

# FIBROMYALGIA IN ADULTS

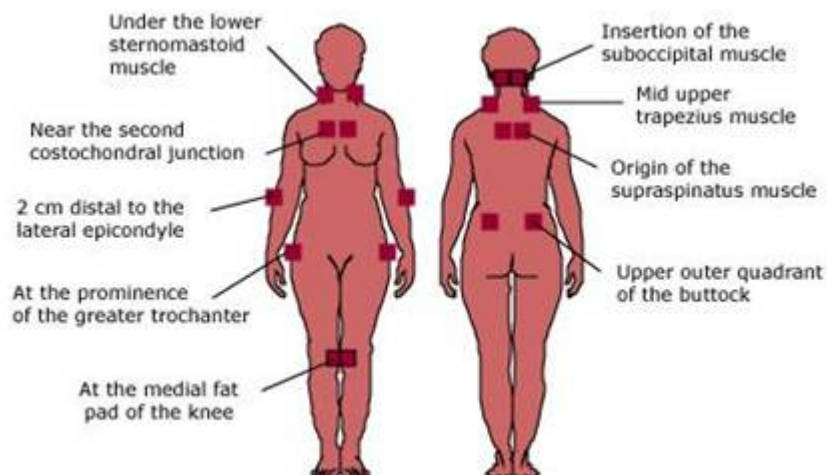
## Diagnosis and Management Advice for GPs

### DIAGNOSIS:

The vast majority of GP referrals for fibromyalgia have the diagnosis correct. In my experience GPs refer primarily because they are afraid of 'missing' alternative diagnoses. .

In a patient presenting with pain, stiffness and lethargy, both fibromyalgia and inflammatory pain need to be mentioned as potential differential diagnoses. Statistically, fibromyalgia is more likely than any of the inflammatory arthritides. It is very important that fibromyalgia is presented as a positive diagnosis, rather than, as is often the case, a negative 'waste basket' diagnosis when all investigations have been exhausted.

- History of pain and stiffness. Fibromyalgic symptoms can mimic inflammatory symptoms, including a history of sometimes severe and debilitating nocturnal pain and morning and inactivity stiffness. In inflammatory arthritides there may be gross swelling in the history as opposed to fibromyalgics who sometimes have sensations of puffiness or fullness in the tissues.
- Other history. Is there any other symptom of auto-immune disease – such as pronounced Raynaudism, marked mouth ulceration, alopecia, malar rash or photosensitive rash? Fibromyalgics, on the other hand, commonly have other features of the chronic fatigue syndrome – including fatigue, post-exertion malaise, poor sleep, poor concentration and memory ('brain fog'), headaches or irritable bowel
- Examination. In pure fibromyalgia there is no hard evidence of inflammation (swelling, redness, heat) but often exquisite tenderness in two or more "trigger spots" (below)



- Inflammatory and auto-immune markers. In fibromyalgia, ESR, CRP, rheumatoid factor (test only if signs of inflammation) and anti nuclear antibodies are generally negative.

## MANAGEMENT:

### Education

- There is now a large body of experimental evidence showing that fibromyalgia is a 'real' condition, and the pain that fibromyalgia patients suffer is real and measurable. For a given stimulus (e.g. to the lateral epicondyle fibromyalgia trigger point), both the amplitude of the pain signal travelling proximally and the degree of activity in the pain centres of the brain (as demonstrated by functional MRI studies) is much greater in fibromyalgic compared to non-fibromyalgic patients. There is a very wide array of additional experimental evidence showing abnormalities in neurotransmitter levels, so that pain promoting neurotransmitters are present in enhanced quantities in the CSF, and pain inhibiting neurotransmitters present in reduced quantities in the CSF.

The simple acknowledgement that fibromyalgics' pain is real and not some imaginary or neurotic condition, is probably the single most beneficial piece of information that you can offer patients

- I believe that reduction, not abolition, of pain is a reasonable goal. The aim is a situation where patients feel in control of rather than controlled by their pain.
- Education about what fibromyalgia isn't is also very important. Often fear about fibromyalgia being a destructive or damaging process that will lead to major disability is a very real concern to patients. You should allay these fears from a very early stage.

### Regular exercise.

- It is important to encourage patients to carry out even a tiny amount of exercise on days when they are feeling terrible, but not to do too much exercise on when they are feeling good as they can overcompensate and suffer for days after. This includes activities such as gardening.
- Aerobic exercise, flexibility exercises and resisted weight exercises have all undergone studies that prove benefit. It should be noted that there have also been negative studies for all forms of exercise.

### Pharmacological treatment

Options in New Zealand are slightly limited.

- *[how about mentioning this later rather than first since it is not available]* The only agent approved by the FDA for fibromyalgia is **Pregabalin**, which there is very limited access to in New Zealand. I do have two patients taking Pregabalin who have found it very beneficial, but it comes at a cost of around \$180.00 - \$220.00 per month to the patient. Nevertheless, this should be discussed with patients as a potential option at the outset. **Gabapentin** obviously has a great many similarities with Pregabalin and I use it reasonably frequently, although not as 'first line' therapy.
- **Amitriptyline** or **Nortriptyline** remain first line treatment for all fibromyalgics. Promotion of the quality and quantity of sleep, as well as direct neurogenic analgesia

are important. Although very few patients can tolerate more than 10mg at night, some can tolerate dose escalation up to a maximum, in my practice, of 50mg nocte (although I rarely use more than 30mg). Drowsiness is one of the most common side effects and I advise patients to take the drug early in the evening to combat this. I also suggest that they try to persist beyond the first two weeks of anticholinergic side effects, which seem to be particularly severe in the fibromyalgic group. Despite all of these measures I would estimate that less than half of fibromyalgic patients are able to tolerate tricyclics.

- The more modern dual re-uptake inhibitors (that block both noradrenaline and serotonin re-uptake) have a very considerable body of evidence for their efficacy. In the literature Duloxetine is the agent with the best evidence for efficacy, but to my knowledge it is not available in New Zealand. **Venlafaxine** has dual re-uptake inhibition properties, but is only indicated in New Zealand for severe treatment resistant depression and thus is not practically available in a very large majority of our patients.
- **Gabapentin** therefore is the next funded option and is well worth a try in patients who have not tolerated, or not responded to, tricyclics. It requires special authority application. GPs can apply for the special authority.
- I usually follow this if gabapentin and amitriptylline are unsuccessful, by asking patients to at least try a month long course of **combination Tramadol and Paracetamol**. Several studies in the pain literature have demonstrated synergism between Paracetamol and Tramadol. Tramadol itself has weak serotonin and noradrenaline re-uptake inhibition, and thus is a logical pain killing choice. My practice has been to prescribe 50mg tds Tramadol given concurrently with 1g tds Paracetamol and then let patients decide after a month whether the \$70.00 or so dollars a month cost is justifiable by any benefit that the combination has brought them. A few patients have needed 100mg tds instead of 50mg tds tramadol. A large majority of patients have elected to continue paying for such treatment, which implies to me at least, that there is considerable efficacy.
- A common mistake is to trial prednisone in patients with morning stiffness. **Even in patients with extraordinarily convincing pain and morning and inactivity stiffness, unless there is unequivocal evidence of inflammation (in the physical examination or laboratory tests), prednisone should be avoided.** Virtually all patients who do receive prednisone feel benefit, and it can be exceedingly difficult to withdraw prednisone from a fibromyalgic patient once this has been trialed. Furthermore, dose escalation is inevitable in this patient group
- Finally, there is no evidence for any efficacy of anti inflammatory medication, and despite the importance of sleep disturbance in the pathogenesis of the condition Benzodiazepine sleeping tablets have no role to play. These are only likely to add to the patients' disease burden.

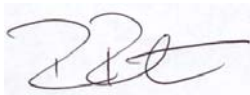
### **Patients with a positive ANA.**

A very common clinical situation is a middle-aged woman with widespread aches and pains, lethargy and a modestly positive ANA. If there are no additional features of connective tissue disease (summarised above) and there is no elevation of inflammatory markers then fibromyalgia is most likely, but a mild connective tissue disease is still possible. My practice is to give a trial of hydroxychloroquine 200mg daily for 6 months (it takes 3 months to “kick in”) and only continue hydroxychloroquine in the absence of side effects and the presence of marked, definite benefit. Only about 5-10% of patients do benefit, but the results can be startling in those lucky few. More aggressive treatment such as with azathioprine cannot be justified unless there is definite evidence of connective tissue disease.

### **NMDHB model of community and hospital care for fibromyalgia**

I am no longer be able to see fibromyalgia patients on an individual basis in my hospital outpatient clinics but am happy to run group sessions. Patients who have a GP diagnosis of fibromyalgia, according to the criteria above, may be referred for a group session with the understanding that they will not be having a one on one consultation and that therefore there will not be strict patient confidentiality. I plan to see ten to fifteen patients in a two and a half hour session.

I am also very happy to come and present to any GP education session about fibromyalgia.



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