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Clinical and electrophysiological
characteristics of peripheral neuropathy in
patients with hematological malignancies

RESEARCH PAPER

Clinical and electrophysiological characteristics of peripheral neuropathy in patients with hematological malignancies on chemotherapy

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Abstract

Background: Chemotherapy-induced peripheral neuropathy (CIPN) is a common complication in cancer patients especially those treated with neurotoxic chemotherapeutic agents. CIPN is usually diagnosed clinically; However, electrophysiologic testing confirms the diagnosis and rules out other potential causes of neuropathic manifestations.

Aims: This study aimed to look for electrophysiological changes and clinical features of peripheral neuropathy among patients with haematological malignancies treated with chemotherapy in Basrah governorate, southern Iraq

Methods: A comparative cross-sectional conducted in Basrah City, southern Iraq. The study involved 109 patients with confirmed diagnoses of multiple myeloma, leukaemia, and lymphoma. Their clinical, and electrophysiological characteristics using nerve conduction studies and needle electromyography have been registered.

Results: The proportion of CIPN among patients with hematological malignancies is 38.53%. All of the cases were axonal polyneuropathy, with 54.76% being sensory followed by 45.24% mixed sensory motor neuropathy. Clinically, 55.96% of the cases reported lower limb paresthesia and numbness and 11.01% reported limb weakness. Sensory nerve conduction studies showed a reduction in the amplitude of sural and ulnar nerves. Moreover, motor nerve conduction studies also showed a reduction in the amplitude of peroneal and ulnar nerves

Conclusions: The chemotherapy regimen induced peripheral neuropathy; the pattern was distal, symmetrical, length-dependent, axonal polyneuropathy of pure sensory mainly followed by mixed sensory-motor type. We recommend early detection, monitoring and appropriate management of CIPN.

Keywords: CIPN; EMG; NCS; haematological malignancies; neurophysiology.

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Introduction

Chemotherapy- induced peripheral neuropathy (CIPN) is a common, painful, dose-limiting complication in cancer patients that occurs as a result of exposure to neurotoxic chemotherapeutic drugs, which become more prevalent as cancer survival improves. CIPN occurs in 30-40% of patients receiving neurotoxic chemotherapy.[1] CIPN is a side effect of certain chemotherapeutic drugs, including molecularly targeted therapeutic agents like (Bortezomib), taxanes (Paclitaxel, Docetaxel), platinum-containing drugs (Oxaliplatin), and vinca alkaloids (Vincristine). Neuropathy develops within weeks or months following the initiation of the chemotherapy and may persist for months or years following the completion of the treatment. Even though its prevalence decreases

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over time, at least 30% of patients continue to suffer from CIPN six months or longer after chemotherapy has been completed, this substantially reduces the quality of life and becomes a significant survivorship concern. [3] Chemotherapy-induced peripheral neuropathy causes axonopathy due to axon injury and neuronopathy affecting the cell bodies of the dorsal root ganglia. The primary axon damage begins at the most vulnerable region of the nerve, which is the end of the longest nerve, and then spreads centrally (length dependent neuropathy).[4] Although motor symptoms are common, sensory symptoms are the most frequently encountered in CIPN patients. Patients may experience paresthesia, discomfort, or numbness because of sensory axonal degeneration. Damage to motor neurons is the most common cause of motor symptoms, which include weakness in the limbs and eventually difficulty in walking, picking up tiny items, and executing delicate motor motions like buttoning clothes[5] Chemotherapy-induced peripheral neuropathy is usually diagnosed clinically; However, electrophysiologic testing confirms the diagnosis and rules out other potential causes of neuropathy. [6] Nerve conduction study (NCS) and electromyography (EMG) may aid in determining the extent of nerve damage from CIPN, which may be utilized as a baseline and monitored for any future symptoms.[7] Unfortunately, there are no agents known to be effective in preventing CIPN. Therefore, routine screening for CIPN is recommended and keeping an eye out for individuals who are at higher risk. However, duloxetine is the only drug confirmed to be effective in the symptomatic treatment of CIPN, and it is the only drug indicated for this condition.[8]

This study aimed to determine the percentage of CIPN and assess the clinical and electrophysiological changes of peripheral neuropathy in adult patients with hematological malignances who are already kept on chemotherapy.

Patients & Methods

A comparative cross-sectional study was conducted in Basrah governorate, south of Iraq, on patients with hematological malignancies who attended the hematological centre at Al-Sadar Teaching Hospital and Al-Sayyab Teaching Hospital during the period of 12 months from the first of October 2022 to the first of October 2023, the patients were referred to the electrophysiological examinations in neurophysiology outpatient clinics of Al-Sadr or Al-Basrah Teaching Hospital. A total of 240 participants were enrolled in the study, including 109 patients of either gender and aged ≥18 years, with confirmed diagnoses of hematological malignancy (multiple myeloma, leukemias, and lymphomas). All patients were on different chemotherapy protocols (UKALL: Cyclophosphamide, Vincristine, Doxorubicin, Methotrexate, Dexamethasone, Etoposide, Cytarabine; CHOP: Cyclophosphamide, Doxorubicin hydrochloride (hydroxy daunomycin), vincristine sulfate (Oncovin), Prednisone; Hyper CVAD: Cyclophosphamide, Vincristine, Dexamethasone, Doxorubicin methotrexate/ cytarabine; ABVD: Doxorubicin, Bleomycin, Vinblastine, Dacarbazine; R-CHOP: Rituximab, Cyclophosphamide, Doxorubicin hydrochloride (hydroxy daunomycin), and Vincristine sulfate (Oncovin), Prednisone; VRD: Bortezomib, Lenalidomide, Dexamethasone; VCD: Bortezomib, Cyclophosphamide, Dexamethasone). The control group was

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comprised of 131 participants including patients' relatives and medical staff age, sex, and BMI match to reduce the bias, they were tested at neurophysiology outpatients' clinics in both Al-Sadr Teaching Hospital and Al-Basrah Teaching Hospital and had normal NCS and EMG study. The study excluded patients with peripheral neuropathy that could be explained by previous diabetes-related neuropathy, chronic kidney disease, thyroid-induced neuropathy, vitamin B12 deficiency, alcoholic, and very ill patients, including those admitted to the ward or intensive care unit (ICU). Children and adolescents (under the age of 18) were also not included in the study. Active surveying, identification of patients receiving chemotherapy, and assessment of their medical records from the statistics unit records served as the first steps in the data recruitment process. The required information is the patients' detailed demography, medical history, drug history, and clinical presentation of the most recent neurological illness, as well as the clinical examination to determine whether they had peripheral neuropathy, which was obtained through direct interviews with the patients. In addition to receiving their agreement to participate in the study, some information for the confirmation of malignancies and the treatment plan was also gathered from the records. Clinical signs and electrophysiological examinations utilizing the Nihon Kohden Neuropack S3 with surface and needle electrodes were used to support the diagnosis of peripheral neuropathy. The results of the tests were either normal or abnormal based on the normal values of the NCS parameters of the studied nerves, [9,10] which is further subclassified according to the pathology into axonopathy or myelinopathy. Or based on the types of nerves damaged (pure motor, pure sensory, or mixed neuropathy).

The study results were analyzed using the computerized Statistical Package for Social Science (SPSS) version 26 (Armonk, NY: IBM Corp.) software. The numerical data were tabulated as mean ± standard deviation (SD). While categorical data were tabulated as number (percentage). Chi-square, independent sample t-test, and Fisher's exact test were used for data analysis. A p-value of ≤0.05 is regarded as statistically significant.

Results

Table-1, presents an overview of the sociodemographic characteristics among the study groups. There was no notable difference in age, gender, smoking status, height, weight and body mass index (BMI) between the patient and the control group.

Table 1. The demographic characteristics of the study groups

Demographic characteristics		Patients with hematological malignancies n=109	Control group n=131	P-value[*]
Age (year)		44.35 ± 18.17	44.06 ±17.32	0.901[##]
Gender	Male	82 (46.9%)	93 (53.1%)	0.471[#]
	Female	27 (41.5%)	38 (58.5%)	
Smoking	Smoker	11 (47.8%)	12 (52.2%)	0.829[#]
	Non-smoker	98 (45.2%)	119 (54.8%)	
Height (cm)		168.34 ± 7.58	167.73± 6.36	0.508[##]
Weight (kg)		74.95 ± 9.35	73.13± 10.16	0.151[##]
BMI		26.48 ± 3.22	25.93 ± 2.84	0.168[##]

[#]chi-square test, [##]independent sample t-test
BMI: Body mass index

Table-2, presents the neurological manifestations of the cases. The majority of the patients (55.96%) exhibited symptoms of paraesthesia and numbness, with neuropathic burning pain being reported in 12.84% of the cases, and only 11.01% presented with limb weakness. Myalgia, on the other hand, was seen in only 9.17% of the studied cases, 10.09% of patients presented with ataxia and loss of vibration and proprioception, and 11% were asymptomatic.

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Table 2. The neurological characteristics of the cases (n=109)

Clinical features	No. (%)
Paresthesia and numbness	61 (55.96)
Limb weakness	12 (11.01)
Neuropathic burning pain	14 (12.84)
Myalgia	10 (9.17)
Asymptomatic	12 (11)
Ataxia	11(10.09)
Loss of vibration and proprioception	11(10.09)
Total	109 (100)

Table-3, presents the comparison between different protocols of hematological malignancies with NCS results, there is no statistically significant difference between them, however, the protocols of multiple myeloma show more NCS abnormality (56.0%, 72.7%) than other hematological malignancies.

Table 3. Nerve conduction study in different types of hematological malignancy and treatment protocol

Hematological malignancy	Nerve conduction study			Total	P- value
	Protocol	Normal No. (%)	Abnormal No. (%)		
Multiple myeloma	VRD	11 (44.0)	14 (56.0)	25(100.0)	0.467
	VCD	3 (27.3)	8 (72.7)	11(100.0)	
Leukemia	UKALL	16 (76.2)	5 (23.8)	21 (100.0)	0.727
	HyperCVAD	12 (70.6)	5 (29.4)	17 (100.0)	
Lymphoma	ABVD	11 (78.6)	3 (21.4)	14 (100.0)	0.712
	R-CHOP	15 (71.4)	6 (28.6)	21 (100.0)	

VRD: Bortezomib, Lenalidomide and Dexamethasone.; VCD: Bortezomib, Cyclophosphamide and Dexamethasone.; UKALL: Cyclophosphamide, Vincristine, Doxorubicin, Methotrexate, Dexamethasone, Etoposide, Cytarabine.; Hyper CVAD: Cyclophosphamide, Vincristine Dexamethasone, Doxorubicin methotrexate/ cytarabine.; ABVD: Doxorubicin, Bleomycin, Vinblastine, Dacarbazine; R-CHOP: Rituximab, Cyclophosphamide, Doxorubicin hydrochloride (hydroxy daunomycin), and Vincristine sulfate (Oncovin), Prednisone.

Table-4, shows the results of the electrophysiological study of the motor nerves among cases and the control group. A significantly lower amplitude was observed in the ulnar and peroneal nerves for both sides of patients compared with the control. Moreover, the F-wave latency of the ulnar nerve and tibial nerve among cases was significantly prolonged bilaterally compared with the control group.

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Table 4. The electrophysiological study results of the motor nerves among cases and controls.

Motor nerves	Parameter		Patients N = 109	Control N = 131	P-value
Median nerve	Distal latency	Rt	3.37 ± 0.32	3.37 ± 0.36	0.996
		Lt	3.24 ± 0.43	3.37 ± 0.38	0.502
	Amplitude	Rt	8.00 ± 1.83	8.29 ± 1.69	0.215
		Lt	8.01 ± 1.99	7.98 ± 1.94	0.912
	Conduction velocity	Rt	57.76 ± 7.27	58.19 ± 5.53	0.608
		Lt	57.08 ± 6.76	58.51 ± 5.37	0.075
Ulnar nerve	Distal latency	Rt	2.38 ± 0.38	2.51 ± 0.33	0.072
		Lt	2.38 ± 0.52	2.50 ± 0.26	0.201
	Amplitude	Rt	7.36 ± 1.26	8.20 ± 1.24	0.001*
		Lt	7.31 ± 1.09	8.43 ± 1.26	0.001*
	Conduction velocity	Rt	59.95 ± 8.14	60.73 ± 6.32	0.415
		Lt	60.61 ± 8.02	61.51 ± 6.53	0.061
	F wave	Rt	27.97 ± 2.24	26.49 ± 1.76	0.001*
		Lt	27.87 ± 2.11	26.51 ± 1.83	0.001*
Peroneal nerve	Distal latency	Rt	3.85 ± 0.79	3.96 ± 0.63	0.255
		Lt	3.85 ± 077	3.96 ± 0.69	0.262
	Amplitude	Rt	3.24 ± 1.44	4.23 ± 1.25	0.001*
		Lt	3.29 ± 1.35	4.37 ± 1.42	0.001*
	Conduction velocity	Rt	51.19 ± 10.69	51. 74 ± 4.87	0.626
		Lt	51.52 ± 10.57	51.76 ± 4.63	0.823
Tibial nerve	Distal latency	Rt	3.88 ± 0.70	3.93 ± 0.57	0.497
		Lt	3.88 ± 0.60	3.95 ± 0.62	0.406
	Amplitude	Rt	8.04 ± 3.05	7.81 ± 2.73	0.537
		Lt	8.05 ± 2.99	7.73 ± 2.17	0.365
	Conduction velocity	Rt	53.16 ± 6.66	54.03 ± 4.67	0.255
		Lt	53.44 ± 6.38	54.25 ± 5.04	0.281
	F wave	Rt	50.56 ± 4.45	46.54 ± 3.12	0.001*
		Lt	50.65 ± 4.62	46.56 ± 3.20	0.001*
Independent sample t- test, Rt= right, Lt= left, *indicates significant difference (p≤0.05)					

Concerning the sensory nerves, the amplitudes of both ulnar and sural nerves were significantly lower among cases compared with the controls (Table-5)

Table 5. The electrophysiological results of the sensory nerves among cases and controls

Sensory nerves	Parameters	Parameter	Patients n=109	Control n=131	p-value
Ulnar nerve	Distal latency	Rt	2.02 ± 0.40	2.09 ± 0.26	0.102
		Lt	2.07 ± 0.38	2.08 ± 0.24	0.657
	Amplitude	Rt	23.77 ± 7.35	34.33 ± 13.73	0.001*
		Lt	24.24 ± 8.92	33.89 ± 13.91	0.001*
	Conduction velocity	Rt	55.38 ± 9.54	57.58 ± 7.10	0.084
		Lt	55.02 ± 9.55	58.28 ± 6.77	0.065
Sural nerve	Distal latency	Rt	2.47 ± 0.72	2.63 ± 0.41	0.213
		Lt	2.48 ± 0.73	2.56 ± 0.41	0.322
	Amplitude	Rt	7.11 ± 3.42	11.64 ± 2.95	0.001*
		Lt	7.07 ± 3.32	11.45 ± 2.86	0.001*
	Conduction velocity	Rt	52.51 ± 16.33	49.91 ± 5.64	0.115
		Lt	52.55 ± 16.22	49.90 ± 5.99	0.108
*Independent sample t-test **Rt: Right, Lt: Left**					

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The results of EMG exams were symmetrical bilaterally. The presence of neurogenic motor unit action potentials (MUAPs) in 17.43% of patients in the EHL muscle and 9.17% of patients in the TA muscle. It was significantly higher than the control group, as determined by needle electromyography (EMG) examination.

However, insignificant changes in Motor Unit Action Potentials (MUAPs) were observed in the VM, First Dorsal Interosseous (FDI), and Triceps muscles, in which neurogenic MUAPs were only detected in 1.83% of the patients. All the investigated patients showed normal findings in their deltoid muscles.

Table 6. Needle electromyography of lower and upper limbs muscles

		Patients N = 109 No. (%)	Control N = 131 No. (%)	Total No. (%)	P-value
EHL	**Neurogenic**	19 (100.0)	0 (0.0)	19 (100.0)	0.001*
	Normal	90 (40.7)	131 (59.3)	221 (100.0)	
TA	**Neurogenic**	10 (100.0)	0 (0.0)	10 (100.0)	0.001*
	Normal	99 (43.0)	131 (57.0)	230 (100.0)	
VM	**Neurogenic**	2 (100.0)	0 (0.0)	2 (100.0)	0.205
	Normal	107 (45.0)	131 (55.0)	238 (100.0)	
FDI	**Neurogenic**	2 (100.0)	0 (0.0)	2 (100.0)	0.205
	Normal	107 (45.0)	131 (55.0)	238 (100.0)	
Triceps	**Neurogenic**	2 (100.0)	0 (0.0)	2 (100.0)	0.205
	Normal	107 (45.0)	131 (55.0)	238 (100.0)	
Deltoid	**Normal**	109 (45.4)	131 (54.6)	240 (100.0)	-
Fisher's exact test, EHL= Extensor halluces longus, TA= Tibialis anterior, VM= Vastus medialis, FDI= first dorsal interosseous, Rt: Right, Lt: Left					

Table-6, presents the result of the electrodiagnostic study function and pathology in haematological malignancies, 63.6% of abnormal NCS in multiple myeloma cases showed mixed motor and sensory neuropathy, while 90% and 60% of lymphoma and leukaemia respectively showing pure sensory neuropathy. None of them show pure motor neuropathy. All of the cases were axonopathy. There were statistically significant in EDX function between lymphoma and multiple myeloma.

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Table 6. The electro-diagnostic study of among cancer patients

EDX		Leukemia (n=38) No. (%)	Lymphoma (n=35) No. (%)	Myeloma (n=36) No. (%)	P-value
Function	**Mixed motor and sensory neuropathy**	4 (40.0)	1 (10.0)	14 (63.6)	**0.121a** **0.212b** **0.005c***
	Pure sensory neuropathy	6 (60.0)	9 (90.0)	8 (36.4)	
	Pure motor neuropathy	0 (0.0%)	0 (0.0)	0 (100.0)	
Pathology	**Axonopathy**	10 (100.0)	10 (100.0)	22 (100.0)	-
	Demyelination	0	0	0	
EDX: Electrodiagnostic study; a: p-value between leukemia and lymphoma **b: p-value between lymphoma and myeloma; c: p-value between leukemia and myeloma; *: p-value <0.05**					

Discussion

With advancements in early detection and treatment of cancer, the number of cancer survivors will continue to rise, leading to an increase in CIPN cases among patients. Numerous studies were carried out to evaluate the prevalence of CIPN, clinical and electrophysiological alterations, antineoplastic medications, and risk factors associated with CIPN. Furthermore, studies have been conducted on specific medications that induce peripheral neuropathy, most frequently vincristine, bortezomib, and platinum-based medications. However, few of these were carried out in our local area. The majority of patients in this study had sensory symptoms (table-2) such as paresthesia and numbness, and a smaller percentage experienced burning pain. Motor symptoms, such as limb weakness and myalgia, were less prevalent. The symptoms initially manifested in the toes and fingers in a pattern resembling gloves and stockings and subsequently spread proximally. The results align with a study conducted by Chu et al. (2015), which proposes that the majority of symptoms

associated with chemotherapy-induced peripheral neuropathy are linked to sensory nerves. These symptoms include numbness and tingling caused by injury to the large-fiber sensory axons, as well as pain arising from damage to the smaller sensory fibers. [11]This phenomenon can be attributed to the presence of fenestrated endothelial cells in the dorsal root ganglion (DRG), which facilitate the unrestricted movement of molecules between the bloodstream and the extracellular fluid. In comparison, the blood-nerve barrier in the DRG is considerably more permeable than in the spinal cord. [12] The nerve endings in the hands and feet are usually the first to be damaged by toxicity, and this happens in a symmetrical and length-dependent manner. The predominant impact is on the larger sensory nerve fibers, while injury to the smaller sensory fibers is infrequent and limited to certain chemotherapeutic treatments. Motor involvement is less prevalent compared to sensory nerve impairment. Additionally, the administration of chemotherapy may lead to the emergence of motor and autonomic neuropathy symptoms,

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which might vary depending on the specific treatment received.[13] We conducted a comparative analysis of electrophysiological changes in two groups: patients diagnosed with hematological malignancies and healthy individuals. The purpose is to evaluate the presence of peripheral neuropathy resulting from chemotherapy treatment. Regarding the nerve conduction study, the motor nerves (median, ulnar, peroneal, and tibial nerves) and sensory nerves (ulnar and sural nerves) were examined. The ulnar and tibial F-wave latencies were also measured. Although all the parameters fell within the normal range, there was a significant decrease in the amplitude of the sural and sensory ulnar nerves (table-5), as well as the peroneal and motor ulnar nerves on both sides (table-4), when compared to healthy individuals. Additionally, there was a significant prolongation of both tibial and ulnar F-wave latencies on both sides. The amplitude of compound muscle action potential (CMAP) correlates to the number of motor nerve axons, while the amplitude of sensory nerve action potential (SNAP) is indicative of the number of sensory nerve axons. According to Chung et al. (2013), when there are lesions that cause loss of axons, it usually leads to a decrease in the amplitudes of CMAP and SNAP. This indicates a likelihood of axonal polyneuropathy involving both the sensory and motor neurons in the lower and upper limbs in a symmetrical manner.[14] Regarding F-wave latency in axonal polyneuropathies, it can be either normal or slightly impacted, with the latter occurring due to the loss of faster-conducting axons. Nevertheless, the latency of F-waves does not approach 125% to 130% of the upper limit of normal (ULN) just as a result of axonal loss.[15] According to Jerath et al. (2015), F-wave minimal latencies can identify even the smallest abnormalities in neuropathies

that primarily affect the axons.[16] Yet, all the values fell within the normal range of electrodiagnostic testing. However, this does not rule out the occurrence of peripheral neuropathy because normative ranges are often large, reflecting the wide range of amplitudes across healthy people. As a result, NCS may remain normal in patients who have experienced substantial axonal loss.[17] These findings are in line with a longitudinal study by Argyriou et al. (2007) that looked at the characteristics of peripheral neuropathy brought on by chemotherapy. Sensory nerve conduction studies of the sural and ulnar nerves revealed a reduction or complete absence (in the sural nerve) of sensory action potentials (SAPs), although the sensory conduction velocity (SCV) remained intact. however, there were no significant alterations observed in the motor conduction studies of the peroneal and ulnar nerves, including the compound muscle action potential (CMAP), motor conduction velocity (MCV), and F wave latencies.[18] Timmins et al. (2020) have also identified axonal sensory predominance in CIPN. It was discovered that the SNAP amplitudes of the sural, superficial peroneal, and ulnar nerves were lower than the normal range in 23–30% of the cohort. However, the SNAP latencies were within the normal range for 93.5–97.8% of the cohort, indicating the presence of axonal neuropathy. with only 4.4% of patients having mean tibial CMAP amplitude abnormalities, which is consistent with sensory predominance.[17] In a prospective study including individuals undergoing chemotherapy in their study, Briani et al. (2014) observed a significant longitudinal drop in both SNAP and SCV of the sural and ulnar nerves from the beginning to the end of treatment. In contrast, there was no significant alteration observed in the motor

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conduction study scores of the peroneal nerve over time.[19] Needle electromyography (EMG) is a crucial component of the electrodiagnostic examination because it provides important details about the distribution and functionality of muscle fibers and motor units that nerve conduction studies (NCS) cannot provide on their own. Needle electromyography (EMG) is highly adept at detecting alterations associated with axonal loss (denervation and reinnervation) and can diagnose motor impairment before its manifestation in clinical symptoms. Analysis of spontaneous activity, as well as changes in the morphology and recruitment of motor unit potentials (MUAPs), is conducted to identify the timing and severity of polyneuropathy as well as the effectiveness of reinnervation. Axonal polyneuropathies are characterized by the presence of neurogenic motor unit action potentials (MUAPs) that have a long duration, large amplitude, and polyphasia. Additionally, there is a reduced recruitment of MUAPs that is disproportionate to the level of activity, and this can occur with or without spontaneous activity.[15] These MUAPs exhibited characteristics such as prolonged duration, high amplitude, polyphasia, and reduced recruitment. Importantly, these changes were found to be statistically significant when compared to healthy controls. These findings corroborate the idea of axonal peripheral neuropathy, wherein the longest nerves are initially afflicted, followed by a gradual advance towards the proximal areas over time. Therefore, the majority of distal muscles will exhibit the largest EMG alterations.[20] These findings are in line with research by Custodio et al. (2017) and Argyriou et al. (2008), which discovered that needle EMG findings in chemotherapy-induced axonal neuropathy include reduced recruitment, fibrillation potentials, and large, polyphasic

MUAPs in the distal limb muscles, with the lower limbs showing the greatest severity of these findings. However, we were unable to detect any fibrillation potential, possibly because the individuals we analyzed were chronic cases.[21,22]

This study found that symmetrical axonal, mostly pure sensory polyneuropathy (table-7), was the most common type. Mixed sensory motor polyneuropathy came in second, and none of the people who were studied had pure motor polyneuropathy. Various chemotherapies exert their effects on distinct parts of the nervous system through diverse mechanisms. Some of them affect the DRG because it is less protected by the blood-nerve barrier and more likely to be damaged by neurotoxins. This may explain why sensory predominance is more common in people with CIPN. Other ones mess up microtubule dynamics, which are important for energy and material delivery. Consequently, there is a length-dependent degeneration of the distal segments of axons (known as Wallerian degeneration) and a modification of the peripheral nerves' axonal membrane.[13,23] These results align with research conducted by Kandula et al. (2017), Winters-Stone et al. (2017), and Argyriou et al. (2012), which indicates that sensory axonal neuropathy is the major neurophysiological manifestation of CIPN, followed by sensorimotor axonal neuropathy. [24-26]

In conclusions & recommendations,
chemotherapy regimens induced peripheral neuropathy in about 38.5% of the treating cases. The pattern is usually distal, symmetrical, length-dependent, axonal polyneuropathy of pure sensory mainly followed by sensory-motor type. In NCS, sural and sensory ulnar nerve amplitude were most affected, while needle EMG showed neuropathic alterations in distal lower limb

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muscles. We recommend a screening schedule for early detection of PN, and further study to compare pre- and post-treatment changes with a larger sample size and longer follow-up duration.

Conflicts of interest

The authors declare no conflict of interest in this study.

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الخصائص السريرية والكهربية للاعتلال العصبي المحيطي في المرضى الذين يعانون من الأورام الخبيثة الدموية على العلاج الكيميائي

المقدمة: الاعتلال العصبي المحيطي الناجم عن العلاج الكيميائي هو أحد المضاعفات الشائعة لدى مرضى السرطان الذي يحدث نتيجة التعرض لأدوية العلاج الكيميائي السامة للأعصاب. عادة ما يتم تشخيصه سريريا. ومع ذلك، تؤكد الاختبارات الفيزيولوجية الكهربية التشخيص وتستبعد الأسباب المحتملة الأخرى لأعراض الاعتلال العصبي.

الاهداف: هدفت هذه الدراسة إلى البحث عن التغيرات الكهربية والسمات السريرية للاعتلال العصبي المحيطي بين المرضى الذين يعانون من الأورام الدموية الخبيثة الخاضعين للعلاج الكيميائي في البصرة.

الطريقة: أجريت دراسة مقطعيه مقارنه في مدينة البصرة، جنوب العراق. شملت الدراسة ١٠٩ مرضى تم تشخيص إصابتهم بأورام الدم الخبيثة بشكل مؤكد. وقد تم تسجيل خصائصها السريرية والفيزيولوجية الكهربية باستخدام دراسات التوصيل العصبي وتخطيط كهربية العضل بالإبرة.

النتائج: نسبة الاعتلال العصبي بين الحالات المصابة بسرطان الدم تبلغ ٣٨,٥٪. سريريًا، أبلغ ٦١ (٥٥,٩٦٪) من الحالات عن تنمل في الأطراف السفلية، و١٤ (١٢,٨٤٪) يعانون من الالام, بينما يشكل الضعف في الأطراف وألالم العضلي نسبة أقل. تظهر دراسات التوصيل العصبي الحسي انخفاضًا في سعة الأعصاب الربلية والزندية. تُظهر دراسات توصيل العصب الحركي أيضًا انخفاضًا في سعة الأعصاب الشظوية والزندية. جميع الحالات كانت عبارة عن اعتلال أعصاب محوري، بنسبة ٥٤,٧٦٪ كانت حسية يليها اعتلال عصبي حسي حركي مختلط.

الاستنتاجات: نظام العلاج الكيميائي يسبب الاعتلال العصبي المحيطي في حوالي ٣٨,٥٪ من حالات العلاج. النمط عادة ما يكون اعتلال الأعصاب الطرفي، المتماثل، المحوري الحسي، يليه النوع الحسي الحركي. نوصي بالكشف المبكر والمراقبة للاعتلال العصبي المحيطي لمساعدة المرضى الذين يعانون من الأورام الدموية الخبيثة على الاستفادة من العلاج ومتابعة حياتهم بشكل طبيعي.

الكلمات المفتاحية: اعتلال الاعصاب الناتج عن العلاج الكيميائي ، تخطيط الاعصاب ، تخطيط العضلات ، الفسلجة العصبية، اورام الدم الخبيثة.