Electrophysiological Markers of Chemotherapy-Induced Polyneuropathy

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Abstract

Introduction. *Electrophysiological testing is the gold standard for diagnosing polyneuropathy. However, its use in oncology practice for patients with chemotherapy-induced polyneuropathy (CIPN) remains limited and the value of its findings is not fully understood.*

The study was aimed *at identifying electrophysiological CIPN markers and evaluting their sensitivity and specificity.*

Materials and methods. *The study included patients (n = 71) over 18 years of age with solid tumor presenting with polyneuritic complaints following neurotoxic therapy with platinum-based agents and taxanes. Patients with known risk factors for polyneuropathy were excluded. Electrophysiological and clinical patient data were evaluated no earlier than 3 months following chemotherapy initiation.*

Results. *The study identified electromyographic markers: SRAR index (sural/radial ratio – the ratio between the action potential amplitudes of the sural and radial nerves) and the sural nerve action potential (SNAP), demonstrating equal sensitivity (73.7%) and high specificity (75% and 84.6%, respectively).*

Conclusion. *Electromyographic parameters such as SRAR and SNAP sural nerve can be utilized for the diagnosis and monitoring of CIPN in daily practice.*

Keywords: *electroneuromyography; chemotherapy-induced polyneuropathy; markers; SRAR; cancer*

Ethics approval. All patients provided their voluntary informed consent to participate in the study. The study protocol was approved by the Ethics Committee of the Clinical Research Center at Immanuel Kant Baltic Federal University (Conclusion No. 35 dated October 27, 2022).

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Электрофизиологические маркеры химиоиндуцированной полинейропатии

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Аннотация

Введение. *Электрофизиологическое исследование является стандартом при диагностике полинейропатии. Пока его использование ограничено в онкологической практике у пациентов с химиоиндуцированной полинейропатией (ХИПН), а ценность полученных результатов не до конца понятна.*

Цель *исследования – выявить электрофизиологические маркеры ХИПН и оценить их чувствительность и специфичность.*

Материалы и методы. *В исследование были включены пациенты (n = 71) старше 18 лет с солидными злокачественными новообразованиями, предъявляющие полиневритические жалобы после нейротоксической терапии с использованием препаратов платины и таксанов. Исключались пациенты с известными факторами риска развития полинейропатии. Изучали электрофизиологические, клинические данные пациентов не ранее чем через 3 мес после старта химиотерапии.*

Результаты. *В ходе исследования выявлены электромиографические маркеры: индекс SRAR (sural/radial ratio – соотношение между амплитудой потенциала действия икроножного и лучевого нервов) и потенциал действия икроножного нерва с равной чувствительностью (73,7%) и высокой специфичностью (75 и 84,6% соответственно).*

Заключение. *Для диагностики и мониторинга ХИПН в ежедневной практике могут использоваться электромиографические показатели, такие как SRAR и потенциал действия икроножного нерва.*

Ключевые слова: *электронейромиография; химиоиндуцированная полинейропатия; маркеры; SRAR; рак*

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Introduction

Chemotherapy-induced peripheral neuropathy (CIPN) is a common neurological complication in patients with malignant neoplasms [1]. CIPN symptoms may be heterogeneous, ranging from mild to severe manifestations that compromise patients' quality of life and require chemotherapy dose reductions or even complete treatment discontinuation, which, in turn, may negatively affect overall survival in cancer patients. CIPN symptoms may build up gradually and not always be overt at early stages, as well as progress after cessation of chemotherapy. CIPN diagnosis is established based on patient complaints and clinical assessment, despite electromyography (EMG), gold standard in diagnosing polyneuropathy (PNP) [2, 3]. Electrophysiological findings are objective markers for monitoring and understanding peripheral nerve disorders. However, most PNP patients with malignancies do not undergo this testing in routine clinical setting. This might be due to the limited availability, lack of CIPN diagnosis algorithms, practical and financial challenges during such testing overburdened oncology settings, and controversial data on the need of neurophysiology studies in this population [4–6]. Therefore, electrophysiological markers should be identified to assess the peripheral nerve status in patients receiving chemotherapy agents and to accumulate sufficient array of reliable data with further implementation in clinical practice.

The study was aimed at identifying electrophysiological CIPN markers and determining their sensitivity and specificity.

Materials and methods

The study included patients (n = 71) with solid tumors of gastrointestinal tract (GIT) (n = 34; 48%), respiratory system (n = 9; 12.6%), and pelvis (n = 28; 39.4%).

Inclusion criteria:
- age > 18 years;
- histologically confirmed solid tumors of GIT, respiratory system, and pelvis;
- polyneuritic complaints;
- first-time chemotherapy.

Exclusion criteria:
- a history of other PNPs and conditions (diabetes, paraproteinemic hemoblastoses, systemic connective tissue disorders, vasculites, hepatitis C, HIV;
- alcohol intake and use of medications (amiodarone, metronidazole, etc.) potentially inducing PNP.

All patients underwent standard neurological examination with assessment of superficial and deep sensation, reflexes, and muscle strength using the MRC scale [7]. PNP severity was assessed using the Neuropathy Dysfunction Score (NDS) [8], and the degree of neurotoxicity was assessed using the National Cancer Institute Common Terminology Criteria for Adverse Events (NCI-CTCAE) version 5.0, 2021 [9]. Nerve conduction studies were performed under temperature-controlled conditions using a Dantec Keypoint electromyograph (Medtronic) [10].

The EMG protocol evaluated long limb nerves, excluding median and ulnar nerves due to susceptibility to entrapment neuropathies. The following sensory nerve action potential (SNAP) parameters were evaluated: amplitude and conduction velocity; compaund muscle action potential (CMAP) included amplitude, distal latency, conduction velocity, F-wave minimal latency, response dispersion, and conduction blocks. The data were compared with normative values [11] and the sural/radial amplitude ratio (SRAR) was calculated, as this marker is reportedly the most senstive to damage of large fibers and independent of age and BMI based on the majority of findings [12–14]. Mean values from bilateral measurements were analyzed.

All participants provided informed consent. The study protocol was approved by the Independent Ethics Committee of the Clinical Research Center at Immanuel Kant Baltic Federal University (Conclusion No. 35 dated October 27, 2022).

Statistical analysis used StatTech v. 4.2.8 (StatTech) and GraphPad Prism v. 8.0.1 (Insightful Science). Quantitative parameters were assessed for normal distribution using Kolmogorov–Smirnov test. Normally distributed quantitative data were described as mean (M) ± SD (SD) with 95% confidence interval (CI); non-normally distributed data – using median (Me) and upper and lower quartiles [Q_1; Q_3]. Categorical variables were described with absolute/percentage values with Clopper–Pearson 95% CIs. Two groups for quantitative variables with normal distribution and equal variances were compared using Student's t-test, while non-normally distributed variables were analyzed with the Mann–Whitney U test.

To assess the diagnostic significance of quantitative predictors for outcome prognosis, ROC analysis was applied with cut-off determination based on the maximum Youden index value. Models with AUC > 0.7 and 95% CI > 0.5 were considered, requiring statistical significance of $p < 0.05$ for the constructed model.

Results

The mean age of the patients (49 women (69%) and 22 men (31%)) was 59.0 ± 10.1 years. Patients were examined 4.50 ± 1.02 months after chemotherapy; the number of chemotherapy courses was 5.2 ± 1.5. Patients predominantly received platinum-based drugs and taxanes (Table 1).

All patients included in the study presented with sensory complaints. During neurological examination with scale-based assessment, convincing changes consistent with CIPN were identified in 52 (73%) patients. Decreased and/or absent brachioradialis and Achilles reflexes of various sensory modalities with lower limb onset were reported. The clinical pattern and electrophysiological data demonstrated a length-dependent predominantly sensory polyneuropathy. Motor function impairment with reduced distal foot extensor strength was observed in only 5 (7%) cases during docetaxel and carboplatin therapy. According to the NDS, neuropathy progression (> 5 points) was seen in 52 (73%) patients, while NCI-CTCAE grade 1–2 neurotoxicity was detected in 62 (87.3%) patients.

The electrophysiology study demonstrated SNAP below normative values in 46 (65%) patients from the superficial peroneal nerve, 29 (41%) from the sural nerve, and 21 (30%) from the radial nerve. The CMAP (distal latency, amplitude, conduction velocity) in peroneal (*n. peroneus*) and tibial (*n. tibialis*) nerves remained within normal limits, thus excluded from further analysis.

Only superficial peroneal nerve SNAP amplitude exceeded normal values at 3.0 μV (Table 2). SRAR of 0.43 ± 0.31 exceeded previously reported parameters. Electromyography revealed no demyelination patterns per EFNS/PNS 2021 criteria [15].

When analyzing the relationship between electrophysiological data and the presence of CIPN and its severity according to the NDS scale, we found significant changes in all parameters ($p < 0.05$). These changes were more pronounced for the superficial peroneal nerve, sural nerve, and SRAR ($p < 0.001$) (Table 3). The data confirm that lower values of the analyzed electrophysiological parameters correlate with greater severity of CIPN.

To evaluate the sensitivity and specificity of sensory nerve action potential (SNAP) parameters in CIPN, ROC analysis was used. All models were statistically significant with acceptable area under the curve (AUC) and 95% CIs, but differed in sensitivity and specificity (Table 4). The most sensitive and specific parameters for assessing CIPN progression were the

Table 1. Treatment regimen

Regimen	Number of patients (%)
CAPOX(XELOX) (oxaliplatin + capecitabine)	17 (23.6)
FLOT (oxaliplatin + docetaxel + calcium folinate + fluorouracil)	6 (8.3)
FOLFOX (oxaliplatin + calcium folinate + fluorouracil)	11 (15.3)
Gemcitabine + cisplatin	1 (1.4)
Doxorubicin + cisplatin/doxorubicin + carboplatin	1 (1.4)
Docetaxel	1 (1.4)
Carboplatin/cisplatin + docetaxel	5 (7.1)
Carboplatin/cisplatin + paclitaxel/etoposide	5 (7.1)
Carboplatin + docetaxel	3 (4.2)
Carboplatin + paclitaxel	17 (23.6)
Carboplatin/cisplatin + paclitaxel	3 (4.2)
Etoposide + cisplatin/docetaxel/paclitaxel + carboplatin/paclitaxel + etoposide/docetaxel	1 (1.4)
Total	71 (100)

SNAP amplitude of the sural nerve and SRAR (Fig. 1), while the SNAP amplitudes of the superficial peroneal and radial nerves demonstrated lower specificity.

Discussion

This study evaluated neurophysiological parameters in patients with malignant neoplasms after chemotherapy, assessing their correlation with clinical manifestations to identify easily reproducible electrophysiological markers for neurophysiologists. Patients with known risk factors for CIPN [16–18] were intentionally excluded from the study.

The enrolled patients with solid tumors primarily received platinum-based agents and taxanes; CIPN was observed in 73% of cases although all patients reported complaints. Therefore, diagnosing CIPN based solely on subjective patient reports is unreliable, as previously indicated in studies [19, 20].

Based on clinical data and electrophysiological findings, we determined that sensory axonal length-dependent peripheral neuropathy predominated in patients, with no signs of demyelination. These results align with prior research [21–24]. According to most literature, CIPN manifests as a length-dependent axonal neuropathy; thus, a reduction amplitude SNAP sural nerve would occur earlier than in the superficial radial nerve. Consequently, changes in the SRAR index may characterize early-stage neuropathy. However, a prospective study by V. Myftiu et al. reported reduced motor nerve conduction velocities alongside axonal damage in platinum- and taxane-induced CIPN [25]. The observed conduction velocity decrease (< 25% of the lower normative limit) in typical axonal patterns might result from rapid loss of large myelinated fibers rather than primary demyelination [3]. Thus, axonal changes may affect conduction velocity without definitive demyelination.

Our mean SRAR values closely matched 1997 data (SRAR = 0.4) [13] but differed from 2005 results (0.21) [11, 12]. When performing ROC analysis and determining the cut-off point adjusted for the Youden index for SRAR, we obtained a value of 0.49, exceeding which was considered a CIPN manifestation, which is also closer to 1997 findings [13] than in the 2005 study. [13] than 2005 ones [11, 12]. This discrepancy

Table 2. Electrophysiological findings

Study nerve	Parameter	M ± SD (95% CI)/Me [Q_1; Q_3]	Normal
Deep peroneal nerve (extensor digitorum brevis muscle)	Distal latency, ms	3.67 [3.37; 4.09]	⩽ 6.5
	Amplitude CMAP, mV	3.41 ± 1.58 (3.03–3.78)	⩾ 2.0
	Conduction velocity, m/s	44.65 [42.75; 46.23]	⩾ 44
Tibial nerve (abductor hallucis muscle)	Distal latency, ms	3.49 ± 0.63 (3.34–3.64)	⩽ 5.8
	Amplitude CMAP, mV	9.07 ± 3.77 (8.17–9.96)	⩾ 4.0
	Conduction velocity, m/s	45.31 ± 3.98 (44.37–46.25)	⩾ 44
	Minimal F-wave latency, m/s	49.40 [46.25; 53.30]	⩽ 56
Superficial peroneal nerve	Amplitude SNAP, μV	3.00 [0.00; 7.15]	⩾ 6
	Conduction velocity, m/s	43.50 [0.00; 47.23]	⩾ 40
Sural nerve	Amplitude SNAP, μV	7.35 [3.67; 12.48]	⩾ 6.0
	Conduction velocity, m/s	46.55 [44.40; 48.42]	⩾ 40
Superficial radial nerve	Amplitude SNAP, μV	19.87 ± 8.39 [17.89–21.86]	⩾ 15
	Conduction velocity, m/s	55.00 [52.95; 57.65]	⩾ 50
SRAR		0.43 ± 0.31 (0.36–0.50)	⩾ 0.21 (0.4)

Table 3. Analysis of correlation between SRAR index/sensory nerve action potential amplitude and CIPN severity using NDS

Parameter	Neuropathy severity (NDS)	M ± SD (95% CI)/Me [Q_1; Q_3]	*p**
SRAR	Normal	0.65 ± 0.36 (0.48–0.83)	< 0.001
	Moderate	0.38 ± 0.25 (0.30–0.46)	
	Severe	0.23 ± 0.18 (0,11–0.35)	
Amplitude SNAP sural nerve, μV	Normal	14.59 ± 6.63 (11.40–17.79)	< 0.001
	Moderate	7.22 ± 5.08 (5.62–8.82)	
	Severe	3.58 ± 3.02 (1.55–5.61)	
Amplitude SNAP radial nerve, μV	Normal	24.15 [20.23; 27.38]	0.004
	Moderate	20.05 [12.20; 24.90]	
	Severe	13.90 [9.12; 16.92]	
Amplitude SNAP superficial peroneal nerve, μV	Normal	7.10 [4.35; 10.00]	< 0.001
	Moderate	2.30 [0.00; 6.45]	
	Severe	0.00 [0.00; 3.22]	

Note. Severity according to the NDS: normal — 0–4 points, moderate — 5–13 points, severe — 14–28 points.

Table 4. ROC analysis of sensory nerve motor potential amplitudes and SRAR index

Parameter	AUC	95% CI	Cut-off, μV	Sensitivity	Specificity
Amplitude SNAP					
superficial peroneal nerve	0.764 ± 0.070	0.628–0.901	4.30	78.9	67.3
sural nerve	0.835 ± 0.061	0.715–0.955	11.65	73.7	84.6
superficial radial nerve	0.705 ± 0.074	0.560–0.851	19.20	84.2	55.8
SRAR	0.778 ± 0.068	0.644–0.911	0.49	73.7	75.0

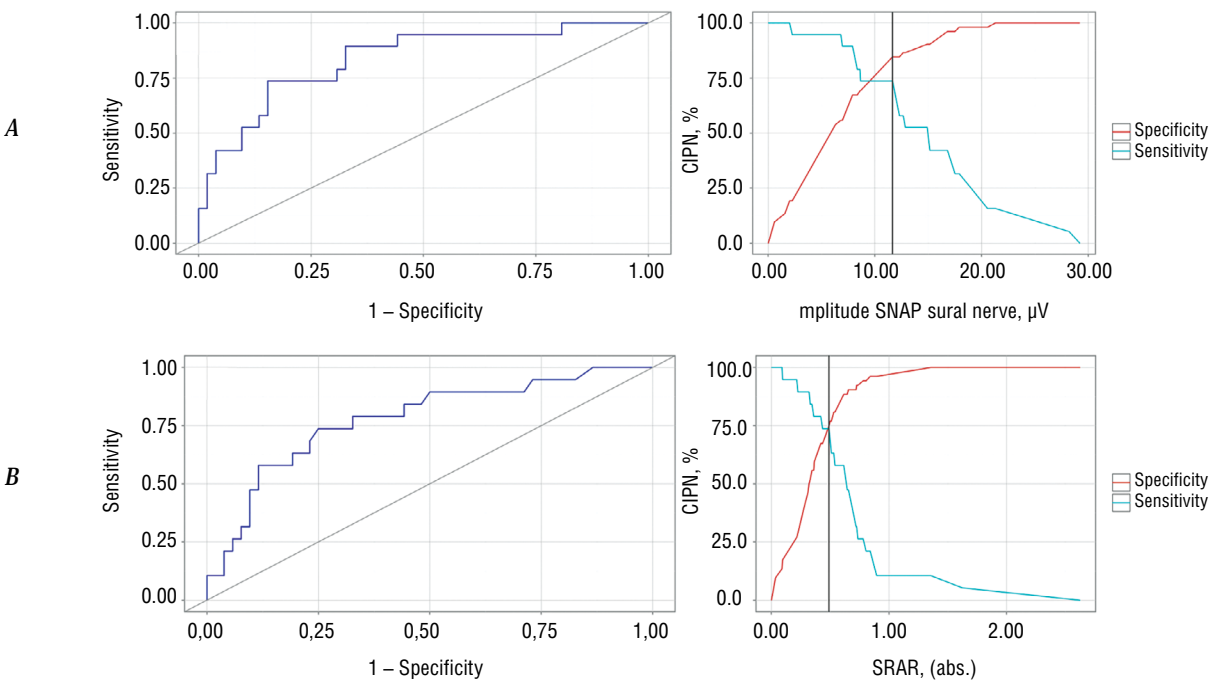

Fig. 1. ROC curve, model specificity and sensitivity, characterizing correlation between CIPN and amplitude SNAP sural nerve (*A*) and SRAR (*B*).

likely stems from study designs and cohorts: the 1997 research evaluated SRAR in patients with polyneuropathy, while the 2005 study involved healthy populations. Earlier studies reported no age-dependent SRAR variability [11–13], but a 2020 Indian study on 146 patients yeilded other normative values and demonstrated age effects [26]. Current literature lacks consensus on SRAR normative values and age influence, highlighting a study limitation requiring further investigation.

Our data show that all models were significant when evaluating sensitivity and specificity of electrophysiological markers for CIPN. SRAR (73.7 and 75.0%, respectively) and sural nerve action potential amplitude (73.7 and 84.6%) had the highest sensitivity and specificity, i.e., the sensitivity of these parameters was equal and specificity was 9.6% higher for the sural nerve. For action potentials of the radial and superficial peroneal nerves, specificity was below 70%, although sensitivity was high. Our findings partially contradict a prior study in patient with malignant neoplasms where SRAR sensitivity/specificity was 56%/77% versus SNAP amplitudes were 64%/70% [27]. That study concluded SRAR was less sensitive than sural SNAP amplitude despite 7% higher specificity, potentially due to unaccounted anamnestic neuropathy risk factors in the cohort.

Conclusion

Clinical CIPN assessment remains limited by patient-reported subjectivity and poor correlation with neurological exams. Electrophysiological markers (SRAR and sural SNAP amplitude) enable objective CIPN evaluation, facilitating early detection of large-fiber neuropathy in cancer patients to optimize treatment strategy and improve their quality of life. The impact of age on SRAR warrants further research in larger cohorts.

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