CLINICAL REVIEW

Beyond the Criteria: Diagnosing Fibromyalgia in Clinical Practice

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Fibromyalgia is a common chronic pain disorder characterized by widespread musculoskeletal pain, fatigue, sleep disturbance, and cognitive difficulties that affects an estimated 4-5% of the general population.[1] In clinical practice, despite the syndrome's common prevalence, the diagnosis remains challenging with delays in diagnosis and treatment.[2] A large-scale international survey of 1,622 physicians reported that, while a majority had seen a patient with fibromyalgia in the last two years, 53% had difficulty diagnosing fibromyalgia, 54% reported their fibromyalgia training was inadequate, and 32% considered themselves not knowledgeable about the condition.[3] This inadequacy is further evidenced in primary care where up to one in 20 patients report FM symptoms but 46% of primary care physicians report uncertainty with diagnosing fibromyalgia.[4-5] Recent publications recommend increased efforts for fibromyalgia education in health care providers.[6-9] The present review discusses the evolution of fibromyalgia diagnosis and provides a guide for the clinical implementation of the latest diagnostic criteria from the American College of Rheumatology (ACR).

Early Descriptions of Fibromyalgia

Early reports of fibromyalgia-like illnesses date back to the 16th century. French physician Guillaume de Baillou introduced the first modern descriptions of "rheumatism" and fibromyalgia in his *Liber de rheumatismo et Pleuritide dorsali*.[10] Over time, symptoms of widespread myofascial pain became known as "muscular rheumatism" as physicians started to differentiate between articular and non-articular musculoskeletal disorders.[11]

In the 19th century, several authors began to identify chronic pain syndromes of muscular origin.[12] One of the earliest to localize widespread pain to the muscles was William Balfour, a surgeon from Edinburgh, who championed the idea that inflammation of muscle and connective tissue was the source of chronic musculoskeletal pain.[13] However, it was Balfour's later findings in 1824 that mark a significant milestone in the development of the fibromyalgia concept and future diagnostic criteria: regions of focal tenderness called tender points.[14]

Following observations that pain could radiate from tender points, the referred pain concept began to draw the attention of those studying muscular rheumatism. In 1841 French pediatrician Francois Louis Isidore Valleix proposed that muscular rheumatism was a form of neuralgia after observing referred pain from tender points ran in close proximity to nerve routes.[15] Others suggested that the pain radiation was independent of nerve trajectory and implicated muscle hypertonicity or nerve hyperreactivity.[16-17] In 1858, Thomas Inman speculated that tender points were caused by localized functional changes such as muscle spasms. Alternatively, Cornelius was one of the earliest to describe a relationship between musculoskeletal pain and psychological factors with the proposition that "nerve points" were hyper-reactive nerve endings reactive to physical and emotional stimuli.

Psychological contributions to myofascial pain, however, would not be explored until after the 19th century due to a majority emphasis on underlying muscle or connective tissue processes. The muscle callous theory claimed that exudative processes led to muscle fiber atrophy and replacement with connective tissue.[18-20] In 1904, British neurologist Sir William Gowers first used the term "fibrositis" to describe fibrous tissue inflammation and studies performed by Stockman identified inflammatory changes in muscular nodules supportive of muscle pathophysiology.[21-22] While not reproducible in later studies, Stockman's findings solidified fibrositis as the favored diagnostic term for chronic myofascial pain and the misnomer persisted throughout fibrositis research in the 20th century.

In the late 1930's and early 1940's interest in psychological factors increased, particularly in response to the mental health consequences of war. Reports documenting the frequent occurrence of fibrositis in 24% to 52% of World War II soldiers resulted in coinage of the term "psychogenic rheumatism" and the theory that fibrositis may be a psychological disorder.[23-24]

As the medical community became increasingly interested in fibrositis, doubt surrounding the syndrome's existence began to dissipate. However, the syndrome remained a neglected miscellany of symptoms despite growing clinical acceptance. In the 1968 article *Fibrositis*, Traut linked together many of fibromyalgia's common features including chronic fatigue, widespread pain, chronic headaches, disturbed sleep, and tender points.[25] At the time, Traut's publication was monumental in unifying many of the syndrome's seemingly unrelated, yet hallmark symptoms and paved the way for fibromyalgia becoming recognized as its own entity.

Emergence of Past and Present Diagnostic Criteria

In 1977 Smythe and Moldolfsky proposed the first diagnostic criteria in their seminal *'Two contributions to the understanding of the "fibrositis" syndrome'* which required "widespread pain lasting longer than three months, disturbed sleep with morning fatigue and stiffness, and a decreased pain threshold" quantified by the presence of at least 12 of 18 possible tender points.[26] The 1977 criteria were the first to objectify diagnosing fibromyalgia with a focus on quantifiable measures and sparked a number of subsequent publications. However, some argued that Smythe and Moldolfsky's level of tenderness needed to achieve a diagnosis was unrealistic in clinical practice and this concern was reflected in other investigators' reduction of the number of tender points required for diagnosis.[27-30]

The term "fibromyalgia" was introduced by Yunus et al. who also proposed the first formal set of diagnostic criteria.[31] The requirements included pain and stiffness in three body regions lasting for at least three months, the presence of four or five tender points, and, dependent on the number of tender points, varying levels of: poor sleep, fatigue, headaches, irritable bowel, anxiety, swelling, tenderness, and symptom exacerbation by weather or physical activity.

Discrepancies in subsequent diagnostic criteria led to debate over the relative contribution of tender points and psychosomatic symptoms. In response, the American College of Rheumatology (ACR) performed a multicenter study to standardize fibromyalgia classification criteria.[32] The 1990 ACR classification criteria deemed 11 of 18 tender points sufficient to differentiate fibromyalgia patients from controls and introduced the requirement of chronic widespread pain (defined as 4-quadrant plus axial pain lasting for a minimum of 3 months) to differentiate fibromyalgia from localized myofascial pain syndromes. It was concluded that primary and secondary-concomitant fibromyalgia were indistinguishable and made a fibromyalgia diagnosis "a valid construct irrespective of other diagnoses." While the 1990 criteria facilitated research advancements, they were meant for classification purposes rather than diagnosis. They were not widely embraced in clinical practice, partly due to difficulty performing the tender point examination.[33]

Twenty years later, the ACR 2010 preliminary diagnostic criteria sought to improve clinical utility.[34] The new criteria replaced the tender point examination with reported painful body regions, measured via the widespread pain index (WPI). New additions addressed the severity of fatigue, unrefreshed sleep, cognitive complaints, and somatic symptoms with the symptom severity scale (SSS). Later amendments in 2011 combined the WPI and SSS forms into the fibromyalgia severity (FS) score and other modifications which allowed self-administration for epidemiologic studies.[35] The 2010/2011 criteria changes enabled the syndrome to be classified on a spectrum rather than as a dichotomous diagnosis.

The absence of definite pathology has led to difficulty differentiating fibromyalgia from other chronic pain conditions sharing the same symptom and diagnostic spaces. The 2016 criteria revisions addressed such concerns to prevent misdiagnosis.[33] The revisions added a generalized pain criterion in place of the chronic widespread pain requirement to prevent misclassification of regional pain syndromes as fibromyalgia. A controversial statement from the 2010/2011 criteria was inclusion of the statement that fibromyalgia diagnosis could not be made if the patient has a "disorder that could otherwise explain the pain." This mistakenly implied that fibromyalgia could not be a comorbidity.[34] The modified 2016 criteria revisions reverted to language used in 1990 and added additional clarifications: "a diagnosis of fibromyalgia is valid irrespective of other diagnoses. A diagnosis of fibromyalgia does not exclude the presence of other clinically important illnesses."[33]

Despite significant advancements, the diagnostic criteria are not without limitations and require further validation. The addition of the WPI and SSS requires a more thorough history and assessment by health care providers. The inherent subjective nature of symptom assessment by both providers and patients also must be taken into account. There are arguments for and against self-report assessments in fibromyalgia diagnosis. Further investigation is needed to determine applicability and appropriateness in both research and clinical practice.[33]

Beyond the Diagnostic Criteria: Pattern Recognition in Clinical Practice

While institution of diagnostic criteria for fibromyalgia has greatly benefited research, the American College of Rheumatology has not recommended definitive use in clinical settings and recommends physician discretion for clinical practice use. In the absence of directly translatable criteria, physicians need to recognize the clinical manifestations of fibromyalgia as part of the wider spectrum of central hypersensitivity syndromes.

One primary challenge in diagnosing fibromyalgia is the syndrome's polysymptomatic nature. Fibromyalgia patients see an average of 3.7 physicians and accumulate numerous disjointed diagnoses prior to receiving a proper diagnosis two years after initial onset of symptoms.[36] This leads to disgruntled patients, frustrated health care providers, and unintentional stigmatization of the disorder which further complicates care.

Growing evidence suggests fibromyalgia is a disorder that results from central sensitization and dysfunctional pain regulation.[37-38] The centralized pain theory best explains the syndrome's wide array of symptoms including multifocal pain, fatigue, insomnia, cognitive difficulties, and mood disturbances.[39-40] Abnormal levels of neurotransmitters such as epinephrine, GABA, serotonin, glutamate, and substance P, may explain the psychiatric and psychosomatic symptoms, as well as sleep disturbances.[41-42] Emerging research reveals the role of neuroinflammation in fibromyalgia.[42] This type of

inflammation is described as neurogenic as it results from the release of neuropeptides from C-fibres with subsequent effects on blood vessels, sensory structures, and regional immune cells. Clinical features may be linked to neurogenic inflammation including tissue swelling, livedo reticularis, dermatographia, and allodynia.[43]

Due to the absence of definitive laboratory or radiographic testing, the diagnosis of fibromyalgia is clinical in nature. The clinician should obtain a detailed history of factors contributing to chronic pain. A central pain prone phenotype is associated with female sex, genetics, early life trauma, family history of chronic pain and mood disorders, personal history of chronic centrally mediated symptoms, behavioral patterns such as catastrophizing, and a lower mechanical pain threshold. [44] Fibromyalgia should be suspected in patients with chronic pain, diffuse hyperalgesia, and/or allodynia with signs of central sensitization. These patients commonly present with a history of multiple functional disorders diagnosed by other specialists including but not limited to irritable bowel syndrome, migraine headaches, temporomandibular joint disorder, interstitial cystitis, and endometriosis.

Laboratory testing to exclude mimickers of fibromyalgia may include: complete blood count (CBC), comprehensive metabolic panel (CMP), thyroid stimulating hormone (TSH), creatinine phosphokinase (CPK), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP).

As emphasized in current diagnostic criteria, the presence of a second disorder does not exclude fibromyalgia or vice versa and all medical problems need to be addressed. Of particular importance is comorbid rheumatologic disorders with fibromyalgia. Analyzing six-months referrals, Fitzcharles and Boulos found only 34% accuracy of fibromyalgia diagnosis in patients referred for rheumatology consultation. Inflammatory rheumatologic conditions accounted for almost one-half of misdiagnoses.[45] While science has yet to prove causality, the rate of comorbid fibromyalgia is high in connective tissue diseases and inflammatory arthritis including: 22.1% with systemic lupus erythematosus, 18-24% with rheumatoid arthritis, 14-16% in axial spondyloarthritis, and 18% in psoriatic arthritis. These are striking findings, when compared to rates of 4-5% in the general population.[1,46-47] The higher comorbidity rates should caution providers to consider rheumatology referral when evaluating patients with joint pain and swelling, unusual rashes, photosensitivity, and family history of autoimmunity. When signs and symptoms of a connective tissue disease or inflammatory arthritis are present, pertinent labs include: CBC, CMP, ESR, CRP, TSH, CPK, as well as antinuclear antibody, rheumatoid factor, and anticyclic citrullinated peptide.

Confirmation of fibromyalgia diagnosis by a specialist may also be beneficial in the face of diagnostic uncertainty or patient concern. Receiving a diagnosis of fibromyalgia commonly brings relief to patients as it both explains and validates their symptoms. Proper diagnosis of fibromyalgia has economic benefits as well as health care costs decrease in fibromyalgia patients following diagnosis by rheumatologists.[48]

Conclusion

The past century has witnessed a significant growth in awareness and interest in fibromyalgia. Despite research advancements, complexities of the clinical diagnosis of fibromyalgia often leads to delays that directly impact patients' quality of life. Fibromyalgia's clinical diagnosis is based on widespread chronic pain, fatigue, and other centrally mediated symptoms. A diagnosis of fibromyalgia can be made appropriately in the primary care setting with careful evaluation including detailed medical history, thorough physical exam, and routine laboratory testing. Due to the high rate of comorbidity with rheumatologic disorders, health care providers should consider rheumatology referral in the appropriate clinical setting.

REFERENCES

1. **Häuser W, Ablin J, Fitzcharles MA, Littlejohn G, Luciano JV, Usui C, Walitt B**. Fibromyalgia. *Nat Rev Dis Primers*. 2015 Aug 13;1:15022. doi: 10.1038/nrdp.2015.22. Review. PubMed PMID: 27189527.
2. **Gendelman O, Amital H, Bar-On Y, Ben-Ami Shor D, Amital D, Tiosano S, Shalev V, Chodick G, Weitzman D**. Time to diagnosis of fibromyalgia and factors associated with delayed diagnosis in primary care. *Best Pract Res Clin Rheumatol*. 2018 Aug;32(4):489-499. doi: 10.1016/j.berh.2019.01.019. Epub 2019 Mar 4. Review. PubMed PMID: 31174818.
3. **Perrot S, Choy E, Petersel D, Ginovker A, Kramer E**. Survey of physician experiences and perceptions about the diagnosis and treatment of fibromyalgia. *BMC Health Serv Res*. 2012 Oct 10;12:356. doi: 10.1186/1472-6963-12-356. PubMed PMID: 23051101; PubMed Central PMCID: PMC3502453.
4. **Arnold LM, Gebke KB, Choy EH**. Fibromyalgia: management strategies for primary care providers. *Int J Clin Pract*. 2016 Feb;70(2):99-112. doi: 10.1111/ijcp.12757. Review. PubMed PMID: 26817567; PubMed Central PMCID: PMC6093261.
5. **Hadker N, Garg S, Chandran AB, Crean SM, McNett MM, Silverman SL**. Efficient practices associated with diagnosis, treatment and management of fibromyalgia among primary care physicians. *Pain Res Manag*. 2011 Nov-Dec;16(6):440-4. PubMed PMID: 22184554; PubMed Central PMCID: PMC3298044.
6. **Kianmehr N, Haghighi A, Bidari A, Sharafian Ardekani Y, Karimi MA**. Are general practitioners well informed about fibromyalgia? *Int J Rheum Dis*. 2017 Dec;20(12):1917-1921. doi: 10.1111/1756-185X.12716. Epub 2015 Jul 22. PubMed PMID: 26200844.
7. **Buskila D, Neumann L, Sibirski D, Shvartzman P**. Awareness of diagnostic and clinical features of fibromyalgia among family physicians. *Fam Pract*. 1997 Jun;14(3):238-41. PubMed PMID: 9201499.

8. **Kamoun S, Elleuch M, Le Lay K, Feki H, Taieb C, André E, Damak J, Elleuch MH**. [Evaluation of knowledge of fibromyalgia in Tunisia]. *Tunis Med*. 2010 Oct;88(10):703-6. French. PubMed PMID: 20890815.
9. **Blotman F, Thomas E, Myon E, Andre E, Caubere JP, Taïeb C**. Awareness and knowledge of fibromyalgia among French rheumatologists and general practitioners. *Clin Exp Rheumatol*. 2005 Sep-Oct;23(5):697-700. PubMed PMID: 16173250.
10. Baillou Gd. Liber de rheumatismo & pleuritide dorsali. 1642.
11. **Inanici F, Yunus MB**. History of fibromyalgia: past to present. *Curr Pain Headache Rep*. 2004 Oct;8(5):369-78. Review. PubMed PMID: 15361321.
12. **Perrot S**. If fibromyalgia did not exist, we should have invented it. A short history of a controversial syndrome. *Reumatismo*. 2012 Sep 28;64(4):186-93. doi: 10.4081/ reumatismo.2012.186. Review. PubMed PMID: 23024963.
13. **Balfour W**. Observations on the Pathology and Cure of Rheumatism. *Edinb Med Surg J*. 1815 Apr 1;11(42):168-187. PubMed PMID: 30329434; PubMed Central PMCID: PMC5743373.
14. **Baldry P**. Myofascial Pain and Fibromyalgia Syndromes: A Clinical Guide to Diagnosis and Management. 1 ed: Churchill Livingstone; 2001.
15. **Valleix FLI**. Traité des névralgies ou Affections douloureuses des nerfs. In. Paris, France: Baillière; 1841; 719.
16. **Inman T**. Remarks on Myalgia, or Muscular Pain. *Br Med J*. 1858;1(73):407–408.
17. **Cornelius A**. "Narben und nerven." *Deutsche Militärärztlische Zeitschrift*.1903; 657-673.
18. **Froriep R**. *Ein Beitrag zur Pathologie des Rheumatismus.* Weimar, 1843.
19. **Virchow R**: Ueber parenchymatose entzundung**.** *Arch Pathol Anat* 1852; 4:261–279.
20. **Reynolds MD**. The development of the concept of fibrositis. *J Hist Med Allied Sci*. 1983 Jan;38(1):5-35. PubMed PMID: 6341451.
21. **Gowers WR**. A Lecture on Lumbago: Its Lessons and Analogues: Delivered at the National Hospital for the Paralysed and Epileptic. *Br Med J*. 1904 Jan 16;1(2246):117-21. PubMed PMID: 20761312; PubMed Central PMCID: PMC2352601.
22. **Stockman R**. The Causes, Pathology, and Treatment of Chronic Rheumatism. *Edinb Med J*. 1904;15(3):223–235.
23. **Boland EW**. Psychogenic Rheumatism: The Musculo-skeletal Expression of Psychoneurosis. *Ann Rheum Dis*. 1947;6(4):195-203. PubMed PMID: 18623768; PubMed Central PMCID: PMC1030553.
24. **Savage O**. Rheumatic Disease in the Forces. *Br Med J*. 1942 Sep 19;2(4263):336-8. PubMed PMID: 20784439; PubMed Central PMCID: PMC2164164.
25. **Traut EF**. Fibrositis. *J Am Geriatr Soc*. 1968 May;16(5):531-8. PubMed PMID: 4230883.
26. **Smythe HA, Moldofsky H**. Two contributions to understanding of the "fibrositis" syndrome. *Bull Rheum Dis*. 1977-1978;28(1):928-31. PubMed PMID: 199304.
27. **Bennett RM**. Fibrositis: misnomer for a common rheumatic disorder. *West J Med*. 1981 May;134(5):405-13. PubMed PMID: 6167073; PubMed Central PMCID: PMC1272765.
28. **Hudson JI, Hudson MS, Pliner LF, Goldenberg DL, Pope HG Jr**. Fibromyalgia and major affective disorder: a controlled phenomenology and family history study. *Am J Psychiatry*. 1985 Apr;142(4):441-6. PubMed PMID: 3856400.
29. **Wolfe F, Hawley DJ, Cathey MA, Caro X, Russell IJ**. Fibrositis: symptom frequency and criteria for diagnosis. An evaluation of 291 rheumatic disease patients and 58 normal individuals. *J Rheumatol*. 1985 Dec;12(6):1159-63. PubMed PMID: 2936884.
30. **Wolfe F, Cathey MA**. The epidemiology of tender points: a prospective study of 1520 patients. *J Rheumatol*. 1985 Dec;12(6):1164-8. PubMed PMID: 3879278.
31. **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 Aug;11(1):151-71. PubMed PMID: 6944796.
32. **Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, Tugwell P, Campbell SM, Abeles M, Clark P, et al**. The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia. Report of the Multicenter Criteria Committee. *Arthritis Rheum*. 1990 Feb;33(2):160-72. PubMed PMID: 2306288.
33. **Wolfe F, Clauw DJ, Fitzcharles MA, Goldenberg DL, Häuser W, Katz RL, Mease PJ, Russell AS, Russell IJ, Walitt B**. 2016 Revisions to the 2010/2011 fibromyalgia diagnostic criteria. *Semin Arthritis Rheum*. 2016 Dec;46(3):319-329. doi: 10.1016/j.semarthrit.2016.08. 012. Epub 2016 Aug 30. PubMed PMID: 27916278.
34. **Wolfe F, Clauw DJ, Fitzcharles MA, Goldenberg DL, Katz RS, Mease P, Russell AS, Russell IJ, Winfield JB, Yunus MB**. The American College of Rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity. *Arthritis Care Res (Hoboken)*. 2010 May;62(5):600-10. doi: 10.1002/acr. 20140. PubMed PMID: 20461783.
35. **Wolfe F, Clauw DJ, Fitzcharles MA, Goldenberg DL, Häuser W, Katz RS, Mease P, Russell AS, Russell IJ, Winfield JB**. Fibromyalgia criteria and severity scales for clinical and epidemiological studies: a modification of the ACR Preliminary Diagnostic Criteria for Fibromyalgia. *J Rheumatol*. 2011 Jun;38(6):1113-22. doi: 10.3899/jrheum. 100594. Epub 2011 Feb 1. PubMed PMID: 21285161.
36. **Choy E, Perrot S, Leon T, Kaplan J, Petersel D, Ginovker A, Kramer E**. A patient survey of the impact of fibromyalgia and the journey to diagnosis. *BMC Health Serv Res*. 2010 Apr 26;10:102. doi: 10.1186/1472-6963-10-102. PubMed PMID: 20420681; PubMed Central PMCID: PMC2874550.

37. **Sluka KA, Clauw DJ**. Neurobiology of fibromyalgia and chronic widespread pain. *Neuroscience*. 2016 Dec 3;338:114-129. doi: 10.1016/j.neuroscience.2016.06.006. Epub 2016 Jun 9. Review. PubMed PMID: 27291641; PubMed Central PMCID: PMC5083139.
38. **Cagnie B, Coppieters I, Denecker S, Six J, Danneels L, Meeus M**. Central sensitization in fibromyalgia? A systematic review on structural and functional brain MRI. *Semin Arthritis Rheum*. 2014 Aug;44(1):68-75. doi: 10.1016/j.semarthrit.2014.01.001. Epub 2014 Jan 8. Review. PubMed PMID: 24508406.
39. **Fukuda K, Dobbins JG, Wilson LJ, Dunn RA, Wilcox K, Smallwood D**. An epidemiologic study of fatigue with relevance for the chronic fatigue syndrome. *J Psychiatr Res*. 1997 Jan-Feb;31(1):19-29. PubMed PMID: 9201644.
40. **Fukuda K, Nisenbaum R, Stewart G, Thompson WW, Robin L, Washko RM, Noah DL, Barrett DH, Randall B, Herwaldt BL, Mawle AC, Reeves WC**. Chronic multisymptom illness affecting Air Force veterans of the Gulf War. *JAMA*. 1998 Sep 16;280(11):981-8. PubMed PMID: 9749480.
41. **Williams DA, Clauw DJ**. Understanding fibromyalgia: lessons from the broader pain research community. *J Pain*. 2009 Aug;10(8):777-91. doi: 10.1016/j.jpain.2009.06.001. Review. PubMed PMID: 19638325; PubMed Central PMCID: PMC2741022.
42. **Littlejohn G, Guymer E**. Neurogenic inflammation in fibromyalgia. *Semin Immunopathol*. 2018 May;40(3):291-300. doi: 10.1007/s00281-018-0672-2. Epub 2018 Mar 19. Review. PubMed PMID: 29556959.
43. **Littlejohn G**. Neurogenic neuroinflammation in fibromyalgia and complex regional pain syndrome. *Nat Rev Rheumatol*. 2015 Nov;11(11):639-48. doi: 10.1038/nrrheum.2015.100. Epub 2015 Aug 4. Review. PubMed PMID: 26241184.
44. **Phillips K, Clauw DJ**. Central pain mechanisms in the rheumatic diseases: future directions. *Arthritis Rheum*. 2013 Feb;65(2):291-302. doi: 10.1002/art.37739. Review. PubMed PMID: 23045168; PubMed Central PMCID: PMC3610409.
45. **Fitzcharles MA, Boulos P**. Inaccuracy in the diagnosis of fibromyalgia syndrome: analysis of referrals. *Rheumatology (Oxford)*. 2003 Feb;42(2):263-7. PubMed PMID: 12595620.
46. **Wolfe F, Petri M, Alarcón GS, Goldman J, Chakravarty EF, Katz RS, Karlson EW**. Fibromyalgia, systemic lupus erythematosus (SLE), and evaluation of SLE activity. **J Rheumatol**. 2009 Jan;36(1):82-8. doi: 10.3899/jrheum.080212. PubMed PMID: 19004039; PubMed Central PMCID: PMC2944223.
47. **Zhao SS, Duffield SJ, Goodson NJ**. The prevalence and impact of comorbid fibromyalgia in inflammatory arthritis. *Best Pract Res Clin Rheumatol*. 2019 Jun;33(3):101423. doi: 10.1016/j.berh.2019.06.005. Epub 2019 Jul 17. Review. PubMed PMID: 31703796.
48. **van Eijk-Hustings Y, Kroese M, Creemers A, Landewé R, Boonen A**. Resource utilisation and direct costs in patients with recently diagnosed fibromyalgia who are offered one of three different interventions in a randomised pragmatic trial. *Clin Rheumatol*. 2016 May;35(5):1307-15. doi: 10.1007/s10067-015-3067-y. Epub 2015 Sep 26. PubMed PMID: 26409883; PubMed Central PMCID: PMC4844631.