

Photobiomodulation for chemotherapy-induced peripheral neuropathy: symptomatic effectiveness in a real-world single-center cohort

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Abstract

Purpose.

Chemotherapy-induced peripheral neuropathy (CIPN) is often chronic, with few treatment options. We assessed the magnitude and durability of symptomatic change following photobiomodulation (PBM) and explored baseline characteristics associated with response.

Methods.

We retrospectively analyzed 41 PBM courses in 37 patients with established CIPN (CTCAE I–III), April 2023–July 2025. A polychromatic light-emitting-diode device (620, 680–760 and 820 nm) treated symptomatic extremities two or three times weekly. Pain (0–10 visual analogue scale, VAS) and DN4 were recorded before and after treatment. Analyses used Wilcoxon signed-rank tests and Wilson 95% confidence intervals (CIs).

Results.

Median VAS fell from 7 to 3 and DN4 from 5 to 2 (median paired reduction 3 points each; $p < 0.001$; $r = 0.82$ and 0.85). Clinical pain response (VAS reduction > 1 point) occurred in 80.5% of courses (95% CI 66.0–89.8), neuropathic-symptom response (DN4 reduction $\geq 50\%$) in 63.4% (48.1–76.4), and durable response at three months in 83.3% (30/36; 68.1–92.1). Single-course-per-patient results were similar; worst-case imputation lowered pain response to 75.0%. No baseline factor survived false-discovery-rate correction; no toxicity was attributed to PBM.

Conclusion.

In this real-world cohort with chronic, largely refractory CIPN, PBM was followed by large, durable symptomatic relief, with no identifiable poor-response subgroup and no attributable toxicity, warranting randomized confirmation.

Introduction

Chemotherapy-induced peripheral neuropathy (CIPN) is a frequent, often dose-limiting toxicity of platinum compounds, taxanes and vinca alkaloids, presenting in a glove-and-stocking distribution with numbness, tingling, burning pain and loss of fine sensation [1]. In a meta-analysis of 4,179 patients, prevalence was 68% within the first month after chemotherapy, 60% at three months and 30% at six months or beyond [2]; a substantial minority retain symptoms that do not resolve. Beyond its effect on quality of life, CIPN may force dose reduction or early discontinuation, with potential consequences for disease control [3].

Therapeutic options remain limited. Duloxetine is recommended in the 2020 American Society of Clinical Oncology guideline [4]; in the supporting trial, 59% of patients receiving duloxetine reported any decrease in pain versus 38% receiving placebo [5]. Gabapentinoids, tricyclic antidepressants and opioids lack consistent supporting evidence [6], while evidence for exercise, acupuncture and cryotherapy remains heterogeneous [7].

Photobiomodulation (PBM), formerly low-level laser therapy, is established in supportive oncology for oral mucositis in defined settings [8]. Red to near-infrared light can modulate mitochondrial ATP production and redox signaling [9, 10]. WALT and MASCC/ISOO guidance support selected oncology applications [11, 12]. Randomized trials have evaluated PBM for CIPN [13, 14], and a systematic review has examined this evidence [15]. Its effectiveness and determinants of response remain uncertain.

This matters clinically: if response could be anticipated from baseline characteristics, patient selection and protocol intensity could be individualized rather than uniform. We therefore treated a consecutive series of patients with established CIPN on a standardized PBM protocol, recording pain and neuropathic symptoms before and after treatment, to quantify the magnitude and durability of symptomatic change and screen baseline patient, disease and treatment characteristics for association with response. We hypothesized that a coexisting non-chemotherapy cause of neuropathy would attenuate the effect of PBM.

Methods

Study design and setting

This retrospective, single-center before–after cohort study, conducted in a hospital department of radiation oncology, is reported according to STROBE. Each patient served as their own pre-treatment reference; without a concurrent control group, the design supports inference about association rather than causation.

Participants

Eligible participants were adults who developed CIPN during or after neurotoxic chemotherapy and were referred for PBM between 24 April 2023 and 23 July 2025 (13 courses in 2023, 19 in 2024, 9 in 2025).

Inclusion required CIPN graded I–III and completion of chemotherapy. Patients were excluded for fewer than five PBM sessions, continuation of care at another center, or neuropathy judged primarily attributable to a non-chemotherapy cause. Diabetes or a remote history of neuropathy was not in itself exclusionary when the clinician considered CIPN the predominant current syndrome.

Forty-one patients were assessed for eligibility. One was not included (pre-existing diabetic and autoimmune neuropathy) and three were excluded after starting PBM (two with fewer than five sessions,

one who continued care elsewhere), leaving 37 patients who underwent 41 treatment courses, four having been treated twice (Fig. 1).

Data collection and baseline assessment

Baseline data were extracted from the medical record before the first PBM session: demographics, smoking and alcohol use, comorbidities, any pre-existing neuropathy, and cancer primary site, histology and stage. Prior neurotoxic exposure was coded for taxanes, oxaliplatin and cisplatin, and the interval from end of chemotherapy to start of PBM calculated. Severity was graded with NCI-CTCAE version 5.0.

Neuropathic symptoms were assessed with the DN4 questionnaire (score ≥ 4 indicates neuropathic pain) and pain intensity with a 0–10 visual analogue scale (VAS) [16]. The symptom inventory captured numbness, tingling, burning, electric-shock sensations, allodynia, cold-induced pain, and tactile or pinprick hypoesthesia. Concurrent analgesics (pregabalin, gabapentin) and other supportive measures (acupuncture, intravenous trace-element infusion) were recorded as present or absent at baseline.

Photobiomodulation protocol

PBM was delivered to symptomatic extremities with a polychromatic light-emitting-diode (LED) device rather than a laser (ATP38; Swiss Bio Inov S.A., Moudon, Switzerland). The standard neuropathy protocol combined three wavelengths per session – red (620 nm, 4 J/cm²), deep-red (680–760 nm, 8 J/cm²) and near-infrared (820 nm, 8 J/cm²) – over approximately 486 s (8 min) per field, the LED panels in a "sandwich" configuration around the affected hands and/or feet. Sessions were given once daily, two or three times weekly, for a median of 18 sessions (interquartile range, IQR, 15–25). Irradiance and spot geometry are set internally by the device and were not recorded separately, so dosimetric reproducibility is defined by the manufacturer's ATP38 protocol rather than independently measured optical parameters.

Two adaptations were available: a refractory-neuropathy protocol adding green (470 or 525 nm, 2–4 J/cm²) and amber (590 nm, 1 J/cm²) light to a modified red and near-infrared combination over approximately 1,220–1,320 s per field, usually with the source at 30 mm from the skin; and a more intensive diabetic-polyneuropathy protocol. Both were reserved for patients whose symptoms had not improved sufficiently under the standard protocol or whose previous course had failed, the decision resting with the supervising physician and not on any baseline characteristic. A course counted as modified if such a switch was documented at any point.

Outcomes

Three outcomes were specified: the primary clinical pain response, a reduction of more than one point on the VAS (≥ 2 points on the integer scale) between baseline and the end of treatment; the secondary

neuropathic-symptom response, a reduction of at least 50% in the DN4 score; and a durable response, persistence of clinical or neuropathic-symptom improvement at three months.

The DN4 was developed and validated to diagnose neuropathic pain at a threshold of 4, not to grade severity or measure change over time [16]; used here as a pragmatic index of overall neuropathic-symptom burden, it yields a symptom signal rather than a validated response criterion.

Statistical analysis

Continuous variables are summarized as medians with IQRs, categorical variables as counts and percentages. Paired change in VAS and DN4 was tested with the Wilcoxon signed-rank test, the effect size r derived from the test statistic and the median paired difference carrying a bootstrap 95% confidence interval (CI; 10,000 resamples). Responder proportions carry Wilson 95% CIs.

Each baseline factor and symptom was screened against both outcomes with Fisher's exact test, p -values within each outcome family adjusted by the Benjamini–Hochberg false-discovery-rate (FDR) method. Continuous predictors of durable response were explored by receiver-operating-characteristic analysis (Online Resource 3). Dose was examined as sessions per week (Fisher's exact test) and total sessions against magnitude of change (Spearman correlation). A penalized (ridge) multivariable logistic model served as a stability check only, the small number of non-responders precluding a conventional model.

Two subgroups were compared: standard protocol throughout versus switched to a modified protocol at any point, and PBM started before versus at or after nine months from the end of chemotherapy – a threshold chosen because CIPN prevalence plateaus around six months, symptoms persisting beyond that being conventionally regarded as chronic [2]. An attrition sensitivity analysis applied worst-case imputation, adding the three courses discontinued after starting PBM to the denominator as non-responders: a full intention-to-treat (ITT) population of 44 and a modified ITT population of 43 excluding the transfer of care as non-informative censoring. Two further sensitivity analyses were restricted to one course per patient and to courses with no neuropathic analgesic introduced or up-titrated during PBM.

Four patients each contributed two courses, treated as independent observations in the primary analysis. The durable-response outcome was analyzed on available cases (36 of 41). Analyses used Python 3 (SciPy, statsmodels, scikit-learn), with two-sided $p < 0.05$ significant and FDR-adjusted values the primary screen.

Safety assessment

Tolerance was assessed clinically at every session by the treating physician or operator, who recorded symptom course and any new event in the record; events were attributed to PBM, the underlying neuropathy or an unrelated cause.

Results

Cohort characteristics

Forty-one treatment courses in 37 patients were analyzed (Table 1). Median age was 64 years (IQR 58–72) and 22 patients (59%) were women; the most frequent primary sites were colorectal (10, 27%) and breast (9, 24%) cancer. Oxaliplatin preceded 20 courses (49%), a taxane 13 (32%) and cisplatin 7 (17%).

Table 1
Baseline characteristics of the study population

Characteristic	n (%) unless otherwise stated
Patient-level characteristics (n = 37 patients)	
Age, years — median (IQR)	64 (58–72)
Sex	
Female	22 (59)
Male	15 (41)
Smoking status	
Never smoker	26 (70)
Former smoker	6 (16)
Current smoker	5 (14)
Diabetes mellitus	6 (16)
Pre-existing peripheral neuropathy	1 (3)
Primary tumor site	
Colon or rectum	10 (27)
Breast	9 (24)
Esophagus	4 (11)
Ampulla of Vater	2 (5)
Lung	2 (5)
Pancreas	2 (5)
Prostate	2 (5)
Other ^b	5 (14)
Not documented	1 (3)
Course-level characteristics (n = 41 treatment courses)	
Disease stage at the start of PBM	
Non-metastatic (M0)	27 (66)
Metastatic (M1)	12 (29)
Not documented	2 (5)

Characteristic	n (%) unless otherwise stated
Neurotoxic chemotherapy received ^a	
Oxaliplatin	20 (49)
Taxane	13 (32)
Cisplatin	7 (17)
Other or not specified	2 (5)
Interval from end of chemotherapy to PBM, months	
Median (IQR), n = 27 documented courses	6.8 (2.3–19.6)
< 9 months	14/27 (52)
≥ 9 months	13/27 (48)
Not documented	14 (34)
CIPN severity (NCI-CTCAE v5.0)	
Grade I	2 (5)
Grade II	17 (41)
Grade III	22 (54)
Symptom distribution	
Distal extremities only	31 (76)
Glove-and-stockings	5 (12)
Stocking (feet) only	3 (7)
Glove (hands) only	2 (5)
Baseline VAS pain score (0–10) – median (IQR)	7 (5–8)
Baseline DN4 score (0–10) – median (IQR)	5 (5–6)
Site treated	
Feet	22 (54)
Hands	18 (44)
Hands and feet	1 (2)
PBM sessions – median (IQR)	18 (15–25)
Sessions per week	
Two	25 (61)

Characteristic	n (%) unless otherwise stated
Three	16 (39)
Concurrent neuropathic analgesic at baseline	
Pregabalin alone	6 (15)
Gabapentin alone	3 (7)
Pregabalin and gabapentin	3 (7)
None	29 (71)

CIPN, chemotherapy-induced peripheral neuropathy; CTCAE, Common Terminology Criteria for Adverse Events; DN4, Douleur Neuropathique en 4 questions; IQR, interquartile range; PBM, photobiomodulation; VAS, visual analogue scale.

Four patients each contributed two treatment courses; patient-level and course-level denominators therefore differ. Percentages are calculated within the stated denominator and may not total 100 owing to rounding.

^a Categories are not mutually exclusive: one course followed exposure to two of the three agents.

^b Other primary sites were stomach, testis, nasopharynx, bladder and skin (one patient each).

Neuropathy was moderate to severe: 22 courses (54%) were graded CTCAE III and 17 (41%) CTCAE II; median baseline VAS was 7 (IQR 5–8) and median DN4 5 (IQR 5–6). Where the chemotherapy end date was documented (27 courses), the median interval to PBM was 6.8 months (IQR 2.3–19.6; range 0.7–66.8), 13 beginning at or beyond nine months. Competing causes were infrequent: one patient (3%) had pre-existing peripheral neuropathy and six (16%) diabetes. The most frequent symptoms were numbness (37 courses, 90%) and tingling (34, 83%).

Change in pain and neuropathic symptoms

Both scores improved after PBM: median VAS fell from 7 to 3 and median DN4 from 5 to 2, a median paired reduction of 3 points each (Wilcoxon signed-rank $p < 0.001$). Effect sizes were large ($r = 0.82$ for VAS, 0.85 for DN4) and the bootstrap 95% CI for the median paired change was -3 to -2 points for both. For comparison, the minimal clinically important difference reported for acute pain is one to two points [17, 18].

Individual trajectories are shown in Fig. 2 and the distribution of paired change in Fig. 3. Pain improved in 36/41 courses and DN4 in 38/41, the remainder showing no change or a slight increase; the two changes were moderately correlated (Spearman $\rho = 0.47$, $p = 0.002$).

Rate of symptomatic response

A clinical pain response occurred in 33/41 courses (80.5%; 95% CI 66.0–89.8) and a neuropathic-symptom response in 26/41 (63.4%; 48.1–76.4). Three-month data were available for 36 courses, of which 30 (83.3%; 68.1–92.1) maintained improvement; of the other six, two relapsed after an initial response and four had never achieved one (Fig. 4).

Sensitivity analyses

Under worst-case imputation the clinical pain-response rate was 76.7% (95% CI 62.3–86.8) in the modified ITT population of 43 and 75.0% (60.6–85.4) in the full ITT population of 44, versus 80.5% per protocol; the neuropathic-symptom rates were 60.5% and 59.1%, versus 63.4% per protocol (Online Resource 1).

Restricted to one course per patient ($n = 37$), the clinical pain response was 83.8% (95% CI 68.9–92.3), the neuropathic-symptom response 64.9% (48.8–78.2) and the durable response 84.4% (68.2–93.1); median changes and effect sizes were unchanged ($r = 0.83$ and 0.86).

Baseline factors and response

Eighteen baseline factors and symptoms were screened against each outcome; two reached nominal significance for the clinical one. Neither course delivered to the single patient with pre-existing neuropathy achieved a pain response, versus 84.6% (33/39) of the rest (Fisher $p = 0.034$), and all 12 courses in metastatic (M1) disease responded, versus 70.4% (19/27) in non-metastatic disease (stage undocumented for 2 courses; $p = 0.042$). Neither survived FDR correction (adjusted $p = 0.40$ for both), and the first came from a single patient. No other factor – including age, sex, diabetes, chemotherapy agent, baseline severity and CTCAE grade – was associated with either outcome (Online Resource 2).

Chemotherapy agent and tumor type

Clinical response rates were similar across neurotoxic agents: 80.0% after oxaliplatin (95% CI 58.4–91.9), 84.6% after a taxane (57.8–95.7) and 85.7% after cisplatin (48.7–97.4), with widely overlapping intervals; the neuropathic-symptom response showed the same pattern. Pain reduction did not differ across tumor types (Kruskal–Wallis $p = 0.82$, comparing breast, colorectal, esophageal and other sites).

Protocol delivered and interval from chemotherapy

Response by protocol delivered and by interval from chemotherapy is shown in Fig. 5a, b. A switch to an intensified regimen occurred in 26/41 courses (63.4%) at a median of session 11 (IQR 9–17) – to the refractory protocol in 20 and the diabetic-polyneuropathy protocol in 6, with reversion to standard in 7 – following insufficient improvement under the standard protocol in 23 and resumption after an unsuccessful previous course in 3. Courses on the standard protocol throughout ($n = 15$) and those switched ($n = 26$) did not differ significantly in pain response (93.3%, 14/15, versus 73.1%, 19/26; Fisher $p = 0.22$) or durable response (92.9%, 13/14, versus 77.3%, 17/22; $p = 0.37$), but the neuropathic-symptom response was higher on the standard protocol (86.7%, 13/15, versus 50.0%, 13/26; $p = 0.023$).

Baseline VAS and DN4 were identical (medians 7 and 5; Mann–Whitney $p = 1.0$ for VAS), though switched courses comprised more sessions (median 24.5 versus 15; $p = 0.002$).

Response rates were numerically higher when PBM started within nine months of chemotherapy, for pain (78.6%, 11/14, versus 69.2%, 9/13; $p = 0.68$), neuropathic symptoms (71.4%, 10/14, versus 46.2%, 6/13; $p = 0.25$) and durability (84.6%, 11/13, versus 66.7%, 8/12; $p = 0.38$), but none was significant and VAS reduction did not differ (median – 2 versus – 3 points; $p = 0.92$).

Treatment intensity

Courses delivered three times weekly had a numerically higher durable-response rate than those given twice weekly (93.8% versus 75.0%; odds ratio 5.0), though not significantly (Fisher $p = 0.20$). More total sessions correlated with smaller VAS improvement (Spearman $\rho = +0.36$, $p = 0.020$); the association with DN4 improvement was not statistically significant ($\rho = +0.28$, $p = 0.072$). No continuous baseline variable discriminated durable responders with clinically useful accuracy (Online Resource 3); in the penalized model pre-existing neuropathy was the only variable retained with a negative coefficient.

Tolerance and treatment interruptions

No adverse event attributable to PBM was recorded: no treatment was discontinued for toxicity and no cutaneous, thermal or systemic complication occurred.

Treatment was interrupted or ended prematurely in 11 courses: four for perceived lack of efficacy, four for logistical reasons such as planned leave, two for an intercurrent oncological event (hepatic surgery; progression requiring resumption of chemotherapy) and one for a six-month equipment failure, during which the initial benefit regressed and resumed only after treatment restarted.

A concurrent neuropathic analgesic was recorded at baseline in 12 courses (29%): pregabalin alone in 6, gabapentin alone in 3, both in 3. One was introduced or up-titrated during PBM in four courses (10%); excluding these, the clinical pain-response rate was 30/37 (81.1%; 95% CI 65.8–90.5) versus 80.5% overall.

Discussion

PBM developed from early observations of low-power laser effects [19]. The term encompasses laser and light-emitting-diode applications [20], including the polychromatic LED device used here.

Evidence in supportive oncology is strongest for oral mucositis in defined treatment settings [8, 12]. WALT guidance addresses additional toxicities [11], and a systematic review has examined tumor safety [21]. This experience provides a rationale for investigating CIPN.

CIPN involves axonal injury, mitochondrial dysfunction and neuroinflammation [22]. Proposed PBM mechanisms include mitochondrial and redox signaling [9, 10], with a biphasic dose–response [23]. In a rat sciatic-nerve crush model, irradiation improved nerve regeneration and functional recovery [24].

PBM was followed by a substantial reduction in CIPN pain and neuropathic symptoms, with a clinical pain response in four of every five courses and most improvements maintained at three months.

The clinical literature uses different populations and endpoints, limiting direct comparison. Argenta et al. randomized 70 patients with CIPN and reported improved modified total neuropathy scores with PBM compared with sham treatment [13]. Teng et al. enrolled 44 survivors with symptoms persisting at least three months after chemotherapy in a randomized, non-comparative phase II trial: response rates were 48% with PBM versus 53% with sham at six weeks, and 45% versus 33% at 12 weeks [14]. That design did not establish superiority over standard care. A systematic review has synthesized PBM studies in CIPN [15], while NEUROLASER examined prevention during chemotherapy [25]. The 59% duloxetine response reported by Smith et al. denotes any pain decrease [5], whereas our pain-response threshold was a reduction of more than one point; these percentages do not establish comparative efficacy.

Durability is rarely reported in this setting. Among courses with three-month data, 83% retained benefit and only two relapsed — persistence beyond the end of treatment being more consistent with nerve repair than with transient analgesia, as a reparative mechanism would predict. The same pattern appeared in the course interrupted by six-month equipment failure, where benefit regressed and returned only with treatment.

This routine-practice cohort included varied primary sites, neurotoxic agents, ages and neuropathy grades. The median interval from chemotherapy was nearly seven months, with half of documented courses beginning at nine months or later. Previous trials also included established CIPN, including symptoms persisting at least three months after chemotherapy in Teng et al. [14]. The present cohort adds observational data from a heterogeneous clinical population.

Prediction of CIPN outcomes has been much investigated: the MASCC neurological complications working group concluded that no clinical, genetic or biochemical marker is yet suitable for routine use [26]. Among clinical factors, diabetes carries the most consistent evidence, a meta-analysis having shown increased risk of developing CIPN [27], while baseline neuropathy, cumulative dose and age have been proposed but not consistently validated [2]. Predictors of response to treatment, as distinct from predictors of developing CIPN, have received little attention.

The present screen of 18 baseline factors was negative, none surviving FDR correction. The single patient with pre-existing neuropathy responded to neither course, consistent with our prior hypothesis that a coexisting metabolic or autoimmune cause renders nerve damage less reversible; but this rests on one patient, did not survive correction, and is hard to reconcile with a randomized trial in which low-level laser therapy reduced pain and sensorimotor disturbance in diabetic polyneuropathy [28]. Pre-existing neuropathy is therefore a candidate selection factor for evaluation in a larger sample, not an established one. The 100% response rate in metastatic disease is similarly imprecise, resting on 12 courses, and most plausibly reflects higher baseline pain and hence greater scope for improvement.

Age, sex, chemotherapy agent, tumor type and baseline severity were uninformative and response rates by agent closely comparable, giving no basis for withholding PBM from any particular subgroup.

Three sessions per week were associated with numerically better durability, a longer total course with less improvement; both patterns are most plausibly confounding by indication, the most refractory patients receiving the longest treatment. The protocol comparison is subject to the same confounding: courses requiring intensification had by definition already failed the standard protocol, and their lower DN4 response rate reflects that selection rather than inferiority of the intensified protocol, identical baseline scores ruling out severity as the explanation. Dose optimization cannot be resolved observationally and requires a trial randomizing schedule and protocol directly.

Limitations

The principal limitation is the absence of a control arm. CIPN resolves spontaneously in an appreciable proportion of patients, so part of the improvement may reflect natural history [2]. Three observations make this less likely without excluding it: most patients had stable symptoms for several months before treatment; response was retained in the subgroup treated at nine months or beyond; and improvement coincided closely with the start of PBM. Worst-case imputation for the discontinued courses cost fewer than six percentage points.

The study was also retrospective, single-center and unblinded, so expectation effects may have inflated the patient-reported VAS, and the DN4 was used outside its validated diagnostic purpose. Adverse events were recorded in free text rather than on a structured form, and concomitant analgesic doses were not systematically captured, though excluding the four courses with an analgesic introduced or up-titrated left the pain-response rate unchanged. Four patients contributed two courses each, so course-level observations are not fully independent, but restricting to one course per patient did not alter the rates. The interval from chemotherapy was available for 27/41 courses and the durable-response estimate rests on 36/41; no quality-of-life, nerve-conduction or balance and gait measures were collected [29]. Certainty of evidence is therefore low by GRADE criteria, and a randomized, sham-controlled trial with blinded assessment and objective endpoints remains necessary.

Conclusion

In a heterogeneous, real-world cohort with chronic and largely refractory CIPN, PBM was associated with large, durable reductions in pain and neuropathic symptoms — a clinical pain response in approximately four of every five courses, no identifiable poor-response subgroup and no attributable toxicity. The benefit persisted under worst-case imputation and in patients whose neuropathy had been established for nine months or longer. These findings support PBM as a promising non-pharmacological strategy in persistent CIPN, warranting prospective randomized confirmation.

Declarations

Competing interests.

The authors have no relevant financial or non-financial interests to disclose.

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Author Contribution

Sami Frikha: data analysis and interpretation, manuscript drafting and writing. Aziz Cherif, Kamel Debbi, Nhu Hanh To, and Noémie Grellier: critical revision of the manuscript and review of important intellectual content. Tarik Rabehi, Tristan Rossy, Sovandeth Ngeth, and Sabine Tebessi: data collection and acquisition. Mohammad Rahmoun, Lahcène Belaidi, Alexander Benassi, Charles Lasar, Gabriele Coraggio, and Yazid Belkacemi: critical review and revision of the manuscript for important intellectual content. All authors reviewed and approved the final version of the manuscript for publication.

Data Availability

The datasets generated and/or analyzed during the current study are not publicly available due to patient privacy and institutional restrictions but are available from the corresponding author on reasonable request.

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Figures

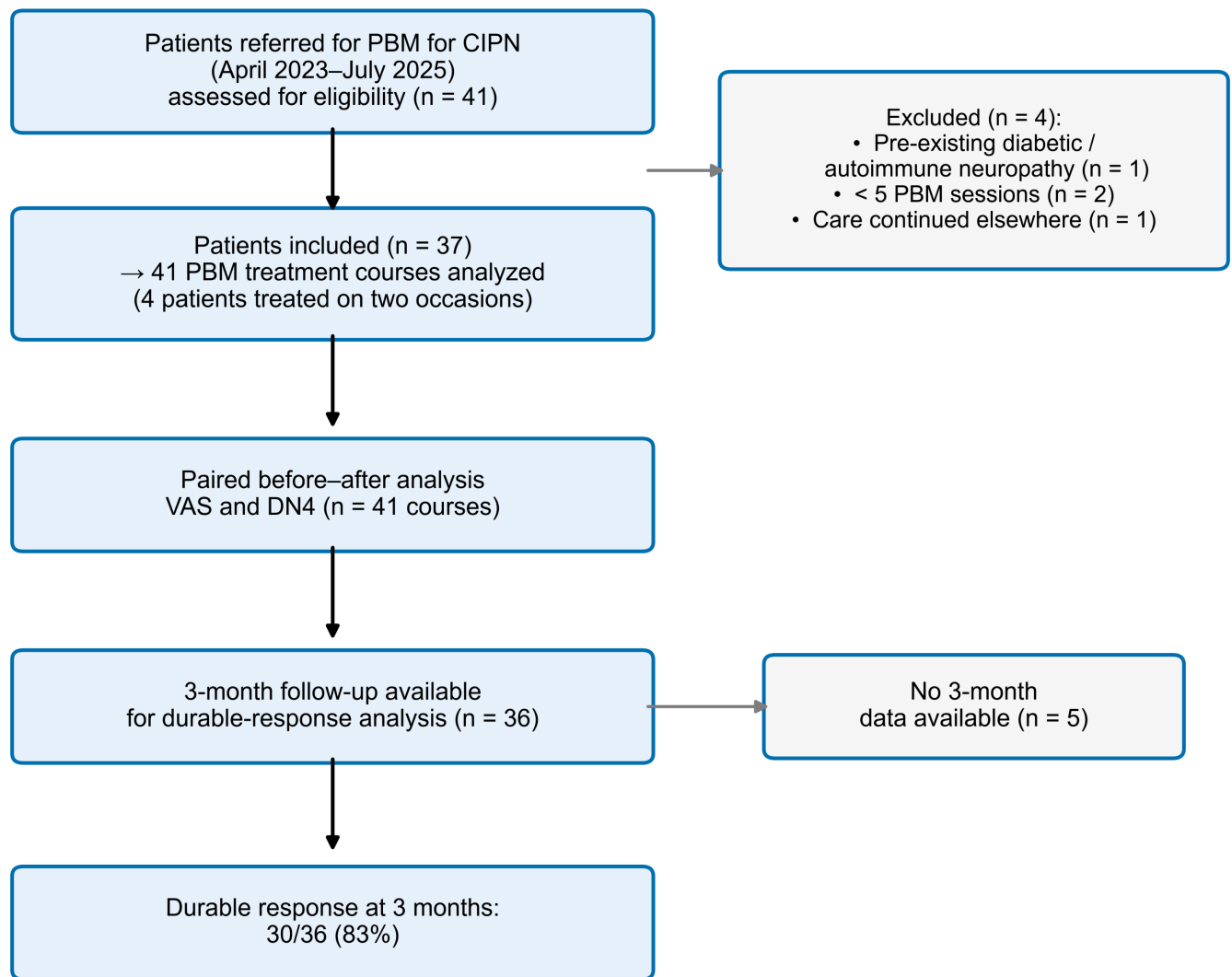


Figure 1

STROBE participant flow, from eligibility assessment through exclusions to the paired analysis and three-month follow-up. Four of the 37 included patients each underwent two treatment courses

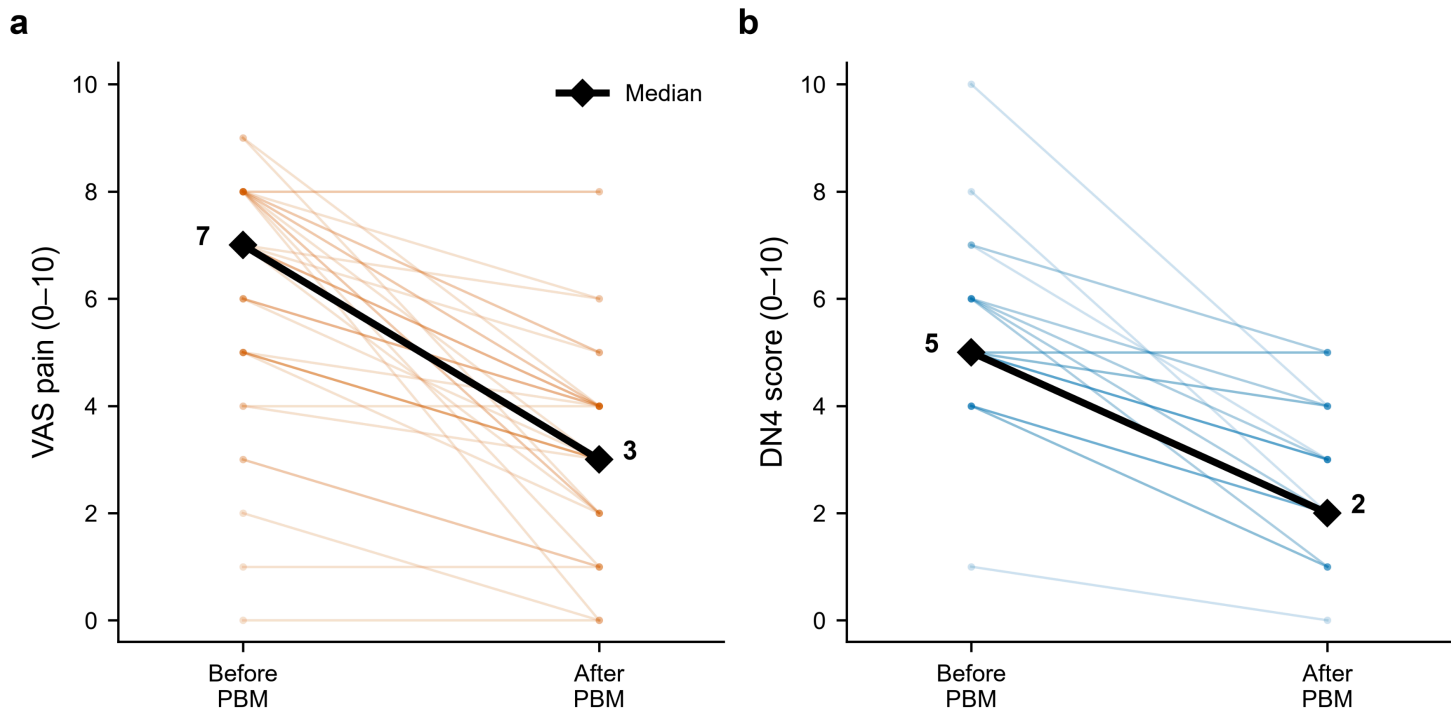


Figure 2

Individual before–after trajectories for a VAS pain and b DN4 score, with the cohort median in bold (n = 41 treatment courses). VAS visual analogue scale, DN4 Douleur Neuropathique en 4 questions, PBM photobiomodulation

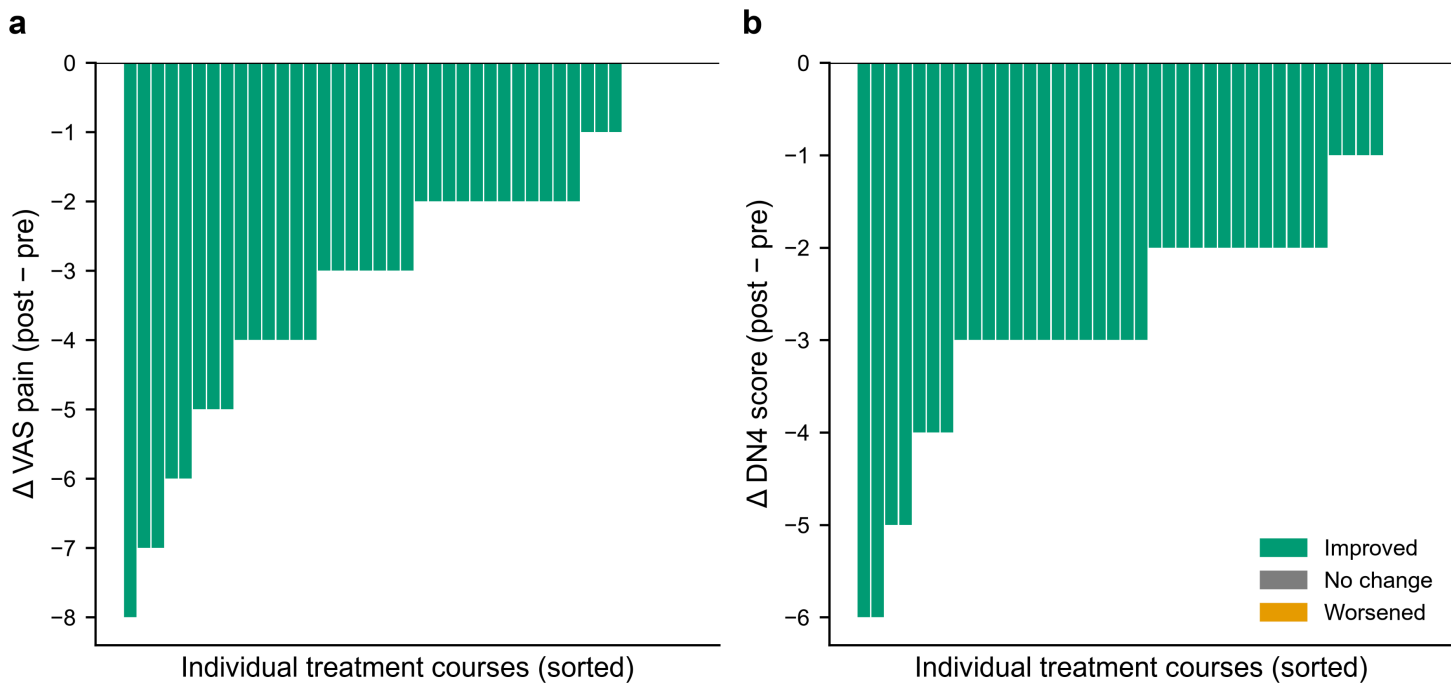


Figure 3

Waterfall plots of paired change for a VAS and b DN4 (post minus pre) across the 41 treatment courses, sorted by magnitude. Green bars denote improvement

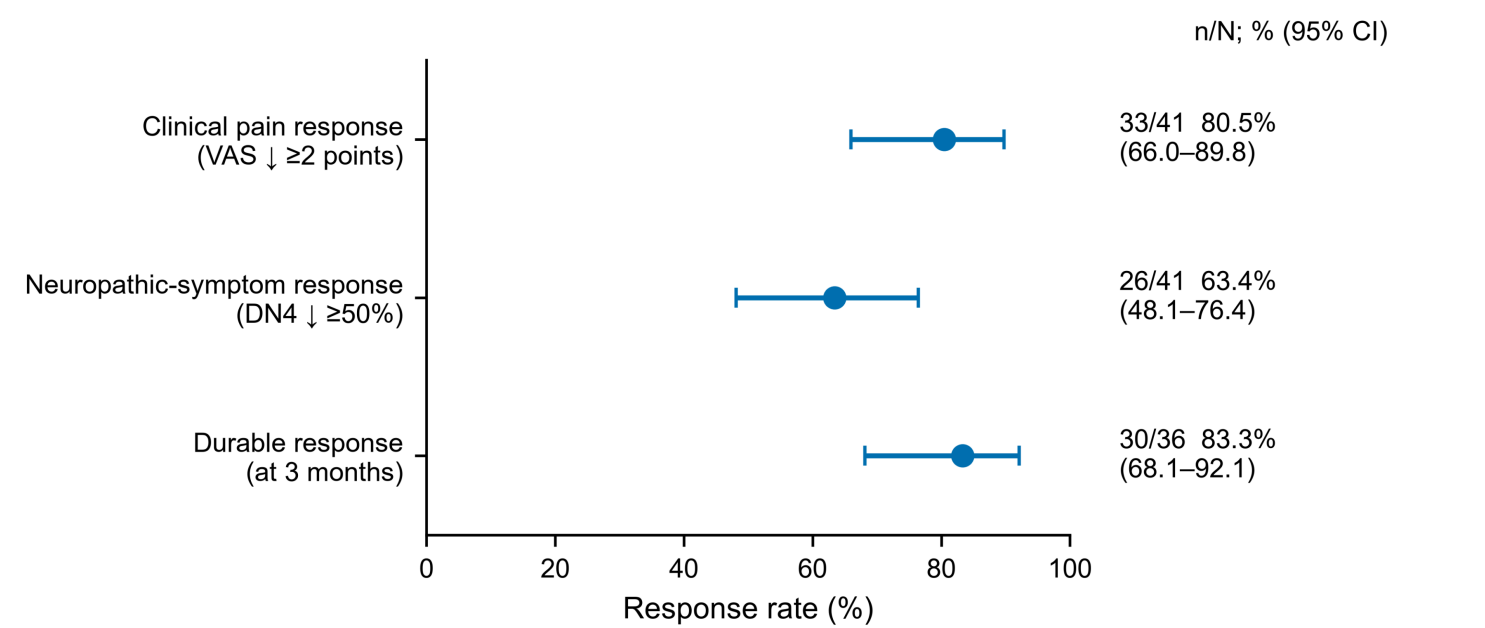


Figure 4

Responder rates with 95% Wilson confidence intervals: clinical (VAS reduction > 1 point), neuropathic-symptom (DN4 reduction ≥ 50%) and durable (three-month) response

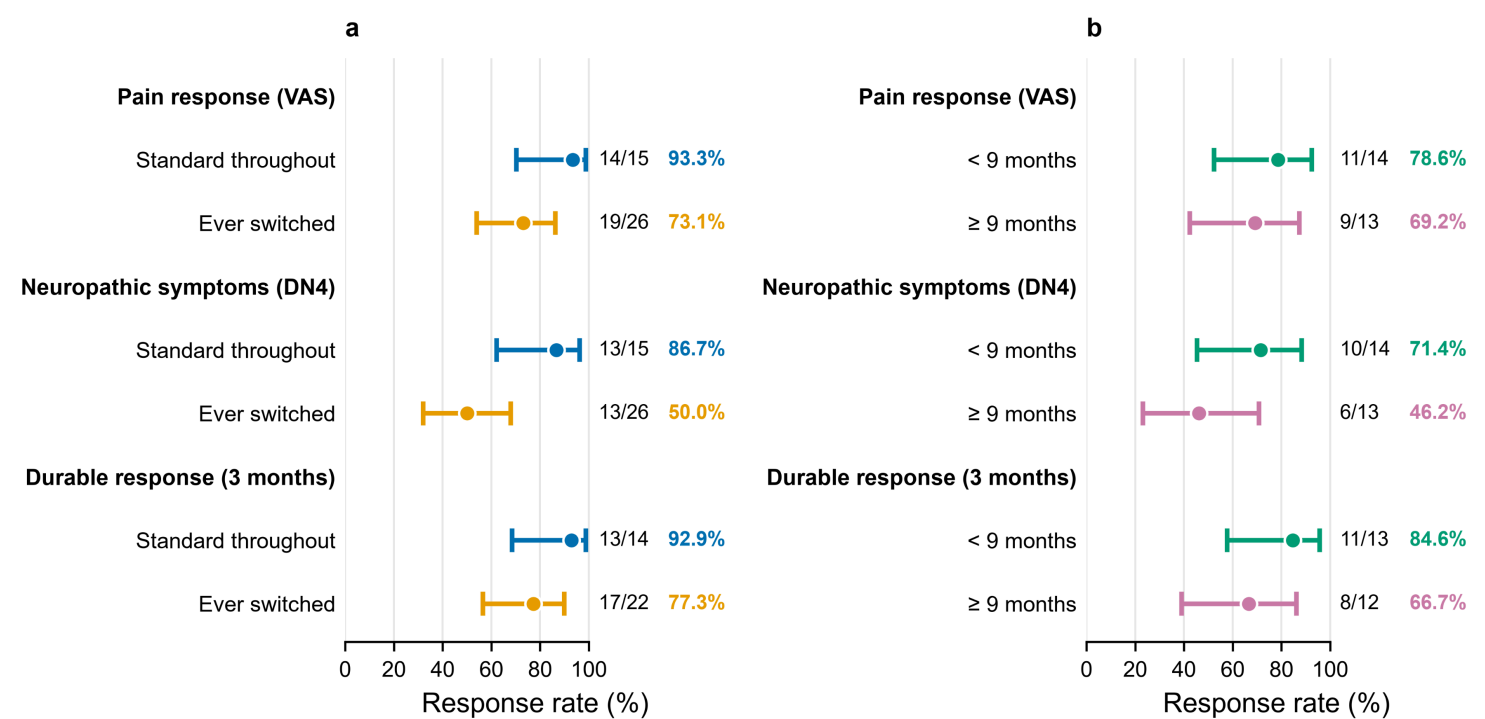


Figure 5

Subgroup analyses with Wilson 95% confidence intervals. a Response by protocol switch during the course (standard throughout versus ever switched to a modified protocol). b Response by interval from the end of chemotherapy to PBM (< 9 versus $\geq$ 9 months)

Supplementary Files

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