Fibromyalgia

Pain Management as a Psychiatrist

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Conflict of Interest

• None
Key Points to Cover

1. Complex etiology involving biological and environmental influences
2. Central Sensitization
3. Pathophysiology supported by imaging studies
4. FM vs SSD and the new DSM-V criteria
5. Traditional treatment and novel therapeutic approaches under investigation
6. OMT and Soft Tissue Techniques
Modern Definition

- Chronic, multifaceted, and widespread musculoskeletal pain syndrome involving both central and peripheral sensitization.
- Assoc w/ spectrum of symptoms
- Affects many domains of functioning: somatic and psychological
- Not same as anxiety and MDD (1)
- Billable Codes:
  - ICD9: 729.1 “Myositis and Myalgia, unspecified”
  - ICD10: M79.7 “Fibromyalgia”
Early Descriptions

“I, too, have been assigned months of futility, long and weary nights of misery. When I go to bed, I think, `When will it be morning?’ But the night drags on, and I toss till dawn...Depression haunts my days. My weary nights are filled with pain as though something were relentlessly gnawing at my bones.’”

- Job 7:3-4; 30:16-17 - NLT
French physician Guillaume de Baillou: FM-like symptoms first given a name: muscular rheumatism

Dr. Hugh Smythe laid the foundation for the modern definition of FM by describing widespread pain and tender points

No evidence of inflammation so changed name from fibrositis to FM (= pain in muscles and tissues)

Sir William Gowers coined the term fibrositis (= inflammation of fibers) to denote the tender points found in patients with muscular rheumatism

The first controlled clinical study with validation of known symptoms and tender points was published

The first sleep EEG study identifying the sleep disturbances that accompany FM was performed

The AMA recognized FM as a real physical condition

Dr. Balfour described tender points

The ACR developed diagnostic criteria for FM for research. Later used in clinical practice

The concept of neurohormonal mechanisms with central sensitization was developed

FDA approves first drug for FM: lyrica (pregabalin)
Epidemiology

• Affects 2-8% of US population (2-4)
• Most common cause of widespread MSK pain in women 20-55yo (5)
• Rheum: second to OA as the most common disorder encountered (6)
• Onset: 35-55yo (7)
• Prevalence increases w/age, but it can also be seen in children (9-10)
Epidemiology

• Children born prematurely - more likely to show tender points and decreased pain threshold vs children delivered term \(^{(10)}\) (Buskila, et. al.)
  • case-control study; 60 pre-term and 60 term adolescents
  • observed in both sexes, but girls had > pain sensitivities than boys
  • Higher somatic pain sensitivities \(\rightarrow\) higher risk of future pain syndromes?
    • more studies needed
Epidemiology

• Buskila, et. al hypotheses:
  • Differences in pain perception d/t:
    • lack of myelination of their sensitive fiber
    • immature cortical organization of the somatosensory system
  
  • Differences in pain threshold d/t:
    • lack of protection of spinal and supraspinal inhibitory pathways
    • abnormal behavioral imprinting 2/2 early painful stimuli → increased plasticity of neonatal brain → future incr vulnerability of CNS to stress and pain d/o

Painful stimuli perceived as widespread and durable

Lower pain threshold
Epidemiology: Painful procedures in the NICU

**Diagnostic:**
- Venipuncture
- Heel lancing
- Lumbar puncture
- ROP examination
- Endoscopy
- Bronchoscopy
- Suprapubic bladder tap

**Therapeutic:**
- Bladder catheterization
- Central line insertion and removal
- Chest tube insertion and removal
- Chest physiotherapy
- Mechanical ventilation
- Dressing change
- Gavage tube insertion
- Intramuscular injection
- Peripheral venous catheterization
- Tracheal intubation and extubation
- Tracheal suctioning
- Suture removal
- Ventricular tap

**Surgical:**
- Circumcision
- Others
Epidemiology

• Long term effects of untreated pain in the neonate:
  • Untreated pain leads to abnormal pain pathways → alteration in cerebral neuroanatomy → ?developmental delays, ?emotional disorders, ?chronic pain d/o
  • MRI of newborns (within first 7 days of life)
    • fMRI’s of newborns: reveal neural activity of pain overlap with adult pain \(^{(33)}\)
    • Areas: primary somatosensory cortices, ACC, bilateral thalamus, insula

“Even if not expressed as conscious memory, memories of pain may be recorded biologically and alter brain development and subsequent behavior” \(^{(26)}\)
Complex Etiology

Genetic factors + Environmental triggers → Alterations in gene expression
Complex Etiology: Genetic factors

- Strong genetic predisposition → d/t marked familial aggregation \(^{(11-15)}\)
- 1st-degree relatives of pts w/FM: 8-fold increase in risk of FM \(^{(16)}\)
- Family members of FM pts: more likely to have lower pain threshold than general pop; often carry dx of other chronic pain syndromes: IBS, TMJ, and chronic HAs. \(^{(18)}\)
- Current research: identified polymorphisms in genes assoc w/ serotonergic, dopaminergic, and catecholaminergic systems in pts w/ FM. \(^{(19)}\)
Complex Etiology: Environmental triggers

Stressors involving: (1)
- acute pain
- motor vehicle traumas
- deployment to war
- infectious diseases
- psychological stress
Pathophysiology and Pathogenesis

• Hallmark of FM = Centralized Pain
  • Process: CNS amplifies sensory input across many organ systems (20-21)
  • But... can’t r/o peripheral nociceptive input as a contributing factor (22-23)
    • Patients will respond with pain to presence of touch or light pressure (i.e. allodynia), or even from normal noxious stimuli (i.e. hyperalgesia)
Pathophysiology and Pathogenesis
Central Sensitization

• A-delta fibers (large myelinated nociceptors)
  • transitory pain, easier to endure
• C-fibers (small unmyelinated nociceptors; free nerve endings)
  • chronic pain, harder to endure
  • most numerous of sensory receptors and nociceptors
  • bombardment of C-fibers in spinal cord → proposed MOA FM central sensitization syndromes (chronically maintained and sympathetically maintained pain syndromes)
Pathophysiology and Pathogenesis

Central Sensitization: BOMBARDMENT OF C-FIBERS

A. 
- **Incr in NMDA receptors + glutamate**
  - Required to processes influx of C-fibers
  - Sprouting occurs (anatomic disorganized change in laminae)

B. 
- **Incr Substance P production**
  - Travels down peripheral receptors
  - Ends at termination of C-fibers
  - Lowers threshold of C-fibers

C. 
- **“Wind Up”**
  - Incr receptor field in periphery
  - Here: receptors more sensitive and more likely to depolarize
  - Widened receptor field allows for A + B to occur
  - Disorganizes processing of pain

- **Wind Up**
  - Lows threshold of C-fibers
Pathophysiology and Pathogenesis

• fMRI studies: \(^{(27,28)}\)
  - Pain when stimulated by mild pressure or a heat stimulus
  - Pain assoc w/activation in brain areas involved in pain processing
    - Decr cortical thickness in the DLPFC, anterior insula, anterior cingulate cortex and somatosensory cortex (SI, SII) \(^{(35)}\)
Pathophysiology and Pathogenesis

**fMRI studies:** (34)

- **Increased brain connectivity:** areas of increased pain processing
- **Decreased brain connectivity:** areas that attenuate pain

Incr intrinsic DMN activity within R. middle and ant. insula
Pathophysiology and Pathogenesis

- Proton spectroscopy studies: \((29-32)\)
  - Increases and decreases in neuromodulators of pain
  - Tx for FM: alter these by targeting their receptors

Figure 1: CNS neurotransmission influencing pain \((34)\)
Pathophysiology and Pathogenesis

- PET imaging studies: \(^{(44,45)}\)
  - Paradoxical increase in endogenous opioid activity and a decrease in mu-opioid receptor availability
Pathophysiology and Pathogenesis

• Glial cell activation and central inflammation
  • modulate pain by:
    • (1) harboring neurotransmitter receptors
    • (2) releasing neuromodulators in response to pain

Acute effects:
Pathophysiology and Pathogenesis

• Glial cell activation and central inflammation
  • When chronically activated:
    • pro-inflammatory cascade $\rightarrow$ neurotoxicity
    • ? explanation for central inflammatory process also involved in pathogenesis
• Glial cell modulating therapy:
  • LDN

Chronic effects:

1. Injured Peripheral Nerves or Brain Injury
2. Chemical and/or Electronic Signals Enter CNS
3. Microglial Activation
4. Neuroinflammation
5. Release of Excess Glutamate/Neurotoxins
6. Cell Death, Apoptosis, Reformation
7. Imprinting of Pain Sensation

Figure 1: Algorithm illustrating how central pain develops. CNS: central nervous system.
Pathophysiology and Pathogenesis

• Small fiber neuropathy
  • Defined: injury to peripheral nerves affecting small fibers
  • Sx: burning, shooting, allodynia, hyperesthesia

• 2013 case-controlled study \(^{(46)}\)
  • FM pts: reduction in intraepidermal innervation and regeneration of C fibers
  • Pts showed neuropathic pain
  • Findings differed from healthy controls and unipolar depression w/o pain

• 2015 Retrospective study \(^{(47)}\)
  • FM vs SFN and controls
  • using EM: reduced axonal diameters of C-fibers (absent in SFN)
Pathophysiology and Pathogenesis

• SFN
  • Better hypothesis based on CNS theory (central sensitization):
    • peripheral abnormalities d/t neuroplasticity or other co-morbid changes in chronic pain (e.g. deconditioning)?
    • just a sequela of the syndrome?
    • incidental finding?
Pathophysiology and Pathogenesis

• Raymond Perrin, DO, PhD
  • Hypothesis:
    • insulin - biofeedback w/hypothalamus; HPA disrupted $\rightarrow$ insulin fx disrupted
    • poor filtering/drainage of CSF in BOTH head and spine $\rightarrow$ toxins build up in CSF
d/t lymph backflow
  • Common findings seen clinically
  • Treatment: massage, OMT
  • Anecdotally tx worked, confirmed findings in Journal of medical engineering and technology
Pathophysiology and Pathogenesis

External Stimuli: temperature, trauma, etc.

From chemoreceptor and mechanoreceptor via afferent nerve to sympathetic nervous system.

Sympathetic Nervous System Overloads

Sympathetic Nervous System Breaks Down

Reduced Blood Flow and Lymphatic Drainage

Build Up of Toxins in Brain and Spinal Cord

Further Damage to Sympathetic Nervous System

Worsening Lymphatic Drainage

Further Toxic Build-up

M.E.
Osteopathic Perspective

5 Domains of Pathology in FM

- Stressors
  - Trauma
  - Nicotine
  - Aging
  - Socio-Economics
  - Work Environment

- Environment
  - OMT
  - 1. Altered posture motion
  - 2. Sleep apnea heart rate variability decreased
  - 3. Metabolic bioenergetic endocrine imbalance
  - 4. Central sensitization autonomic imbalance
  - 5. Inactivity depression circadian rhythm disturbance

Somatic Dysfunction
Diagnosis

• Clinical Manifestation\(^7\):
  • Multifocal pain with low suspicion for a single etiology
  • Amplified pain in other areas, usually earlier in life
  • Symptoms suggestive of CNS origins
  • Symptoms suggestive of global sensory hyper-responsiveness
  • Pertinent past med hx:
    • Co-morbid: chronic regional pain syndromes, rheumatic disease, psych
    • Fam h/o FM
    • Patient Self-Report Survey based on 2011 ACR Criteria \(\rightarrow\) Helpful for diagnosis
      • Score of \(\geq 13\) is consistent with FM.
Diagnosis: ACR 2011 modified fibromyalgia criteria (7)
Diagnosis: ACR 2011 modified fibromyalgia criteria (7)
Diagnosis

• Physical Exam Findings
  • Diffuse tenderness assessed by tenderpoint exam
  • Blood pressure cuff sensitivity from insufflation
  • Low pain threshold to firm pressure of upper extremities
Diagnosis

• Perrin’s Signs:
  • head forward posture
  • hyper mobility in cervical and lumbar region
  • commonly thoracic problems
  • incr in thoracic kyphosis and lordosis (T1-L2), mechanically SNS is over-irritated → symptoms
  • lymphatic varicosity
Diagnosis

Spinal Cord Abnormality
Diagnosis

- Flattened Area
- Kyphotic Segment
Diagnosis
Diagnosis

Varicose Lymphatic Vessels

Varicose Lymphatic Vessels
Diagnosis

• Perrin’s Symptoms:
  • “Perrin’s points” - tender points (stars in image)
  • Tenderness at celiac plexus
  • Abnml Cranial Rhythm
Diagnosis

- "Fibro Fog"
  - Complaint: “I feel like I’ve been taking cold medicine constantly”
  - Symptoms: difficulty concentrating, finding words, holding conversations, feeling alert and remembering things
  - Pathophysiology: (43)
    1. Central processing abnormalities?
    2. Morphologic abnormalities in the frontoparietal network?
    3. Dysfunctional dopamine system?
Diagnosis

• “Fibro Fog”
  • Diagnosis: \(^{(43)}\)
    • Neurocognitive testing \(\rightarrow\) (1) effects of distraction on memory (2) speed of word retrieval
  • Treatment: \(^{(43)}\)
    • Acknowledge its existence and reassure
    • Co-occurrence of ADHD (inattentive) and fibro is high \(\rightarrow\) common neurobiology and rationale for stimulants \(\rightarrow\) ongoing open-label trials (methylphenidate 10-60mg)
    • Better sleep (FM often have insomnia)
    • Best option thus far: CBT+physical activity+meds
Diagnosis

- **Laboratory Tests**
  - **Blood Tests**
    - Results: non-specific
    - If low suspicion for other causes: CBC, CMP, TSH, Vitamin D, ESR, and/or CRP.
    - Rheum labs: (only if necessary): ANA and Rheumatoid factor.
  - **Ancillary Test**
    - Pts w/neuropathic signs (e.g. from SFN), or to r/o other causes: ENFD testing with skin punch biopsies
  - **Further W/U**
    - Imaging studies, muscle biopsies, EMGs, and mm enzyme assays
    - Not required
Differential

• Somatic Symptom Disorder (SSD), specifier: with predominant pain (DSM-V)
  • Previously: Somatoform Disorders (DSM-IV)

• Myofascial Pain Disorder

• Inflammatory Myopathies

• Polymyalgia Rheumatica
Differential: SSD

Changes in new DSM-5:

- Removal of DSM-IV disorders:
  - somatization d/o, hypochondriasis, pain d/o, undifferentiated somatiform d/o →
    many can now be diagnosed under SSD
  - Somatization d/o required specific number of complaints from among 4 symptom groups
    - SSD no longer requires this
  - SSD does not require the presence of “medically unexplained symptoms” as an inclusion criteria
• CFS/ME
  • controversial: unique or on spectrum?
  • overlap with FM: sleep, mood, pain, fatigue
  • Differences:
    • CFS/ME → fatigue primary c/o
    • FM → pain primary c/o
  • Tx:
    • same for both
    • so… doesn’t matter what you call it
    • Dr. Perrin and OMT
Diagnosis: FM vs SSD

- It is difficult to differentiate the two b/c of similarities
- See functional somatic illnesses elsewhere in medicine, w/ overlapping sx and tx (CBT and meds)
  - Rheumatology: Fibromyalgia
  - Infectious disease: Chronic fatigue syndrome
  - Gastroenterology: Irritable bowel syndrome
  - Neurology: Chronic headaches
  - Cardiology: Noncardiac chest pain
  - Urology: irritable bladder syndrome
  - Gynecology: Vulvodynia and chronic pelvic pain
  - Allergy: Multiple chemical sensitivity
  - Oral surgeons: Temporomandibular joint syndrome
  - Physical medicine: Myofascial pain syndrome
Diagnosis: FM vs SSD

• Result of confusion?
  • Skepticism among the medical community that FM as a disease, truly exists

“I used to think the worst thing in life was to end up all alone, it’s not. The worst thing in life is to end up with people that make you feel alone.”
- Robin Williams
Diagnosis: FM vs SSD

• What we know today:
  • FM is NOT SSD
  • Need to establish diagnosis of FM - essential to tx (17)

• Key clinical differences to remember:
  • Pts usually do not focus on their condition to the exclusion of other life interests, should not have markedly diminished social, sexual, and vocational functioning, and should not be profoundly physically deconditioned
Diagnosis: FM vs SSD

• Result of incorrect dx:
  • Categorizing as having only SSD \(\rightarrow\) psychiatrization \(^{(25)}\)
  • Conversely, treating a patient for only FM pain \(\rightarrow\) ignoring sx of SSD and other psych factors \(\rightarrow\) poorer outcomes
Diagnosis: Solution

• A more nuanced method:
  • use a multidimensional approach:
    • patient’s somatic, psychosocial, and functional status
    • categorizing on a spectrum of severity (e.g. mild to moderate) along with co-
morbidities (e.g. FM plus IBS) to individualize treatment (25)
Proposed method for diagnosing FM

Suspected FM $\rightarrow$ begin a trial of treatment while evaluation of other co-morbidities $^{(24)}$
Chronic Pain Rehabilitation and FM

• Admission:
  • Pain: moderate-severe
  • Functional impairment: severe
  • Depression: moderate
  • Anxiety: severe

• Discharge:
  • Pain: mild-moderate
  • Functional impairment: moderate
  • Normalization of anxiety/depression
  • Improvements were clinically and statistically significant (p < .01)


Courtesy of Sara Davin, Psy.D, MPH
Do improvements last?

12 month follow-up
- 27.32% (n = 100) returned follow up surveys
- Pain: moderate
- Functional impairment: moderate
- Depression: moderate
- Anxiety: mild
- In comparison to admission, improvements remained clinically and statistically relevant (p < .01)

Vij et al. Poster to be presented at American Pain Society (2014)

Courtesy of Sara Davin, Psy.D, MPH
Treatment

• No treatment alters pathogenesis of disease
• Focus on symptoms relief and functional restoration
  ➢ Conservative Management
  ➢ Oral pharmacotherapy
  ➢ Non-invasive treatments
  ➢ Invasive treatments
Treatment: Conservative Management

- *Patient education
  - Confirm FM label -- NO judgement
  - Emphasize multimodal tx + active patient involvement
- *CBT
  - Pain based CBT in 1-1 sessions, groups, or via telemedicine
- *Graded Exercise
  - Aerobic exercise, strength training, stretching
- Nutrition
  - Weight loss (for those overweight or obese)
- Complementary medicine
  - Music: specifically water and wave sounds
  - Other: Yoga, acupuncture, tai chi, chiropractic manipulation, myofascial release therapy, and osteopathic manipulation

* 3 best studies non-pharm tx for FM
Components of CBT-based Skill Building

- Education
- Coping skills
- Activity pacing
- Problem solving skills
- Assertiveness training
- Emotional modulation
- Relaxation training
- Sleep hygiene
- Relapse prevention

Courtesy of Sara Davin, Psy.D, MPH
Overarching goal of oral pharmacotherapy:

- Excitatory neurotransmission via:
  - Glutamate, substance P, and nerve growth factor activity

- Inhibitory neurotransmission via:
  - Norepinephrine, dopamine, and GABA activity

Facilitate pain transmission
Inhibit pain transmission
Treatment: Oral pharmacotherapy

Figure 1: CNS neurotransmission influencing pain (34)
Treatment: Oral pharmacotherapy

**TCAs and SNRIs**
- Amitriptyline: 10-70mg qhs
- Cyclobenzaprine: 5-20mg qhs
- Duloxetine: 30-120mg qd (FDA approved)
- Milnacipran: 100-200mg qd (FDA approved)
- Venlafaxine XR: 75-225mg qd

**Gabapentinoïds and GABA agents**
- Gabapentin: up to 3600mg qd in divided doses
- Pregabalin: up to 600mg qd in divided doses (FDA approved)
- Gamma-hydroxybutyrate (sodium oxybate): 4.5-6.0 mg qd in divided doses

First line - Level 1A evidence
Treatment: Oral pharmacotherapy

SSRIs
- Fluoxetine
- Sertraline
- Paroxetine

Second line – no convincing evidence

Cyclobenzaprine
- Monotherapy: 10-40mg qd
- Combination: cyclobenzaprine 10mg qd + fluoxetine 20mg qd
Treatment: Oral pharmacotherapy

Opioids (full and weak opioid agonist):

• History:
  • Nidus: very poor 1986 retrospective review of 38 pts
• Fact:
  • poor outcomes → not recommended
• Unfortunately
  • (1) use in FM is widespread, (2) those given opioids were unlikely to receive evidence based pharmacotherapy,(3) less likely to respond well opioids (41-42)
• Interestingly:
  • patients have elevated CSF levels of endogenous opioids (38) + decreased binding capacity to mu-opioid receptors in pain areas of brain (39) → ? account for poor outcomes (40)
Treatment: Oral pharmacotherapy

Future treatment options: promising results, but lack sufficient data

- **Low dose naltrexone: 4.5mg qhs** (36)
  - Promise in treating chronic pain conditions that involve inflammatory processes → but FM doesn’t have inflammation in FM??
  - Rationale: FM may involve chronic glial activation and subsequent production of pro-inflammatory cytokines
  - Benefits: low SE profile, no abuse potential, low cost (on avg, $35/month vs >$100/month for conventional oral meds)

- **Synthetic Cannabinoids** (37)
  - Nabilone: 0.5mg qhs to 1.0mg bid
  - Limited available data with studies showing a non-significant trend for analgesia, but they may be a consideration in those with significant sleep disturbances
Treatment: Other treatments

• Non-invasive:
  • rTMS
  • rTDCS and HD-rTDCS
  • Hyperbaric O2 (prelim study)
  • TENS unit (mixed results)?
  • OMT and Soft Tissue Techniques

• Invasive:
  • ONS
  • TP injections?
Treatment: Non-invasive treatments

• rTMS and HD-rTDCS
  • Target: left primary motor cortex and dorso-lateral prefrontal cortex
  • Study outcomes: \(^{(48,49)}\)
    • reduction in FM pain and effects outlasting the duration of stimulation
    • outcomes similar to medications
  • Benefits: minimal SE (most common: transient HA and scalp irritation)

• Studies:
  • on-going phase-II open label trial for HD-rTDCS \(^{(50)}\)
    • more targeted, more tolerable, higher specificity
Treatment: Non-invasive treatments

- ESWT
  - Concept: high energy acoustic waves (i.e. shock waves) \(\rightarrow\) delivers mechanical force
  - Use: MSK pain (e.g. MPD)
  - How it works in MPD (MOA still elusive)
    - Shock waves elicit referred pain (from TPs) \(\rightarrow\) induces energy \(\rightarrow\) stimulates healing through inflammation and incr blood flow to damaged tissues \(\rightarrow\) promote healing
  - In FM: (only 1 study to date) improvements 3-mo post tx, no SE \(^{(51)}\)
    - improved: VAS and algometer
    - 2000 pulses in 5 sessions
    - ?effective adjunct early in d/o
Treatment: Invasive treatments

• **ONS**
  • Use: migraine headaches, chronic primary HAs
  • MOA: unclear
  • SE: high risk of complications d/t invasive
  • 2007 study (lacked placebo; co-morbid chronic HAs) \(^{(52)}\)
    • C2 scalp area; VAS, BDI, QOL (SF-36)
    • HAs, widespread bodily pain, mood, and fatigue improved
  • 2013 study (double-blind, placebo controlled) \(^{(53)}\)
    • subsensory threshold stimulation
    • psych d/o excluded (except mild depression), no HAs, intractable to PT, meds, psych therapy
    • decr (40-60%) in pain 6 mo post-implant (VAS and PCS, not BDI), decr in tender points
Treatment: Perrin’s Plan

- cranial massage
- stimulating blood blood flow in shoulders with cold and heat
- draining motion down nose
- working fingers up chest
- rotating body around upper waist
- rotating shoulders
- marching up and down, shaking arms back and forth
- avoid stimulants - coffee and etoh
- watch for bad posture
- keeping spine straight while sleeping; pillow between knees
- proper sleep
Treatment: Perrin’s Plan

• Video excerpt
Treatment: OMT Overview

- Counterstrain of various bilateral tenderpoints assoc w/FM
- Rib raising
- Myofascial Release
- Muscle energy
- Balance Ligamentous Tension
- Facilitated Positional Release
- TMJ treatments
Treatment: OMT Specifics

- Seated lumbosacral functional technique
- Seated lumbosacral functional technique
- Sidelying rib functional technique
- Seated upper thoracic spine muscle energy technique
- Seated or supine diaphragm myofascial release technique
- Supine cervical functional and strain/counterstrain techniques
- Supine TMJ muscle energy techniques
Take Home Points

• FM-like symptoms described in texts dating back 100s of years
• First given a name in the 1600’s; 2007 FDA approved 1st drug
• Very common in rheum practices
• Onset between 35-55yo
• Symptoms increase with age, but see in children also
• Life long sx - mimic other pain d/o
• Defined on a spectrum of central sensitization d/o
• Active research - glial cell activation, central inflammation, and small fiber neuropathy, LDN, TMS, TDCS, ECWT, ONS
• Many options for management
• Left untreated - devastating for pt, provider and health care system
References


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