Clinical pain phenotypes in breast cancer survivors: the role of neuropathic pain and central sensitization

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Abstract

Introduction: Persistent pain is a common long-term sequela in breast cancer survivors treated with chemotherapy and may involve neuropathic pain and central sensitization (CS). However, few studies have examined these mechanisms together and their association with specific sensory symptoms.
Material and methods: A multicenter cross-sectional study was conducted in four Spanish hospitals between September 2022 and January 2025. A total of 254 women with surgically treated breast cancer who had completed chemotherapy were included. Neuropathic pain was assessed using the Spanish version of the Douleur Neuropathique 4 (DN4), CS using the Central Sensitization Inventory (CSI), and pain intensity and sensory symptoms (paresthesia, tingling, and thermal sensitivity) using visual analog scales. Between-group comparisons according to the presence of neuropathic pain and central sensitization, as well as analyses of their independent and combined effects on pain intensity and sensory symptoms, were performed.
Results: Participants reported moderate pain intensity, with clinically relevant neuropathic pain features and CS. Patients with neuropathic pain (DN4 $\geq$ 4) showed significantly higher pain intensity and greater severity of all sensory symptoms (all $p < 0.001$). Similarly, participants with CS reported higher pain intensity and greater severity of tingling and thermal sensitivity. Neuropathic pain and CS were independently associated with increased symptom burden and frequently coexisted.
Conclusions: Neuropathic pain and CS are common and overlapping components of persistent pain in breast cancer survivors treated with chemotherapy and are associated with greater pain intensity and sensory symptom severity. Concurrent assessment of both mechanisms may improve the clinical characterization and management of post-chemotherapy pain.

Key words: neuropathic pain, central sensitization, breast cancer, sensory symptoms.

Introduction

Over recent decades, cancer has become one of the leading causes of death worldwide, and its global burden is expected to continue increas-

ing substantially in the coming years [1]. Advances in early detection and the development of new therapeutic strategies have improved the prognosis of many cancers, meaning that advanced cancer is no longer necessarily considered a terminal condition. As a result, clinical care has shifted toward addressing the long-term sequelae associated with both the disease and its treatments, of which pain represents a major concern [2, 3].

Breast cancer exemplifies this clinical scenario. It is the most frequently diagnosed cancer among women worldwide and is associated with 5-year survival rates exceeding 90%, leading to a growing population of survivors experiencing persistent consequences of the disease and its treatment [4, 5]. Among these sequelae, pain is one of the most prevalent symptoms, affecting more than 40% of women following cancer treatments [6]. Several studies have shown that persistent pain is more common in women who have received chemotherapy and/or radiotherapy, and that a substantial proportion of these patients experience signs consistent with central nervous system hypersensitivity even more than 1 year after treatment completion [4, 7, 8].

Pain is classified into two types: nociceptive and neuropathic pain. Nociceptive pain arises from actual or threatened damage to non-neural tissue and is mediated by the activation of peripheral nociceptors, which usually reflects ongoing tissue injury or inflammation [9]. However, this framework does not fully explain persistent pain conditions in which pain severity is disproportinate to peripheral nociceptive input or persist after tissue healing. In this context, central sensitization (CS) has been proposed as a key mechanism to explain certain forms of persistent pain that are not proportional to peripheral nociceptive inputs. It is defined as a sustained increase in the excitability and synaptic efficacy of neurons involved in central nociceptive pathways [10, 11]. This phenomenon is associated with upregulation of the nociceptive system and may involve multiple levels of the central nervous system, including the spinal cord and supraspinal structures, the brainstem, thalamus, limbic system, and the cerebral cortex [12]. CS typically manifests as pain hypersensitivity, characterized by signs and symptoms such as tactile allodynia, secondary pressure hyperalgesia, increased temporal summation, and persistence of pain sensations after stimulus removal [10, 13]. These manifestations reflect underlying neurophysiological changes, including lower activation thresholds, exaggerated responses to harmful stimuli, and amplification of central nociceptive processing [13].

When CS is present, the central nervous system can modify or amplify the pain experience, increasing its intensity, duration, and spatial extent in a way that no longer directly corresponds to the characteristics of the original peripheral stimulus [13, 14]. As a result, nociceptive neurons may become activated by innocuous low-threshold stimuli, leading to painful perceptions in the absence of tissue damage. In this context, pain becomes disproportionate to the severity of the underlying lesion [15]. Experimental studies in humans have shown that the presence of allodynia, secondary hyperalgesia, increased temporal summation, and persistent sensory side effects is indicative of central amplification related to increased excitation or reduced inhibitory control [10, 12, 13]. Clinically, these changes may lead to lowered pain thresholds, exaggerated responses to harmful stimuli, pain persisting after stimulus cessation, and spread of sensitivity to adjacent or distant tissues [11].

Neuropathic pain represents another characteristic component of cancer-related pain and is associated with specific clinical classification criteria. According to standards established by the Neuropathic Pain Special Interest Group (NeuPSIG) of the International Association for the Study of Pain, neuropathic pain is defined as pain arising as a direct consequence of a lesion or disease affecting the somatosensory system, at either the central or peripheral level [7, 11]. Its identification requires a neuroanatomically plausible pain distribution and consistency between etiology, pain distribution, and clinical examination findings [16]. Therefore, assessment should not be limited to pain localization alone, but should also consider the distribution of hyperalgesia and other sensory abnormalities.

Clinically, neuropathic pain is characterized by the coexistence of positive and negative somatosensory phenomena, reflecting both gains and losses in sensory function. Positive symptoms include spontaneous or evoked pain, as well as burning sensations, tingling, stabbing pain, electric sensations, and mechanical or thermal allodynia or hyperalgesia. In contrast, negative symptoms manifest as hypoesthesia or reduced sensitivity to light touch, vibration, or pain [7]. In oncology, neuropathic pain frequently develops as chemotherapy-induced peripheral neuropathy (CIPIN), due to the effects on peripheral nerves [16].

The mechanisms involved in neuropathic pain are not exclusive and frequently overlap with sensitization processes. Hyperexcitability may affect both central and peripheral pathways, which helps explain why cancer survivors may simultaneously exhibit neuropathic pain features and signs of CS, resulting in mixed pain phenotypes [11]. This overlap has important clinical implications, as cancer-related neuropathic pain has been associated

with higher analgesic requirements, poorer functional status, and greater interference with activities of daily living compared to nociceptive pain [16]. Furthermore, symptoms such as numbness, tingling, or electric sensations have been linked to increased disability, balance disturbances, loss of manual dexterity, and limitations in physical and occupational functioning [17].

In women living beyond breast cancer, sustained nociceptive input and treatment-related physiological stress may drive neuroplastic changes that contribute to development of CS [18]. Consequently, neuropathic sensory features may coexist with signs of generalized pain amplification, resulting in a more complex pain presentation [14, 19]. This interaction between peripheral and central mechanisms helps explain the observed clinical variability and reinforces the need to jointly assess both dimensions to characterize the pain phenotype in this population.

Although current evidence highlights the relevance of both neuropathic pain and CS in oncology, important gaps remain in understanding how these mechanisms interact and contribute to the clinical pain profile in breast cancer patients. Few studies have simultaneously examined neuropathic pain features and symptoms related to CS, as well as their relationship with pain intensity and specific sensory manifestations [6]. Therefore, the aim of this study was to describe the clinical profile of neuropathic pain and CS in breast cancer survivors treated with chemotherapy and to analyze differences in pain intensity and the severity of specific sensory symptoms (paresthesia, tingling, and thermal sensitivity) according to the presence or absence of neuropathic pain and CS. It was hypothesized that patients with neuropathic pain and/or CS would report higher pain intensity and more severe sensory symptoms compared with patients without neuropathic pain and without CS.

Material and methods

Study design

A multicenter cross-sectional observational study was conducted following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for observational studies [20].

Participants

The study population consisted of women diagnosed with breast cancer who had undergone surgical treatment and subsequently received chemotherapy. Only patients who had completed chemotherapy within the first year after treatment completion were considered eligible for inclusion. Patient recruitment was performed between September 2022 and January 2025 in four Spanish hospitals. Chemotherapy regimens were varied and reflected routine oncology practice. Treatments included taxane-based, platinum-based, anthracycline-based, and combination regimens, with or without targeted or hormonal therapies. All eligible patients attending routine follow-up visits after completion of chemotherapy were invited to participate, regardless of the presence or absence of pain or sensory symptoms.

Participants were eligible for inclusion if they met the following criteria: female sex, age $\geq$ 18 years, history of breast cancer treated surgically, chemotherapy treatment, and completion of chemotherapy treatment prior to study participation. Patients were excluded if they had not undergone prior surgical treatment for breast cancer or if they had received pharmacological treatments other than chemotherapy (e.g., excluding hormonal or targeted therapies) as part of their cancer treatment. Patients who did not receive chemotherapy were excluded from the sample.

Outcomes assessment

CS was assessed using the Spanish version of Central Sensitization Inventory (CSI), a screening tool that evaluates 25 health-related symptoms and is scored using a five-point Likert scale ranging from 0 ("never") to 4 ("always"), yielding a total score between 0 and 100 [21]. Higher scores indicate a greater degree of self-reported symptomatology related to CS and can be classified into different severity levels [10, 22]. The CSI has demonstrated solid clinicmetric properties, including high test–retest reliability and adequate internal consistency, and has been validated in multiple clinical populations, including breast cancer patients [5, 23].

Neuropathic pain was evaluated using the Spanish version of the Douleur Neuropathique 4 (DN4) questionnaire, a clinical instrument designed to identify clinicmetric and psychometric neuropathic pain features [24]. The DN4 consists of ten items, seven of which correspond to symptom descriptors assessed through patient interview, including cold or burning pain, electric sensations, tingling, pins and needles, numbness, and itching. The remaining three items are based on clinical examination and include the assessment of hypoesthesia to light touch, hypoesthesia to pinprick, and the presence of brush-evoked allodynia [25]. The total score ranges from 0 to 10, with a score of 4 or higher considered indicative of neuropathic pain [26]. This instrument has shown adequate discriminative performance for differentiating neuropathic from non-neuropathic pain and high sensitivity for identifying neuropathic pain in oncological populations [4].

Pain intensity was measured using the Visual Analog Scale (VAS), a tool consisting of a continuous 10-cm line anchored by two verbal descriptors at each end. For pain assessment, the anchors were defined as "no pain" and "the worst pain imaginable". Participants marked the point on the scale that best represented their pain intensity at the time of assessment. The VAS allows continuous quantification of pain intensity and has demonstrated good test–retest reliability, supporting its use in clinical research settings [27].

The severity of specific sensory symptoms was assessed using independent visual analog scales, following the same format as the pain VAS. Separate scales were used to evaluate paresthesia, tingling, and thermal sensitivity, allowing a symptom-specific assessment. Each scale consisted of a continuous 10-cm line, with anchors defined as absence of the symptom and the maximum imaginable intensity of that symptom. Participants rated the perceived intensity of each symptom independently, enabling differentiated quantification of the magnitude of the various sensory manifestations associated with pain [28, 29].

Procedure

Data collection was performed in person at the participating centers. Questionnaires were completed under the supervision of a member of the research team to ensure data collection. Depending on patient preference and logistical considerations, questionnaires were completed either in paper format or electronically using a secure online form (Google Forms). All variables were assessed at a single time point after completion of chemotherapy.

Statistical analysis

Descriptive analyses were performed to summarize participant characteristics and study variables, and results are presented as mean and standard deviation (SD) or median and interquartile range, as appropriate. Normality of continuous variables was assessed using the Shapiro–Wilk test; variables were considered normally distributed when $p \geq 0.05$; all continuous variables departed from normality (all $p < 0.001$). Between-group comparisons were performed according to the presence of neuropathic pain, CS independent samples *t*-tests were used to compare continuous variables between groups, and effect sizes were reported as Cohen's *d*. Categorical variables were compared using χ^2 tests or Fisher's exact test where appropriate; log odds ratios with 95% confidence intervals were reported. Additionally, two-way analysis of variance (ANOVA) was conducted to examine the main and interaction effects of neuropathic pain (yes/no) and CS (yes/no) on pain intensity and sensory symptom severity. Effect sizes were expressed as partial eta squared. Statistical significance was set at $p < 0.05$. Effect sizes were interpreted according to Cohen's conventional thresholds, with values of 0.2, 0.5, and 0.8 representing small, moderate, and large effects, respectively [30]. All analyses were performed using JASP (version 0.95.4).

Results

Sociodemographic and clinical characteristics of the study sample (n = 254) are summarized in Table I. Overall, participants reported moderate pain intensity together with clinically relevant neuropathic pain features and symptoms consistent with central sensitization. Based on DN4 and CSI scores, a substantial proportion of the sample met criteria for neuropathic pain and CS, while the inclusion of participants without these conditions allowed estimation of their incidence in this population. As illustrated in Figures 1 A–D, patients with neuropathic pain reported significantly higher pain intensity and greater severity of sensory symptoms, including paresthesia, tingling, and

Table I. Sociodemographic and descriptive data

Variable	Mean ± SD	Median [IQR]	Min–max
Age [years]	52.2 ±10.0	50.0 [13.0]	36.0–83.0
Height [cm]	162.6 ±6.9	161.0 [9.0]	148.0–189.0
Weight [kg]	66.5 ±14.6	64.0 [17.5]	44.0–128.0
BMI [kg/m²]	25.13 ±5.7	24.00 [6.0]	17.9–53.3
Neuropathic pain (DN4)	4.55 ±2.5	4.00 [3.0]	0.0–10.0
Central sensitization (CSI)	40.23 ±19.2	40.0 [26.00]	4.0–91.0
VAS pain	4.11 ±3.2	5.00 [7.0]	0.0–10.0
VAS paresthesia	5.16 ±3.1	6.00 [5.0]	0.0–10.0
VAS tingling	4.4 ±3.3	5.0 [6.7]	0.0–10.0
VAS thermal sensitivity	4.4 ±3.7	4.5 [8.0]	0.0–10.0

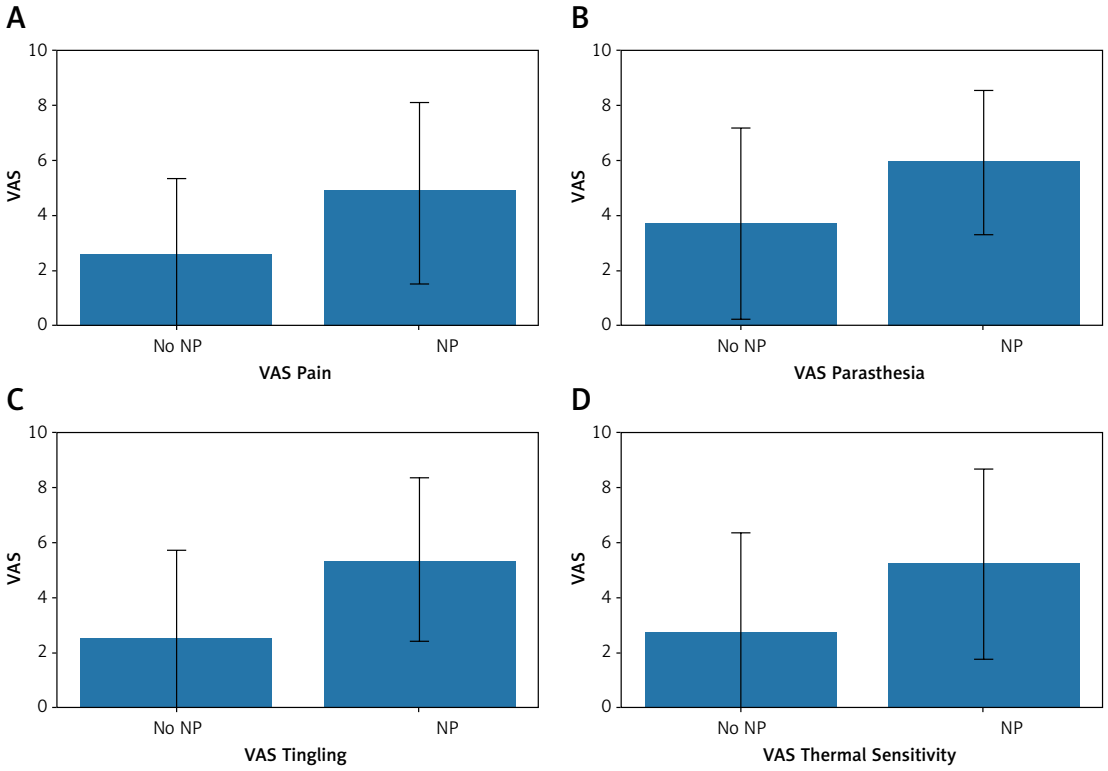

Figure 1. Pain intensity and sensory symptoms according to the presence of neuropathic pain (NP). Participants with neuropathic pain (DN4 ≥ 4) showed significantly higher scores in pain intensity (**A**) and sensory symptoms, including paresthesia (**B**), tingling (**C**), and thermal sensitivity (**D**), compared with those without neuropathic pain (all $p < 0.001$). Bars represent mean values

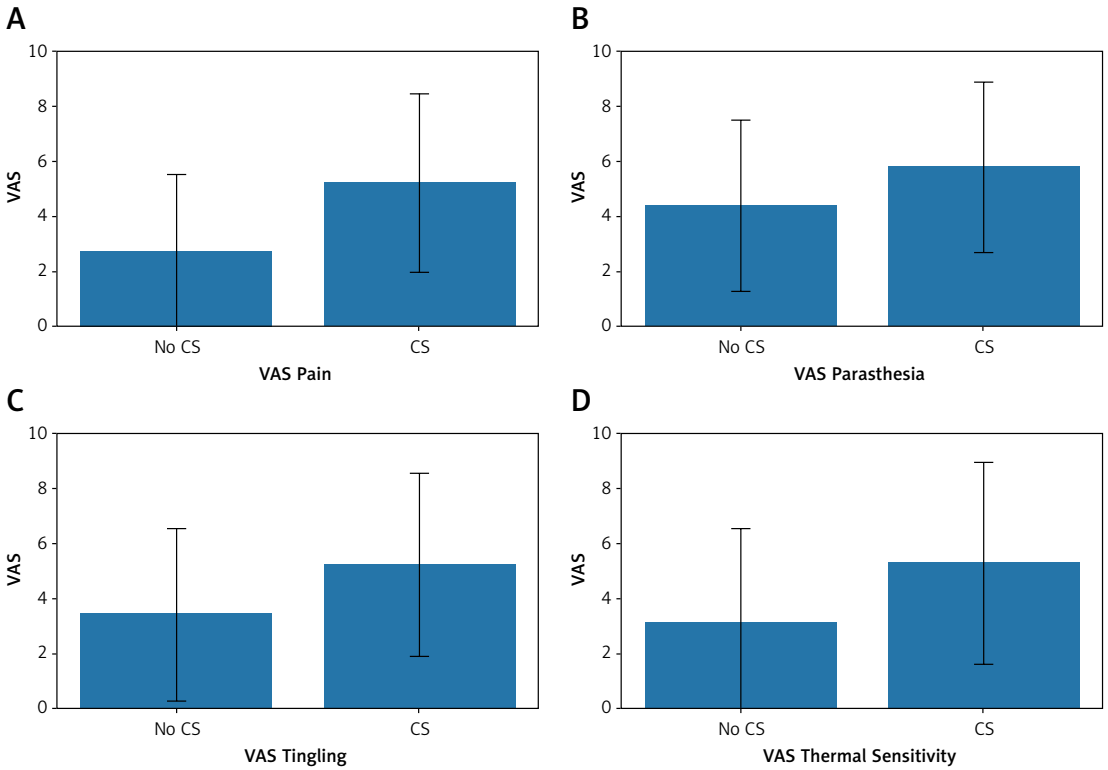

Figure 2. Pain intensity and sensory symptoms according to the presence of central sensitization (CS). Participants with central sensitization reported higher pain intensity and more severe sensory symptoms than those without central sensitization. Differences were significant for pain (**A**), tingling (**C**), and thermal sensitivity (**D**) ($p < 0.001$), while paresthesia (**B**) showed a smaller effect size. Bars represent mean values

thermal sensitivity, compared with those without neuropathic pain (all $p < 0.001$), with large effect sizes (Table II). Similarly, higher pain intensity and more severe sensory symptoms were observed in participants with central sensitization, as shown in Figures 2 A–D, with small-to-moderate effect sizes (Table III). Categorical analyses further demonstrated that neuropathic pain was strongly associated with a higher proportion of sensory symptoms, particularly paresthesias, tingling, and thermal sensitivity (Table IV), whereas central sensitization showed more modest but significant associations with pain intensity and selected sensory manifestations, notably tingling and thermal sensitivity (Table V).

Discussion

The aim of the present study was to characterize neuropathic pain and CS in women with breast cancer treated with chemotherapy and to examine their relationship with pain intensity and specific sensory symptoms. The findings suggest that both neuropathic pain and CS are associated with a greater symptom burden [30].

In our sample, participants showed mean DN4 and CSI scores consistent with the presence of neuropathic pain and central sensitization, with values comparable to those previously reported in breast cancer populations assessed using the same instruments. This finding suggests that, in a substantial proportion of women with breast cancer, persistent pain cannot be explained solely by peripheral nociceptive mechanisms. The coexistence of neuropathic pain and CS points to the involvement of nociceptive signal amplification and altered sensory processing [14, 18].

When differences were analyzed according to the presence of neuropathic pain, patients exhibited significantly higher scores for pain intensity as well as for all evaluated sensory symptoms (paresthesia, tingling, and thermal sensitivity), with effect sizes ranging from moderate to large (d = 0.73–0.94). These results are consistent with

Table II. Clinical features according to the presence of neuropathic pain

Variable	No neuropathic pain (mean ± SD)	Neuropathic pain (mean ± SD)	*P*-value	Effect size (Cohen's *d*)
VAS pain	2.62 ±2.72	4.87 ±3.26	< 0.001	–0.726
VAS paresthesia	3.68 ±3.46	5.91 ±2.61	< 0.001	–0.760
VAS tingling	2.52 ±3.19	5.37 ±2.96	< 0.001	–0.936
VAS thermal sensitivity	2.66 ±3.69	5.23 ±3.41	< 0.001	–0.734

Table III. Clinical features according to the presence of central sensitization

Variable	No central sensitization (mean ± SD)	Central sensitization (mean ± SD)	*P*-value	Effect size (Cohen's *d*)
VAS pain	2.741 ±2.796	5.197 ±3.214	< 0.001	–0.809
VAS paresthesia	4.384 ±3.053	5.768 ±3.022	< 0.001	–0.456
VAS tingling	3.420 ±3.118	5.190 ±3.290	< 0.001	–0.551
VAS thermal sensitivity	3.179 ±3.404	5.303 ±3.684	< 0.001	–0.596

Table IV. Neuropathic pain associations

Predictor	NP No *N* (No)	NP No *N* (Yes)	NP Yes *N* (No)	NP Yes *N* (Yes)	χ^2 *p*; log(OR) [95% CI]
Pain	38	40	48	128	< 0.001; 0.930 [0.375, 1.484]
Paresthesia	31	10	55	158	< 0.001; 2.187 [1.411, 2.963]
Tingling	47	17	39	151	< 0.001; 2.371 [1.714, 3.028]
Thermal sensitivity	52	35	34	133	< 0.001; 1.760 [1.189, 2.330]

Table V. Central sensitization associations

Predictor	CS No *N* (No)	CS No *N* (Yes)	CS Yes *N* (No)	CS Yes *N* (Yes)	χ^2 *p*; log(OR) [95% CI]
Pain	49	29	63	113	< .001; 1.109 [0.556, 1.662]
Paresthesia	22	19	90	123	0.178; 0.459 [–0.212, 1.130]
Tingling	38	26	74	116	0.004; 0.829 [0.251, 1.407]
Thermal sensitivity	50	37	62	105	0.002; 0.828 [0.300, 1.356]

the clinical definition of neuropathic pain, which is characterized by the presence of both positive and negative sensory symptoms [7]. Moreover, previous evidence indicates that cancer-related neuropathic pain is associated with greater functional interference and a worse clinical course compared with other types of cancer-related pain [17]. In a cross-sectional study conducted in breast cancer, the prevalence of neuropathic pain exceeded 40% and was associated with greater limitations in activities of daily living compared with patients without neuropathic features [31]. Additionally, a multicenter cross-sectional observational study reported a prevalence of cancer-related neuropathic pain of 36%, with a higher frequency of this type of pain among patients with higher VAS pain scores [32].

Our results also show that CS was associated with greater pain intensity and increased severity of sensory symptoms. Pain, tingling, and thermal sensitivity were significantly associated with the presence of CS; however, paresthesia did not show a statistically significant association. This finding suggests that certain sensory symptoms, such as tingling and alterations in thermal sensitivity, may more accurately reflect processes of central pain amplification and dysregulation of sensory processing. These results are consistent with previous studies demonstrating that CS contributes to the prevalence of chronic pain following breast cancer treatment, with patients exhibiting moderate to high levels of CS reporting greater pain intensity and a more complex sensory experience [5]. Similarily, a cohort study in breast cancer patients with pain lasting more than one year found that approximately 38% of patients showed signs of central nervous system hypersensitivity, suggesting that CS may persist even after the completion of cancer treatment [33]. Furthermore, chemotherapy treatments have been associated with a higher likelihood of developing persistent pain, potentially leading to central mechanisms involved in pain maintenance [4].

A relevant finding of this study is that neuropathic pain and CS can coexist, providing complementary information on how symptoms manifest in patients. These results highlight that an assessment based solely on pain intensity may be insufficient and reinforce the need for more multidimensional evaluation approaches. From a clinical perspective, the use of tools that identify both neuropathic features and symptoms related to CS may facilitate more personalized treatment strategies [15].

This study has several limitations. First, its cross-sectional design precludes establishing causal relationships between neuropathic pain, CS, and the sensory symptoms evaluated. Second, CS and neuropathic pain were assessed using self-reported instruments, which do not allow for objective confirmation of the underlying neurophysiological mechanisms. In addition, the assessment of sensory symptoms using VAS may be influenced by psychological or contextual factors. Third, detailed chemotherapy regimens were heterogeneous and not controlled for drug-specific effects. However, participants were included based on exposure to potentially neurotoxic chemotherapy, reflecting real-world oncology practice rather than a drug-comparison design. Finally, the exclusive inclusion of women with breast cancer treated with chemotherapy limits the generalizability of the findings to other oncological populations. Another limitation is the lack of imaging, nerve conduction studies, and electromyography data, as well as the absence of analysis regarding cancer stage and clinico-pathologic type in relation to neuropathic pain. These factors will be considered in future research to further strengthen the findings. Further longitudinal research is needed to confirm the observed associations and to explore their temporal evolution.

In conclusion, neuropathic pain and CS in breast cancer patients treated with chemotherapy are associated with greater pain intensity and more severe sensory symptoms, suggesting that peripheral neuropathic features and central pain processing alterations represent complementary components of the post-chemotherapy pain phenotype. The co-occurrence of neuropathic pain and central sensitization was associated with greater pain severity and sensory symptom burden, supporting the need for their concurrent assessment to improve the clinical characterization and management of pain in this population.

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Ethical approval

The study protocol was reviewed and approved by the Ethics Committee of the Hospital Príncipe de Asturias (approval code: OE 17/2022), which granted authorization for the conduct of the study across multiple hospitals throughout Spain. All procedures involving human participants were conducted in accordance with the ethical standards of the responsible institutional committees and with the principles of the Declaration of Helsinki. Prior to participation, all patients received detailed oral and written information about the study and provided written informed consent. Participation was voluntary, and all participants were

informed of their right to withdraw from the study at any time without any consequences for their clinical care.

Conflict of interest

The authors declare no conflict of interest.

References

1. Kalita M, M Devaraja, Saha I, Chakrabarti A. Global burden of cancer pattern in 2020 & prediction to 2040 among older adults. Indian J Med Res 2024; 160: 397-406.
2. Wagle NS, Nogueira L, Devasia TP, et al. Cancer treatment and survivorship statistics, 2025. CA Cancer J Clin 2025; 75: 308-40.
3. Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021; 71: 209-49.
4. Yoon SY, Oh J. Neuropathic cancer pain: prevalence, pathophysiology, and management. Korean J Intern Med 2018; 33: 1058-69.
5. Nishigami T, Manfuku M, Lahousse A. Central sensitization in cancer survivors and its clinical implications: state of the art. J Clin Med 2023; 12: 4606.
6. Hurth A, Nijzink-Ter Steege J, Scheepbouwer P, et al. Assessment of central sensitization in breast cancer survivors: convergent validity and use of the Central Sensitization Inventory (CSI) and its short-form as a clustering tool. Clin Pract 2021; 11: 607-18.
7. Mulvey MR, Paley CA, Schuberth A, King N, Page A, Neoh K. Neuropathic pain in cancer: what are the current guidelines? Curr Treat Options Oncol 2024; 25: 1193-202.
8. Singh S, Dhiraaj S, Shamshery C, et al. Incidence of different characters of neuropathic pain in cancer patients coming to tertiary care centre in north india over a period of 1 year – an observational study. Indian J Palliat Care 2024; 30: 27-33.
9. Dubin AE, Patapoutian A. Nociceptors: the sensors of the pain pathway. J Clin Invest 2010; 120: 3760-72.
10. Neblett R, Cohen H, Choi Y, et al. The Central Sensitization Inventory (CSI): establishing clinically significant values for identifying central sensitivity syndromes in an outpatient chronic pain sample. J Pain 2013; 14: 438-45.
11. Nijs J, Leysen L, Adriaenssens N, et al. Pain following cancer treatment: Guidelines for the clinical classification of predominant neuropathic, nociceptive and central sensitization pain. Acta Oncol 2016; 55: 659-63.
12. Smart KM, Blake C, Staines A, Thacker M, Doody C. Mechanisms-based classifications of musculoskeletal pain: part 1 of 3: symptoms and signs of central sensitisation in patients with low back (± leg) pain. Man Ther 2012; 17: 336-44.
13. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain 2011; 152 (3 Suppl): S2-15.
14. Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. J Pain 2009; 10: 895-926.
15. Nijs J, Torres-Cueco R, van Wilgen CP, et al. Applying modern pain neuroscience in clinical practice: criteria for the classification of central sensitization pain. Pain Physician 2014; 17: 447-57.
16. Edwards HL, Mulvey MR, Bennett MI. Cancer-related neuropathic pain. Cancers 2019; 11: 373.
17. Tofthagen CS, McMillan SC. Pain, neuropathic symptoms, and physical and mental well-being in persons with cancer. Cancer Nurs 2010; 33: 436-44.
18. Fallon MT. Neuropathic pain in cancer. Br J Anaesth 2013; 111: 105-11.
19. Treede RD, Jensen TS, Campbell JN, et al. Neuropathic pain: redefinition and a grading system for clinical and research purposes. Neurology 2008; 70: 1630-5.
20. Cuschieri S. The STROBE guidelines. Saudi J Anaesth 2019; 13: 31.
21. Cuesta-Vargas AI, Roldan-Jimenez C, Neblett R, Gatchel RJ. Cross-cultural adaptation and validity of the Spanish central sensitization inventory. Springerplus 2016; 5: 1837.
22. Mayer TG, Neblett R, Cohen H, et al. The development and psychometric validation of the central sensitization inventory. Pain Pract 2012; 12: 276-85.
23. Roldán-Jiménez C, Pajares B, Iglesias Campos M, et al. Structural validity and reliability of the Spanish Central Sensitization Inventory in breast cancer survivors. Pain Pract 2021; 21: 740-6.
24. Perez C, Galvez R, Huelbes S, et al. Validity and reliability of the Spanish version of the DN4 (Douleur Neuropathique 4 questions) questionnaire for differential diagnosis of pain syndromes associated to a neuropathic or somatic component. Health Qual Life Outcomes 2007; 5: 66.
25. Bouhassira D, Attal N, Alchaar H, et al. Comparison of pain syndromes associated with nervous or somatic lesions and development of a new neuropathic pain diagnostic questionnaire (DN4). Pain 2005; 114: 29-36.
26. Baek SK, Shin SW, Koh SJ, et al. Significance of descriptive symptoms and signs and clinical parameters as predictors of neuropathic cancer pain. PLoS One 2021; 16: e0252781.
27. Hawker GA, Mian S, Kendzerska T, French M. Measures of adult pain: Visual Analog Scale for Pain (VAS Pain), Numeric Rating Scale for Pain (NRS Pain), McGill Pain Questionnaire (MPQ), Short-Form McGill Pain Questionnaire (SF-MPQ), Chronic Pain Grade Scale (CPGS), Short Form-36 Bodily Pain Scale (SF-36 BPS), and Measure of Intermittent and Constant Osteoarthritis Pain (ICOAP). Arthritis Care Res 2011; 63 Suppl 11: S240-52.
28. Cruccu G, Anand P, Attal N, et al. EFNS guidelines on neuropathic pain assessment. Eur J Neurol 2004; 11: 153-62.
29. Backonja MM, Attal N, Baron R, et al. Value of quantitative sensory testing in neurological and pain disorders: NeuPSIG consensus. Pain 2013; 154: 1807-19.
30. Cohen J. A power primer. Psychol Bull 1992; 112: 155-9.
31. Oh SY, Shin SW, Koh SJ, et al. Multicenter, cross-sectional observational study of the impact of neuropathic pain on quality of life in cancer patients. Support Care Cancer 2017; 25: 3759-67.
32. Rayment C, Hjermstad MJ, Aass N, et al.; EPCRC. Neuropathic cancer pain: prevalence, severity, analgesics and impact from the European Palliative Care Research Collaborative-Computerised Symptom Assessment study. Palliat Med 2013; 27: 714-21.
33. Pineda-Moncusí M, Servitja S, Tusquets I, et al. Assessment of early therapy discontinuation and health-related quality of life in breast cancer patients treated with aromatase inhibitors: B-ABLE cohort study. Breast Cancer Res Treat 2019; 177: 53-60.