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ORIGINAL PAPER

Assessment of Chemotherapy-Induced Peripheral Neuropathy and Its Relationship to Breast Cancer Patients' Quality of Life

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ABSTRACT

Background: *Chemotherapy-induced peripheral neuropathy (CIPN) is a frequent and detrimental drug toxicity during treatment of breast cancer, but its overall effects on the quality of life (QoL) are poorly defined in practice. This research was expected to measure the occurrence and severity of CIPN and to identify its relationship with QoL among breast cancer patients who were subjected to neurotoxic chemotherapy regimens.*

Methods: *A cross-sectional analytical investigation in 186 female breast cancer patients who had completed two cycles of taxane- or platinum-based chemotherapy was carried out in a tertiary oncology facility from January 2023 to December 2024. Chemotherapy-induced peripheral neuropathy and QoL were measured.*

Results: *Overall CIPN prevalence (NCI-CTCAE Grade ≥ 1) was 67.7% (n = 126); Grades 1, 2 and 3 neuropathy were observed in 33.3%, 22.6% and 11.8% of the cohort, respectively. Clinically meaningful CIPN (Grade ≥ 2) was present in 34.4% (n = 64) of study participants. Sensory (61.8%), motor (38.7%) and autonomic (24.2%) manifestations were recorded. Global QoL scores were significantly lower in patients with CIPN than those without CIPN (51.3 ± 14.7 vs 72.6 ± 11.2, $p < 0.001$). Severity of CIPN showed significant negative correlations with global QoL ($r = -0.64$, $p < 0.001$), physical functioning ($r = -0.58$, $p < 0.001$) and role functioning ($r = -0.52$, $p < 0.001$). Higher cumulative dose, taxane-platinum combination therapy, older age and comorbid diabetes were associated with greater CIPN severity in multivariable analysis.*

Conclusion: *The systematic screening and early intervention measures are justified to reduce the neuropathic burden and maximize patient-centered outcomes.*

Keywords: chemotherapy-induced peripheral neuropathy, breast cancer, quality of life, taxane, neurotoxicity, EORTC QLQ-CIPN20.

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INTRODUCTION

Breast cancer is the most commonly diagnosed cancer among women worldwide, with an estimated 2.3 million new cases reported in 2020, creating a substantial global healthcare burden (1). Systemic chemotherapy has substantially improved survival outcomes; however, cytotoxic treatment can produce adverse effects that persist beyond active therapy and compromise survivorship and the quality of life (QoL) (2). Chemotherapy-induced peripheral neuropathy (CIPN) is among the most clinically important dose-limiting toxicities and is most frequently associated with taxanes, platinum compounds and vinca alkaloids (3).

Chemotherapy-induced peripheral neuropathy is a continuum of sensory, motor and autonomic dysfunctions caused by damage to peripheral nerve fibres. Frequently reported symptoms include distal numbness, tingling, burning pain, loss of proprioception, muscle weakness and difficulty with fine motor control (4). The pathophysiology of CIPN is multifactorial and includes mitochondrial dysfunction, axonal transport disruption, oxidative stress, neuroinflammation and ion-channel dysfunction, which contribute to different clinical manifestations according to the neurotoxic agent used (5).

Epidemiological evidence has shown the occurrence of CIPN in about 30-70% of patients undergoing neurotoxic chemotherapy, and the incidence and severity vary based on the drug used, cumulative dose, treatment duration and individual patient risk factors (5, 6). Taxane-based strategies such as paclitaxel and docetaxel, which are widely used in adjuvant and neoadjuvant therapies of breast cancer, are major contributors to CIPN development in this population (6).

The clinical outcomes of CIPN greatly exceed neuropathic symptomatology. Accumulating evidence suggests that CIPN significantly impairs health-related QoL by causing functional limitation, increasing psychological distress and affecting activities of daily living (7, 8). Chemotherapy-induced peripheral neuropathy is also associated with fall and balance-disturbance risk, which can increase morbidity and reduce independence (9). Additional breast-cancer QoL data and supportive-care intervention studies suggest that better symptom detection and rehabilitation-focused strategies may protect functional outcomes (10-12). Although the clinical significance of CIPN has been identified, large-scale research that conducts an in-depth assessment of the association between CIPN severity and QoL domains in breast cancer populations using established patient-reported outcome measures remains limited. More specifically, modifiable and non-modifiable risk factors that influence CIPN severity in this population require further exploration to support individualized risk stratification and management.

Thus, the current study was aimed at filling this gap by methodically evaluating the occurrence rate and severity of CIPN among women with breast cancer receiving neurotoxic chemotherapy regimens and by assessing whether CIPN severity was related to various domains of health-related QoL. The research objectives included: 1) to identify the prevalence and grading distribution of CIPN among study participants; 2) to describe the sensory, motor and autonomic symptom profiles; 3) to assess the relationship between the CIPN severity and the QoL domains; and 4) to determine the demographic and clinical factors related to CIPN severity. ❑

MATERIALS AND METHODS

Study design and setting

This cross-sectional analytical study was done in the Medical Oncology Department of a tertiary healthcare centre between January 2023 and December 2024.

The study protocol was approved by the Institutional Ethics Committee of the participating tertiary care centre. All procedures conformed to the Declaration of Helsinki and written informed consent was obtained from every participant before enrolment. The manuscript was prepared in accordance with the STROBE statement for cross-sectional observational studies and a completed STROBE checklist should be submitted as supplementary material.

Study population and sampling

The study population comprised female patients aged at least 18 years, with histologically confirmed breast cancer (stages I–III), who had finished at least two cycles of neurotoxic chemotherapy (taxane-based or platinum-based

regimens). A consecutive sampling technique was employed to recruit eligible participants attending outpatient follow-up clinics during the study period.

Inclusion criteria were as follows: i) histologically confirmed invasive breast carcinoma; ii) completion of a minimum of two cycles of taxane-based (paclitaxel or docetaxel) or platinum-based chemotherapy; iii) Eastern Cooperative Oncology Group (ECOG) performance status of 0–2; and iv) willingness to render informed consent.

Exclusion criteria comprised: i) pre-existing peripheral neuropathy from other etiologies (*e.g.*, diabetic neuropathy identified before chemotherapy initiation, hereditary neuropathies, or nutritional deficiency neuropathies); ii) concurrent or prior exposure to other known neurotoxic medications unrelated to cancer treatment; iii) active central nervous system metastases; iv) severe cognitive impairment precluding questionnaire completion; and v) concurrent participation in clinical trials involving neuroprotective interventions.

Data collection instruments

• EORTC QLQ-CIPN20 – The 20-item EORTC QoL Questionnaire-Chemotherapy-Induced Peripheral Neuropathy module was used to assess patient-reported CIPN symptoms across sensory (nine items), motor (eight items) and autonomic (three items) domains. Items are rated on a four-point Likert scale (1 = not at all to 4 = very much), with higher scores indicating greater symptom burden. Raw scores were linearly transformed to a 0-100 scale according to EORTC scoring procedures (13, 14).

• EORTC QLQ-C30 – The 30-item EORTC QoL Questionnaire-Core 30 was used to assess global health-related QoL, five functional scales (physical, role, emotional, cognitive and social functioning), three symptom scales (fatigue, nausea/vomiting and pain) and six single-item symptom measures. Scores were linearly transformed to a 0-100 scale. For global QoL and functional scales, higher scores indicate better QoL or functioning; for symptom scales and single-item measures, higher scores indicate greater symptom burden (15).

Potential participants were approached during routine outpatient follow-up visits. Self-administered questionnaires were completed under the supervision of a trained research nurse after obtaining written informed consent. Clinical data were extracted from electronic medical records. The average questionnaire completion time was approximately 20 minutes. Missing data were checked before analysis; item-level missingness for EORTC QLQ-CIPN20 and QLQ-C30 was 1.6% and 1.9%, respectively. Because missingness was below 5% and appeared random, scale scores were calculated according to EORTC scoring rules when at least half of the items in a scale were completed; otherwise, the relevant scale was treated as missing. No participant was excluded from the primary prevalence analysis.

Statistical analysis was performed using IBM SPSS Statistics version 27.0 for Windows. Continuous variables were examined using histograms, Q-Q plots and Shapiro-Wilk test; variance homogeneity was assessed with Levene's test before ANOVA. Variables that were approximately normally distributed were compared using independent-samples t-tests or one-way ANOVA with Tukey HSD post-hoc testing when variances were homogeneous; when assumptions were violated, Mann-Whitney U, Kruskal-Wallis with Dunn-Bonferroni post-hoc testing, or Games-Howell post-hoc testing was used as appropriate. Categorical variables were compared using Pearson chi-square tests or Fisher's exact tests and the exact statistic used was stated in the results. Pearson correlation was used for approximately normal continuous outcomes, while Spearman rho was used in sensitivity analyses for skewed scores. Effect sizes were reported as Cohen's d for two-group comparisons, partial eta-squared for ANOVA, Cramer's V for chi-square tests and standardized beta coefficients for regression. Multiple testing across QoL domains and symptom scales was controlled using the Holm method, with global QoL and CIPN20 total score treated as pre-specified primary outcomes and other domain analyses interpreted as secondary/exploratory. Multiple linear regression was used to identify independent predictors of CIPN severity. Chemotherapy regimen was dummy-coded with platinum-only therapy as the reference category; cumulative dose was operationalised as the percentage of the planned regimen-specific neurotoxic dose received to allow comparison across non-equivalent drug classes. Regression diagnostics included variance

inflation factor (VIF), residual normality, residuals-*versus*-fitted assessment, Breusch-Pagan testing for heteroscedasticity, leverage values and Cook's distance. A two-sided p-value < 0.05 was considered statistically significant. ❑

RESULTS

A total of 186 breast cancer patients with a mean age of 52.4 ± 10.8 years (range: 28-74 years) were enrolled in the present study. Most participants were postmenopausal (62.9%), married (78.0%) and had completed secondary education or higher (66.1%). The mean body mass index (BMI) was 27.3 ± 4.6 kg/m². Regarding clinical characteristics, 43.0% of patients had stage II disease. Taxane-only regimens were administered to 132 (71.0%) patients, while total taxane exposure, including the taxane-platinum combination group, was 157/186 (84.4%). The mean number of chemotherapy cycles completed was 5.8 ± 1.4 and the median time since the last chemotherapy cycle was 4.2 months. Comorbid diabetes mellitus was present in 35 (18.8%) patients and hypertension was identified in 58 (31.2%) subjects. Table 1 provides specific clinical and demographic details.

TABLE 1. Demographic and clinical traits of study sample (N = 186)

Variable	n (%) or mean ± SD
Age (mean ± SD) years	52.4 ± 10.8
< 45 years	48 (25.8%)
45–60 years	94 (50.5%)
> 60 years	44 (23.7%)
BMI (kg/m²), mean ± SD	27.3 ± 4.6
Normal (18.5–24.9)	58 (31.2%)
Overweight (25.0–29.9)	74 (39.8%)
Obese (≥ 30.0)	54 (29.0%)
Menopausal status	
Premenopausal	69 (37.1%)
Postmenopausal	117 (62.9%)
Education level	
Primary or below	63 (33.9%)
Secondary or higher	123 (66.1%)
Cancer stage	
I	34 (18.3%)
II	80 (43.0%)
III	72 (38.7%)
Chemotherapy regimen	
Taxane-based (paclitaxel/docetaxel)	132 (71.0%)
Platinum-based	29 (15.6%)
Taxane + Platinum combination	25 (13.4%)
Number of cycles, mean ± SD	5.8 ± 1.4
Months since last cycle, median (IQR)	4.2 (2.1–7.8)
Comorbid diabetes mellitus	35 (18.8%)
Comorbid hypertension	58 (31.2%)

SD=standard deviation; BMI=body mass index; IQR=interquartile range

The overall prevalence of CIPN (NCI-CTCAE Grade ≥1) was 67.7% (n = 126). Grade 1 neuropathy was noted in 33.3% (n = 62) of patients, Grade 2 in 22.6% (n = 42) and Grade 3 in 11.8% (n = 22). No patient had Grade 4 neuropathy. Clinically meaningful CIPN (Grade ≥2) was present in 34.4% (n = 64). Sensory symptoms were the most frequently reported manifestations (61.8%), followed by motor symptoms (38.7%) and autonomic symptoms (24.2%). Among patients with CIPN, the mean EORTC QLQ-CIPN20 subscale scores were 42.7 ± 18.3, 28.4 ± 15.6 and 21.8 ± 14.2 for sensory, motor and autonomic symptoms, respectively. Using the cell counts reported in Table 2, Pearson's chi-square test showed that CIPN prevalence differed by chemotherapy regimen ($\chi^2 = 8.11$, df = 2, p = 0.017; Cramer's V = 0.21), with the highest prevalence (88.0%) being found in the taxane-platinum combination group. The prevalence of CIPN was numerically higher among

TABLE 2. Prevalence and severity of CIPN by chemotherapy regimen (N = 186)†

Variable	Taxane-based (n = 132)	Platinum-based (n = 29)	Taxane+Platinum (n = 25)	p-value
CIPN prevalence, n (%)	89 (67.4%)	15 (51.7%)	22 (88.0%)	0.017*
NCI-CTCAE Grade				
Grade 0 (no CIPN)	43 (32.6%)	14 (48.3%)	3 (12.0%)	0.011*
Grade 1	46 (34.8%)	9 (31.0%)	7 (28.0%)	
Grade 2	28 (21.2%)	4 (13.8%)	10 (40.0%)	
Grade 3	15 (11.4%)	2 (6.9%)	5 (20.0%)	
CIPN20 sensory score, mean ± SD	40.2 ± 17.9	32.6 ± 16.4	52.8 ± 19.1	< 0.001**
CIPN20 motor score, mean ± SD	27.1 ± 14.8	22.3 ± 13.7	38.6 ± 17.4	< 0.001**
CIPN20 autonomic score, mean ± SD	20.4 ± 13.6	18.7 ± 12.8	29.5 ± 16.1	0.004**

†The regimen-level CIPN prevalence statistic was recalculated using Pearson's chi-square test from the displayed cell counts ($\chi^2 = 8.11$, df = 2, p = 0.017).
CIPN=chemotherapy-induced peripheral neuropathy; SD=standard deviation

patients with diabetes (82.9%) than non-diabetic ones (64.2%), but the unadjusted comparison was borderline and did not reach conventional statistical significance ($\chi^2 = 3.70$, df = 1, p = 0.055; Cramer's V = 0.14). Prevalence and severity of CIPN by chemotherapy regimen are summarized in Table 2.

Patients with CIPN demonstrated significantly lower global QoL scores than those without CIPN (51.3 ± 14.7 *vs* 72.6 ± 11.2, p < 0.001; Cohen's d = 1.53). Significant differences were observed across all five functional domains and key symptom scales. After Holm correction for multiple comparisons, global QoL and all functional domains remained statistically significant; symptom-scale findings were interpreted as secondary/exploratory. Pearson correlation showed significant negative correlations between CIPN severity scores (EORTC QLQ-CIPN20 total) and global QoL (r = -0.64, p < 0.001), physical functioning (r = -0.58, p < 0.001), role functioning (r = -0.52, p < 0.001), emotional functioning (r = -0.41, p < 0.001), cognitive functioning (r = -0.33, p < 0.001) and social functioning (r = -0.47, p < 0.001). Positive correlations were reported between CIPN severity scores and symptom scales including fatigue (r = 0.51, p < 0.001), pain (r = 0.59, p < 0.001) and insomnia (r = 0.43, p < 0.001). Spearman sensitivity analyses produced the same direction and statistical interpretation. Table 3 presents the comparison of EORTC QLQ-C30 domain scores between patients with and without CIPN and the correlation coefficients between CIPN severity and QoL domains.

Multiple linear regression revealed the following independent predictors of higher CIPN severity scores (QLQ-CIPN20 total): taxane-platinum combination regimen (β = 0.31, p < 0.001), higher cumulative chemotherapy dose (β = 0.27, p = 0.001), comorbid diabetes mellitus (β = 0.22, p = 0.003), older age (β = 0.18, p = 0.012) and higher BMI (β = 0.14, p = 0.041). The overall regression model was statistically significant (F = 14.82, p < 0.001) and explained 38.4% of variance in CIPN severity scores (adjusted $R^2 = 0.384$). Regression diagnostics supported model acceptability: VIF values ranged from 1.08 to 2.21, indicating no serious multicollinearity; residual plots did not show major heteroscedasticity; the Breusch-Pagan test was not significant (p = 0.18); and no influential observations exceeded Cook's D > 1.0. ❑

DISCUSSION

The reported CIPN prevalence of 67.7% is within the range recorded in prior large-scale studies of taxane-associated neuropathy among breast cancer survivors (5, 6). In a multicentre prospective Chinese cohort of patients with breast cancer, Mo *et al* also showed that patient-reported CIPN varied according to taxane type, supporting the clinical relevance of regimen-specific risk assessment (6). Recent studies examining exercise, inflammatory and behavioural risk factors, and multiethnic survivorship cohorts have further shown that CIPN may persist after treatment and was influenced by clinical, behavioural and psychosocial va-

TABLE 3. EORTC QLQ-C30 domain scores by chemotherapy-induced peripheral neuropathy (CIPN) status and correlations with CIPN severity†

EORTC QLQ-C30 domain	CIPN present (n = 126), mean ± SD	CIPN absent (n = 60), mean ± SD	p-value^a	Correlation with CIPN20 total (r)^b	p-value
Global QoL	51.3 ± 14.7	72.6 ± 11.2	< 0.001	−0.64	< 0.001
Functional scales					
Physical functioning	58.4 ± 16.3	79.8 ± 12.7	< 0.001	−0.58	< 0.001
Role functioning	54.7 ± 19.2	76.3 ± 14.5	< 0.001	−0.52	< 0.001
Emotional functioning	60.2 ± 18.6	73.1 ± 15.4	< 0.001	−0.41	< 0.001
Cognitive functioning	68.9 ± 17.4	78.5 ± 13.8	< 0.001	−0.33	< 0.001
Social functioning	55.8 ± 20.1	74.2 ± 15.9	< 0.001	−0.47	< 0.001
Symptom scales					
Fatigue	52.6 ± 19.8	31.4 ± 14.3	< 0.001	0.51	< 0.001
Pain	48.3 ± 21.4	22.7 ± 13.6	< 0.001	0.59	< 0.001
Insomnia	44.1 ± 22.7	26.8 ± 16.2	< 0.001	0.43	< 0.001
Nausea/vomiting	24.6 ± 18.3	18.2 ± 14.7	0.017	0.19	0.009

†p-values shown are unadjusted; Holm correction was applied for interpretation across multiple QoL domain and symptom-scale comparisons.
CIPN=chemotherapy-induced peripheral neuropathy; QoL=quality of life

riables (16-18). The slightly higher prevalence observed in the present cohort may be explained by the inclusion of patients who received combination taxane-platinum regimens, which may increase neurotoxic risk through complementary mechanisms of peripheral nerve injury.

Our observation that sensory symptoms dominate when compared with motor and autonomic manifestations is consistent with the known clinical pattern of CIPN. Taxanes and platinum compounds can damage dorsal root ganglion neurons and long sensory axons, resulting in length-dependent sensory neuropathy with distal numbness, paraesthesia and neuropathic pain (3, 5, 6). Previous survivorship studies have also affirmed that sensory symptoms, especially numbness and tingling in the limbs, are the most commonly reported neuropathic symptoms, whereas motor impairments are reported less frequently (6, 18). However, the fact that almost one-quarter of our patients manifested autonomic symptoms explains the clinical significance of investigating beyond sensory complaints and asking about orthostatic symptoms, constipation and urinary dysfunction during follow-up visits.

The substantial relationship between CIPN severity and symptom scales, especially pain, fatigue and insomnia, deserves attention. The association between CIPN and pain is mechanistically plausible because neuropathic pain can arise from peripheral nerve injury and central sensitization mechanisms (4). The correlations with fatigue, insomnia and emotional distress suggest broader systemic effects on patient well-being beyond neuropathic symptoms alone (20). These multidimensional effects support multimodal management strategies that include evidence-based neuropathic pain treatment, physical rehabilitation, sleep-hygiene interventions and psychological support.

The apparent relationship between older age and higher CIPN severity in our study is consistent with evidence that patient characteristics can influence CIPN severity in clinical trial populations (21). Age-related physiological changes in peripheral nerve function, including reduced nerve fibre density, impaired axonal regeneration, altered neurotrophic support and pharmacokinetic changes, may prolong exposure to neurotoxic agents. Higher BMI was a statistically significant predictor with a small effect size, possibly because metabolic dysregulation, chronic low-grade inflammation and insulin resistance may increase peripheral nerve vulnerability and impair repair pathways. These findings justify the development of personalised risk assessment tools to identify patients at high risk of severe CIPN before chemotherapy initiation.

Our results have several clinical implications. Firstly, the high prevalence and substantial QoL impact of CIPN support routine standardized screening using the EORTC QLQ-CIPN20, a patient-reported instrument developed and validated for sensory, motor and autonomic neuropathy domains (13, 14). Embedding CIPN assessment into routine oncology workflow may enable earlier identification and timely treatment before symptoms become persistent. Secondly, potentially modifiable risk variables, such as BMI and glycaemic control, suggest opportunities for prehabilitation and metabolic optimization before and during chemotherapy. Thirdly, ASCO guideline recommendations support dose modification when clinically necessary and duloxetine for established painful CIPN, while emphasizing that evidence for most preventive pharmacological agents remains limited (22). The EORTC QLQ-C30 provides a validated framework for longitudinal monitoring of treatment impact across functional and symptomatic domains (15).

A number of weaknesses of the current research may be identified. The cross-sectional design makes it impossible to establish temporal or causal links between CIPN and QoL outcomes; prospective longitudinal studies are required to describe CIPN development, persistence and resolution, and to assess how its effect on QoL changes over time. The use of self-reported symptom scales is patient-friendly and relevant to clinical practice, but it may introduce subjective bias and does not provide objective neurophysiological measurements of nerve function. The single-centre design can also restrict the applicability of the findings to wider and more diverse populations with different treatment procedures and demographic variables. Future studies including objective neurophysiological measures such as nerve conduction studies, quantitative sensory testing, intraepidermal nerve fibre density, fall-risk assessment and long-term follow-up would enhance the evi-

dence base and guide targeted neuroprotective and neurorehabilitative strategies (9, 21, 22). ❑

CONCLUSION

The present study shows that chemotherapy-induced peripheral neuropathy is a common complication among breast cancer patients receiving neurotoxic chemotherapy, affecting 67.7% of the cohort at Grade ≥1 and 34.4% at Grade ≥2. The severity of CIPN was significantly and negatively related to health-related QoL across functional domains, particularly physical functioning, role functioning and global QoL. Sensory symptoms predominated, although motor and autonomic dysfunction were also clinically relevant. Taxane-platinum combination therapy, higher cumulative dose, comorbid diabetes mellitus, older age and higher BMI were independent predictors of CIPN severity. These findings emphasize the need for routine patient-reported CIPN screening, individualized risk stratification and timely evidence-based pharmacological and non-pharmacological interventions to reduce neuropathic burden and preserve QoL in breast cancer care. Further longitudinal research is warranted to define the temporal course of CIPN and evaluate targeted neuroprotective and neurorehabilitative strategies. ❑

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