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STAGE-SPECIFIC ELECTRONEUROMYOGRAPHIC PATTERNS AND LABORATORY MARKERS OF UREMIC NEUROPATHY SEVERITY

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Objective. To characterize electroneuromyographic (ENMG) abnormalities and evaluate laboratory markers associated with peripheral neuropathy severity across chronic kidney disease (CKD) stages. **Material and methods.** This cross-sectional observational study included 65 patients with CKD (stage I, n=9; stage II, n=14; stage III, n=8; stage IV, n=8; stage V, n=26) and a healthy control group for electrophysiological comparison. Peripheral neuropathy was classified into stages 0–3 using an adapted Dyck staging approach. Bilateral motor nerve conduction studies assessed nerve conduction velocity (NCV) and compound muscle action potential (M-response) amplitude in the median, ulnar, tibial, and peroneal nerves. Receiver operating characteristic (ROC) analysis evaluated creatinine, urea, potassium, erythrocyte sedimentation rate (ESR), ferritin, and parathyroid hormone (PTH). **Results.** Among 65 patients, 24.6% had no neuropathy, 36.9% had subclinical neuropathy, 33.8% had symptomatic neuropathy, and 4.6% had disabling neuropathy. Axonal and mixed patterns predominated. Patients receiving hemodialysis had lower NCV and M-response amplitudes in all examined nerves (all $p<0.001$). Potassium showed the strongest discrimination for symptomatic neuropathy (cut-off 5.08 mmol/L; sensitivity 95.0%; specificity 78.3%; AUC 0.868), whereas PTH showed high discrimination for subclinical neuropathy (cut-off 146.05 pg/mL; sensitivity 88.9%; specificity 86.5%; AUC 0.898). **Conclusion.** ENMG abnormalities increased with neuropathy severity. Serum potassium and PTH were associated with clinically relevant neuropathy stages and may be useful candidate markers; however, the proposed cut-offs require prospective external validation.

Keywords: chronic kidney disease, uremic neuropathy, electroneuromyography, nerve conduction studies, biomarkers, potassium, parathyroid hormone, hemodialysis

Introduction. Globally, the burden of chronic kidney disease (CKD) continues to increase, representing a major medical and social challenge [1, 2]. A systematic review and meta-analysis estimated the global prevalence of CKD at approximately 11–13% [3]. CKD also represents an important chronic disease in pediatric populations, with substantial long-term morbidity [4, 5]. Neurological complications are common in advanced CKD and have been reported in up to 70% of patients before dialysis and up to 90% of patients receiving dialysis [6, 7]. Peripheral neuropathy is among the most frequent neurological manifestations, supporting the need for combined clinical, electrophysiological, and biochemical assessment to improve early detection and characterization [8, 9].

The aim of this study was to characterize ENMG patterns and assess laboratory markers associated with peripheral neuropathy severity across CKD stages.

Material and methods. *Study design and participants.* This cross-sectional observational study was conducted at Tashkent State Medical University, Department of Neonatology, Tashkent, Uzbekistan. The analysis included 65 patients with CKD who underwent clinical, laboratory, and ENMG evaluation. According to CKD stage, 9 patients (13.8%) had stage I disease, 14 (21.5%) stage II, 8 (12.3%) stage III, 8 (12.3%) stage IV, and 26 (40.0%) stage V. A healthy control group was used for comparison of electrophysiological parameters.

Clinical assessment and neuropathy classification. All participants underwent neurological examination with attention to symptoms and signs of peripheral nerve involvement. Peripheral neuropathy severity was classified using a four-stage scheme adapted from the Dyck staging approach and previously applied in CKD cohorts [10]: stage 0, no neuropathy; stage 1, subclinical neuropathy; stage 2, symptomatic neuropathy; and stage 3, disabling neuropathy. Clinical manifestations were additionally categorized as astheno-neurotic

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syndrome, encephalopathy syndrome, or peripheral neuropathy syndrome.

Electroneuromyographic assessment. Motor nerve conduction studies were performed bilaterally in the median, ulnar, tibial, and peroneal nerves using standard surface-electrode techniques. The principal electrophysiological parameters were nerve conduction velocity (NCV, m/s) and compound muscle action potential (M-response) amplitude (mV). Bilateral measurements were obtained for each nerve, and averaged bilateral values were used for analysis. Electrophysiological involvement was categorized as axonal, demyelinating, or mixed. ENMG parameters were analyzed according to neuropathy severity stage and hemodialysis status.

Laboratory assessment. Laboratory evaluation included serum creatinine, urea, potassium, ferritin, parathyroid hormone (PTH), and erythrocyte sedimentation rate (ESR). Associations between laboratory parameters and neuropathy severity were evaluated, with particular attention to symptomatic neuropathy (stage 2) and subclinical neuropathy (stage 1).

Statistical analysis. Categorical variables were summarized as numbers and percentages and were assessed using the Pearson chi-square test where applicable. Continuous ENMG variables were summarized as mean values with dispersion estimates and compared across neuropathy severity groups and by hemodialysis status. Receiver operating characteristic (ROC) curve analysis was used to evaluate the discriminative ability of creatinine, urea, potassium, ESR, ferritin, and PTH. Areas under the curve (AUCs), standard errors, 95% confidence intervals, cut-off values, sensitivity, and specificity were reported. All tests were two-sided, and $p<0.05$ was considered statistically significant.

Ethical considerations. The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the institutional ethics committee. Written informed consent was obtained from all participants before inclusion in the study.

Results. Among the 65 patients, 16 (24.6%) were categorized as stage 0, 24 (36.9%) as stage 1, 22 (33.8%) as stage 2, and 3 (4.6%) as stage 3. ENMG findings stratified by predominant clinical syndrome and CKD stage are presented in Table 1.

Table 1.

Distribution of neuropathy stages by clinical syndrome and CKD stage

Neuropathy Stages	Astheno-neurotic Syndrome (n=4)	Encephalopathy Syndrome (n=26)	Peripheral Neuropathy Syndrome (n=35)
CKD Stage I (n=9) $\chi^2=9$; $p>0.05$			
Stage 0	2 (50%)	2 (7.7%)	-
Stage 1	-	-	4 (11.4%)
Stage 2	-	-	1 (2.9%)
CKD Stage II (n=14) $\chi^2=11.2$; $p<0.02$			
Stage 0	2 (50%)	3 (11.5%)	-
Stage 1	-	1 (3.8%)	4 (11.4%)
Stage 2	-	-	4 (11.4%)
CKD Stage III (n=8) $\chi^2=3.7$; $p>0.05$			
Stage 0	-	2 (7.7%)	-
Stage 1	-	4 (15.4%)	1 (2.9%)
Stage 2	-	-	1 (2.9%)
CKD Stage IV (n=8) $\chi^2=3.4$; $p>0.05$			
Stage 0	-	2 (7.7%)	-

Table 1. Continued ...			
Stage 1	-	3 (11.5%)	1 (2.9%)
Stage 2	-	-	2 (5.7%)
CKD Stage V (n=26) χ^2=26; p<0.001			
Stage 0	-	3 (11.5%)	-
Stage 1	-	6 (23.2%)	-
Stage 2	-	-	14 (40%)
Stage 3	-	-	3 (8.5%)

Note: CKD – chronic kidney disease; n – number of patients; χ^2 – Pearson chi-square statistic; p – statistical significance level; "–" – no cases.

Patients presenting with astheno-neurotic syndrome had no ENMG conduction abnormalities. Among patients with encephalopathy syndrome, 12 (46.1%) had preserved nerve conduction, whereas 14 (53.9%) had conduction abnormalities despite the absence of clinical manifestations of peripheral nerve involvement. Among patients with peripheral neuropathy syndrome, 10 (28.6%) had subclinical neuropathy, 22 (62.8%) had symptomatic neuropathy, and 3 (8.6%) had disabling neuropathy. ENMG parameters did not differ significantly between the control group and patients at neuropathy stage 0. In contrast, NCV and M-response amplitude differed significantly between controls and patients at stages 1, 2, and 3 (all p<0.001). Significant differences were also observed between stage 1 and stages 2 and 3 (p<0.001; Table 2).

Table 2.

NCV and M-response values for median and ulnar nerves according to neuropathy stage

	n.medianus		**n.ulnaris**	
	NCV, m/s	M-response, mV	NCV, m/s	M-response, mV
Stage 0 (n=16)	49.13±0.55	5.48±0.05	49.28±0.57	5.4±0.09
Stage 1 (n=24)	45.86±0.34*^	3.65±0.05*^	45.67±0.34*^	3.7±0.05*^
Stage 2 (n=22)	43.55±0.42*^#	3.38±0.04*^#	43.3±0.4*^#	3.37±0.04*^#
Stage 3 (n=3)	37.9±1.57*^#&	1.9±0.06*^#&	35.77±1.4*^#&	1.98±0.09*^#&
Control	50.35±0.48	5.8±0.15	50.5±0.43	5.6±0.14

Note: NCV – nerve conduction velocity; m/s – meters per second; M-response – compound muscle action potential (CMAP) amplitude; mV – millivolts; n – number of patients. Significant differences: *p<0.001 versus control; ^p<0.001 versus stage 0; #p<0.001 versus stage 1; &p<0.001 versus stage 2. Values represent averaged bilateral nerve measurements.

ENMG findings in the upper extremities demonstrated mixed (25; 51.0%) and axonal (24; 49.0%) patterns of nerve damage. Assessment of the tibial and peroneal nerves similarly showed no significant differences in NCV or M-response amplitude between controls and patients at neuropathy stage 0, whereas progressively greater abnormalities were observed at more advanced neuropathy stages (Table 3).

Table 3.

NCV and M-response values for tibial and peroneal nerves according to neuropathy stage

	n.tibialis		**n.peroneus**	
	NCV, m/s	M-response, mV	NCV, m/s	M-response, mV
Stage 0 (n=16)	50.8±0.48	5.54±0.06	50.26±0.29	5.6±0.06
Stage 1 (n=24)	45.4±0.54*	3.92±0.04*	46.05±0.37*	4.2±0.07*
Stage 2 (n=22)	40.7±0.58*^#	3.08±0.07*^#	40.8±0.72*^#	3.05±0.07*^#
Stage 3 (n=3)	36.3±1.45*^#&	1.97±0.09*^#&&	34.7±0.9*^&	2.17±0.18*^#&&
Control	51.03±0.74	5.8±0.14	50.6±0.67	5.8±0.15

Note: NCV – nerve conduction velocity; m/s – meters per second; M-response – compound muscle action potential (CMAP) amplitude; mV – millivolts; n – number of patients. Significant differences: *p<0.001 versus control; ^p<0.001 versus stage 0; #p<0.001 versus stage 1; &p<0.001 and &&p<0.02 versus stage 2. Values represent averaged bilateral nerve measurements.

In the lower extremities, axonal (20; 40.8%) and mixed (29; 59.2%) patterns were observed, with mixed involvement predominating at advanced neuropathy stages. Patients undergoing hemodialysis had significantly lower NCV and M-response amplitudes across all examined nerves (all p<0.001; Table 4).

Table 4.

ENMG parameters according to hemodialysis status

ENMG parameters by nerve	**Not receiving hemodialysis**	**Receiving hemodialysis**	**P**
Tibial nerve			
NCV, m/s	47.3±0.56	40.2±1.08	<0.001
M-response amplitude, mV	4.6±0.14	3.04±0.18	<0.001
Peroneal nerve			
NCV, m/s	47.2±0.5	40.19±1.1	<0.001
M-response amplitude, mV	4.7±0.14	3.15±0.2	<0.001
Median nerve			
NCV, m/s	47.46±0.13	42.2±0.97	<0.001

Table 4. Continued ...			
M-response amplitude, mV	4.6±0.13	2.99±0.17	<0.001
Ulnar nerve			
NCV, m/s	47.4±0.38	41.57±1.15	<0.001
M-response amplitude, mV	4.56±0.13	3.02±0.18	<0.001

Note: ENMG – electroneuromyography; NCV – nerve conduction velocity; m/s – meters per second; M-response – compound muscle action potential (CMAP) amplitude; mV – millivolts; p – statistical significance level.

ROC analysis of laboratory markers for symptomatic neuropathy is presented in Table 5.

Table 5.

ROC analysis of laboratory markers for symptomatic neuropathy

	AUC ± SE	p	95% Confidence Interval	
			Lower	Upper
Creatinine	0.759±0.059	0.001	0.643	0.875
Urea	0.824±0.05	0.001	0.721	0.928
Potassium	0.868±0.04	0.001	0.789	0.947
ESR	0.808±0.066	0.001	0.664	0.908
Ferritin	0.786±0.06	0.05	0.675	0.919
Parathyroid hormone	0.799±0.069	0.013	0.664	0.934

Note: ROC – receiver operating characteristic; AUC – area under the ROC curve; SE – standard error; CI – confidence interval; ESR – erythrocyte sedimentation rate; p – statistical significance level.

Potassium had the largest AUC (0.868), followed by urea (0.824) and ESR (0.808). The corresponding cut-off values, sensitivity, and specificity are shown in Table 6.

Table 6.

Diagnostic performance of laboratory markers for symptomatic neuropathy

Laboratory Parameters	Cut-off Point	Sensitivity	Specificity
Creatinine	95 μmol/L	86.4%	56.5%
Urea	9.2 mmol/L	86.4%	61.3%
Potassium	5.08 mmol/L	95%	78.3%
ESR	32 mm/hour	77.8%	72.5%
Ferritin	267.02	90%	78.6%
Parathyroid hormone	330.55 pg/mL	71.4%	86.5%

Note: ESR – erythrocyte sedimentation rate; mmol/L – millimoles per liter; μmol/L – micromoles per liter; mm/hour – millimeters per hour; pg/mL – picograms per milliliter.

For symptomatic neuropathy, serum potassium showed the strongest discriminative performance. At a cut-off of 5.08 mmol/L, sensitivity was 95.0% and specificity was 78.3%. For subclinical neuropathy (stage 1), PTH was the only laboratory marker with significant discriminative performance: AUC 0.898±0.05, cut-off 146.05 pg/mL, sensitivity 88.9%, specificity 86.5%, $p<0.001$.

Discussion. Electrophysiological evidence of peripheral neuropathy was present in 75.4% of the CKD cohort, including a substantial proportion of patients with subclinical disease. Uremic neuropathy is typically described as a distal, symmetric, length-dependent polyneuropathy with predominantly axonal involvement, and its frequency increases as renal function declines [11]. In a hemodialysis cohort, Bakre et al. identified neuropathy electrophysiologically in 65% of patients, with axonal and mixed sensorimotor patterns predominating [12]. These observations are broadly consistent with the axonal and mixed patterns found in the present study.

The lower NCV and M-response amplitudes observed in patients undergoing hemodialysis should be interpreted cautiously. Because dialysis status is closely linked to advanced CKD, the cross-sectional comparison cannot establish that hemodialysis itself causes the electrophysiological differences. Experimental and clinical neurophysiological studies have shown that axonal membrane depolarization in advanced kidney disease correlates strongly with serum potassium [13], and a potassium-clamp study demonstrated a causal relationship between hyperkalemia and axonal dysfunction in end-stage kidney disease [14]. In addition, a randomized trial in stages 3–4 CKD suggested that sustained potassium restriction may help preserve nerve function [15]. Therefore, the relatively high AUC for potassium in the present cohort is biologically plausible; however, it should be regarded as a discriminative association rather than a validated prognostic predictor.

The association between PTH and subclinical neuropathy also warrants cautious interpretation. Earlier clinical work demonstrated an inverse relationship between elevated PTH levels and motor nerve conduction velocity in uremic patients [16]. Conversely, a more recent study by Bakre et al. found no significant correlation of potassium or PTH with electrophysiological neuropathy indices [12]. The high AUC observed for PTH in stage 1 neuropathy in our cohort therefore represents a potentially relevant finding that requires confirmation in independent samples.

Although axonal involvement is considered characteristic of uremic neuropathy, mixed axonal–demyelinating electrophysiological patterns are not specific to CKD and may also occur in other acquired polyneuropathies, including variants of chronic inflammatory demyelinating polyneuropathy [17].

Limitations. This study has several limitations. First, the overall sample size was modest, and the number of patients in some neuropathy severity subgroups was small, particularly in stage 3 (n=3), which limits the precision of subgroup comparisons. Second, the cross-sectional design precludes causal and prognostic inferences regarding the relationships between CKD, electrophysiological abnormalities, and laboratory markers. Third, the ROC-derived cut-off values for potassium and PTH were developed and evaluated in the same cohort and were not externally validated; therefore, their diagnostic performance may be overestimated. In addition, comparisons according to hemodialysis status may be confounded by CKD stage because dialysis is closely associated with advanced renal disease. The size and demographic characteristics of the healthy control group, as well as the numbers of participants in the hemodialysis and non-hemodialysis subgroups, were not reported in the available dataset, limiting independent verification and interpretation of these comparisons. Larger prospective studies with independent validation cohorts are required to confirm the proposed laboratory thresholds and determine their incremental value beyond clinical and electrophysiological assessment.

Conclusion. Uremic neuropathy was common in this CKD cohort and was characterized by progressively greater ENMG abnormalities with increasing neuropathy severity. Axonal and mixed patterns predominated. Serum potassium showed the strongest discrimination for symptomatic neuropathy, whereas PTH showed high discrimination for subclinical neuropathy. These findings support further evaluation of potassium and PTH as candidate markers of neuropathy severity; the proposed thresholds should not be considered validated clinical cut-offs until confirmed in independent prospective cohorts.

Conflict of interest. The authors declare no conflict of interest.

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Author contributions. S.N. Saidkhodjaeva: conceptualization, methodology, supervision,

interpretation of clinical and electrophysiological data, review and editing. Sh.Sh. Kudratkhujaeva: investigation, data collection, data curation, formal analysis, and preparation of the original draft. Both authors reviewed and approved the final version of the manuscript.

Use of artificial intelligence. Generative AI-assisted tools were used for language editing only. No AI tool was used to generate, modify, or analyze the study data. The authors remain fully responsible for the accuracy and integrity of the manuscript.

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XÜLASƏ

UREMİK NEYROPATİYANIN AĞIRLIĞI ÜZRƏ MƏRHƏLƏ-SPESİFİK ELEKTRONEYROMİOQRAFİK XÜSUSİYYƏTLƏR VƏ LABORATOR MARKERLƏR

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Məqsəd. Xroniki böyrək xəstəliyinin (XBX) müxtəlif mərhələlərində periferik neyropatiyanın ağırlığı ilə əlaqəli elektroneyromioqrafik (ENMQ) xüsusiyyətləri və laborator markerləri qiymətləndirmək. **Material və metodlar.** XBX olan 65 pasiyentdə (I mərhələ – 9, II – 14, III – 8, IV – 8, V – 26) ENMQ müayinəsi aparılmış və göstəricilər sağlam nəzarət qrupu ilə müqayisə edilmişdir. Periferik neyropatiya Dyck yanaşmasının adaptasiya olunmuş

variantından istifadə edilməklə 0–3-cü mərhələlər üzrə təsnif edilmişdir. Sinir üzrə impulsun yayılma sürəti və M-cavabın amplitudası orta, dirsək, böyük qamış və kiçik qamış sinirlərində bilateral qiymətləndirilmişdir. ROC (receiver operating characteristic) təhlilinə kreatinin, sidik cövhəri, kalium, eritrositlərin çökmə sürəti (EÇS), ferritin və paratireoid hormon (PTH) daxil edilmişdir. **Nəticələr.** Pasiyentlərin 24,6%-də neyropatiya aşkar edilməmiş (0-cı mərhələ), 36,9%-də subklinik (1-ci mərhələ), 33,8%-də simptomatik (2-ci mərhələ), 4,6%-də isə əlillik yaradan neyropatiya (3-cü mərhələ) müəyyən edilmişdir. Aksonal və qarışıq zədələnmə tipləri üstünlük təşkil etmişdir. Hemodializ alan pasiyentlərdə bütün müayinə olunan sinirlər üzrə impulsun yayılma sürəti və M-cavab amplitudası daha aşağı olmuşdur ($p<0,001$). Kalium simptomatik neyropatiyanın fərqləndirilməsində ən yüksək diskriminativ qabiliyyət göstərmişdir (kəsim nöqtəsi 5,08 mmol/l; həssaslıq 95%; spesifiklik 78,3%; ROC əyrisi altında sahə (AUC) 0,868), PTH isə subklinik neyropatiya üçün ən yüksək göstəriciyə malik olmuşdur (kəsim nöqtəsi 146,05 pg/ml; həssaslıq 88,9%; spesifiklik 86,5%; AUC 0,898). **Yekun.** Neyropatiyanın ağırlığı artdıqca ENMQ dəyişiklikləri də daha ifadəli olmuşdur. Kalium və PTH klinik baxımdan əhəmiyyətli neyropatiya mərhələləri ilə əlaqəli olmuş və potensial laborator markerlər kimi nəzərdən keçirilə bilər; təklif olunan kəsim nöqtələrinin daha geniş prospektiv tədqiqatlarda təsdiqlənməsinə ehtiyac vardır.
Açar sözlər: xroniki böyrək xəstəliyi, uremik neyropatiya, elektroneyromioqrafiya, sinir keçiriciliyinin tədqiqi, biomarkerlər, kalium, paratireoid hormon, hemodializ

РЕЗЮМЕ

СТАДИЙНО-СПЕЦИФИЧЕСКИЕ ЭЛЕКТРОНЕЙРОМИОГРАФИЧЕСКИЕ ПАТТЕРНЫ И ЛАБОРАТОРНЫЕ МАРКЕРЫ ТЯЖЕСТИ УРЕМИЧЕСКОЙ НЕЙРОПАТИИ

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Цель. Оценить электронейромиографические (ЭНМГ) паттерны и лабораторные маркеры, ассоциированные с тяжестью периферической нейропатии на различных стадиях хронической болезни почек (ХБП). **Материал и методы.** ЭНМГ-обследование выполнено у 65 пациентов с ХБП (I стадия – 9, II – 14, III – 8, IV – 8, V – 26) с сопоставлением показателей со здоровым контролем. Периферическая нейропатия классифицировалась на стадии 0–3 с использованием адаптированного подхода Дайка. Скорость проведения по нерву (СПН) и амплитуда М-ответа оценивались билатерально в срединном, локтевом, большеберцовом и малоберцовом нервах. Анализ характеристической кривой (receiver operating characteristic, ROC) включал креатинин, мочевину, калий, скорость оседания эритроцитов (СОЭ), ферритин и паратиреоидный гормон (ПТГ). **Результаты.** У 24,6% пациентов нейропатия отсутствовала (стадия 0), у 36,9% выявлена субклиническая (стадия 1), у 33,8% – симптоматическая (стадия 2), у 4,6% – инвалидизирующая нейропатия (стадия 3). Преобладали аксональные и смешанные паттерны поражения. У пациентов на гемодиализе СПН и амплитуда М-ответа были ниже во всех исследованных нервах ($p<0,001$). Калий имел наибольшую дискриминационную способность для симптоматической нейропатии (порог 5,08 ммоль/л; чувствительность 95%; специфичность 78,3%; площадь под ROC-кривой (area under the curve, AUC) 0,868), а ПТГ – для субклинической нейропатии (порог 146,05 пг/мл; чувствительность 88,9%; специфичность 86,5%; AUC 0,898). **Заключение.** ЭНМГ-изменения нарастали по мере увеличения тяжести нейропатии. Калий и ПТГ были ассоциированы с клинически значимыми стадиями нейропатии и могут рассматриваться как потенциальные лабораторные маркеры; предложенные пороговые значения требуют подтверждения в более крупных проспективных исследованиях.
Ключевые слова: хроническая болезнь почек, уремическая нейропатия, электронейромиография, исследования проводимости нервов, биомаркеры, калий, паратиреоидный гормон, гемодиализ

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