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REVIEW ARTICLE

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Polymyalgia Rheumatica and Fibromyalgia: Similarities and Distinctions

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Abstract

This review explores the similarities and distinctions between polymyalgia rheumatica (PMR) and fibromyalgia (FM), focusing on pain mechanisms, clinical features, diagnostic challenges, and individualized treatment strategies. Although both disorders manifest as musculoskeletal pain syndromes with overlapping features, PMR is characterized by inflammation and glucocorticoid responsiveness, while FM is dominated by central sensitization mechanisms. This article emphasizes the importance of differentiating between the two for accurate diagnosis and tailored therapy.(**International Journal of Biomedicine. 2026;16(2):145-150.**)

Keywords: polymyalgia rheumatica • fibromyalgia • central sensitization • inflammation • glucocorticoids • differential diagnosis

Main Points

- PMR and FM share symptoms but differ in that PMR is inflammatory, and FM is centrally mediated.
- Distinguishing PMR from FM is very important to avoid misdiagnosis and inappropriate glucocorticoid use.
- Diagnosis depends on symptoms, inflammation markers, imaging (for PMR), and classification criteria.
- PMR is treated with glucocorticoids, while FM requires neuromodulators and non-drug therapies.
- PMR and FM can coexist, especially in older adults, requiring tailored multidisciplinary care.

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Abbreviations

CBT, cognitive behavioral therapy; **CRP**, C-reactive protein; **ESR**, erythrocyte sedimentation rate; **FM**, fibromyalgia; **FMS**, fibromyalgia syndrome; **GCA**, giant cell arteritis; **PMR**, polymyalgia rheumatica; **RA**, rheumatoid arthritis.

Introduction

Fibromyalgia (FM) is a chronic condition marked by pervasive musculoskeletal pain, accompanied by symptoms that significantly impact daily activities and diminish quality of life. The hallmark symptoms of FM encompass pervasive pain throughout the body, debilitating fatigue, sleep disturbances, and cognitive impairment, often termed "fibro-fog." Alongside psychological issues such as anxiety and depression, patients frequently endure headaches, irritable bowel syndrome, muscular and joint stiffness, and heightened pain sensitivity (allodynia and hyperalgesia). It is estimated that between 0.2% and 6.6% of the global population is afflicted by this condition, with a marked prevalence among women compared to men. A significant percentage of patients with inflammatory rheumatic diseases meet the fibromyalgia syndrome (FMS) criteria, complicating diagnosis, treatment, and follow-up. The coexistence of FMS may lead to superfluous laboratory and radiological evaluations.[1,2]

Polymyalgia rheumatica (PMR) is the most common inflammatory rheumatological disease in adults over 50,

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occurring 2-3 times more frequently in women. PMR results in significant disabilities if inadequately managed. Symptoms include muscle rigidity and discomfort, predominantly in the cervical or shoulder regions and pelvic girdle, along with low-grade fever, depression, fatigue, anorexia, and weight reduction. Individuals affected often experience significant mobility restrictions due to discomfort, typically exacerbated in the mornings or after periods of inactivity. PMR is distinguished by increased inflammatory markers, including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). It is typically administered with medium or low doses of oral glucocorticoids. The diagnosis of PMR depends on a synthesis of clinical assessment, medical history, physical examination, and laboratory tests. To diagnose PMR, conditions that may elicit similar symptoms must be excluded, according to the guidelines set forth by the American College of Rheumatology and the European League Against Rheumatism. The core exclusion disorders encompass infections, cancer, and giant cell arteritis (GCA), along with FM, hypothyroidism, rheumatoid arthritis, and drug-induced myalgia.[3]

The symptoms of FM and PMR often coincide, leading to potential misinterpretation or misdiagnosis, especially in elderly individuals presenting with fatigue and musculoskeletal pain. Muscle pain, stiffness, and fatigue are prevalent symptoms of both conditions, and distinguishing between them can be challenging because FM lacks definitive diagnostic tests, and symptoms vary. Certain criteria facilitate the differentiation between PMR and other pain disorders, such as FM, which typically manifests at a younger age than PMR. Furthermore, laboratory markers typically do not show elevation to indicate an inflammatory condition in FM. It is essential to differentiate between PMR and FM for accurate diagnosis and effective treatment, as well as to prevent the unwarranted administration of glucocorticoids in FM patients.[4]

This review paper examines pain mechanisms, clinical features, diagnostic techniques, and individualized therapies for PMR and FM. Furthermore, to demonstrate the similarities, intersections, and distinctions between the two conditions.

Pain Mechanisms

Polymyalgia Rheumatica

Common symptoms may include fatigue, fever, and weight loss. Pain is induced by systemic inflammation, primarily involving IL-6 and various cytokines. In certain studies, arthroscopic biopsies from the glenohumeral joints of untreated patients with PMR demonstrated synovitis characterized by leukocyte infiltration and vascular proliferation. Proximal pain is associated with synovitis and bursitis in the hip and shoulder regions. Most infiltrating cells were macrophages and memory T cells, with a minor presence of B cells. T cells exhibited elevated levels of major histocompatibility complex class II molecules, indicating activation. Inflamed tissues contain activated macrophages and T cells that sensitize peripheral nociceptors. Recent studies suggest that PMR patients experiencing persistent pain may exhibit low-grade central sensitization. Vascular endothelial activation may contribute to pathogenesis, as elevated expression of vascular endothelial growth factor (VEGF) in synovial biopsies is associated with GCA in 10-20% of cases, with vascular inflammation exacerbating pain. Elevated expression of adhesion molecules in endothelial and synovial lining cells may facilitate the recruitment of inflammatory cells to these lesions. Levels of vasoactive intestinal peptide (VIP) were elevated in the shoulder synovium of patients with PMR compared to those with rheumatoid arthritis (RA) or osteoarthritis. Nociception linked to local VIP production may contribute to the distinctive shoulder pain in PMR. A recent observation indicates that PMR may manifest as an adverse effect of cancer therapy utilizing checkpoint inhibitors.[5-9]

Fibromyalgia

Fibromyalgia is predominantly attributed to heightened pain signaling within the central nervous system. Neuroimaging research indicates modified brain connectivity in pain-processing areas, such as the insula and anterior cingulate cortex (10). Discrepancies in serotonin, norepinephrine, and substance P all enhance pain perception. Emerging evidence indicates low-grade inflammation and small-fiber neuropathy in certain FM patients, although it is less common than in polymyalgia rheumatica. Fibromyalgia-related pain has been linked to modifications in brain regions involved in pain processing, reduced activity of pain-inhibitory pathways, and heightened activity of pain-facilitating pathways. The precise pathogenetic mechanism underlying FM pain remains incompletely elucidated, yet it is thought to engage multiple peripheral and central pathways. Conditions such as FM can lead to sustained activation of glial cells and the ensuing release of proinflammatory agents.[10]

Thus, PMR pain is primarily inflammatory and peripheral, driven by cytokine-mediated activation of nociceptors. FM pain is centralized, with minimal inflammatory contribution, focusing on CNS amplification and neurochemical changes.

Clinical Differences

Polymyalgia rheumatica

The predominant symptoms include proximal pain and morning stiffness in the pelvic girdle and shoulders persisting for over 45 minutes, which may be acute or progress over several days to weeks, a swift response to glucocorticoids, and elevated ESR/CRP levels. Common symptoms may encompass fatigue, fever, and weight reduction. The nonspecific clinical presentation and absence of distinctive laboratory or serologic findings often result in diagnostic delays. PMR imposes a considerable burden on the daily lives of elderly individuals.[3,4]

Fibromyalgia

Fibromyalgia symptoms are persistent functional manifestations. Individuals exhibit a spectrum of symptoms and severity, ranging from mild and intermittent to persistent and debilitating. Clinical manifestations encompass diffuse pain, tender points, fatigue, sleep disturbances, and non-restorative sleep, alongside a form of cognitive impairment characterized by concentration difficulties and mental fog (“fibro fog”); headaches (e.g., migraines), paresthesia, memory deficits, abdominal discomfort or cramps, autonomic dysregulation,

xerostomia, xerophthalmia, visual disturbances, restless leg syndrome, and normal inflammatory markers. Fibromyalgia and other conditions with overlapping symptoms are not mutually exclusive and may coexist simultaneously.[2,5]

Demographics

Polymyalgia rheumatica

Polymyalgia rheumatica is the most common inflammatory rheumatic condition in individuals over 50, with a prevalence in women that is 2-3 times greater than in men. In Sweden, the prevalence of PMR among individuals aged 50 and older varies from 34 to 50 per 100,000. One review established that PMR is more prevalent among individuals of Northern European descent, while GCA and PMR are less prevalent in Asian populations. Polymyalgia rheumatica is 2 to 3 times more prevalent than GCA and manifests in approximately 50% of individuals diagnosed with GCA. Polymyalgia rheumatica may precede, coincide with, or succeed GCA.[4,11-13]

Fibromyalgia

This disease impacts individuals across all age groups and typically exacerbates with advancing age, reaching its zenith between 50 and 60 years old. Women are three times more likely to be affected than men. Global estimates vary from 0.2% to 6.6%. It is the third most prevalent musculoskeletal condition, following lumbar pain and osteoarthritis. The prevalence in Europe is 2.31%. Countries like the United States, Canada, and Japan report prevalence rates of 6.4%, 1.5%, and 2.1%, respectively. Y. Bawazir conducted a systematic review and meta-analysis regarding the prevalence of FMS in Saudi Arabia.[14] It disclosed that FMS is more prevalent among women, especially in Saudi Arabia, where its occurrence is 13.4%. Factors correlated with a heightened probability of developing FM encompass diagnoses of major depressive disorder, irritable bowel syndrome, and restless legs syndrome. A study conducted by AlOmair et al.[15] revealed that 15.2% (n= 47) of the 310 seropositive RA patients met the diagnostic criteria for FMS.[2,14,15]

Diagnostic Approaches

Polymyalgia Rheumatica

The diagnosis of PMR is difficult because there are no definitive findings on clinical evaluation, laboratory analysis, or imaging to validate the condition. Increased ESR (>40 mm/h) and CRP are prevalent but may be normal in 10-20% of instances. Ultrasound or MRI can identify bursitis or synovitis; PET-CT may be used to assess for GCA association; and a response to glucocorticoids (e.g., prednisone 15-20 mg/day) can serve as a diagnostic criterion. Chuang et al. and Healey et al. delineated diagnostic criteria for PMR, with the latter incorporating a swift response to less than 20 mg/day of prednisolone. Nevertheless, these criteria are not extensively utilized in standard clinical practice. The American College of Rheumatology and the European League Against Rheumatism recommend excluding conditions that mimic PMR symptoms, including core exclusion conditions (GCA, cancer, infections), rheumatoid arthritis (RA), FM, hypothyroidism, and drug-induced myalgia. The American College of Rheumatology and the European League Against Rheumatism recommend excluding conditions that mimic the symptoms of PMR, including core exclusion conditions (GCA, cancer, infections), RA, FM, hypothyroidism, and drug-induced myalgia. In 2012, the European League Against Rheumatism (EULAR) proposed new classification criteria for PMR. The American College of Rheumatology (ACR) developed a scoring algorithm incorporating factors such as morning stiffness exceeding 45 minutes (2 points), hip pain or restricted range of motion (1 point), absence of rheumatoid factor and/or anti-citrullinated protein antibodies (ACPA) (2 points), and absence of peripheral joint pain (1 point). A score of ≥4 demonstrates 68% sensitivity and 78% specificity for PMR. The inclusion of ultrasound results yields a score of ≥5, increasing sensitivity to 66% and specificity to 81%. Patients aged 50 and above presenting with bilateral shoulder pain of unexplained origin may be diagnosed with PMR if they exhibit morning stiffness exceeding 45 minutes, elevated CRP and/or ESR, and newly developed hip pain. These classification criteria mitigate heterogeneity among positive cases by emphasizing typical manifestations, thereby reducing the likelihood of false-positive diagnoses. Nonetheless, they may not be applicable in atypical instances of PMR. The majority of diagnostic criteria for PMR exhibit low sensitivity and specificity and were formulated for populations with a high prevalence of the disorder. Consequently, comprehensive clinical assessment and the exclusion of alternative potential causes are imperative for precise diagnosis. The functions of imaging studies in the diagnosis, treatment, and prognosis of PMR are inadequately documented and necessitate further elucidation.[16-20]

Fibromyalgia

The diagnosis is predicated on the persistence and extensive distribution of pain. The pain must persist for at least 3 consecutive months and be widespread. Sleep disturbances, fatigue, and mood disorders are typically evident. The diagnosis is established only after a comprehensive history and clinical examination have excluded peripheral pain of structural, inflammatory, or neuropathic origin, and after specific laboratory tests and clinical evaluations have excluded other conditions, such as normal inflammatory markers and negative autoimmune serologies. In 1990, the American College of Rheumatology (ACR) published the inaugural diagnostic criteria for fibromyalgia. In 2010, the ACR acknowledged "The American College of Rheumatology Preliminary Diagnostic Criteria for Fibromyalgia and Measurement of Symptom Severity" as "an alternative diagnostic method." The 1990 classification and 2010 diagnostic criteria were formulated among rheumatology patients and were physician-centric. Due to its nonspecific nature, it is no longer recommended to assess fibromyalgia based on pain at 11 of the 18 tender points. The tender point count relies on the patient's self-report, the physician examiner's assessment and interpretation, and the natural decrease in pain threshold. Gracely et al. characterized the tender count as "some unspecified combination of tenderness and distress" and referred to it as "a sedimentation rate for distress."[11] The 2011 criteria incorporated a fibromyalgia

severity (FS) score, derived from the sum of the Widespread Pain Index (WPI) and the Symptom Severity Scale (SSS), facilitating a quantitative evaluation of fibromyalgia symptom severity. Application of the 2010/2011 criteria to regional pain syndromes resulted in misclassification. Nevertheless, the incorporation of a modified widespread pain criterion, termed the "generalized pain criterion," eradicated misclassification. Fibromyalgia and other conditions with overlapping symptoms are not mutually exclusive and may coexist simultaneously. Consider testing for alternative conditions in patients with the appropriate history and distinctive clinical features. ACR 2016 criteria, highlighting the intensity of symptoms and extensive pain. This is an update to the fibromyalgia criteria from 2010 and 2011. Emerging tools for assessing fibromyalgia patients encompass Quantitative Sensory Testing and neuroimaging for research applications. A diagnosis of fibromyalgia is legitimate regardless of other diagnoses.[21-23] Adults can now be diagnosed with fibromyalgia if they meet all of the following criteria:

- There is widespread pain, which is characterized by pain in at least four out of five areas.
- For at least three months, the symptoms have persisted at a comparable level.
- Symptom severity scale (SSS) score ≥5 and widespread pain index (WPI) ≥7 or SSS score ≥9 and WPI of 4-6.
- A diagnosis of fibromyalgia is valid irrespective of other diagnoses. A diagnosis of fibromyalgia does not exclude the presence of other clinically important illnesses.

Diagnostic Challenges

Overlap: Both may present stiffness, fatigue, and generalized pain, leading to misdiagnosis.

Tools: Polymyalgia rheumatica diagnosis relies on clinical criteria (e.g., EULAR/ACR 2012 classification) and inflammatory markers. FM diagnosis uses ACR 2016 criteria (widespread pain index, symptom severity scale).

Comorbidity: Fibromyalgia may coexist with PMR, complicating pain assessment. Up to 20% of PMR patients may meet FM criteria. Yu Yokota et al reported a 92-year-old woman with polymyalgia rheumatism who had been treated with prednisolone for 18 years and was later diagnosed with coexisting fibromyalgia based on two physical examinations and her personal history of general pain. Pregabalin was prescribed, and her pain gradually disappeared.[24]

Differentiation

Inflammatory markers and glucocorticoid response are key to distinguishing PMR from FM. Fibromyalgia requires assessment of non-pain symptoms (headaches, paresthesia, memory deficits, abdominal discomfort or cramps, autonomic dysregulation, xerostomia, xerophthalmia, visual disturbances, restless leg syndrome) to ensure an accurate diagnosis. Table 1 for summarizing diagnostic features of PMR vs. FM.

Table 1.

Diagnostic Features of PMR vs. FM.

Diagnostic Feature	PMR	FM
Inflammatory markers	Elevated CRP, ESR	Normal
Age relevance	>50 years	Any age (peak 50–60)
Imaging	Ultrasound, MRI (bursitis/synovitis)	Not useful clinically
Classification criteria	EULAR/ACR 2012	ACR 2016 (WPI + SSS)
Response to glucocorticoids	Rapid and significant	None

Treatment Approaches

Polymyalgia Rheumatica

The management of PMR emphasizes symptom relief, reduction in inflammation, and enhancement of overall function. Corticosteroid therapy is the foundation of PMR treatment. Nonsteroidal anti-inflammatory drugs (NSAIDs) may be prescribed to augment corticosteroid therapy and alleviate symptoms. Physical therapy is crucial for managing PMR. Glucocorticoids generally elicit a rapid response to all PMR symptoms. The latest ACR/EULAR guideline for the initial daily dosage of prednisolone in PMR is 12.5–25 mg. The diagnosis is corroborated by the patient's rapid response to treatment and alleviation of pain within 1-2 weeks. It is recommended to gradually reduce dosage over a period of 6 to 12 months to mitigate side effects. A suboptimal treatment response should necessitate a reevaluation of alternative diagnoses, especially malignancy or other rheumatic disorders. To mitigate long-term toxicity, supportive measures must be employed alongside GC treatment. Calcium, vitamin D, and bisphosphonate supplements are utilized to prevent osteoporosis. A daily dosage of 10 mg or greater is associated with an increased risk of injury. When evaluating the extent of harm associated with a daily dose of 5–10 mg, it is essential to consider patient characteristics and comorbidities. The prognosis for PMR is generally positive, and the condition usually resolves within a few years. A meta-analysis by Floris et al. included 21 studies, revealing that 77%, 51%, and 25% of participants remained on glucocorticoids at 1, 2, and 5 years post-diagnosis, respectively. While further research is necessary, intramuscular MP is probably an effective alternative to oral GC for specific patients, including the elderly and those with adherence issues. Patients with PMR necessitate GC-sparing strategies due to the myriad adverse effects associated with prolonged GC therapy. The ACR/EULAR panel conditionally recommended the early initiation of MTX, particularly for patients at elevated risk of relapse and/or those necessitating extended treatment. Numerous case reports and series indicate the effectiveness of intravenous tocilizumab in individual patients with PMR. TNF inhibitors are contraindicated for the management of PMR. The most prevalent comorbidity reported following a PMR diagnosis is vascular disease. The vascular disorders encompass strokes, myocardial infarction, and peripheral vascular disease.[25-28] Table 2 presents a treatment comparison of PMR and FM.

Table 2.

Treatment Comparison of PMR and FM.

Treatment Feature	PMR	FM
First-line therapy	Glucocorticoids (prednisone)	SNRIs, anticonvulsants, TCAs
Treatment response time	Rapid (days)	Gradual (weeks to months)
Non-pharmacologic approaches	Physical therapy, taper monitoring	CBT, exercise, sleep hygiene
Other medications	Methotrexate, tocilizumab	Low-dose naltrexone, ketamine (experimental)
Steroid use	Essential for remission	Ineffective and not recommended
Comorbidity focus	Osteoporosis, GCA, vascular risk	Depression, anxiety, IBS, sleep disorders

Fibromyalgia

Individualized and multidisciplinary treatment is necessary. It is crucial to manage comorbid conditions like psychiatric disorders and sleep disturbances and to educate patients. Aerobic cardiovascular fitness exercises are beneficial. For certain patients, tai chi and/or water exercise may be more beneficial. FDA-approved drugs include a dual uptake inhibitor, such as duloxetine 20 to 30 mg at breakfast, gradually increased to 60 mg/day, or milnacipran 12.5 mg each morning, gradually increased as tolerated to 50 mg twice daily. Pregabalin (anticonvulsant) for centralized pain taken at bedtime. Treatment is initiated at a dose of 25 to 50 mg at bedtime and is adjusted upwards as tolerated to 300 to 450 mg/day. Gabapentin is an acceptable alternative for patients for whom cost or regulatory constraints limit the availability of pregabalin. Amitriptyline (low dose) and/or cyclobenzaprine can also be used. Emerging treatment low-dose naltrexone (1-4.5 mg/day) shows promise in reducing pain and fatigue. Lidocaine infusions could give an alternative option for patients who have not responded well to conventional therapies. A 2024 systematic review by de Carvalho and de Sena[30] demonstrates the effectiveness and safety of ketamine in FM patients in the short term. More studies, including long-term follow-up studies, are still needed. Recently, the FDA approved cyclobenzaprine HCL sublingual (Tonmya) as a treatment for fibromyalgia in adults. Non-pharmacological options also include cognitive behavioral therapy (CBT) and mindfulness-based stress reduction for pain coping.[29-31]

Comparison

PMR treatment targets inflammation, with a rapid response to steroids. FM treatment focuses on neuromodulation and symptom management, with a slower, variable response. Overlap management: If PMR and FM coexist, combine anti-inflammatory and neuromodulation strategies.

Conclusion

It is important to diagnose both FM and PMR accurately and early. Additionally, clinicians should be aware of the distinct pain mechanisms in both conditions (inflammatory vs. central), differences in clinical features, and the possibility of both conditions coexisting. Tailored therapy is needed to improve the outcomes. Integrating rheumatology, pain medicine, and psychology is needed for holistic management. Longitudinal studies to understand the development of FM in PMR patients are needed.

Author Contribution Statement

Abdulrahman Ali M. Khormi confirms sole responsibility for all aspects of the research.

Conflict of Interest

The author has declared no conflict of interest.

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