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Original Research Article

THE SEVERITY OF NEUROPATHY IN PATIENTS OF CHRONIC KIDNEY DISEASE IN VARIOUS STAGES

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ABSTRACT

Background: Peripheral neuropathy is a common yet under-recognized complication of chronic kidney disease (CKD), particularly in patients with coexisting diabetes mellitus. This study aimed to evaluate the severity of peripheral neuropathy in CKD patients across different stages and to determine the influence of diabetes mellitus on neuropathy severity using nerve conduction studies (NCS). **Materials and Methods:** A cross-sectional study was conducted among 96 CKD patients attending the Departments of General Medicine and Neurology at Government Villupuram Medical College and Hospital. Clinical evaluation, laboratory investigations, and nerve conduction studies were performed to assess neuropathy severity. Statistical analysis included Chi-square and ANOVA tests, with $p < 0.05$ considered statistically significant. **Results:** The mean age of participants was 54.3 ± 14.27 years, with 62.5% males and 37.5% females. Diabetes mellitus was present in 65.6% of the study population. Stage 3 CKD was the most common stage (33.3%), followed by Stage 2 and Stage 5 (22.9% each), Stage 4 (15.6%), and Stage 1 (5.2%). Peripheral neuropathy was highly prevalent, with 90.6% of patients showing electrophysiological evidence of neuropathy. Moderate neuropathy was the most common (36.5%), followed by mild (29.2%) and severe neuropathy (25.0%), while only 9.4% had no neuropathy. Diabetic patients showed a significantly higher proportion of moderate and severe neuropathy compared to non-diabetics ($p = 0.0496$). Neuropathy severity increased with CKD progression, with Stage 5 showing the highest proportion of severe neuropathy, and the association between CKD stage and neuropathy severity was highly significant ($p < 0.001$). **Conclusion:** Peripheral neuropathy is highly prevalent among CKD patients and is strongly associated with diabetes mellitus and advancing CKD stage. Routine nerve conduction studies may facilitate early detection and timely management of neuropathy in CKD populations.

INTRODUCTION

Chronic Kidney Disease (CKD) is a progressive, irreversible condition marked by the gradual loss of kidney function over months or years. Globally, CKD poses a growing public health concern, contributing significantly to morbidity, mortality, and healthcare burden. As the disease advances, patients are susceptible to a variety of systemic complications, including cardiovascular events, mineral-bone disorders, and hematologic abnormalities. Among these, neurologic complications such as peripheral neuropathy remain under-recognised but clinically significant.[1]

Peripheral neuropathy, particularly uremic neuropathy, is a common manifestation in patients with advanced CKD, particularly in those undergoing dialysis. It is typically characterised by symmetrical, distal sensorimotor symptoms, including numbness, tingling, burning pain, and weakness, primarily affecting the lower limbs. The underlying pathophysiology involves the retention of uremic toxins, electrolyte imbalances, and oxidative stress, which collectively impair neuronal integrity and conduction. Early diagnosis and appropriate

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interventions can reduce disability and enhance quality of life.[2,3]

Nerve conduction studies (NCS) are the cornerstone for diagnosing and quantifying peripheral neuropathy. They allow for the objective assessment of both sensory and motor nerve fibres, distinguishing between axonal degeneration and demyelination patterns. In CKD, NCS can reveal neuropathic changes even in asymptomatic individuals, aiding in timely clinical decision-making and monitoring of disease progression. Regular electrophysiological screening may thus hold significant value in nephrology practice.[4]

Diabetes mellitus is a well-established cause of both CKD and peripheral neuropathy, and their coexistence often accelerates the onset and severity of nerve damage. Diabetic peripheral neuropathy is one of the most prevalent chronic complications of diabetes, affecting nearly half of long-standing diabetic individuals. When superimposed on CKD, the neurotoxic burden appears to be compounded, with synergistic mechanisms involving hyperglycaemia-induced microvascular damage, inflammation, and mitochondrial dysfunction.[5,6]

The progression of CKD has been positively correlated with increased neuropathic severity. Several studies have reported that patients in later stages of CKD exhibit more profound nerve conduction abnormalities compared to those in early stages. The worsening of renal clearance allows for the accumulation of neurotoxins such as guanidino compounds and middle molecules, which disrupt nerve membrane potentials and metabolic homeostasis, further exacerbating neuronal damage.

Peripheral neuropathy in patients with CKD is a recognised but underdiagnosed complication in the primary care practice. Despite the fact that older age is a well-recognised risk factor for neuropathy in the general population, there is a great deal of controversy over age's role as an independent risk factor for neuropathy in CKD. Age-related neuronal degeneration has been linked to a decline in nerve conduction parameters. On the other hand, research has shown that these nerve conduction variables are not significantly different between older and younger people with CKD, supporting the idea that renal failure is a more significant etiological factor in the development of neuropathy in these populations. It is possible that advancing age may be a risk factor that should be accounted for when making treatment decisions, particularly in cases where nerve conduction findings are borderline.

Peripheral neuropathy in patients with CKD in India and other LMICs may frequently go undetected because a neurophysiological evaluation is not a standard aspect of routine CKD patient care. This study is being done to determine whether nerve conduction studies (NCS) are able to detect neuropathy in CKD patients. The widespread availability of NCS will provide nephrologists with a dependable and non-invasive approach for assessing nerve damage in CKD patients at all stages of disease severity. This will aid in the development of early intervention treatment regimens for a variety of diseases that result in peripheral neuropathy and CKD.

Clinicians will be able to prioritise their high-risk patients for early diagnosis and management based on their understanding of how CKD stage, diabetes status, and the severity of peripheral neuropathy relate to one another. By evaluating nerve conduction parameters in CKD patients with and without diabetes at various stages of renal impairment, the study hopes to fill in some gaps in the body of existing literature. Hence, the study aims to evaluate the severity of peripheral neuropathy in patients with CKD across various stages and to determine the influence of coexisting diabetes mellitus on neuropathic severity.

Objective

To assess the severity of peripheral neuropathy in patients diagnosed with CKD across different stages using nerve conduction studies, to compare the severity of peripheral neuropathy between CKD patients with and without diabetes mellitus, to analyse the association between CKD stage progression and the degree of peripheral neuropathy, and to investigate the demographic and clinical characteristics such as age and sex in relation to neuropathic changes among CKD patients.

MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Departments of General Medicine and Neurology at Government Villupuram Medical College and Hospital (GVMCH), Mundiyambakkam, Tamil Nadu, over a period of one year. Ethical approval for the study was obtained from the Institutional Ethics Committee of Government Villupuram Medical College prior to the commencement of the study, and written informed consent was obtained from all participants before enrollment.

Inclusion Criteria

Patients aged 18 years and above who were diagnosed with CKD with serum creatinine >2 mg/dL, at any stage (Stage 1–Stage 5) according to KDIGO guidelines, with or without coexisting diabetes mellitus and already receiving treatment for either or both conditions, and who provided informed written consent were included in the study.

Exclusion Criteria

Patients with previously diagnosed peripheral neuropathy before the onset of CKD (such as neuropathy due to alcoholism, vitamin B12 deficiency, or hereditary causes), those who had already been initiated on dialysis therapy, patients with known neuromuscular disorders, cervical or lumbar radiculopathies, or stroke affecting nerve conduction parameters, and patients with limb injuries or amputations preventing nerve conduction testing were excluded from the study.

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Methods: 96 Adult patients attending the outpatient departments of General Medicine and Neurology or admitted to the respective wards, who fulfilled the inclusion criteria, were consecutively recruited using a non-probability sampling technique. Based on sample size estimation using the formula for population proportion and considering a 10% non-response rate, a final sample size of 96 patients was included in the study. A detailed clinical history was recorded in a structured proforma, including demographic details such as age, sex, occupation, and educational status; duration and stage of CKD based on serum creatinine and estimated glomerular filtration rate (eGFR); presence and duration of diabetes mellitus and glycemic control status using HbA1c levels; neuropathic symptoms such as numbness, tingling, burning sensation, foot ulcers, muscle cramps, and weakness; and associated comorbid conditions including hypertension, hypothyroidism, alcoholism, smoking, or vitamin B12 deficiency.

All patients underwent a detailed physical examination with particular emphasis on neurological assessment, including evaluation of superficial and deep sensations, muscle strength and tone, deep tendon reflexes (ankle jerk, knee jerk, biceps, and triceps), and examination for foot ulcers or deformities such as Charcot foot. Laboratory investigations included serum urea, serum creatinine, fasting blood sugar, postprandial blood sugar, HbA1c, serum electrolytes (sodium, potassium, and calcium), and estimation of eGFR using the CKD-EPI formula. Additional investigations such as serum vitamin B12 levels and thyroid function tests were performed when necessary to exclude secondary causes of neuropathy.

All enrolled participants underwent nerve conduction studies (NCS) using a digital EMG-NCV machine in the Department of Neurology. Motor nerve conduction studies were performed for the median and ulnar nerves in the upper limbs and the tibial and common peroneal nerves in the lower limbs, while sensory nerve conduction studies were performed for the median and ulnar nerves in the upper limbs and the sural nerve in the lower limbs. Parameters recorded included distal latency, compound muscle action potential (CMAP) or sensory nerve action potential (SNAP) amplitude, nerve conduction velocity, and F-wave latency where applicable. The obtained values were interpreted using standardized reference norms adjusted for age and temperature. Peripheral neuropathy was categorized as mild, moderate, or severe based on the degree of abnormality in latency, amplitude, and conduction velocity, and further classified as axonal, demyelinating, or mixed neuropathy according to the pattern of nerve conduction abnormalities.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS software version 26.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The one-way analysis of variance (ANOVA) was used to compare continuous variables between groups, and the Chi-square test was applied to assess associations between categorical variables. A p-value of less than 0.05 was considered statistically significant.

RESULTS

The mean age of the participants was 54.3 ± 14.27 years. Males constituted 60 (62.5%) of the study population, while 36 (37.5%) were females. Diabetes mellitus was present in 63 (65.6%) patients, whereas 33 (34.4%) did not have diabetes. [Table 1]

Table 1: Baseline Demographic and Clinical Characteristics

Parameters		Values
Mean Age (Years)		54.3 ± 14.27
Sex	Male	60 (62.5%)
	Female	36 (37.5%)
Diabetes Status	Yes	63 (65.6%)
	No	33 (34.4%)

Among the study participants, Stage 3 CKD was the most common (32; 33.3%), followed by Stage 2 and Stage 5 (22; 22.9% each), Stage 4 (15; 15.6%), and Stage 1 (5; 5.2%). Regarding neuropathy severity, moderate neuropathy was most frequent (35; 36.5%), followed by mild (28; 29.2%), severe (24; 25.0%), while 9 (9.4%) patients had no neuropathy. [Table 2]

Table 2: Distribution of CKD Stages and Neuropathy Severity

Parameters		n (%)
CKD Stage	Stage 1	5 (5.2%)
	Stage 2	22 (22.9%)
	Stage 3	32 (33.3%)
	Stage 4	15 (15.6%)
	Stage 5	22 (22.9%)
Neuropathy Severity	None	9 (9.4%)
	Mild	28 (29.2%)
	Moderate	35 (36.5%)
	Severe	24 (25.0%)

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Among patients with diabetes, moderate (25; 26.0%) and severe neuropathy (20; 20.8%) were more common compared to non-diabetic patients, where moderate (10; 10.4%) and severe neuropathy (4; 4.2%) were less frequent. This association between diabetes status and neuropathy severity was statistically significant (p = 0.0496).

Regarding CKD stages, Stage 1 patients predominantly had no neuropathy (4; 4.2%), while Stage 2 and Stage 3 showed increasing mild and moderate neuropathy. Severe neuropathy was most common in Stage 5 (12; 12.5%), indicating worsening neuropathy with disease progression. The association between CKD stage and neuropathy severity was highly significant (p < 0.001). [Table 3]

Table 3: Association of Diabetes Status and CKD Stage with Neuropathy Severity

Parameters		Neuropathy Severity				p- value
		None n (%)	Mild n (%)	Moderate n (%)	Severe n (%)	
Diabetes Status	Yes	4 (4.2%)	14 (14.6%)	25 (26.0%)	20 (20.8%)	0.0496
	No	5 (5.2%)	14 (14.6%)	10 (10.4%)	4 (4.2%)	
CKD Stage	Stage 1	4 (4.2%)	1 (1.0%)	0 (0%)	0 (0%)	< 0.001
	Stage 2	3 (3.1%)	9 (9.4%)	8 (8.3%)	2 (2.1%)	
	Stage 3	1 (1.0%)	10 (10.4%)	14 (14.6%)	7 (7.3%)	
	Stage 4	1 (1.0%)	4 (4.2%)	7 (7.3%)	3 (3.1%)	
	Stage 5	0 (0%)	4 (4.2%)	6 (6.3%)	12 (12.5%)	

The one-way ANOVA analysis showed no statistically significant difference in mean age across the neuropathy severity groups (F = 0.475, p = 0.7). [Table 4]

Table 4: One-Way ANOVA Showing Association Between Age and Neuropathy Severity

Source	Sum of Squares	df	F	p-value
Neuropathy Severity	295.2	3	0.475	0.7
Residual (Within)	19061	92	—	—

DISCUSSION

Peripheral neuropathy is a well-recognised but often underdiagnosed complication in patients with CKD, particularly when associated with metabolic disorders such as diabetes mellitus. In the present cross-sectional study, nerve conduction studies were used to evaluate the prevalence and severity of peripheral neuropathy in CKD patients across different stages and to examine the influence of coexisting diabetes mellitus. The findings demonstrate that neuropathy severity is significantly associated with CKD stage and diabetic status, whereas age did not show a statistically significant association.

The mean age of the study population was 54.3 ± 14.27 years, indicating that most participants were middle-aged to elderly. However, the analysis revealed no statistically significant relationship between age and neuropathy severity (p = 0.7). Although advancing age is often considered a risk factor for peripheral neuropathy due to cumulative metabolic and vascular insults, our findings suggest that in CKD populations, disease-related metabolic disturbances may play a more dominant role than age alone.[1] Similar observations have been reported in large population-based studies, which indicated that metabolic dysregulation and uremic toxicity may contribute more significantly to nerve damage in CKD patients than chronological ageing.[2]

The study population showed a male predominance (62.5%), with females accounting for 37.5% of participants. This finding is consistent with regional epidemiological data from South India, where CKD is reported more frequently among males, possibly due to differences in healthcare-seeking behaviour, occupational exposure, or referral patterns to tertiary care centres.[3] Despite this male predominance, gender did not appear to significantly influence neuropathy severity in the present study, suggesting that neuropathic changes in CKD may be driven primarily by metabolic and renal factors rather than demographic characteristics.

Another important finding of this study was the high prevalence of diabetes mellitus (65.6%) among CKD patients, highlighting the strong interrelationship between these two conditions. Diabetes is widely recognised as both a major cause and a frequent comorbidity of CKD worldwide.[4] In the present study, diabetic patients demonstrated a significantly higher proportion of moderate to severe neuropathy compared to non-diabetic patients (p = 0.0496). This supports the well-established pathophysiological link between chronic hyperglycemia and peripheral nerve damage, often manifested as distal symmetric polyneuropathy due to chronic metabolic and microvascular injury.[5]

Several previous studies corroborate this association. The UK Prospective Diabetes Study (UKPDS) demonstrated that prolonged duration of diabetes and poor glycemic control are strong predictors of both renal and neurological complications.[6] Similarly, research by Jasti et al. reported that diabetic patients with concurrent renal impairment exhibit more pronounced electrophysiological abnormalities and higher neuropathy prevalence compared with non-diabetic CKD patients.[7] Some investigators suggest that uremic toxins alone can induce neuropathy even

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in non-diabetic CKD patients, emphasising the multifactorial nature of neuropathy in renal disease.[8] The progression of CKD was strongly associated with increasing neuropathy severity in this study ($p < 0.001$). Stage 3 CKD was the most frequently observed stage, while Stage 5 patients showed the highest proportion of severe neuropathy, indicating a clear relationship between worsening renal function and peripheral nerve damage. This finding supports the concept of uremic neuropathy, which results from the accumulation of neurotoxic metabolic waste products, electrolyte imbalances, and chronic inflammatory processes that progressively impair nerve function.[9] Previous studies similarly demonstrated that advanced CKD stages are associated with worsening neuropathic symptoms and reduced nerve conduction velocities.[10,11]

The underlying pathophysiology of uremic neuropathy involves multiple mechanisms, including axonal degeneration, impaired mitochondrial energy metabolism, and Schwann cell dysfunction. As renal failure progresses, the accumulation of uremic toxins and metabolic derangements exacerbates neuronal damage, resulting in progressive loss of peripheral nerve fibres and increasing clinical severity.[12] Our findings are consistent with these mechanisms and highlight the importance of early detection and management of CKD to prevent neurological complications.

Despite the documented age-related slowing of nerve conduction in the general population, the present study found no significant age-related differences in neuropathy severity. It is possible that the metabolic and uremic factors associated with CKD overshadow the contribution of age in determining neuropathic changes. However, Wang et al. observed that advanced age, increased body weight, duration of DM, male gender for all ages and HbA1c were the significant predictors of neuropathy severity in diabetic patients with impaired renal function.[13]

In this study, over 90% of patients exhibited some degree of neuropathy, emphasising the substantial burden of peripheral nerve involvement in CKD populations. This highlights the importance of routine NCS for early detection, particularly among diabetic patients and those with advanced CKD stages. Comparable prevalence rates have been reported in previous Indian studies, including research by Ramachandran et al., which documented a high frequency of neuropathy among CKD patients attending tertiary care centres.[14]

The clinical implications of these findings are significant. Incorporating routine neurological evaluation and NCS into CKD management protocols may facilitate earlier detection of neuropathy. Strategies aimed at tight glycemic control, slowing CKD progression through renoprotective therapies such as renin–angiotensin system blockade, and patient education on foot care and physiotherapy may help prevent or delay neuropathic complications and improve overall quality of life.[15]

The present study demonstrates that peripheral neuropathy is highly prevalent among CKD patients and is strongly associated with both diabetes mellitus and advancing CKD stage, while age does not appear to significantly influence neuropathy severity. These findings suggest that neuropathy in CKD is primarily driven by disease-related metabolic and uremic factors rather than demographic characteristics. Early screening and timely intervention may therefore play an important role in reducing the neurological burden in CKD populations. Future longitudinal studies with larger sample sizes are required to further clarify the progression and long-term outcomes of neuropathy in patients with CKD.

Limitations

Being a single-centre study with a relatively small sample size, the findings may not be fully generalizable to a wider population. The cross-sectional study design limits the ability to establish causal relationships or assess the progression of neuropathy over time. Certain potential confounding factors, such as vitamin B12 deficiency, alcohol consumption, and other comorbid conditions, were not comprehensively evaluated for their possible influence on peripheral nerve function. The electrophysiological findings were not correlated with symptom-based scoring systems or quality-of-life assessments, which could have provided a more comprehensive evaluation of the clinical impact of neuropathy.

CONCLUSION

The severity of peripheral neuropathy is significantly correlated with the stage of chronic kidney disease and the presence of diabetes mellitus, according to this study. The risk and severity of neuropathy significantly rise with the progression of CKD, with diabetic CKD patients bearing a disproportionately greater burden. These results highlight the significance of early neuropathy screening and treatment in CKD populations, particularly in diabetics. Although age has historically been thought of as a risk factor for neuropathy, it had no discernible effect on severity in our cohort, indicating that uremic toxicity and metabolic disturbances are the main causes in this clinical setting.

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