Review

Pain in myositis: prevalence, assessment, potential mechanisms and considerations for management

S. Glaubitz[1], Y.C. Lee[2], D. Saygin[3]

[1]Department of Neurology, Neuromuscular Center, University Medical Center Göttingen, Germany;
[2]Department of Medicine, Division of Rheumatology, Northwestern University, Chicago, IL, USA;
[3]Department of Medicine, Division of Rheumatology, Rush University Medical Center, Chicago, IL, USA.

Stefanie Glaubitz, MD*
Yvonne C. Lee, MD, MMSc
Didem Saygin, MD

*S. Glaubitz is a member of the European Reference Network for Rare Neuromuscular Diseases (ERN EURO-NMD).

Please address correspondence to:
Didem Saygin
Department of Medicine,
Division of Rheumatology,
Rush University Medical Center,
1611 W Harrison Street, Suite 510,
Chicago, IL 60612, USA.
E-mail: didem_saygin@rush.edu

Received on September 30, 2025; accepted in revised form on January 19, 2026.

Clin Exp Rheumatol 2026; 44: 000-000.

Key words: pain, myalgia, myositis, muscle pain, inflammatory myopathies

Competing interests: S. Glaubitz has received speakers' honorariafrom Argenx, Alexion and UCB, and travel expenses from Alexion, Bayer, Grifols, Octapharma and UCB. Y.C. Lee served on an advisory board for Tonix Pharmaceuticals and holds stock in GE Healthcare. She previously held stock in CVS Health. D. Saygin has served as consultant for ArgenX (with no direct payment).

ABSTRACT

Pain is a prevalent symptom experienced by over 80% of patients with idiopathic inflammatory myopathies (IIM). Pain severity ranges between 3 to 4 (out of 10) assessed by various tools in the literature. Myositis-related pain could be affected by underlying disease activity and other biological mechanisms, including muscle and joint inflammation, fasciitis, and central pain sensitisation. Pain is associated with a high disease activity, fatigue and poor functional outcomes among patients with IIM. In this narrative review, we provide an overview of the current knowledge on pain and pain assessment tools in IIM and discuss potential mechanisms underlying myositis-related pain and considerations for management.

Introduction

Idiopathic inflammatory myopathies (IIM) are a rare, heterogeneous group of systemic autoimmune diseases primarily characterised by immune-mediated inflammation of the skeletal muscles. IIM subgroups include dermatomyositis (DM), polymyositis (PM), inclusion body myositis (IBM), immune-mediated necrotising myopathy (IMNM), overlap myositis (OM), anti-synthetase syndrome (ASyS) and juvenile myositis (JM) (1, 2). As a systemic disease, IIM frequently presents with a range of extra-muscular symptoms, including interstitial lung disease, rash, and arthritis besides muscle involvement (3, 4).

IIM can lead to a substantial morbidity, particularly long-term functional limitations due to decreased muscle strength and/or endurance (5-7). Therefore, the clinical care of these patients has historically focused on muscle weakness as the primary symptom of patients living with IIM. However, increasing evidence highlights symptoms other than muscle weakness that contributes to poor health-related quality of life among patients with IIM (8, 9). One of the key symptoms that contributes to poor health-related quality of life among patients with IIM is pain (10). For many years, pain has been considered as rare or absent in the clinical course of myositis. However, multiple international surveys, qualitative interviews and focus group studies over the last decade repeatedly show that the pain is not only common, but also one of the most important symptoms of the disease for patients with IIM (10-13). Interestingly, pain was one of the significant drivers of discordance between physician- and patient-reported assessments of global disease activity in IIM (14). This finding suggests that pain likely continues to be an overlooked symptom by clinicians in routine clinical practice. Further, pain is not being adequately captured or reported in myositis clinical trials (15).

The primary aim of this narrative review is to provide a comprehensive overview of pain in myositis to advance the knowledge about myositis-related pain among clinicians caring for patients with myositis. First, we review the prevalence and intensity of pain among patients with IIM. Second, we summarise pain assessment tools and potential underlying pathophysiological mechanisms that may lead to pain in myositis. Finally, we provide considerations for management of myositis-related pain with the aim of providing patient-centred care.

Prevalence of pain in IIM

Clinical studies across various patient groups consistently report prevalence of pain as over 80% ranging from 64% (16) to 86% among patients with IIM (13). For instance, a randomised con-

trolled trial (RCT) including patients with newly diagnosed myositis showed that 81% of patients reported muscle pain at baseline (17). Similar findings were also observed in a US-based prospective registry study with 86% of patients with IIM reporting pain (13). Two studies reported the frequency of muscle and joint pain separately (13,18). The first study reported similar rates of muscle and joints pains at 61.7% and 63.2% (13), while the second study reported higher rates of muscle pains (48%) than joint pains (28%) among patients with IIM (18). This variability could be due to differences in the cohort characteristics and how these questions were asked, as it could be hard for patients to precisely pinpoint if their pain is localized to their joints or muscles.

Current evidence suggests that most patients affected by IIM experience pain at some timepoint during the course of their disease. One international survey reported that over 91% of patients had pain at some point, with almost all attributing their pain to their myositis (19). However, even though pain is common among patients with IIM, it is rarely the sole presenting symptom. Presentation of a patient with myalgias without accompanying muscle weakness or other relevant symptoms should raise the concern for non-IIM diseases.

Pain assessment in myositis

The intensity of pain experienced by patients with IIM has been reported in several studies using different scales (11, 13, 16-18, 20-41) (Table I). Most studies used unidimensional scales, such as the visual analogue scale (VAS) or numerical rating scale (NRS) (13, 17,1 8, 23, 25, 29, 35, 36, 38-44). With VAS, patients are asked to rate their overall pain by placing a mark on a horizontal line in a paper form or using a slider scale on an electronic form. These scales often range from 0 to 10 centimetres (cm) with no pain at one end and the worst imaginable pain at the other end. In contrast, NRS asks patients to choose a discrete number from 0 to 10, often with annotations provided to aid participants with interpretation of different points in the scale. The recall period for both scales is not standardised and unfortunately not frequently specified in studies which limits its comparability between the studies. The most common recall period used in pain scales are 24-hours and 7 days ("past week").

Other questionnaires used in the assessment of pain in IIM include the Short Form 36 Bodily Pain (SF-36 BP) (17, 18, 20-23, 25, 26, 28-30, 34, 39, 45-47), the Health Assessment Questionnaire Pain VAS (HAQ) (16), the Individualised Neuromuscular Quality of Life Questionnaire (INQOL) (33) and the Nottingham Health Profile (NHP) (31, 37):

- SF-36 BP is one of eight subscales of SF-36 health-related quality of life tool which includes two questions on pain (48). These questions ask participants to categorise their level of pain from "none" to "very severe" and their level of pain interference from "not at all" to "extremely" over the past 4 weeks. The scores for SF-36 BP range from 0 to 100 with higher scores indicating less bodily pain.
- HAQ is a patient reported outcome measure that primarily intends to assess physical function but also includes a separate 100 mm VAS that asks the participants to mark their level of pain over the past week (49). Higher scores indicate higher levels of pain.
- INQOL is a patient reported outcome measure that assesses health-related quality of life in patients with neuromuscular diseases (50). It consists of 45 items within 11 subscales, one of which is pain with 3 items. These items focus on both the severity of pain as well as the impact of the pain on daily life and emotions. Each item is rated on a 7-point Likert scale ranging from "not at all" to "extremely". The recall period is two weeks. The scores range from 0 to 100 with higher scores indicating a higher pain severity and impact.
- NHP is another patient reported outcome measure that aims to assess health-related quality of life "at the present time" (51). Pain is one of six sections of the NHP and includes eight items related to impact of pain with each item answered as yes or no. The scores range from 0 to 100 with higher scores indicating more pain-related problems.

Even though all of these tools have been used to report on myositis-related pain in literature, the measurement properties of these tools have not been studied in patients with IIM. Currently, the most well-studied patient reported outcome measure for pain assessment in patients with IIM is PROMIS Pain Interference 6a, v1. The tool includes six questions focusing on the impact of pain on different aspects of one's life in the past 7 days. Response to each question is a Likert scale ranging from "not at all" to "very much". The raw score obtained from the PROMIS tool gets converted to a standardised T score based on U.S. general population. A T score of 50 represents the population mean with higher scores indicating higher pain interference. The content validity, construct validity, test-retest reliability and responsiveness of this tool have been established in adults with IIM by the OMERACT Myositis Working Group (52, 53).

Pain intensity in myositis

The mean pain intensity reported by patients with IIM range between 1.3–3.9 (out of 10) (13, 18, 23, 36). Using the SF-36 BP questionnaire (0-100), mean pain intensity ranged from 42 to 88 in cohorts with stable or no disease activity (23, 54).

A number of studies compared the intensity of pain experienced by patients with myositis with the general population (11, 20-22, 25-31, 54). In the majority of studies, patients with IIM reported higher levels of pain than the general population (20, 21, 26, 27, 29, 30, 54).

Several studies also compared pain intensity reported by myositis patients with other neuromuscular and rheumatic diseases (11, 22, 25, 30, 31, 33). With regard to neuromuscular diseases, IBM appears to have a comparable pain intensity to facioscapulohumeral dystrophy, Charcot-Marie-Tooth type 1

Table I. Characteristics of the instruments used in IIM studies to assess pain.

Instrument	Type	Items/format	Recall period	Scoring range and direction
Visual Analogue Scale (VAS)	Unidimensional pain intensity scale	Single horizontal line marked by patient (paper/electronic)	Not standardised (commonly 24h or 7 days)	0–10 cm or 0–100 mm; higher = more pain intensity
Numerical Rating Scale (NRS)	Unidimensional pain intensity scale	Selection of a discrete number (verbal/paper/electronic)	Not standardised (commonly 24h or 7 days)	Higher = more pain intensity
SF-36 Bodily Pain (SF-36 BP)	Subscale of HRQoL PROM	2 questions (pain intensity and interference)	4 weeks	0–100 mm; higher = less pain intensity and interference
Health Assessment Questionnaire (HAQ) – Pain VAS	Physical function PROM with an additional VAS for pain intensity	Single horizontal 100 cm line	1 week	Higher = more pain intensity
Individualised Neuromuscular Quality of Life Questionnaire (INQOL) – Pain Subscale	Subscale of neuromuscular disease specific HRQoL PROM	3 items on severity & impact of pain; 7-point Likert	2 weeks	0–100; higher = more pain severity and impact
Nottingham Health Profile (NHP) – Pain Section	Subscale of HRQoL PROM	8 items with yes/no answers on pain impact	"At present" (point-in-time)	0–100; higher = more pain-related problems
PROMIS Pain Interference 6a v1	PROMIS measure of pain impact, validated in IIM	6 items; Likert responses ("not at all" to "very much")	Past 7 days	Higher = more pain interference (T-scores available)

PROM: patient reported outcome measure; HRQoL: health related quality of life; IIM: idiopathic inflammatory myopathies.

and myotonic dystrophies (22), whereas patients with DM and PM report higher pain intensities than those with facioscapulohumeral dystrophy, limb-girdle muscular dystrophy and myotonic dystrophies (33). IIM patients had pain levels significantly lower than those with rheumatoid arthritis and osteoarthritis in one study (31) and comparable to those with rheumatoid arthritis and systemic lupus erythematosus in other studies (25, 30).

Pain intensity and prevalence in different subgroups of myositis

Several studies compared pain intensity, prevalence or burden among different IIM subgroups (18, 19, 21, 26, 33, 34, 36, 54). While the majority of studies found no differences between the IIM subgroups (21, 26, 34, 54), some studies showed significant differences (18, 19, 33, 36). It is important to cautiously interpret these findings due to significant differences between studies on the criteria used to subclassify these patients. For example, IMNM was possibly included under PM in studies that used Bohan-Peter or 2017 EULAR-ACR criteria, and ASyS was likely included under DM or other subgroups given that the majority of the criteria do not recognize these diseases as distinct IIM subgroups.

In general, patients with IBM consistently reported lower pain levels than other IIM subgroups. In one study, IBM patients reported average pain levels of 22±27 on a 100-mm VAS, compared to patients with PM (39±29), DM (37±28) and OM (38±33) (36). The prevalence of pain was the lowest among IBM patients (80.9%), compared to patients with DM (97.2%) and PM (94.5%) (19). Similarly, pain-related problems were also lower in IBM patients than in patients with DM/PM with mean INQOL scores of 46 *versus* 70, respectively (33). The literature seems to be relatively unanimous regarding the lower pain levels in patients with IBM compared to other IIM subgroups.

Studies on pain levels between different IIM subgroups other than IBM showed variable results. One study showed patients with OM were more likely to cluster with higher pain interference group than patients with DM, PM, ASyS, and IMNM (32). Another study showed that patients with OM and ASyS had higher pain levels than DM, PM, IMNM and IBM (11). A higher prevalence of pain was noted in patients with DM compared to those with PM in one study (19), while another study showed comparable pain levels between patients with PM, DM and OM (36). More frequent joint involvement in OM and a higher rate of fasciitis in DM (55) could explain the higher pain intensity in patients with OM and DM, respectively. However, the results of the studies to date are mixed which could be due to small sample size for each subtype, differences in disease activity levels of patients enrolled as well as the classification criteria used in these studies.

Association between pain, fatigue, physical function and quality of life among patients with IIM

Pain is often associated with poor outcomes including worse fatigue, physical functioning and health-related quality of life among patients with IIM (11, 13, 18). Higher levels of pain intensity were consistently associated with higher levels of fatigue and poorer performance across several outcome measures, at baseline and over time (18). IIM patients with pain levels higher than 3 out of 10 had significantly worse fatigue, functional impairment, lower scores in both the physical and mental components of the SF-36 indicating worse health-related quality of life and lower health satisfaction scores than patients with pain levels <3 (13).

In a large international cohort, nearly 70% of patients with IIM reported pain that interferes with their daily activities. More than 90% of patients indicated that their pain affected their daily functioning (32). Household chores

were the most commonly affected activity, with 94.5% of patients with IIM reporting some degree of disruption. This was followed by disruption of general day-to-day activities in 91.3% of patients, and social engagement in 78.3%. Pain interference was strongly correlated with fatigue and physical function levels. These findings highlight that pain is a burdensome symptom among patients with IIM, and although the intensity of pain experienced by patients with myositis may vary, a significant proportion of patients experience levels of pain that may have a substantial impact on their quality of life and daily activities.

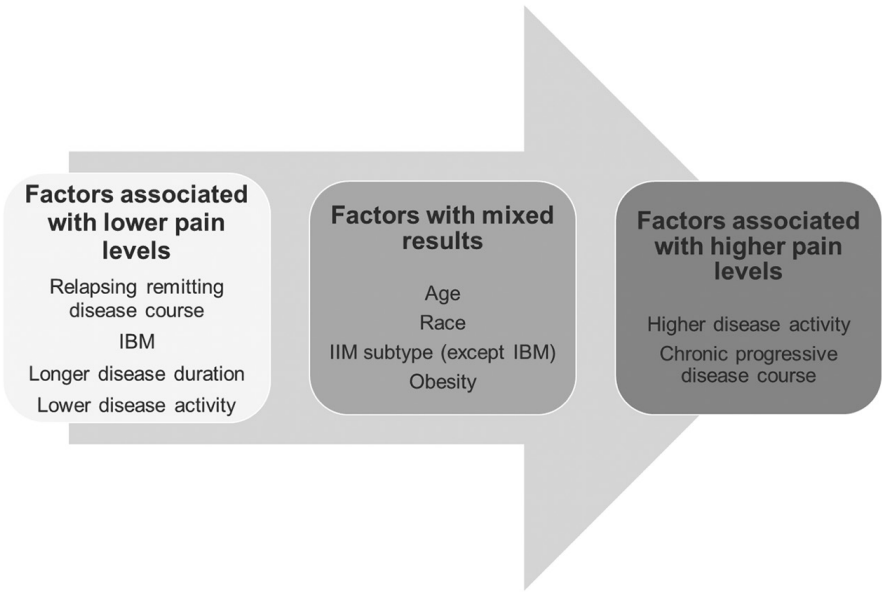

Fig. 1. Factors associated with pain levels among patients with IIM.

Factors associated with pain in IIM

The experience and intensity of pain in myositis are likely influenced by a complex interplay of clinical and disease-specific factors (56). Several studies examined factors that contribute to the frequency or severity of myositis-related pain with no definitive conclusions on these factors except disease activity (Fig. 1). For instance, results regarding the impact of age on pain are variable. Older age appears to be a relevant factor in myositis-related pain, with older individuals reporting greater pain intensity, regardless of their disease activity status (11). In contrast, younger age has also been identified as a risk factor for pain in studies. For example, one study found that IIM patients over 60 years of age were significantly less likely to report pain (19), while another study found that younger age was associated with a higher risk of experiencing more severe pain in the univariate analysis. However, this was not confirmed in the multivariate analysis (13). Furthermore, no correlation was found between pain and age in further studies (22, 27).

Sex-based differences remain inconsistent across the studies as well. While some studies reported significantly higher pain levels among women (21), others found no differences between men and women with IIM (13, 26). Women are generally thought to suffer from pain more commonly than men due to differences in the genetic, cellular and molecular mechanisms involved in the processing of acute and chronic pain (57); however, further studies are required to better understand this relationship in patients with IIM.

Studies on the impact of disease course on pain show that chronic progressive disease course is associated with significantly more severe pain than relapsing-remitting course in patients with IIM (20), while another study found that active myositis is associated with higher levels of pain (16). These results are in line with other studies that report close association between disease activity and pain levels even after accounting for several factors including age, sex, race, income, education level, body mass index, and comorbidities such as osteoarthritis, depression and anxiety (32).

The heterogeneity of findings between studies highlights the complex and subjective nature of pain experience and pain reporting behaviour among patients with IIM and suggests that the subjective burden of pain may depend less on subgroup classification and more on patient-perceived disease activity and other unknown factors.

Potential mechanisms of myositis-related pain

The International Association for the Study of Pain (IASP) defines three pain mechanism categories: nociceptive, neuropathic and nociplastic (58). Nociceptive pain arises from damage to non-neuronal tissues due to stimuli such as inflammation, while neuropathic pain is caused by nerve damage. A more recently recognised pain category, nociplastic pain, results from altered central pain processing rather than tissue or nerve damage (59). Central sensitisation, which is characterised by increased excitability of neurons in the central nervous system, is one of the primary mechanisms underlying nociplastic pain (60).

The mechanisms underlying pain in myositis remain incompletely understood. Herein, we provide an overview of the potential factors that may contribute to the development of pain in myositis. In IIM, pain likely arises from a complex interplay of immune-mediated inflammation and non-immune tissue damage (nociceptive pain) and sensitisation of nociceptive pathways (nociplastic pain) in muscles and joints in patients with inflammatory arthritis as a manifestation of IIM.

Nociceptive pain. Muscle pain is perceived through the activation of specialised receptors known as nociceptors. These receptors are free nerve endings that connect to the nervous system via unmyelinated (group IV) or thinly myelinated (group III) fibres (61, 62). Activation of these receptors can occur through a strong mechanical stimulus,

as well as through endogenous mediators such as bradykinin, serotonin (63), and prostaglandins (64), which are typically found in inflammatory tissues. ATP (65), which is present in all cells of the body and particularly abundant in muscle fibres, can be released upon any type of tissue injury and activate nociceptors. Similarly, acidic pH and lactate, especially when they occur together, can activate these nociceptors and cause muscle pain (66).
Fascia, the connective tissue layer surrounding the skeletal muscles, is richly innervated and inflammation of fascia, seen particularly in DM, can also contribute to pain in these patients. In fact, one retrospective study showed significantly higher rates of muscle pain among IIM patients with fasciitis compared to those without fasciitis (55).

Nociplastic pain. In addition to local activation and peripheral sensitisation of the nociceptors, central sensitisation is an important mechanism which plays a crucial role in the persistence of pain. Central sensitisation due to muscle pain can occur when the influx of nerve impulses from muscle nociceptors increases the excitability of dorsal horn neurons in the spinal cord (67, 68). Even a low-frequency and low-threshold of activation of nociceptors can cause hyperexcitability in dorsal horn cells (69). However, most of the research in this field has been conducted in rats and mice. The inflammatory environment was mostly induced by injecting formalin into the muscle (70, 71) rather than through autoimmune inflammation, which significantly limits the generalisability of the findings to IIM in humans. However, intramuscular injections of TNF-alpha in rats (71) and IL-6 in mice (72) have also led to hyperalgesia, suggesting that inflammation in muscle tissue, as also seen in IIM, may be a potential cause of pain.
Another underlying mechanism for nociplastic pain is dysfunctional conditioned pain modulation. Unlike central sensitisation where a bottom-up pain facilitation is observed, conditioned pain modulation is an endogenous pain modulatory pathway that involves descending inhibitory signals from brainstem to spinal cord (top-down) to help limit pain. Poor sleep, physical inactivity and depression can cause impairment in conditioned pain modulation that leads to inefficient descending pain inhibition and can contribute to chronic pain in these patients (60).
In a prospective cohort study, approximately one-third of patients with IIM met the modified criteria for fibromyalgia which is comparable to other systemic autoimmune diseases (73). High prevalence of fibromyalgia observed in patients with IIM overall supports the role of nociplastic pain in pathogenesis of myositis-related pain.

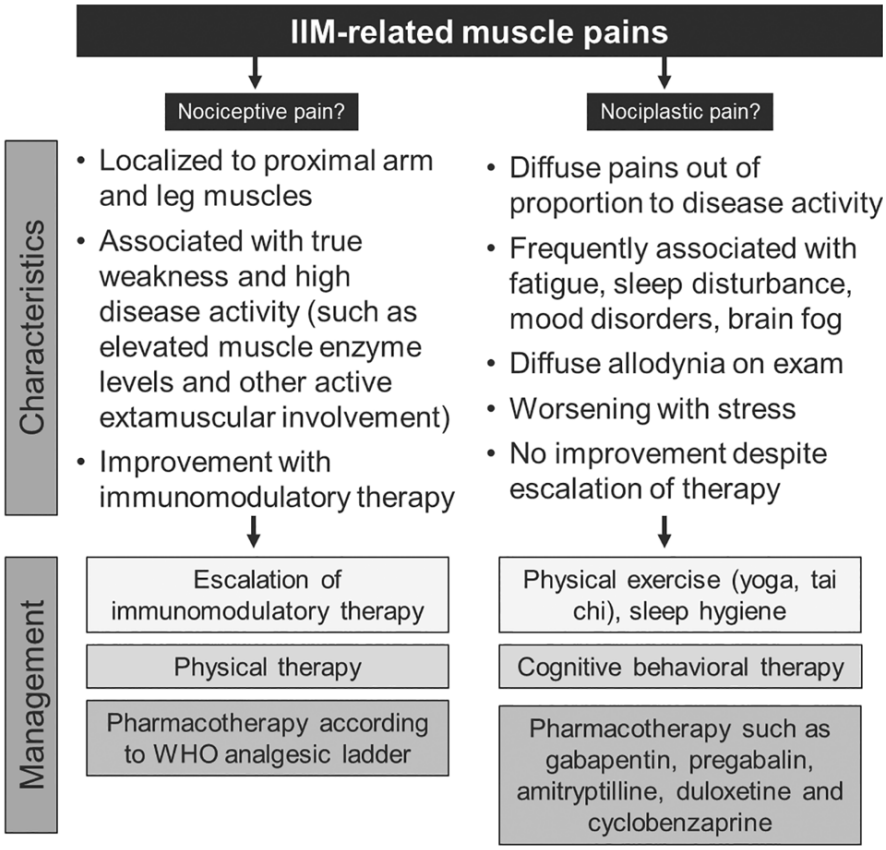

Fig. 2. Approach to assessment and management of IIM-related muscle pains.

Considerations for management of pain in patients with IIM

Although pain is a frequently reported symptom among patients with IIM, there is a notable lack of evidence regarding management of pain. This may be partly due to the incomplete understanding of the underlying mechanisms of myositis-related pain. Besides muscle pain, patients with IIM can also experience pain related to inflammatory arthritis, cutaneous ulcerations, Raynaud's phenomenon, or calcinosis as extra-muscular manifestations of IIM as well as pain related to comorbidities such as osteoarthritis or treatment side effects (such as headaches from intravenous immunoglobulin infusions). In this section, we will focus on IIM-related muscle pains as they are most commonly reported by these patients (Fig. 2).
There is growing evidence to suggest that adequate control of disease activity is crucial for pain management. Several studies demonstrated a significant correlation between pain intensity and disease activity markers, as summarized above (11, 18). In a prospective, observational, longitudinal analysis, changes in disease activity were also significantly correlated with changes in pain interference over time (32). Additionally, a cohort study observed a decline in the prevalence of moderate to severe pain, from 53% in the year 1997 to 27% in 2017 which was potentially attributed to improved disease control with newer therapeutics (42). Similar-

ly, in a RCT involving early untreated myositis, pain levels significantly improved over an 18-month treatment period (17). Therefore, pain can be a symptom of active disease and intensified immunosuppressive therapy can provide relief in these cases.

Besides better control of disease activity, general pain management recommendations for musculoskeletal pain support a multimodal approach combining pharmacological and non-pharmacological strategies and can also be applied in patients with IIM. Alongside the use of analgesics, aligned with the World Health Organization (WHO) pain ladder and including adjuvant therapies, non-drug therapies such as heat therapy and physical therapy are recommended (74).

The efficacy of analgesics in reducing pain among patients with IIM is currently unknown. Available data suggest that a high proportion of patients with IIM use analgesics. In a large cohort study, 92.8% of patients reporting pain were on non-opioid analgesics, while 69% were on opioid medications (19). Non-steroidal anti-inflammatory drugs (NSAIDs) and acetaminophen were the most commonly used non-opioid analgesics. Anticonvulsants and antidepressants were also prescribed in some patients. Another study found that 29.1% of patients with IIM were on NSAIDs, while 22.8% were on opioids for pain management (13). Among patients with more severe pain (>3 out of 10), 35.3% were on opioids in this study. However, it is unclear whether these medications effectively reduced pain or how long their effect lasts in these patients.

In patients whose disease activity does not match the severity of their muscle pain, nociplastic pain, as mentioned above, should be considered (Figure 2). Typical indications of nociplastic pain include diffuse pain, frequent association with fatigue, sleep disturbance, mood disorder and brain fog, worsening due to psychosocial stress, no improvement despite escalation of immunomodulatory therapy, and diffuse allodynia on exam (75). If there is a concern for nociplastic pain, patient education, physical exercise (such as tai chi, yoga), cognitive behavioural therapy (CBT) along with medications used in fibromyalgia management (such as gabapentin) could be considered in the management. For the prototypical nociplastic condition, fibromyalgia, CBT is used as an effective treatment. The results of a RCT demonstrated that fibromyalgia patients who underwent CBT experienced significantly greater improvement in their symptoms including reduced pain interference (76) and pain catastrophising.

Influence of physical activity on pain

A number of studies examined the influence of physical activity interventions on pain in myositis (23, 24, 37-40, 44, 46, 47, 77). Even though most physical activity interventions did not lead to significant changes in pain scores, some studies reported improvements in pain with physical activity (56). Overall, the cohort sizes of these studies were small ranging between 9 (24) and 37 (77). The interventions conducted were mostly home-based exercise programs (23, 37, 46, 77) and included resistance training, such as isotonic training, as well as training of the respiratory muscles (39). The duration of the intervention varied from 3 weeks (38, 39) to 1 year (44). Of the 10 studies, three studies showed a positive effect of physical activity on pain (44, 46, 47).

In an open-label trial (n=13) involving a 12-week program of low-intensity resistance exercises, patients with mildly active DM/PM reported a significant improvement in their pain levels (47). A randomised controlled trial (n=21) found that patients with IIM who participated in a hospital-based exercise program showed a significant decrease in pain intensity after 12 months (44). A 12-week resistance home-based exercise program in patients with active DM and PM also achieved a significant improvement in pain levels (n=11) (46). Only one study found a worsening in SF-36 BP score, despite stable VAS scores after a home-based program (n=10) (23). A recent systematic review highlighted that physical exercise may be a safe and effective way to improve muscle strength, fatigue and, in some cases, pain in myositis (78). However, patients may report muscle pain with physical activity, which can make it difficult to participate in physical exercise programs (10). Nevertheless, more research with larger sample size is needed to better understand the true efficacy of exercise intervention on pain in patients with myositis.

Conclusion

Recent studies focusing on the prevalence and the impact of pain in myositis highlighted pain as a common symptom with a substantial burden. Despite growing recognition of its importance, critical knowledge gaps remain in understanding the mechanisms, risk factors and management of myositis-related pain. Current evidence is limited by the scarcity of mechanistic studies in IIM and the lack of clinical trials that focus on pain as a primary outcome. Future studies addressing these aspects may significantly improve our understanding and approach to this important symptom. Until we have more evidence on specific management strategies, a multimodal, patient-centred approach remains essential.

References

1. GLAUBITZ S, SAYGIN D, LUNDBERG IE: Current efforts and historical perspectives on classification of idiopathic inflammatory myopathies. *Curr Opin Rheumatol* 2024; 36(6): 473-80. https://doi.org/10.1097/BOR.0000000000001042
2. FATTORINI F, CONTICINI E, DOURADO E *et al*.: Idiopathic inflammatory myopathies: one year in review 2024. *Clin Exp Rheumatol* 2025; 43(2): 167-77. https://doi.org/10.55563/clinexprheumatol/yizkja
3. SCHMIDT J: Current Classification and Management of Inflammatory Myopathies. *J Neuromuscul Dis* 2018 May; 5(2): 109-29. https://doi.org/10.3233/JND-180308
4. CONTICINI E, DOURADO E, BOTTAZZI F *et al*.: Idiopathic inflammatory myopathies: one year in review 2023. *Clin Exp Rheumatol* 2024; 42(2): 213-24. https://doi.org/10.55563/clinexprheumatol/dh5o6c
5. SHU XM, LU X, XIE Y, WANG GC: Clinical characteristics and favorable long-term outcomes for patients with idiopathic inflammatory myopathies: a retrospective single center study in China. *BMC Neurol* 2011; 11(1): 143. https://doi.org/10.1186/1471-2377-11-143
6. JANARDANA R, KN S, BHAT V *et al*.: Long Term Outcomes in Idiopathic Inflammatory Myositis: An Observational Epidemiologic Study over 15 Years. *Mediterr J Rheumatol* 2023; 34(4): 513-24. https://doi.org/10.31138/mjr.280823.lto
7. COX FM, TITULAER MJ, SONT JK, WINTZEN AR, VERSCHUUREN JJGM, BADRISING UA:

A 12-year follow-up in sporadic inclusion body myositis: an end stage with major disabilities. *Brain* 2011; 134(11): 3167-75. https://doi.org/10.1093/brain/awr217
8. BAIG S, PAIK JJ: Inflammatory muscle disease – An update. *Best Pract Res Clin Rheumatol* 2020; 34(1): 101484. https://doi.org/10.1016/j.berh.2019.101484
9. GUPTA L, CHINOY H: Monitoring disease activity and damage in adult and juvenile idiopathic inflammatory myopathy. *Cur Opin Rheumatol* 2020; 32(6): 553-61. https://doi.org/10.1097/BOR.0000000000000749
10. OLDROYD A, DIXON W, CHINOY H, HOWELLS K: Patient insights on living with idiopathic inflammatory myopathy and the limitations of disease activity measurement methods – a qualitative study. *BMC Rheumatol* 2020; 4(1): 47. https://doi.org/10.1186/s41927-020-00146-3
11. SHINJO SK, KIM M, HOFF LS *et al*.: Pain in individuals with idiopathic inflammatory myopathies, other systemic autoimmune rheumatic diseases, and without rheumatic diseases: A report from the COVAD study. *Int J of Rheum Dis* 2023; 26(4): 727-39. https://doi.org/10.1111/1756-185X.14636
12. MECOLI CA, PARK JK, ALEXANDERSON H *et al*.: Perceptions of Patients, Caregivers, and Healthcare Providers of Idiopathic Inflammatory Myopathies: An International OMERACT Study. *J Rheumatol* 2019; 46(1): 106-11. https://doi.org/10.3899/jrheum.180353
13. SAYGIN D, MALFAIT A, RITZ EM, WIPFLER K, MICHAUD K, LEE YC: Prevalence of Pain and Factors Associated with Pain in Patients with Idiopathic Inflammatory Myopathies. *Arthritis Care Res* 2025; acr.25571. https://doi.org/10.1002/acr.25571
14. KERET S, SAYGIN D, MOGHADAM-KIA S, REN D, ODDIS CV, AGGARWAL R: Discordance between patient- and physician-reported disease activity in adult idiopathic inflammatory myopathy. *Rheumatology* 2023; 62(12): 3957-61. https://doi.org/10.1093/rheumatology/kead316
15. SAYGIN D, WERTH V, PAIK JJ *et al*.: Current myositis clinical trials and tribulations. *Ann Rheum Dis* 2024; 83(7): 826-9. https://doi.org/10.1136/ard-2023-224652
16. CHRISTOPHER-STINE L, WAN GJ, KELLY W, MCGOWAN M, BOSTIC R, REED ML: Patient-reported dermatomyositis and polymyositis flare symptoms are associated with disability, productivity loss, and health care resource use. *J Manag Care Pharm* 2020; 26(11): 1424-33. https://doi.org/10.18553/jmcp.2020.26.11.1424
17. VAN DE VLEKKERT J, HOOGENDIJK JE, DE HAAN RJ *et al*.: Oral dexamethasone pulse therapy versus daily prednisolone in subacute onset myositis, a randomised clinical trial. *Neuromuscul Disord* 2010; 20(6): 382-9. https://doi.org/10.1016/j.nmd.2010.03.011
18. CHANDRASEKHARA PILLAI A, BIJOY GEORGE T, DIANXU R *et al*.: Pain is common in myositis and associated with disease activity. *Rheumatology* 2025;64(2): 780-8. https://doi.org/10.1093/rheumatology/keae126
19. BHASHYAM A, LUBINUS M, FILMORE E *et al*.: Pain profile and opioid medication use in patients with idiopathic inflammatory myopathies. *Rheumatology* 2022; 62(1): 264-9. https://doi.org/10.1093/rheumatology/keac271
20. SULTAN SM, IOANNOU Y, MOSS K, ISENBERG DA: Outcome in patients with idiopathic inflammatory myositis: morbidity and mortality. *Rheumatology* 2002; 41(1): 22-6. https://doi.org/10.1093/rheumatology/41.1.22
21. PONYI A, BORGULYA G, CONSTANTIN T, VÁNCSA A, GERGELY L, DANKÓ K: Functional outcome and quality of life in adult patients with idiopathic inflammatory myositis. *Rheumatology* (Oxford) 2005; 44(1): 83-8. https://doi.org/10.1093/rheumatology/keh404
22. SADJADI R, ROSE MR, MUSCLE STUDY GROUP: What determines quality of life in inclusion body myositis? *J Neurol Neurosurg Psychiatry* 2010; 81(10): 1164-6. https://doi.org/10.1136/jnnp.2009.183863
23. ALEXANDERSON H, STENSTRÖM CH, LUNDBERG I: Safety of a home exercise programme in patients with polymyositis and dermatomyositis: a pilot study. *Rheumatology* (Oxford) 1999; 38(7): 608-11. https://doi.org/10.1093/rheumatology/38.7.608
24. ALEXANDERSON H, DASTMALCHI M, ESBJÖRNSSON-LILJEDAHL M, OPAVA CH, LUNDBERG IE: Benefits of intensive resistance training in patients with chronic polymyositis or dermatomyositis. *Arthritis Rheum* 2007; 57(5): 768-77. https://doi.org/10.1002/art.22780
25. GORESHI R, CHOCK M, FOERING K *et al*.: Quality of life in dermatomyositis. *J Am Acad Dermatol* 2011; 65(6): 1107-16. https://doi.org/10.1016/j.jaad.2010.10.016
26. REGARDT M, WELIN HENRIKSSON E, ALEXANDERSON H, LUNDBERG IE: Patients with polymyositis or dermatomyositis have reduced grip force and health-related quality of life in comparison with reference values: an observational study. *Rheumatology* 2011; 50(3): 578-85. https://doi.org/10.1093/rheumatology/keq356
27. XU A, SUN C, METCALF R, LIMAYE V: Health-related quality of life and work impairment in idiopathic inflammatory myopathies in South Australia. *Int J of Rheum Dis* 2021; 24(6): 809-14. https://doi.org/10.1111/1756-185X.14120
28. CLEARY LC, CROFFORD LJ, LONG D *et al*.: Does Computed Tomography-Based Muscle Density Predict Muscle Function and Health-Related Quality of Life in Patients with Idiopathic Inflammatory Myopathies? *Arthritis Care Res* 2015; 67(7): 1031-40. https://doi.org/10.1002/acr.22557
29. POULSEN KB, ALEXANDERSON H, DALGÅRD C, JACOBSEN S, WEILE L, DIEDERICHSEN LP: Quality of life correlates with muscle strength in patients with dermato- or polymyositis. *Clin Rheumatol* 2017; 36(10):2 289-95. https://doi.org/10.1007/s10067-017-3706-6
30. FELDON M, FARHADI PN, BRUNNER HI *et al*.: Predictors of Reduced Health-Related Quality of Life in Adult Patients with Idiopathic Inflammatory Myopathies. Arthritis Care Res (Hoboken) 2017; 69(11): 1743-50. https://doi.org/10.1002/acr.23198
31. CHUNG YL, MITCHELL HL, HOUSSIEN DA, AL-MAHROUKI H, CARR AJ, SCOTT DL: A comparative study of outcome in myositis and other musculoskeletal disorders assessed using the Nottingham health profile. *Clin Exp Rheumatol* 2001; 19(4): 447-50.
32. SAYGIN D, ALEXANDERSON H, DIRENZO D *et al*.: The impact of pain on daily activities in patients with idiopathic inflammatory myopathies: Report from the OMERACT myositis working group. *Semin Arthritis Rheum* 2024; 67: 152476. https://doi.org/10.1016/j.semarthrit.2024.152476
33. ROSE MR, SADJADI R, WEINMAN J *et al*.: Role of disease severity, illness perceptions, and mood on quality of life in muscle disease. *Muscle Nerve* 2012; 46(3): 351-9. https://doi.org/10.1002/mus.23320
34. LANDON-CARDINAL O, BACHASSON D, GUILLAUME-JUGNOT P *et al*.: Relationship between change in physical activity and in clinical status in patients with idiopathic inflammatory myopathy: A prospective cohort study. *Semin Arthritis Rheum* 2020; 50(5): 1140-9. https://doi.org/10.1016/j.semarthrit.2020.06.014
35. MAHLER EAM, BLOM M, VOERMANS NC, VAN ENGELEN BGM, VAN RIEL PLCM, VONK MC: Rituximab treatment in patients with refractory inflammatory myopathies. *Rheumatology* 2011; 50(12): 2206-13. https://doi.org/10.1093/rheumatology/ker088
36. OPINC AH, BRZEZINSKA OE, MAKOWSKA JS: Disability in idiopathic inflammatory myopathies: questionnaire-based study. *Rheumatol Int* 2019; 39(7): 1213-20. https://doi.org/10.1007/s00296-019-04302-y
37. ALEXANDERSON H, MUNTERS LA, DASTMALCHI M *et al*.: Resistive Home Exercise in Patients with Recent-onset Polymyositis and Dermatomyositis - A Randomized Controlled Single-blinded Study with a 2-year Followup. *J Rheumatol* 2014; 41(6): 1124-32. https://doi.org/10.3899/jrheum.131145
38. HEIKKILLÄ S, VIITANEN JV, KAUTIAINEN H, RAJAMÄKI T, MÄNTYVOU P, HARJU T: Rehabilitation in Myositis. *Physiotherapy* 2001; 87(6): 301-9. https://doi.org/10.1016/S0031-9406(05)60764-X
39. VARJÚ C, PETHÖ E, KUTAS R, CZIRJÁK L: The effect of physical exercise following acute disease exacerbation in patients with dermato/polymyositis. *Clin Rehabil* 2003; 17(1): 83-7. https://doi.org/10.1191/0269215503cr572oa
40. WALLACE A, PIETRUSZ A, DEWAR E *et al*.: Community exercise is feasible for neuromuscular diseases and can improve aerobic capacity. *Neurology* 2019; 92(15). https://doi.org/10.1212/WNL.0000000000007265
41. ROCKETTE-WAGNER B, SAYGIN D, MOGHADAM-KIA S *et al*.: Reliability, validity and responsiveness of physical activity monitors in patients with inflammatory myopathy. *Rheumatology* 2021; 60(12): 5713-23. https://doi.org/10.1093/rheumatology/keab236
42. ALBRECHT K, HUSCHER D, CALLHOFF J *et al*.: Trends in idiopathic inflammatory myopathies: cross-sectional data from the German National Database. *Rheumatol Int* 2020; 40(10): 1639-47. https://doi.org/10.1007/s00296-020-04634-0
43. VAN DER MEULEN MFG, HOOGENDIJK JE, WOKKE JHJ, DE VISSER M: Oral pulsed high-

dose dexamethasone for myositis. *J Neurol* 2000; 247(2): 102-5. https://doi.org/10.1007/PL00007789

44. TIFFREAU V, RANNOU F, KOPCIUCH F *et al*.: Postrehabilitation Functional Improvements in Patients with Inflammatory Myopathies: The Results of a Randomized Controlled Trial. *Arch Phys Med Rehabil* 2017; 98(2): 227-34. https://doi.org/10.1016/j.apmr.2016.09.125
45. ALEXANDERSON H, REGARDT M, OTTOSSON C *et al*.: Muscle Strength and Muscle Endurance During the First Year of Treatment of Polymyositis and Dermatomyositis: A Prospective Study. *J Rheumatol*.2018 Apr; 45(4): 538-46. https://doi.org/10.3899/jrheum.161183
46. ALEXANDERSON H, STENSTRÖM CH, JENNER G, LUNDBERG I: The safety of a resistive home exercise program in patients with recent onset active polymyositis or dermatomyositis. *Scand J Rheumatol* 2000; 29(5): 295-301. https://doi.org/10.1080/030097400447679
47. MATTAR MA, GUALANO B, PERANDINI LA *et al*.: Safety and possible effects of low-intensity resistance training associated with partial blood flow restriction in polymyositis and dermatomyositis. *Arthritis Res Ther* 2014; 16(5): 473. https://doi.org/10.1186/s13075-014-0473-5
48. WARE JE, SHERBOURNE CD: The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992; 30(6): 473-83.
49. FRIES JF, SPITZ P, KRAINES RG, HOLMAN HR: Measurement of patient outcome in arthritis. *Arthritis Rheum* 1980; 23(2): 137-45. https://doi.org/10.1002/art.1780230202
50. VINCENT KA, CARR AJ, WALBURN J, SCOTT DL, ROSE MR: Construction and validation of a quality of life questionnaire for neuromuscular disease (INQoL). *Neurology* 2007; 68(13): 1051-7. https://doi.org/10.1212/01.wnl.0000257819.47628.41
51. WIKLUND I: The Nottingham Health Profile--a measure of health-related quality of life. *Scand J Prim Health Care Suppl* 1990; 1: 15-8.
52. SAYGIN D, DIRENZO D, RAAPHORST J *et al*.: Responsiveness and meaningful thresholds of PROMIS pain interference, fatigue, and physical function forms in adults with idiopathic inflammatory myopathies: Report from the OMERACT Myositis Working Group. *Semin Arthritis Rheum* 2024; 64: 152339. https://doi.org/10.1016/j.semarthrit.2023.152339
53. DIRENZO D, SAYGIN D, DR GROOT I *et al*.: Reliability and validity of PROMIS physical function, pain interference, and fatigue as patient reported outcome measures in adult idiopathic inflammatory myopathies: International study from the OMERACT myositis working group. *Semin Arthritis Rheum* 2023; 58: 152111. https://doi.org/10.1016/j.semarthrit.2022.152111
54. VAN DE VLEKKERT J, HOOGENDIJK JE, DE VISSER: Long-term follow-up of 62 patients with myositis. *J Neurol* 2014; 261(5): 992-8. https://doi.org/10.1007/s00415-014-7313-z
55. NODA K, YOSHIDA K, UKICHI T *et al*.: Myalgia in Patients with Dermatomyositis and Polymyositis Is Attributable to Fasciitis Rather Than Myositis: A Retrospective Study of 32 Patients who Underwent Histopathological Examinations. *J Rheumatol* 2017; 44(4): 482-7. https://doi.org/10.3899/jrheum.160763
56. LECLAIR V, TSUI H, HUDSON M: Pain in autoimmune inflammatory myopathies: a scoping review. *RMD Open* 2023; 9(1): e002591. https://doi.org/10.1136/rmdopen-2022-002591
57. MOGIL JS: Qualitative sex differences in pain processing: emerging evidence of a biased literature. *Nat Rev Neurosci* 2020; 21(7): 353-65. https://doi.org/10.1038/s41583-020-0310-6
58. INTERNATIONAL ASSOCIATION FOR THE STUDY OF PAIN (IASP): Task Force on Taxonomy. IASP Terminology Updated from "Part III: Pain Terms, A Current List with Definitions and Notes on Usage". Classification of Chronic Pain, 2nd ed. Seattle: IASP. 2017, 209-14.
59. INTERNATIONAL ASSOCIATION FOR THE STUDY OF PAIN (IASP): IASP Terminology. Available from: https://www.iasp-pain.org/resources/terminology/
60. KOSEK E, COHEN M, BARON R *et al*.: Do we need a third mechanistic descriptor for chronic pain states? *Pain* 2016; 157(7): 1382-6. https://doi.org/10.1097/j.pain.0000000000000507
61. GRAVEN-NIELSEN T, MENSE S: The peripheral apparatus of muscle pain: evidence from animal and human studies. *Clin J Pain* 2001; 17(1): 2-10. https://doi.org/10.1097/00002508-200103000-00002
62. MENSE S: Muscle Pain. *Dtsch Arztebl Int* 2008; 105(12): 214-9. https://doi.org/10.3238/artzebl.2008.0214
63. BABENKO V, GRAVEN-NIELSEN T, SVENSSON P, DREWES AM, JENSEN TS, ARENDT-NIELSEN L: Experimental human muscle pain and muscular hyperalgesia induced by combinations of serotonin and bradykinin. *Pain* 1999; 82(1): 1-8. https://doi.org/10.1016/S0304-3959(99)00026-3
64. JANG Y, KIM M, HWANG SW: Molecular mechanisms underlying the actions of arachidonic acid-derived prostaglandins on peripheral nociception. *J Neuroinflammation* 2020; 17(1): 30. https://doi.org/10.1186/s12974-020-1703-1
65. REINÖHL J, HOHEISEL U, UNGER T, MENSE S: Adenosine triphosphate as a stimulant for nociceptive and non-nociceptive muscle group IV receptors in the rat. *Neurosci Lett* 2003; 338(1): 25-8. https://doi.org/10.1016/s0304-3940(02)01360-5
66. GREGORY NS, WHITLEY PE, SLUKA KA: Effect of Intramuscular Protons, Lactate, and ATP on Muscle Hyperalgesia in Rats. *PLoS One* 2015; 10(9): e0138576. https://doi.org/10.1371/journal.pone.0138576
67. WALL PD, WOOLF CJ: Muscle but not cutaneous C-afferent input produces prolonged increases in the excitability of the flexion reflex in the rat. *J Physiol* 1984; 356: 443-58. https://doi.org/10.1113/jphysiol.1984.sp015475
68. HOHEISEL U, KOCH K, MENSE S: Functional reorganization in the rat dorsal horn during an experimental myositis. *Pain* 1994; 59(1): 111-8. https://doi.org/10.1016/0304-3959(94)90054-x
69. HOHEISEL U, UNGER T, MENSE S: Sensitization of rat dorsal horn neurons by NGF-induced subthreshold potentials and low-frequency activation. A study employing intracellular recordings in vivo. *Brain Res* 2007; 1169: 34-43. https://doi.org/10.1016/j.brainres.2007.06.054
70. MEOTTI FC, CAMPOS R, DA SILVA K, PASZCUK AF, COSTA R, CALIXTO JB: Inflammatory muscle pain is dependent on the activation of kinin B1 and B2 receptors and intracellular kinase pathways. *Br J Pharmacol* 2012; 166(3): 1127-39. https://doi.org/10.1111/j.1476-5381.2012.01830.x
71. SCHÄFERS M, SORKIN LS, SOMMER C: Intramuscular injection of tumor necrosis factor-alpha induces muscle hyperalgesia in rats. Pain 2003; 104(3): 579-88. https://doi.org/10.1016/S0304-3959(03)00115-5
72. MANJAVACHI MN, MOTTA EM, MAROTTA DM, LEITE DFP, CALIXTO JB: Mechanisms involved in IL-6-induced muscular mechanical hyperalgesia in mice. *Pain* 2010; 151(2): 345-55. https://doi.org/10.1016/j.pain.2010.07.018
73. SAYGIN D, SCHMUCKLER J, WIPFLER K *et al*.: Prevalence and Impact of Fibromyalgia in Patients with Idiopathic Inflammatory Myopathies (Abstract). *Arthritis Rheumatol* 2024; 76 (Suppl. 9). Available from: https://acrabstracts.org/abstract/prevalence-and-impact-of-fibromyalgia-in-patients-with-idiopathic-inflammatory-myopathies/
74. EL-TALLAWY SN, NALAMASU R, SALEM GI, LEQUANG JAK, PERGOLIZZI JV, CHRISTO PJ: Management of Musculoskeletal Pain: An Update with Emphasis on Chronic Musculoskeletal Pain. *Pain Ther* 2021; 10(1): 181-209. https://doi.org/10.1007/s40122-021-00235-2
75. SCHUBINER H, LOWRY WJ, HEULE M *et al*.: Application of a Clinical Approach to Diagnosing Primary Pain: Prevalence and Correlates of Primary Back and Neck Pain in a Community Physiatry Clinic. *J Pain* 2024; 25(3): 672-81. https://doi.org/10.1016/j.jpain.2023.09.019
76. LEE J, LAZARIDO A, PASCHALI M *et al*.: A Randomized Controlled Neuroimaging Trial of Cognitive Behavioral Therapy for Fibromyalgia Pain. *Arthritis Rheumatol* 2024; 76(1): 130-40. https://doi.org/10.1002/art.42672
77. CHUNG YL, ALEXANDERSON H, PIPITONE N *et al*.: Creatine supplements in patients with idiopathic inflammatory myopathies who are clinically weak after conventional pharmacologic treatment: Six-month, double-blind, randomized, placebo-controlled trial. *Arthritis Rheum* 2007; 57(4): 694-702. https://doi.org/10.1002/art.22687
78. MISSE RG, BORGES IBP, DOS SANTOS AM, GUPTA L, SHINJO SK: Effect of exercise training on fatigue and pain in patients with systemic autoimmune myopathies: A systematic review. *Autoimmun Rev* 2021; 20(10): 102897. https://doi.org/10.1016/j.autrev.2021.102897