Narrative review

A Contemporary Approach to Chronic Pain Management

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Abstract

Chronic pain has traditionally been defined mainly by duration. That definition explains little about why patients with similar diagnoses differ in disability and treatment response. A contemporary approach shifts focus to the underlying pain mechanism. ICD-11 supports this shift by separating chronic primary from chronic secondary pain. Management follows the same logic: rigid analgesic escalation gives way to multimodal, individualized treatment guided by function and patient goals. Personalized analgesia, including pharmacogenomics, extends this individualization further, though its role remains supportive rather than decisive. This review outlines this mechanism-based, patient-centered shift as an alternative to the traditional duration-based model.

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Introduction

Chronic pain is commonly defined as pain persisting or recurring beyond three months (1). It affects a substantial share of adults, though prevalence estimates differ with the population studied, the definition used, and the method of assessment (2,3). Duration alone, however, tells us little about the clinical complexity behind that threshold.

A contemporary approach to chronic pain requires assessment beyond pain intensity. It considers the predominant pain mechanism, functional impairment, psychological burden, comorbidities, treatment response, and risks related to long-term analgesic therapy (1,3,4). This shift is reflected in mechanism-based pain classification and in the ICD-11 framework, which distinguishes chronic primary pain from chronic secondary pain (1,4).

This narrative review provides a concise clinical overview of chronic pain from a contemporary perspective. It focuses on current classification, mechanism-based assessment, and individualized treatment strategies as a basis for more rational pain management.

Methods

This article is a narrative review based on selected peer-reviewed literature addressing the classification, mechanisms, and management of chronic pain. The literature was chosen to support a concise clinical overview of contemporary chronic pain concepts. The review included publications on the ICD-11 framework, mechanism-based pain classification, multimodal management, interventional pain treatment, and pharmacogenetic aspects of analgesic therapy. Publications were identified by searching PubMed/MEDLINE and Google Scholar for English-language articles published between 2016 and 2026, supplemented by screening the reference lists of key articles. Preference was given to recent peer-reviewed publications, including current guidelines and systematic reviews. No formal systematic search strategy, risk-of-bias assessment, or meta-analysis was performed.

Discussion

The contemporary view of chronic pain has moved beyond a simple time-based definition. Pain duration remains useful, but it does not explain why patients with similar diagnoses often differ in symptoms, disability, and treatment response (1,3,5). Chronic pain therefore requires assessment of the predominant pain mechanism, functional impact, psychological context, and individual treatment risks (3–5). This perspective supports more rational treatment selection and avoids reducing chronic pain care to repeated investigations or nonspecific analgesic escalation (3,6).

Mechanism-based assessment and classification of chronic pain

A mechanism-based assessment helps connect clinical presentation with treatment selection. It considers whether pain is predominantly nociceptive, neuropathic, nociplastic, or mixed. These categories are clinically useful, but they are not absolute. Mixed pain phenotypes are common, and the dominant mechanism may change over time (4,5,7).

This distinction carries the most weight in a few settings. Structural findings may not explain the severity. Pain may outlast tissue healing. Widespread symptoms may cluster with sleep disturbance, fatigue, and low mood (1,3–5). In this context, chronic pain assessment should include more than pain intensity. Functional limitation, psychological burden, comorbidities, previous treatment exposure, medication risks, and patient goals all influence clinical decision-making.

Nociceptive pain arises from actual or threatened damage to non-neural tissue. It is usually associated with inflammatory, traumatic, degenerative, or visceral pathology. Somatic nociceptive pain is often relatively well localized. Visceral pain is more diffuse and less anatomically precise. Sensory abnormalities

such as numbness, paresthesia, or dysesthesia are not dominant features (5,7). Neuropathic pain results from a lesion or disease of the somatosensory nervous system. It may involve peripheral nerves, nerve roots, the spinal cord, or central sensory pathways. Patients typically describe burning, shooting, or electric shock-like pain, often with tingling, numbness, allodynia, or hyperalgesia (8,9).

Nociplastic pain is the most recent of the three mechanistic categories, and it has changed how we understand chronic pain. It describes pain that arises from altered nociceptive processing, without clear evidence of ongoing tissue damage or a defined lesion of the somatosensory nervous system. It is most relevant when regional or widespread pain persists out of proportion to structural findings. These patients often report hypersensitivity, fatigue, sleep disturbance, cognitive symptoms, and increased sensitivity to normally non-painful stimuli (4,10,11). Recognizing nociplastic pain matters clinically. It explains presentations that fit neither a nociceptive nor a neuropathic model, and it can spare patients repeated structural investigations. Treatment then shifts toward central sensitization, function, sleep, mood, and self-management (4,10).

ICD-11 classification and clinical implications

These mechanistic categories should not be confused with the ICD-11 classification. The mechanistic labels describe how the pain is generated. ICD-11 describes its relationship to an underlying condition. The two are complementary, not interchangeable. The same patient can be described by a predominant mechanism and by an ICD-11 category (1,4,5). Chronic pain is now structured within ICD-11 for the first time. ICD-11 distinguishes between chronic primary pain (pain is the disease itself) and chronic secondary pain (pain is the result of another disease or pathological process) (1). The difference is clinically useful. It separates pain that requires investigation for an underlying cause from pain that can be treated as a chronic, ongoing problem. It also supports clearer documentation, better communication with patients, and a more thoughtful selection of treatment. In everyday practice, ICD-11 should not become a purely administrative label. Its value lies in the clinic: it pushes toward a more precise description of the pain and lines up diagnosis, mechanism-based assessment, and treatment planning.

From analgesic escalation to multimodal management

Chronic pain is often mechanistically heterogeneous, influenced by comorbidity, psychological factors, previous treatment exposure, and long-term medication risks. Traditional analgesic escalation remains a useful clinical concept, particularly in cancer pain. The classic WHO analgesic ladder has been modified to include interventional methods alongside its pharmacological steps (11,12). However, it is too simple for many patients with chronic non-cancer pain. For this reason, treatment should not follow a rigid stepwise model alone (3,6,12).

Mild or predominantly nociceptive pain may be managed with education, physical activity, non-opioid analgesics, and treatment of the underlying condition. Neuropathic or mixed pain often requires mechanism-based adjuvant therapy, such as antidepressants, gabapentinoids, or topical agents, depending on patient characteristics and tolerability (6,11,13,14).

A contemporary approach favors multimodal management. Treatment should be selected according to the presumed pain mechanism, functional impact, patient comorbidities, treatment goals, and the risk of adverse effects.

Opioids retain an important role in acute pain and cancer-related pain. When considered, opioid therapy should be individualized, time-conscious, and regularly reassessed. It should have clear functional goals and should not be used as an isolated long-term strategy (3,15,16). Their role in chronic non-cancer pain is more limited.

Nonpharmacological treatment is a core component of chronic pain care. Exercise and physical therapy, cognitive-behavioral

strategies, self-management programs, acupuncture, and related physical or behavioral interventions can improve function, coping, and quality of life. None of this should wait until medication has failed. In many patients these approaches work best introduced early, alongside pharmacological treatment (17,18).

Interventional pain management is not reserved for the point where pharmacological escalation has run its course. In selected patients it can be considered earlier, especially when the source of pain is clinically identifiable or can be reasonably targeted. Used well, it provides targeted relief, reduces reliance on long-term pharmacotherapy, and helps avoid unnecessary opioid escalation (6,12,19).

These procedures cover a broad spectrum. Minimally invasive techniques include peripheral nerve blocks, epidural injections, and radiofrequency procedures. More complex interventions include neuromodulation and intrathecal drug delivery. As invasiveness rises, so do the demands: patient selection becomes stricter, perioperative planning more careful, and follow-up more structured. The decision stays individualized, grounded in pain mechanism, clinical findings, previous treatment response, patient risk profile, and functional goals (6,12,19,20).

Function, quality of life, and the biopsychosocial model

In chronic pain, success is not measured by pain intensity alone. Assessment therefore looks wider: physical activity, sleep, mood, work ability, social participation, and how well the patient can self-manage (3,7,17,18). A lower pain score matters, but it does not always mean better function or a better life.

The biopsychosocial model gives this wider assessment a practical shape. In it, persistent pain reflects biological mechanisms together with psychological factors, behavior, expectations, and social context (3,7). This does not make the pain "psychological". It means that the experience of pain, and the response to treatment, arise from several factors acting at once.

Treatment goals should therefore stay realistic and functional. For many patients the aim is not to erase the pain, but to move more during the day, sleep better, fear movement less, use medication more safely, and take a fuller part in rehabilitation. Goals set this clearly also spare the patient repeated ineffective treatments and needless escalation of analgesics (3,17,18).

Personalized analgesia and pharmacogenetics

Response to treatment differs widely from one patient to the next. One patient gets adequate relief from an analgesic, another only limited benefit, a third unacceptable adverse effects. Behind this sit pain mechanism, age, comorbidities, organ function, drug interactions, previous treatment, and genetics (21,22).

Pharmacogenetics matters more and more in personalized analgesia. The idea is not new, but its clinical role in pain medicine is still taking shape. The genetic variability may influence the metabolism of the drug, its effectiveness and its toxicity. Well-established examples include CYP2C9 for several non-steroidal anti-inflammatory drugs (NSAIDs), CYP2D6 for codeine and tramadol, and CYP2C19 or CYP2D6 for tricyclic antidepressants (TCAs) (21,23,24). A small number of pharmacogenetic markers may help identify patients at risk of poor response or drug-related toxicity (21,23,24).

Eventually, such markers may help to direct safer drug selections, spare opioids, and help personalize prescription choices (21,24,25). At this time, most patients with chronic pain are not routinely tested for pharmacogenetics (21,24). Its usefulness depends on the drug, the clinical setting, test availability, how the result is interpreted, and the strength of evidence for each gene–drug association (21,24–26).

Personalized analgesia is, therefore, broader than genetic testing. It is based on mechanism-based assessment, attention to comorbidities, clear treatment goals, medication safety, and periodic reassessment (3,5,21). As the evidence and its application in clinical practice evolve,

pharmacogenetics could become a key component of this approach. For now, its role supports rather than decides (21,23–25).

Conclusion

Chronic pain is best treated as a complex clinical condition, not simply as pain that outlasts a set time threshold. Modern management begins with the recognition of the key mechanism, accurate diagnosis of the nature of the pain and preparation of a treatment plan for the patient. The care is typically multimodal. Pharmacological, non-pharmacological, and interventional measures are used in conjunction with each other and guided by function, safety, and the individual's goals. In practice, this changes what is done. Treatment is matched to the presumed mechanism rather than escalated by default. Investigations that will not change management can be avoided. Opioid use stays more selective. The focus remains on function rather than on pain intensity alone. Pharmacogenetics can add to this, but its role stays complementary. The approach is thus mechanism oriented, multimodal and patient-oriented.

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Author contribution.
GE: Conceptualization, literature interpretation, supervision, validation, writing – review and editing.
AV: Literature search, literature selection, data curation, writing – original draft.
MNP: Literature search.
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SK: Editing.
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Suvremeni pristup liječenju kronične boli

Sažetak

Kronična bol tradicionalno se definirala ponajprije prema trajanju. Takva definicija malo govori o tome zašto se bolesnici sa sličnim dijagnozama razlikuju u stupnju onesposobljenosti i odgovoru na liječenje. Suvremeni pristup pomiče naglasak prema osnovnom mehanizmu boli. ICD-11 podupire taj pomak razlikovanjem kronične primarne i kronične sekundarne boli. Liječenje slijedi istu logiku: kruta eskalacija analgetika ustupa mjesto multimodalnom, individualiziranom liječenju usmjerenom na funkciju i ciljeve bolesnika. Personalizirana analgezija, uključujući farmakogenomiku, dodatno proširuje tu individualizaciju, premda njezina uloga ostaje potporna, a ne odlučujuća. Ovaj pregled prikazuje taj mehanistički i bolesniku usmjeren pomak kao alternativu tradicionalnom pristupu temeljenom na trajanju.

Ključne riječi: kronična bol; liječenje boli; analgetici; farmakogenetika; nociplastična bol