small dosages at bedtime. SSRIs could be tried in selected patients but should be evaluated for effectiveness and reconsidered at follow-up.

9. Encourage physical exercise

Physical exercise is important and besides aerobic fitness it may also have an effect on the descending inhibitory pathway with elevated levels of endorphins and serotonin. The first step is to encourage the patient to take a daily walk. There is often an initial barrier where the patients experience a worsening of the symptoms. The trick is to start gently and gradually increase the walking distance and speed. The exercise should gradually be combined with training of muscle flexibility, strength and endurance under the supervision of a physiotherapist.

10. Massage therapy, acupuncture, and transcutaneous electric nerve stimulation could be adjuvant

Massage therapy can contribute to muscle relaxation and pain reduction. The tactile stimuli may also have a pain-reducing effect through the gate-control and increased activity in descending inhibitory pathways. Acupuncture and transcutaneous elec-

tric nerve stimulation (TENS) may reduce pain perception and nociceptive flow, and could be beneficial together with other therapeutic interventions.¹⁸ There is some evidence that all these therapies may benefit patients.

11. Consider cognitive behavioural therapy

Cognitive behavioural therapy (CBT) has been proven successful in both the short- and the long-term treatment of FM.¹⁹ The main problem is a shortage of experienced therapists.

12. Educate and motivate the patient

This should be an integrated part of assessment, treatment, and rehabilitation. The Arthritis Research Campaign (arc) booklet 'Fibromyalgia' is a useful patient aid.

13. Get yourself a multidisciplinary team or create one in your Primary Care Trust

If several psychosocial risk factors are identified a multidisciplinary team approach has been shown to be beneficial and should be considered early in the course of management. In primary care a useful mini-team would consist of a physician, physio-therapist and psychologist. A nurse, occupational therapist and social worker would complete it.

CONCLUSION

The GP should adopt a strategic approach to management of patients with chronic widespread pain and fibromyalgia based on current knowledge of pathogenesis. This, together with patient education, leads to better control of pain and other symptoms and less frustration for both the patient and the GP.

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REFERENCES

1. Yunus M, Masi AT, Calabro JJ, Miller KA, Feigenbaum SL. Primary fibromyalgia (fibrositis): clinical study of 50 patients with matched normal controls. *Semin Arthritis Rheum* 1981;11(1):151-71.

2. Wolfe F, Smythe HA, Yunus MB et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the Multicenter Criteria Committee. *Arthritis Rheum* 1990;33(2):160-72.

3. Croft P, Schollum J, Silman A. Population study of tender point counts and pain as evidence of fibromyalgia. *BMJ* 1994;309(6956):696-9.

4. Wolfe F. The relation between tender points and fibromyalgia symptom variables: evidence that fibromyalgia is not a discrete disorder in the clinic. *Ann Rheum Dis* 1997;56(4):268-71.

5. Croft P, Burt J, Schollum J, Thomas E, Macfarlane G, Silman A. More pain, more tender points: is fibromyalgia just one end of a continuous spectrum? *Ann Rheum Dis* 1996;55(7):482-5.

6. MacFarlane GJ, Croft PR, Schollum J, Silman AJ. Widespread pain: is an improved classification possible? *J Rheumatol* 1996;23(9):1628-32.

7. Hunt IM, Silman AJ, Benjamin S, McBeth J, Macfarlane GJ. The prevalence and associated features of chronic widespread pain in the community using the 'Manchester' definition of chronic widespread pain. *Rheumatology (Oxford)* 1999;38(3):275-9.

8. Bergman S, Herrström P, Högström K, Petersson IF, Svensson B, Jacobsson LT. Chronic musculoskeletal pain, prevalence rates, and sociodemographic associations in a Swedish population study. *J Rheumatol* 2001;28(6):1369-77.

9. Croft P, Rigby AS, Boswell R, Schollum J, Silman A. The prevalence of chronic widespread pain in the general population. *J Rheumatol* 1993;20(4):710-3.

10. Wolfe F, Ross K, Anderson J, Russell IJ, Hebert L. The prevalence and characteristics of fibromyalgia in the general population. *Arthritis Rheum* 1995;38(1):19-28.

11. White KP, Speechley M, Harth M, Ostbye T. The London Fibromyalgia Epidemiology Study: the prevalence of fibromyalgia syndrome in London, Ontario. *J Rheumatol* 1999;26(7):1570-6.

12. Lindell L, Bergman S, Petersson IF, Jacobsson LT, Herrström P. Prevalence of fibromyalgia and chronic widespread pain. *Scand J Prim Health Care* 2000;18(3):149-53.

13. Pillemer SR, Bradley LA, Crofford LJ, Moldofsky H, Chrousos GP. The neuroscience and endocrinology of fibromyalgia. *Arthritis Rheum* 1997;40(11):1928-39.

14. Bennett GJ. Update on the neurophysiology of pain transmission and modulation: focus on the NMDA-receptor. *J Pain Symptom Manage* 2000;19(1 Suppl):S2-6.

15. Bergman S, Herrström P, Jacobsson LT, Petersson IF. Chronic widespread pain: a three year followup of pain distribution and risk factors. *J Rheumatol* 2002;29(4):818-25.

16. McBeth J, Macfarlane GJ, Benjamin S, Silman AJ. Features of somatization predict the onset of chronic widespread pain: results of a large population-based study. *Arthritis Rheum* 2001;44(4):940-6.

17. Verhagen AP, Peeters GG, de Bie RA, Oostendorp RA. Conservative treatment for whiplash. *Cochrane Database Syst Rev* 2001;(4):CD003338.

18. Offenbacher M, Stucki G. Physical therapy in the treatment of fibromyalgia. *Scand J Rheumatol Suppl* 2000;113:78-85.

19. Williams DA, Cary MA, Groner KH et al. Improving physical functional status in patients with fibromyalgia: a brief cognitive behavioral intervention. *J Rheumatol* 2002;29(6):1280-6.