Section: Neurology

Original Research Article

PERIPHERAL NEUROPATHY IN CHRONIC KIDNEY DISEASE: AN ELECTROPHYSIOLOGICAL CORRELATION BY USING NERVE CONDUCTION STUDY FROM A TERTIARY CARE CENTRE

Ilakkiya Venu[1], Arun Raj Ezhumalai[2], Rajasekaran .M[3]

[1]Final Year Resident, Department of Neurology, Kapv Government Medical College & Hospital, Trichy, Tamilnadu, India.
Professor and Head, Department of Neurology, Kapv Government Medical College & Hospital, Trichy, Tamilnadu, India.
Assistant Professor, Department of Neurology, Kapv Government Medical College & Hospital, Trichy, Tamilnadu, India.

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Corresponding Author:
Dr. Arun Raj Ezhumalai,
Email: dr.e.arunraj@gmail.com

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ABSTRACT

Background: Chronic Kidney Disease (CKD) often causes peripheral neuropathy that may be undiagnosed early, leading to sensory and motor deficits that affect daily functioning and quality of life. Early detection through electrophysiological evaluation can guide timely interventions to prevent progression. This study assessed the prevalence, patterns, and electrophysiological changes and correlated findings with CKD stage, dialysis status, and serum creatinine levels. **Materials and Methods:** This prospective observational study was conducted over six months at Mahatma Gandhi Government General Hospital, including 36 CKD patients aged ≥18 years. NCS were performed on the median, ulnar, tibial, peroneal, and sural nerves using the MEDICAID NEUROSTIM NS4 system, recording distal latency, amplitudes, and conduction velocities. **Result:** Abnormal NCS findings were observed in 20 (100%) on dialysis versus 12 (75%) non-dialysis patients (P=0.018). Across CKD stages, abnormalities increased with severity, present in 2 (40%) at stage 3, 12 (85.7%) at stage 4, and all 14 (100%) at stage 5 (P<0.0001). Dialysis patients showed significantly prolonged distal latency in median, ulnar, peroneal, tibial, and sensory nerves (P<0.05), with reduced amplitudes in ulnar motor, peroneal, tibial, and all sensory nerves (P<0.05). Conduction velocities were significantly slower in sensory nerves (P<0.001). Amplitudes negatively correlated with CKD severity, strongest in the sural nerve (ρ = -0.964, P<0.001). Mixed neuropathy predominated (56.3%), followed by axonal (31.3%) and demyelinating (12.5%), with sensory involvement in 62.5%. **Conclusion:** Peripheral neuropathy is highly prevalent in CKD, increasing with disease severity and dialysis dependency. NCS is valuable for early detection and classification of neuropathy patterns.

INTRODUCTION

Chronic Kidney Disease (CKD) is a progressive condition marked by a sustained reduction in the glomerular filtration rate, often accompanied by structural kidney changes or markers of renal damage.[1] As kidney function deteriorates, the accumulation of uraemic toxins leads to systemic complications, among which neurological involvement is particularly notable. Peripheral neuropathy, frequently referred to as uremic neuropathy, develops insidiously in CKD and may remain clinically silent until advanced stages.[2]

Electrophysiological assessment through Nerve Conduction Studies (NCS) has been established as the gold standard for diagnosing peripheral neuropathy. It offers quantitative evaluation of latency, amplitude, and conduction velocity, enabling detection of both axonal and demyelinating changes even in subclinical stages.[3] Prior studies report a high prevalence of electrophysiologically detected neuropathy: for example, nearly 90% of pre-dialysis CKD patients exhibit NCS abnormalities, often before the emergence of clinical symptoms.[4,5]

Empirical evidence indicates that both the prevalence and severity of peripheral neuropathy increase with advancing CKD stage and greater renal impairment. In pre-dialysis populations, peripheral neuropathy increases markedly as renal function declines, with axonal and mixed sensorimotor patterns being the most commonly observed forms.[6] Moreover, CKD patients on maintenance hemodialysis demonstrate

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even higher rates of electrophysiological abnormalities prevalence up to 98% highlighting the correlation between treatment modality, disease severity, and neuropathy burden.[7]

Beyond mere prevalence, kidney function markers, such as serum creatinine and urea levels, have shown significant correlations with neurophysiological parameters. Studies demonstrate that higher serum creatinine levels are associated with decreased compound muscle action potential (CMAP) amplitudes and slowed conduction velocities.[8,9]

Rapid therapeutic interventions, particularly renal transplantation, have the potential to reverse or markedly improve uraemic neuropathy. Sustained clinical and electrophysiological recovery, such as improved motor and sensory nerve conduction velocities, following successful renal transplantation, particularly when neuropathy has not yet reached irreversible stages.[2,10]

Despite the growing body of literature, data from tertiary care settings in India, particularly those employing standardised NCS protocols to correlate CKD stage, dialysis dependency, and peripheral nerve involvement, remain sparse. There is a clear need for prospective observational studies that explore nerve conduction changes across CKD stages and assess the potential modulatory effects of haemodialysis and renal replacement therapy modalities. Therefore, the present study aimed to evaluate the utility of electrophysiological evaluation via NCS in diagnosing peripheral neuropathy among CKD patients (both conservatively managed and on maintenance haemodialysis), correlate neuropathy severity with serum creatinine levels and CKD stage, and assess the effect of haemodialysis on peripheral nerve dysfunction among these patients.

MATERIALS AND METHODS

This prospective observational study was conducted over six months at the Department of Neurology in collaboration with the Department of Nephrology at Mahatma Gandhi Government General Hospital. A total of 36 patients who fulfilled the diagnostic criteria for CKD were enrolled. The Institutional Ethics Committee approved the study protocol, and written informed consent was obtained from all the patients.

Inclusion Criteria

Patients aged ≥ 18 years diagnosed with CKD based on abnormal serum urea and creatinine levels and confirmed with ultrasonography (USG) findings were included. Only patients willing to provide informed consent and undergo NCS were considered for eligibility.

Exclusion Criteria

Patients with other known causes of peripheral polyneuropathy, including diabetes mellitus, autoimmune diseases such as systemic lupus erythematosus (SLE), connective tissue disorders such as vasculitis, malignancy, inherited peripheral neuropathies, chronic alcoholism, and those unwilling to participate were excluded from the study.

Methods

All included patients underwent a detailed clinical evaluation, including demographic data, duration of CKD, and treatment. After obtaining consent, NCS was performed using the MEDICAID NEUROSTIM NS4 machine. Motor and sensory conduction studies were performed on the upper and lower limbs. For each selected nerve, the distal latency, CMAP amplitude, sensory nerve action potential (SNAP) amplitude, and conduction velocity were measured. Motor conduction included the median, ulnar, tibial, and peroneal nerves, whereas sensory conduction included the median, ulnar, and sural nerves. Based on these findings, patients were classified into normal, axonal, demyelinating, or mixed sensorimotor neuropathy patterns using established electrophysiological criteria.

Statistical Analysis

Data were recorded in Excel and analysed using SPSS v23. Continuous variables were expressed as mean ± SD, and categorical variables as frequency and percentage, with chi-square and t-tests applied where appropriate. Pearson's correlation was used to assess the relationships between NCS parameters, serum creatinine, and CKD stages. Statistical significance was set at $P < 0.05$.

RESULTS

In patients undergoing dialysis, distal latency was significantly higher in the median ($P = 0.001$), ulnar ($P = 0.002$), peroneal ($P = 0.001$), tibial ($P < 0.001$), median sensory ($P < 0.001$), ulnar sensory ($P < 0.001$), and sural ($P < 0.001$) nerves than in non-dialysis patients. The amplitudes were significantly reduced in the ulnar motor ($P = 0.02$), peroneal ($P < 0.001$), tibial ($P < 0.001$), median sensory ($P < 0.001$), ulnar sensory ($P < 0.001$), and sural ($P < 0.001$) nerves. Conduction velocities were also significantly slower in the median sensory, ulnar sensory, and sural nerves (all $P < 0.001$), whereas other motor nerve velocities showed no significant difference ($P > 0.05$). [Table 1]

Table 1: Comparison of NCS parameters between dialysis and non-dialysis patients

Parameter	Dialysis		P-value
	Yes	No	
Age	58.00 ± 2.97	54.50 ± 4.35	0.002
Amp median motor	3.07 ± 0.99	3.54 ± 0.60	0.178
DL median motor	4.66 ± 0.58	4.03 ± 0.07	0.001
CV median motor	45.30 ± 5.94	48.00 ± 3.18	0.262

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Amp ulnar motor	4.24 ± 1.62	5.45 ± 0.87	0.02
DL ulnar motor	3.25 ± 0.24	3.03 ± 0.05	0.002
CV ulnar motor	46.70 ± 5.06	50.13 ± 2.22	0.062
Amp peroneal	0.95 ± 0.55	1.86 ± 0.57	<0.001
DL peroneal	6.70 ± 0.45	5.96 ± 0.39	0.001
CV Peroneal	39.60 ± 0.99	41.31 ± 3.55	0.987
Amp tibial	1.58 ± 0.53	2.68 ± 0.91	<0.001
DL tibial	5.07 ± 0.09	5.76 ± 0.28	<0.001
CV tibial	38.10 ± 1.21	36.00 ± 6.47	0.539
Amp median sensory	8.02 ± 5.68	16.94 ± 3.91	<0.001
DL median sensory	3.55 ± 0.26	3.08 ± 0.09	<0.001
CV median sensory	44.35 ± 4.31	49.31 ± 1.99	<0.001
Amp ulnar sensory	7.03 ± 4.85	13.03 ± 3.60	<0.001
DL ulnar sensory	3.56 ± 0.49	2.93 ± 0.16	<0.001
CV ulnar sensory	43.90 ± 3.58	50.63 ± 0.81	<0.001
Amp sural	1.50 ± 0.56	4.53 ± 0.91	<0.001
DL sural	4.37 ± 0.78	3.43 ± 0.49	<0.001
CV sural	33.20 ± 2.61	39.69 ± 1.30	<0.001

Footnotes: Data are presented as mean ± standard deviation (SD). Amp: Amplitude; DL: Distal Latency; CV: Conduction Velocity. Statistical comparisons between dialysis and non-dialysis groups were performed using the independent samples t-test. $P < 0.05$ was considered significant.

The negative correlation was observed in sural amplitude ($\rho = -0.964$, $P < 0.001$), followed by median sensory ($\rho = -0.883$, $P < 0.001$), tibial ($\rho = -0.880$, $P < 0.001$), peroneal ($\rho = -0.880$, $P < 0.001$), ulnar sensory ($\rho = -0.875$, $P < 0.001$), ulnar motor ($\rho = -0.659$, $P < 0.001$), and median motor ($\rho = -0.572$, $P < 0.001$) amplitudes. [Table 2]

Table 2: Correlation between NCS amplitude parameters and CKD severity

Parameter	Correlation coefficient (Spearman's ρ)	P-value
Amp median motor	-0.572	<0.001
Amp ulnar motor	-0.659	<0.001
Amp peroneal	-0.880	<0.001
Amp tibial	-0.880	<0.001
Amp median sensory	-0.883	<0.001
Amp ulnar sensory	-0.875	<0.001
Amp sural	-0.964	<0.001

Footnotes: HR (heart rate), RR (respiratory rate). Data were presented in mean and standard deviation. The chi-square test was used for comparison, and a p-value < 0.05 was considered significant.

The mean SBP was similar in both groups, but it decreased from pre-induction to 5 min post-induction (125.7 ± 8.9 and 123.8 ± 7.3 vs. 110.5 ± 8.3 and 111.1 ± 8.5, $p > 0.05$). The mean DBP was also comparable between groups; there was a reduction in values from pre-induction to 5 min post-induction (81.8 ± 7.1 and 79.6 ± 7.5 vs.76.4 ± 6.9 and 74.6 ± 7.9, $p > 0.05$). [Table 3]

Table 3: Comparison of NCS findings by dialysis status and CKD stage

Variable	Category	NCS findings		P-value
		Normal	Abnormal	
Dialysis	Yes	0 (0.0%)	20 (100.0%)	0.018
	No	4 (25.0%)	12 (75.0%)	
CKD Stage	3	6 (60.0%)	2 (40.0%)	<0.0001
	4	2 (14.3%)	12 (85.7%)	
	5	0 (0.0%)	14 (100.0%)	

Footnotes: Data were presented as frequency and percentage (%). NCS: Nerve Conduction Study; CKD: Chronic Kidney Disease. Statistical comparisons were performed using the Chi-square test or Fisher's exact test as appropriate. $P < 0.05$ was considered significant.

Mixed neuropathy was the most common type observed in 18 (56.3%), followed by axonal neuropathy in 10 (31.3%) and demyelinating neuropathy in 4 (12.5%). Abnormal lower limb NCS findings were present in 17 (53.1%), while 15 (46.9%) showed normal results. Sensory involvement was noted in 20 (62.5%), whereas 12 (37.5%) had no sensory deficits. [Table 4]

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Table 4: Distribution of neuropathy type, lower limb NCS findings, and sensory involvement

Parameter	Category	N (%)
Neuropathy type	Demyelinating	4 (12.5%)
	Axonal	10 (31.3%)
	Mixed	18 (56.3%)
Lower limb NCS findings	Abnormal	17 (53.1%)
	Normal	15 (46.9%)
Sensory involvement	Yes	20 (62.5%)
	No	12 (37.5%)

Footnotes: Data were presented as frequency and percentage (%). NCS: Nerve Conduction Study.

DISCUSSION

In our study, patients undergoing dialysis showed prolonged distal latencies, reduced amplitudes, and slower sensory conduction velocities than non-dialysis individuals, whereas motor conduction velocities remained similar. Similarly, Fatima et al. reported that patients undergoing dialysis had higher latencies, lower amplitudes, and slower conduction velocities than those not undergoing dialysis. Median motor latency was 3.57 ± 0.85 ms vs. 3.44 ± 0.71 ms, amplitudes 6.47 ± 1.99 vs. 6.78 ± 2.06, and conduction velocities 50.75 ± 5.10 vs. 53.92 ± 4.42, with similar findings in ulnar, peroneal, tibial, median sensory, and sural nerves.9 Gondhali et al. reported more pronounced nerve conduction abnormalities in dialysis patients than pre-hemodialysis patients, including reduced median amplitude (5.66 ± 1.47 vs. 6.53 ± 1.72, $P < 0.05$), slower common peroneal (38.60 ± 8.84 vs. 43.32 ± 7.97, $P < 0.05$) and posterior tibial conduction (37.41 ± 8.78 vs. 41.01 ± 7.44, $P < 0.05$), and increased distal latency in posterior tibial (4.68 ± 1.52 vs. 4.16 ± 0.92, $P < 0.05$) and sural nerves (3.73 ± 1.70 vs. 2.89 ± 1.31, $P < 0.05$).[11]

Khadir et al. reported contrast findings, showing higher SNAP amplitudes in dialysis patients: median nerve 19.45 ± 5.19 µV vs. 14.53 ± 5.99 µV, ulnar 14.60 ± 6.75 µV vs. 10.87 ± 2.41 µV, and sural 10.18 ± 3.35 µV vs. 7.61 ± 3.51 µV, while distal latencies were slightly prolonged in pre-dialysis patients and conduction velocities were comparable ($P > 0.05$).[12] Similarly, Shikur et al. studied 90 CKD patients (60 pre-HD, 30 HD) and found neuropathy in 80% of HD vs. 55% of pre-HD patients ($P = 0.02$), with sural (60%), ulnar sensory (55.55%), median sensory (50%), and common peroneal (43.33%) being most affected.[13] Likewise, Azad et al. reported high neuropathy prevalence among 50 PD patients: 62% clinically and 80% electrophysiologically, with 100% in diabetics and 60% in non-diabetics.[14] Overall, dialysis patients show frequent neuropathic changes with prolonged latencies, reduced amplitudes, and slowed conduction, though some studies report variations in sensory responses.

In our study, a strong negative correlation was observed between CKD severity and nerve amplitudes, indicating that nerve amplitudes decreased with increasing CKD severity. Similarly, Gondhali et al. demonstrated that worsening renal dysfunction was associated with progressive declines in nerve function, with the highest abnormalities in the sural, ulnar sensory, and median nerves. They reported reduced median motor amplitudes in dialysis patients (5.66 ± 1.47) compared to pre-hemodialysis patients (6.53 ± 1.72, $P < 0.05$), along with notable slowing of conduction velocity in the common peroneal and posterior tibial nerves.[11]

Similarly, Shikur et al. reported that neuropathy worsened with disease progression, affecting 80% of HD patients versus 55% of pre-HD patients, with sural (60%), ulnar sensory (55.55%), and median sensory (50%) nerves being the most involved. Longer CKD duration (>5 years) accounted for 40.35% of cases, showing a progressive relationship between disease duration and nerve dysfunction.[13] Azad et al. reported that the prevalence and severity of neuropathy were higher among diabetic PD patients (100%) than among non-diabetic PD patients (60%). Electrophysiological abnormalities were more pronounced in patients with diabetes, with markedly reduced amplitudes in the common peroneal (0.4 ± 1.1 mV), posterior tibial (1.1 ± 1.7 mV), sural (0.5 ± 1.1 mV), ulnar sensory (3.0 ± 1.9 mV), and median sensory nerves (4.4 ± 5.2 mV).[14] Our findings and previous reports highlight that CKD advances, nerve amplitudes decline, neuropathy becomes more frequent, and patients experience the most severe impairments.

In our study, abnormal lower limb NCS findings were more frequent in patients undergoing dialysis than in those not undergoing dialysis, and the occurrence of abnormalities increased progressively with advancing CKD stages. For example, Jasti et al. studied 200 patients with CKD on maintenance haemodialysis (100 diabetic, 100 non-diabetic) and reported a 98% prevalence of peripheral neuropathy by electrophysiology, while clinically it was 45%. Neuropathy prevalence was 100% in diabetic and 96% in non-diabetic patients, with sural (88%), ulnar sensory (70%), and median sensory (64%) nerves most affected.[15]

Similarly, Jasti et al.'s study reported that the most common pattern was mixed sensorimotor axonal (42%), followed by pure axonal sensorimotor (28%). Lower limbs are more commonly affected (length-dependent), and sensory nerves are more often involved than motor nerves.[15] Gondhali et al. reported 80% of dialysis patients had neuropathy compared to 55% of pre-hemodialysis patients ($P < 0.05$), with an overall prevalence of 63.33% (57/90), lower than our dialysis group but consistent with the

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finding that dialysis patients are more frequently affected.[11]

Shikur et al. found 80% of dialysis patients had peripheral neuropathy versus 55% of pre-HD patients (P = 0.02), with most showing mixed sensorimotor neuropathy (82.46%), a smaller proportion with pure sensory involvement (17.54%), and no pure motor cases.13 Similarly, Aggarwal et al. studied 100 CKD patients and found that peripheral neuropathy prevalence increased with serum creatinine: 35% (2–3.4 mg/dL), 89.19% (3.5–4.9 mg/dL), and 100% (≥5 mg/dL) (P < 0.0001). Sensory-motor neuropathy, mainly affecting peroneal and tibial nerves, was most common, with NCVs decreasing as creatinine and T-NSS scores increased.4 Therefore, these findings highlight that dialysis patients are at substantially higher risk of developing peripheral neuropathy, with abnormalities increasing in frequency and severity as CKD progresses.

Limitations

This study was limited by its single-centre design and small sample size, which may affect the generalisability of the findings. Additionally, the short study duration restricted the long-term assessment of neuropathy progression.

CONCLUSION

Peripheral neuropathy is highly prevalent among patients with CKD, with mixed sensorimotor neuropathy being the most common pattern, followed by axonal and demyelinating neuropathy. Abnormal NCS findings were observed in most patients and were strongly associated with advancing CKD stage and dialysis dependency. Sensory involvement was frequent, and there was a significant reduction in nerve amplitudes and conduction parameters that correlated with disease severity. Future studies should focus on early interventions to prevent or slow the progression of neuropathy in patients with CKD.

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