Narrative Review

Cancer-Related Neuropathic Pain: A Review of Contemporary Diagnostic and Therapeutic

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Abstract

Aim of the study: The aim of this review article is to summarize current knowledge on the etiology, pathophysiology, diagnosis, clinical characteristics, and treatment options for cancer-related neuropathic pain (CRNP), with an emphasis on recent advances in pharmacological and non-pharmacological management strategies.

Methods: A narrative review of contemporary literature and clinical recommendations on cancer-related neuropathic pain was conducted. Relevant evidence addressing the etiology, pathophysiology, diagnosis, clinical characteristics, and pharmacological and non-pharmacological management of CRNP was examined. Particular attention was given to diagnostic criteria recommended by the International Association for the Study of Pain (IASP), validated assessment tools such as the DN4 questionnaire, neuroimmune mechanisms underlying neuropathic pain, and contemporary therapeutic approaches.

Results: The reviewed evidence indicates that cancer-related neuropathic pain is a complex, multifactorial condition associated with tumor invasion, nerve compression, chemotherapy- and radiotherapy-induced nerve injury, neuroinflammation, and central sensitization. Diagnosis relies on recognizing characteristic sensory symptoms and demonstrating nervous system involvement, with validated tools such as the DN4 questionnaire supporting clinical assessment. Current evidence supports multimodal pharmacological management, including opioids combined with adjuvant agents such as gabapentinoids, tricyclic antidepressants, and serotonin-norepinephrine reuptake inhibitors. Emerging approaches, including monoclonal antibodies, cannabinoids, intrathecal analgesics, and targeted molecular therapies, show promising but still limited evidence. Non-pharmacological interventions, including acupuncture, cognitive-behavioral therapies, and rehabilitation, may provide additional benefits for symptom control and quality of life.

Conclusion: Cancer-related neuropathic pain remains underrecognized and difficult to treat, significantly impairing patients' quality of life and treatment outcomes. Early diagnosis, individualized multimodal therapy, and integration of pharmacological and supportive interventions are essential for effective management. Future advances in molecular biology, immunology, and pharmacogenomics may enable more targeted and personalized therapeutic strategies for patients with cancer-related neuropathic pain.

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Introduction

Although neuropathic pain and cancer-related neuropathic pain may arise from different underlying causes, they share several fundamental pathophysiological mechanisms, including peripheral nerve injury, abnormal ectopic neuronal activity, peripheral and central sensitization, neuroinflammatory responses, and maladaptive neuroplastic changes (1). Relatively few studies specifically investigate neuropathic pain in patients with cancer, and even fewer focus exclusively on cancer-related neuropathic pain (CRNP) (2). Neuropathic pain in patients with cancer may result from direct tumor-related nerve injury or from treatment-related nerve damage, including surgery, radiation therapy, and neurotoxic chemotherapies. If pain is not assessed and treated promptly, it can become more intense and distressing, negatively affect patients' physical and psychosocial functioning, and increase the burden on families and health care professionals (3). Chronic cancer pain is more often directly related to the tumor or to an inadequate treatment strategy. Pain may be the first symptom of malignant disease in an otherwise healthy patient, or it may appear as the disease progresses or as a treatment-related complication. Neuropathic cancer pain is particularly challenging to identify because neuropathic mechanisms may coexist with nociceptive and visceral mechanisms, and the neuropathic component is not always assessed consistently in clinical practice. Pain related to neurological lesions in patients with advanced cancer may occur in a substantial proportion of patients, although estimates vary according to the diagnostic criteria and assessment methods used (1). In a prospective observational study of 350 patients with cancer, the prevalence of neuropathic cancer pain ranged from 20.0% using the DN4 questionnaire to 28.6% based on retrospective specialist classification, highlighting the impact of the diagnostic method on prevalence estimates (2). Pain from advanced cancer remains a significant clinical problem, with inadequate analgesia still reported in a substantial proportion of patients (4). CRNP, which represents approximately 20% of cancer pain when purely neuropathic pain is considered and a substantially higher proportion when mixed neuropathic–nociceptive pain is included, is associated with greater pain intensity, higher analgesic requirements, poorer functional outcomes, and increased disability (5). Over the past two decades, efforts have focused on standardizing the treatment of CRNP to improve care. Strong opioids remain the cornerstone of pharmacologic treatment for moderate to severe cancer-related pain, and adjuvant analgesics may be added based on underlying pain mechanisms and individual clinical characteristics (6). Co-analgesics used or investigated in cancer pain management include gabapentinoids (gabapentin and pregabalin), antidepressants (tricyclics, duloxetine, and venlafaxine), corticosteroids, bisphosphonates, N-methyl-D-aspartate (NMDA) receptor antagonists, and cannabinoids. The complexity and difficulty of treating CRNP arise from the fact that neuropathic pain is not isolated but is accompanied by nociceptive and visceral pain, ischemia, and inflammatory processes (3). This combination produces mixed pain phenotypes that often require individualized, multimodal treatment (1,3).

Materials and methods

This study was designed as a narrative review to synthesize and critically examine current evidence on cancer-related neuropathic pain (CRNP), with emphasis on its epidemiology, etiology, pathophysiology, clinical characteristics, diagnosis, and therapeutic management. Recent evidence was prioritized, while selected earlier publications were retained when they provided foundational support for diagnostic criteria, validated assessment instruments, pathophysiological mechanisms, or established therapeutic approaches. Evidence directly addressing CRNP was prioritized, and studies from broader neuropathic pain populations were considered when cancer-specific evidence was limited.

A comprehensive literature search was conducted using PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. The primary search covered publications from

january 2019 to may 2026. The search strategy combined Medical Subject Headings (MeSH) and relevant free-text terms with the Boolean operators AND and OR. The main concepts included cancer-related pain, neuropathic pain, diagnosis, pathophysiology, and treatment. The principal MeSH terms included Cancer Pain, Neuralgia, Pain, Pain Management, Analgesics, Opioid, Anticonvulsants, Antidepressive Agents, Cannabinoids, Chemotherapy-Induced Peripheral Neuropathy, Central Nervous System Sensitization, Inflammation, Neoplasms, Antineoplastic Agents, Radiotherapy, Drug-Related Side Effects and Adverse Reactions, and Pharmacogenomics. Search strategies were adapted to the indexing systems of individual databases. Reference lists of relevant reviews, guidelines, and key publications were manually screened to identify additional studies.

Publications were eligible if they addressed CRNP, including its epidemiology, etiology, pathophysiology, diagnosis, or therapeutic management. Eligible publications included original research articles, randomized controlled trials, observational studies, systematic reviews, meta-analyses, clinical guidelines, consensus statements, and narrative reviews deemed relevant. Publications were restricted to full-text articles in English. Publications were excluded if they focused exclusively on non–cancer-related neuropathic pain without direct relevance to CRNP, were duplicates, were conference abstracts without an available full-text article, or were preclinical studies without sufficient clinical relevance.

Titles and abstracts were screened for relevance, followed by full-text assessment of potentially eligible publications. The final selection was based on relevance to the objectives and thematic scope of the review. Evidence was synthesized descriptively according to the main thematic areas of the manuscript. Given the narrative design, no quantitative meta-analysis or statistical pooling was performed. Ethics statement: Ethical approval and informed consent were not required because this study involved only the review and synthesis of previously published literature and did not involve human participants, identifiable patient data, or experimental interventions.

Results

Epidemiology and Clinical Burden

Cancer-related pain is common among patients with malignancies, and a clinically meaningful proportion of these patients experience a neuropathic component (5). Neuropathic cancer pain may arise as a direct consequence of tumor involvement of the nervous system or as a complication of cancer treatment (5). Its clinical presentation is often complex because neuropathic mechanisms may coexist with nociceptive, visceral, inflammatory, and bone pain (7). This mixed phenotype can complicate diagnosis and treatment and is associated with substantial impairment in physical functioning, sleep, psychological well-being, and overall quality of life (5). The burden of cancer-related neuropathic pain is particularly high in patients with advanced disease and in those exposed to neurotoxic chemotherapy, radiotherapy, or extensive surgical procedures (5,8). Early recognition and precise characterization of neuropathic mechanisms are crucial for selecting appropriate treatments and initiating timely, mechanism-based pain management (9). Chemotherapy-induced peripheral neuropathy (CIPN) is a common side effect of chemotherapeutic agents such as cisplatin and paclitaxel (10). CIPN is characterized by substantial loss of primary afferent sensory axonal fibers, leading to sensory disturbances and neuropathic symptoms (10). An estimated 19–85% of patients develop CIPN during chemotherapy, although incidence varies by chemotherapeutic agent, cumulative dose, and assessment method (10). Animal and cell models are often used to study the pathogenesis of CIPN and, more importantly, to assess the efficacy of drugs for preventing or reversing CIPN (11,12).

Pathophysiology and Etiology

CRNP may develop following direct or indirect injury to peripheral or central components of the somatosensory nervous system (13). Tumor

infiltration, perineural invasion, nerve compression, metastatic lesions, and treatment-related neural injury can disrupt normal sensory signaling (14). Cancer treatment may also cause peripheral neuropathy through mitochondrial dysfunction, oxidative stress, ion channel dysregulation, microtubule disruption, and neuroinflammatory mechanisms (13). tumor nerve fibers and T-cell responses across several human cancers (19).

Emerging evidence indicates that neuro-tumor interactions within the tumor microenvironment form a bidirectional signaling network in which neuronal activity and tumor-derived signals influence immune cell function, while tumor-associated neural remodeling and neuroimmune crosstalk contribute to tumor-induced neuropathic pain and treatment-related neurotoxicity (20).

Macrophages, lymphocytes, neutrophils, and other immune cells may contribute to the initiation and maintenance of neuropathic pain (19). Regulatory T cells and T-helper 17 cells appear to modulate persistent cancer pain, although most of the available evidence is still preclinical (19,21). These findings support the concept of CRNP as a neuroimmune disorder in which tumor biology, treatment-related neural injury, inflammation, and maladaptive neural plasticity interact (17) (Table 1).

Table 1. Inflammatory and neuroimmune mechanisms implicated in CRNP, including macrophage-mediated pain, neuroinflammation, sensory neuron–immune interactions, and tumor–nerve signaling

Study	Study type / model	Key mechanism	Hypothesis / aim	Study conclusion
Tang et al., 2022	Experimental; scRNA-seq	Neuron-like macrophages	To identify macrophage subpopulations associated with cancer pain	A distinct neuron-like macrophage subpopulation is associated with cancer pain
Scheff & Saloman, 2021	Review	Neuroimmune signaling	To describe neuroimmune mechanisms underlying cancer-related symptoms	Interactions between tumors, the immune system, and the nervous system contribute to cancer-related symptoms, including pain
Mardelle et al., 2024	Review	Sensory neurons and tumor immunity	To analyze the role of peripheral neurons in tumor immunity	Sensory neurons modulate the tumor microenvironment, immune responses, and nociception
Yaniv et al., 2024	Review	Neural–tumor microenvironment	To analyze interactions between peripheral nerves and tumors	Neural–tumor interactions represent potential therapeutic targets
Zou et al., 2024	Experimental study	TRIM25–CXCL13 axis; neuroinflammation	To investigate the role of Shh signaling in CIPN	Modulation of the TRIM25–CXCL13 axis may attenuate neuropathic changes in CIPN

Table 1. The studies summarize inflammatory and neuroimmune mechanisms implicated in CRNP, including macrophage-mediated pain, neuroinflammation, sensory neuron–immune interactions, and tumor–nerve signaling. Zou et al. specifically examined chemotherapy-induced peripheral neuropathy (CIPN), a treatment-related form of cancer-associated neuropathic pain. (Abbreviations: CIPN, chemotherapy-induced peripheral neuropathy; scRNA-seq, single-cell RNA sequencing; Shh, Sonic hedgehog).

Diagnosis and Clinical Characteristics

CRNP is characterized by pain arising from a lesion or disease affecting the somatosensory nervous system (22). According to the International Association for the Study of Pain (IASP), diagnostic assessment involves establishing a plausible neurological lesion or disease, determining whether the pain distribution is neuroanatomically plausible, identifying sensory signs within the relevant distribution, and obtaining objective evidence of the lesion or disease when required. Based on these criteria, neuropathic pain may be classified as possible, probable, or definite according to the level of diagnostic certainty (Table 2) (22,23).

Table 2. Diagnostic criteria for neuropathic pain according to the IASP.

IASP criteria	Clinical assessment
Lesion or disease of the somatosensory nervous system	Plausible tumor-, treatment-, or disease-related neurological lesion
Neuroanatomically plausible pain distribution	Pain corresponds to the affected nerve, root, plexus, or central pathway
Sensory signs and symptoms	Allodynia, hyperalgesia, hypoesthesia, paresthesia, dysesthesia
Objective evidence, when required	Imaging, neurophysiological studies or other appropriate investigations

Patients may describe CRNP as burning, shooting, electric shock–like, stabbing, or tingling pain, often accompanied by numbness, dysesthesia, allodynia, or hyperalgesia (5,22). Depending on the anatomical structures involved, motor deficits, sensory loss, radiculopathy, plexopathy, or autonomic dysfunction may also occur (22). In oncology patients, neuropathic symptoms frequently coexist with nociceptive or visceral pain, producing a mixed pain phenotype (5,24). Assessment should include onset, location, distribution, intensity, temporal pattern, aggravating and relieving factors, associated sensory and neurological symptoms, previous analgesic treatment, comorbidities, and functional consequences (22). Psychological factors, including anxiety, depression, sleep disturbance, social isolation, and fear related to cancer progression, should also be considered because they may amplify pain-related disability (25,26).

Standard pain intensity measures include the visual analog scale (VAS), verbal rating scale (VRS), and numerical rating scale (NRS). Neuropathic pain screening instruments include the Douleur Neuropathique 4 (DN4), pain DETECT, and the Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) (27,28). These tools support clinical phenotyping but do not replace a comprehensive clinical and neurological assessment (29). The DN4 questionnaire consists of seven patient-reported sensory descriptors and three items derived from clinical sensory examination. A score of ≥4/10 supports the presence of a neuropathic component. Its widespread validation and ease of administration make it useful in routine clinical assessment, although diagnostic performance may vary across populations (30).

Pharmacological treatment

Pharmacological management of CRNP should be individualized and generally follow a multimodal strategy that combines treatment of the underlying malignancy with analgesic and adjuvant therapies targeting neuropathic mechanisms (31). Opioids remain important for

moderate-to-severe cancer pain, particularly when nociceptive and neuropathic components coexist; however, neuropathic pain may respond incompletely to opioid monotherapy (32).

Recent evidence from a systematic review and network meta-analysis suggests that methadone warrants further consideration and promotion as a first-line opioid for this purpose (6,33).

Adjuvant agents include gabapentinoids, antidepressants, and selected topical therapies or N-methyl-D-aspartate (NMDA) receptor–targeted treatments (32). Gabapentin and pregabalin are commonly used for neuropathic pain, although evidence specifically addressing CRNP is less robust than evidence from noncancer neuropathic conditions (34). Tricyclic antidepressants and serotonin–norepinephrine reuptake inhibitors may be considered in selected patients, with treatment choice guided by comorbidities, concomitant medications, and adverse-effect profiles (35,36). Duloxetine is particularly relevant for chemotherapy-induced peripheral neuropathy (37).

Methadone may have a role in selected patients because of its μ-opioid receptor agonism and NMDA receptor antagonism, although its complex pharmacokinetics require experienced prescribing (6,38). Ketamine has theoretical benefits in pain states involving central sensitization, but current clinical evidence does not support its routine use for chronic cancer-related pain or CRNP (39,40). Topical agents such as lidocaine or capsaicin may be useful for selected localized neuropathic pain syndromes (41). Cannabinoids and other emerging pharmacological approaches remain investigational or are supported by limited clinical evidence (42,43). Treatment should therefore be individualized and regularly reassessed, with attention to analgesic efficacy, adverse effects, functional outcomes, and interactions with anticancer therapy (33).

Interventional treatment

Interventional procedures may be considered for patients with severe or refractory CRNP when pharmacological treatment provides insufficient relief or causes unacceptable adverse effects (44,45). Depending on the pain mechanism and anatomical distribution, options include peripheral nerve or plexus blocks, neuraxial techniques, epidural or intrathecal drug delivery, and selected neurolytic procedures (46). Intrathecal therapy may involve opioids and selected adjuvant agents (47). Ziconotide, a selective N-type voltage-gated calcium channel blocker, may be considered as an intrathecal option for appropriately selected patients with severe or refractory cancer-related pain (48,49). Interventional approaches require specialized expertise and careful patient selection, and the evidence base specifically for CRNP remains limited (44). Intrathecal drug delivery has an established role in selected patients with refractory cancer pain and continues to be investigated, particularly in those with severe or treatment-resistant malignant pain (50).

Non-pharmacological treatment

Non-pharmacological interventions should be considered as part of a multidisciplinary approach to CRNP (51,52). Psychological interventions, including cognitive-behavioral strategies, relaxation techniques, and supportive therapies, may help reduce pain-related distress, anxiety, and functional impairment (51). Physical rehabilitation and occupational therapy may support mobility and independence, particularly in patients with treatment-related neuropathy or functional limitations (52,53). Complementary approaches, including acupuncture and other integrative techniques, have been investigated in cancer-related and chemotherapy-induced neuropathic pain (54,55). However, the available evidence remains heterogeneous, and limitations in study quality, sample size, and intervention protocols prevent recommending these approaches as standalone treatments (54). Their use should therefore complement, rather than replace, evidence-based pharmacologic and oncologic management (52).

Future perspectives

Future research in CRNP is increasingly focused on personalized, mechanism-based approaches (56). Genetic variability in opioid receptors, drug-metabolizing enzymes, transporters, ion channels, and inflammatory pathways may contribute to interindividual differences in analgesic response and adverse effects (57,58). Pharmacogenomic approaches, including investigation of CYP2D6 and OPRM1 variability, may ultimately support individualized analgesic selection, although routine clinical implementation remains limited (59).

Advances in single-cell sequencing and other molecular technologies may provide greater insight into interactions among tumor cells, immune cells, glial cells, and sensory neurons (60,61). These approaches may help identify biomarkers for neuropathic pain phenotypes and novel neuroimmune therapeutic targets (14,62). Further research is also needed to evaluate targeted neuromodulation, new analgesic agents, and interventions directed at tumor–nerve interactions (18).

Despite growing understanding of the biological mechanisms underlying CRNP, high-quality clinical evidence remains limited (57,62). Future studies should use standardized diagnostic criteria, clearly defined neuropathic phenotypes, patient-centered outcomes, and adequately powered prospective designs (56,57).

Discussion

Cancer-related neuropathic pain (CRNP) is a heterogeneous, multifactorial condition that often coexists with nociceptive, visceral, inflammatory, and bone pain (5). The reviewed evidence suggests that direct tumor-related nerve injury, nerve compression or infiltration, treatment-induced neurotoxicity, neuroinflammation, and peripheral and central sensitization may all contribute to its development and persistence (19). This complexity presents a major challenge for both diagnosis and treatment (59). CRNP should therefore not be considered a single pain entity but rather a heterogeneous clinical phenotype in which the relative contribution of individual mechanisms may change with tumor progression and treatment exposure (5). This heterogeneity may partly explain the variable response to analgesic therapy and the limited applicability of treatment recommendations in the broader population of patients with CRNP (19). A mechanism-based assessment is therefore essential to determine whether treatment should primarily target nerve injury, tumor-related nociceptive pain, inflammation, or central sensitization (59). In clinical practice, this supports the use of multimodal treatment strategies rather than reliance on a single class of analgesic (5).

Neuroinflammation appears to play a central role in the transition from acute to chronic pain (19). Tumor cells, immune cells, sensory neurons, and glial cells form a dynamic network in which inflammatory mediators increase neuronal excitability and promote peripheral and central sensitization (17). Neuroinflammation may therefore link tumor-related nerve injury and treatment-induced neurotoxicity to persistent pain (21), highlighting neuroimmune processes as potential targets for future mechanism-based therapies (20).

Early recognition of the neuropathic component is crucial because clinical manifestations may be masked by other cancer-related symptoms or may occur as part of a mixed pain phenotype (8). Clinical assessment based on established neuropathic pain criteria, neurological examination, and validated screening tools such as the Douleur Neuropathique 4 (DN4) can facilitate identification and guide treatment selection (63).

Opioids remain important for moderate to severe cancer pain, but their efficacy in CRNP may vary based on the underlying pain mechanisms (6,33). Neuropathic mechanisms, central sensitization, opioid tolerance, and opioid-induced hyperalgesia may contribute to incomplete analgesic responses in some patients (32). Opioid signaling may also interact with immune and inflammatory pathways within

the tumor microenvironment and nervous system. Therefore, opioid therapy should be individualized, regularly reassessed, and, when appropriate, combined with mechanism-specific adjuvant therapies (33). Gabapentinoids, tricyclic antidepressants, and serotonin–norepinephrine reuptake inhibitors may be selected according to the pain phenotype, comorbidities, concomitant medications, and risk of adverse effects (64). Interventional procedures, rehabilitation, and psychological interventions may provide additional benefit in refractory or complex cases (51).

A major limitation of the current evidence is the lack of high-quality clinical studies specifically addressing CRNP (52). Much of the available evidence has been extrapolated from larger populations with neuropathic pain or from studies of chemotherapy-induced peripheral neuropathy, limiting the certainty of CRNP-specific recommendations (57). Emerging research into neuroimmunological mechanisms, molecular targets, and pharmacogenomics may contribute to more personalized treatment, but such approaches still require clinical validation (65). The heterogeneity of CRNP highlights the need to advance personalized treatment (59). Differences in tumor biology, treatment exposure, neurological injury, inflammatory responses, and genetic determinants of analgesic metabolism may contribute to significant interindividual variability in both pain phenotype and response to treatment (58). Pharmacogenomic data, including variations in opioid receptors and drug-metabolizing enzymes, may ultimately help identify patients at increased risk of inadequate analgesia or side effects (65). However, these approaches should currently be considered investigational rather than routine clinical tools (57). Their future value will depend on prospective validation and integration with clinical phenotyping and objective assessment of neuropathic mechanisms (59).

Future studies should focus on developing clinically meaningful phenotypes of CRNP that integrate anatomical, neurological, inflammatory, and molecular characteristics. Such phenotyping could improve patient stratification in clinical trials and help determine which patients are most likely to benefit from opioids, adjuvant analgesics, interventional procedures, or novel mechanism-specific therapies (66). Integrating clinical assessments with molecular profiling, neuroimaging, and pharmacogenetic data could ultimately enable the transition from predominantly symptom-based treatment to mechanism-based therapy selection.

Limitations

This review has several limitations. As a narrative review, it did not follow a systematic review protocol or include a quantitative meta-analysis. The available evidence on CRNP is limited and heterogeneous, with many recommendations based on studies of broader neuropathic pain populations or chemotherapy-induced peripheral neuropathy rather than CRNP-specific cohorts. Differences in patient populations, cancer types, diagnostic approaches, and treatment protocols restrict direct comparisons between studies and limit the generalizability of the findings.

Conclusion

CRNP is a complex, heterogeneous condition that requires early recognition and individualized multimodal management. Current treatment combines appropriate oncologic therapy with pharmacological, interventional, rehabilitative, and psychological approaches. Although advances in neuroimmunology and pharmacogenomics offer promising therapeutic opportunities, CRNP-specific clinical evidence remains limited. Further well-designed prospective studies are needed to establish effective mechanism-based, personalized treatment strategies and improve pain control and quality of life in patients with cancer. Rather than focusing solely on reducing pain intensity after CRNP has become established, future therapeutic strategies should aim to modify the biological processes responsible for nerve injury, neuroinflammation, peripheral and

central sensitization, and subsequent pain chronification.

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Neuropatska bol povezana s karcinomom: pregled suvremenih dijagnostičkih i terapijskih pristupa

Sažetak

Cilj: Cilj ovoga preglednog članka jest sažeti dosadašnja saznanja o etiologiji, patofiziologiji, dijagnostici, kliničkim obilježjima i mogućnostima liječenja neuropatske boli povezane s rakom (CRNP), s naglaskom na novijim spoznajama o farmakološkim i nefarmakološkim strategijama liječenja.

Metode: Proveden je narativni pregled suvremene literature i kliničkih preporuka o neuropatskoj boli povezanoj s rakom. Analizirani su relevantni dokazi o etiologiji, patofiziologiji, dijagnostici, kliničkim obilježjima te farmakološkom i nefarmakološkom liječenju CRNP-a. Posebna je pozornost posvećena dijagnostičkim kriterijima koje preporučuje Međunarodno udruženje za proučavanje boli (IASP), validiranim alatima za procjenu, poput upitnika DN4, neuroimunološkim mehanizmima u podlozi neuropatske boli te suvremenim terapijskim pristupima.

Rezultati: Pregledani dokazi pokazuju da je neuropatska bol povezana s rakom složen, multifaktorski poremećaj povezan s tumorskom invazijom, kompresijom živaca, oštećenjem živaca izazvanim kemoterapijom i radioterapijom, neuroinflamacijom i centralnom senzibilizacijom. Dijagnoza se temelji na prepoznavanju karakterističnih osjetnih simptoma i dokazu zahvaćenosti živčanog sustava, pri čemu validirani alati, poput upitnika DN4, mogu dodatno poduprijeti kliničku procjenu. Suvremeni dokazi podupiru multimodalno farmakološko liječenje, uključujući opioide u kombinaciji s adjuvantnim lijekovima, kao što su gabapentinoidi, triciklički antidepresivi te inhibitori ponovne pohrane serotonina i noradrenalina. Novi terapijski pristupi, uključujući monoklonska protutijela, kanabinoide, intratekalne analgetike i ciljanu molekularnu terapiju, pokazuju obećavajuće, ali još uvijek ograničene dokaze o učinkovitosti. Nefarmakološke intervencije, uključujući akupunkturu, kognitivno-biheviоralne terapije i rehabilitaciju, mogu pružiti dodatnu korist u kontroli simptoma i poboljšanju kvalitete života.

Zaključak: Neuropatska bol povezana s rakom i dalje je nedovoljno prepoznata i teško liječiva te značajno narušava kvalitetu života bolesnika i ishode liječenja. Rana dijagnoza, individualizirano multimodalno liječenje te integracija farmakoloških i suportnih intervencija ključni su za učinkovito zbrinjavanje. Budući napredak u molekularnoj biologiji, imunologiji i farmakogenomici mogao bi omogućiti ciljane i personaliziranije terapijske strategije za bolesnike s neuropatskom boli povezanoj s rakom.

Ključne riječi: analgetici; novotvorine; neuroinflamacija