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Research Article

An Observational Study of Nerve Conduction Abnormalities in Patients of Chronic Kidney Disease Attending a Tertiary Care Centre

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ABSTRACT
Relevance: Metabolic neuropathy includes a wide spectrum of peripheral nerve disorders associated with systemic diseases of metabolic origin. These diseases include diabetes mellitus, uraemia, hypothyroidism, hepatic failure, hypoglycaemia, polycythaemia, amyloidosis, and porphyria, disorders of lipid/glycolipid metabolism, nutritional /vitamin deficiencies, and mitochondrial disorders. Diabetic mellitus is the most common cause of metabolic neuropathy, followed by uraemia.
Aims & objectives: To study the prevalence and patterns of Peripheral Neuropathy in patients with chronic kidney disease on maintenance Haemodialysis
Materials & Methods: This Study of Nerve Conduction Abnormalities in Metabolic Disorders, a cross sectional study was conducted in Narayana Medical College & Hospital between 1st April 2025 and 31st July 2025. Uremic patients were collected from department of nephrology who have come with neuropathy symptoms.
Results: Out of the 50 patients with CRF polyneuropathy was evident in 39(78%). Of these 21(42%) had clinical symptoms suggestive of polyneuropathy and 10(20%) patients had only objective evidence of neuropathy without symptoms of polyneuropathy and 8 (16%) patients had only electro physiological evidence.
Conclusions: The prevalence of polyneuropathy in CRF patients is Eighty percent out of which 25% were asymptomatic. Axonopathic changes were predominantly seen in affected patients. among nerves involved Sural SNAP and peroneal CMAP amplitudes were more sensitive parameters for detection of PN.

Keywords: Chronic Kidney Disease, Nerve Conduction Studies, Peripheral Neuropathy, Dialysis.

INTRODUCTION

Metabolic neuropathy includes a wide spectrum of peripheral nerve disorders associated with systemic diseases of metabolic origin. These diseases include diabetes mellitus, uraemia, hypothyroidism, hepatic failure, hypoglycaemia, polycythaemia, amyloidosis, and porphyria, disorders of lipid/glycolipid metabolism, nutritional /vitamin deficiencies, and mitochondrial disorders. Diabetic mellitus is the most common cause of metabolic neuropathy, followed by uraemia. [1,2]

Peripheral neuropathy is a chronic and common disease, affecting 2 to 7% of the population, according to estimates from population-based studies in India. [3] Not only is neuropathy a widespread condition, but it is also quite disabling. sensory deficits lead to balance difficulties and frequent falls, with resulting musculoskeletal injuries, including fractures. [4] Neuropathy is also a risk factor for foot ulcerations and lower extremity amputations, all of these manifestations of neuropathy have a profound effect on an individual's quality of life. [5]

Symptoms of polyneuropathy associated with chronic renal failure are restless legs (51%), cramps (65%), weakness (12%), paresthesias (40%), dysesthesia (24%), pain (7%), and burning feet (8%). Other descriptors include painful tingling or electric feelings, unpleasant sensations induced by touching, band like constrictive feelings around the feet and ankles, and sensations of swelling or twisting feelings in the limbs. [6] Signs of polyneuropathy in patients with renal failure include muscle atrophy (13%), weakness (14%), loss of deep tendon reflexes (28%), loss of vibration perception (38%), and hypaesthesia (16%). Sensory loss develops to two-point discrimination, position sensation, and light touch, pain, and temperature sensation. [7]

The development of uremic neuropathy is exceedingly common in CKD, with prevalence of 60-90% in patients undergoing haemodialysis. Several electrophysiological

studies demonstrated that almost 80% of CRF patients had electrophysiological evidence of impaired nerve function half of these patients were symptomatic. [8]

The electrophysiological features of uremic neuropathy include prolonged distal motor latencies, conduction velocity slowing, and declines in the amplitudes of CMAPs and SNAPs. [9] While these abnormalities suggest axon loss, there is additional evidence of demyelination: slowing of conduction velocity that is greater than that expected with large axon loss alone, and proximal CMAP dispersion. [10] In some instances, motor conduction velocity may fall to 50% of the mean control value. While the relationship between the prevalence of nerve conduction abnormalities and clinical signs of polyneuropathy is relatively poor, the relationship between peroneal motor nerve conduction velocity and creatinine clearance is more robust. [11] Sural nerve conduction slowing may be the most sensitive electrophysiological parameter of early polyneuropathy, [12] and there may be slowing of conduction velocity before clinical symptoms and signs of polyneuropathy appear. [13]

The mechanism of nerve dysfunction in renal failure is not established. There are reductions in nerve excitability, loss of nodal sodium permeability, and resistance to ischemic conduction failure. [14] Experimental renal failures in rats is associated with an early (within 48 hours) decline in motor conduction velocity. [15] Identifying the particular components that drive neuropathy is essential in framing future clinical trials. There is no much information on the interactions between the different metabolic disorders and neuropathy. It is possible that a specific combination of components is needed to cause neuropathy or that the effects of the individual components are not additive but synergistic. Much also remains to be learned about the underlying causes and potential treatments of metabolic neuropathy, and by contending that targeting inflammation offers a novel and likely effective treatment strategy. as all metabolic disorders components have currently available treatments. Hopefully, this new knowledge will also help us develop novel therapeutics with the potential to prevent, halt, or reverse this common, disabling disease.

MATERIALS & METHODS

This Study of Nerve Conduction Abnormalities in uremic Disorders, a cross sectional study was conducted in Narayana Medical College & Hospital between 1st April 2025 and 31st July 2025. Uremic patients were collected from department of nephrology outpatient department who have come with neuropathy symptoms.

Inclusion Criteria

Patient with single metabolic factor as a cause of neuropathy were included in the study. Total 50 patients of Chronic Kidney Disease(CKD)were included in this study, CKD was defined as abnormalities of kidney structure or function and presence for more than 3 months with implication for health including albuminuria more than 3 mg/mmol, structural abnormalities were detected through imaging, abnormalities detected through histology and with decreased GFR less than 60mL/min/1.73m2.

Exclusion Criteria

Patients having more than one metabolic factor for neuropathy were excluded from the study. Patients with a family history of inherited neuropathies, occupational or environmental history of heavy metal exposure, history of lumbar or cervical radiculopathy as well as patients using medications which could cause polyneuropathy and HIV were excluded.

Procedure

Ethical committee approval was taken for this study having Ethical committee approval: IEC/NMC/07/03/25-8.Detailed History taken with special emphasis on cause, onset, duration of kidney disease and duration of haemodialysis. Detailed neurological history taken with particular reference to presence of risk factors for polyneuropathy. Each patient was alert, fully oriented, cooperative and responsive during all phases of testing. Complete neurological examination was done with special emphasis on peripheral nervous system examination.

Laboratory investigations:

Electro physiologic evaluation was done by using Nicolet Viking Quest Machine. Motor nerve conduction studies were done on Median, Ulnar, Common Peroneal and Tibial nerves on both sides. The assessed parameters were distal latency (DL), amplitude & duration of the compound muscle action potential (cMAP), conduction velocity (CV), F wave latency. Sensory nerve conduction studies were done on Ulnar, Median and Sural nerves on both sides. The assessed parameters were amplitude of sensory nerve action potential (SNAP) and conduction velocity (CV). For patients who are on haemodialysis electrophysiological studies

were done on the next day of haemodialysis. Data was analysed statistically. Nerve conduction study values are compared with our standard lab values.

Table 1. Depicting Motor Conduction Parameters

Nerve	Distal Latency (ms)	Amplitude (mV)	Segment	Distance (cm)	F Waves (ms)	Conduction Velocity (m/s)
Median	4.2	5	APB	6.5	31	50
Ulnar	3	5	ADM	7	31	50
Peroneal	5.1	5	EDB	9	57	45
Tibial	5	5	AH	10	57	45

Table 2. Depicting Sensory Conduction Parameters

Nerve	Peak latency (ms)	Amplitude(uV)	Distance(cm)
Median	4.2	10	14
Ulnar	3.2	5	14
Sural	4.4	10	14

Criteria followed in this study

Axonal Neuropathy (Electromyography and Neuromuscular Disorders clinical Electrophysiological correlations 3rd editions book by David C Preston, Barbara E Shapiro)

A. Reduced Amplitude
B. Conduction velocities are normal or slightly decreased but never below 75 percent of lower limit of normal
C. Distal latencies are normal or slightly prolonged but never greater than 130 percent of the upper limit of normal

Demyelinating Neuropathy

A. It is associated with marked slowing of the conduction velocity (Slower than 75 percent lower limit of normal) Marked prolongation of distal latency (greater than 130 percent of upper limit of normal) or both

CIDP (Adhoc Subcommittee of American Academy of Neurology AIDS Task Force 1991)

Must meet 3 of the following criteria

1. CV in two or more nerves
2. <80% LLN if amplitude >80% of LLN
3. <70% LLN if amplitude <80of LLN
4. Partial conduction block or abnormal TD ≥1 motor nerves (not over compression sites)
5. Distal latency in >2 nerves
6. Absent F waves or increased minimum latencies ≥2 nerve

Carpal Tunnel Syndrome (AANEM Criteria)

1. Distal Median motor latency > 4.2 ms
2. Difference between Distal motor latency of Median and ulnar nerves >1.1 ms
3. Difference between Distal sensory latency of Median and ulnar nerves>0.2ms
4. Difference between Median and ulnar sensory latencies on stimulating fourth digit and recording from the wrist is at equal distance >0.2ms
5. Palm wrist conduction difference between median and ulnar sensory latencies across 8 cm >0.4 ms
6. Inching technique latency jump> 0.2ms
7. Comparison of lumbrical (Median nerve) and interosseous (ulnar) latencies ≥0.6ms

RESULTS

Table 3. Showing Age and Sex Distribution of the Study Group

Age	Total number of patients-50		
	Males (30)	Females(20)	Total(50)
11-20	08	02	10
21-30	05	06	11
31-40	07	05	12
41-50	04	07	11
51-60	06	0	06

Total 50 patients with chronic kidney disease met the inclusion criteria, out of which 30(60%) were males and 20(40%) were females. Mean age of the patients was 34.5 years ranging from 11yrs to 58yrs.(table 3)

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Table 4. Distribution of Neuropathy in Different Age Groups

Age	No. of Patients with Neuropathy(39)
11-20	07
21-30	09
31-40	10
41-50	08
51-60	05

Table 5. Distribution of Neuropathy among Male and Female Patients

sex	Neuropathy on EDX		Total
	Absent	Present	
Female	7(35%)	13(65%)	20(100%)
Male	4(13.3%)	26(86.7%)	30 (100%)
Total	11(22.0%)	39(78%)	50(100%)

Electrophysiological evidence of neuropathy was found in 13(65%) females and in 26 (86.7%) males. Neuropathy was more common among males than females. (table 5)

Table 6. Neuropathy in Relation to Presence or Absence of Symptoms

Neuropathy	Symptoms		Total
	Present	Absent	
	21(63.8%)	18(36.2%)	39(100%)

Out of the 50 patients with CRF polyneuropathy was evident in 39 (78%). Of these 21(42%) had clinical symptoms suggestive of polyneuropathy and 10(20%) patients had only objective evidence of neuropathy without symptoms of polyneuropathy and 8 (16%) patients had only electrophysiological evidence. (table 6)

Table 7. Motor Conduction in Uremic Poly Neuropathy

Tested nerve	No of Cases (%) with abnormal Electrophysiological findings		
	Prolongation of Distal latency (ms)	Reduced CMAP amplitude(mV)	Reduced Conduction velocity(m/s)
Median	3(6%)	17(34%)	17(34%)
Ulnar	3(6%)	18(36%)	18(36%)
Peroneal	24(48%)	38 (76%)	35(70%)
Tibial	21(42%)	34(68%)	33(66%)

The most common nerve affected being peroneal followed by tibial, ulnar, median in that order. The most common parameters affected were CMAP amplitude and conduction velocity. (table 7)

Table 8. Sensory Conductions in Uremic Poly Neuropathy

Tested nerve	No of Cases (%) with abnormal Electrophysiological findings		
	Prolongation of Distal latency (ms)	Reduced CMAP amplitude(mV)	Reduced Conduction velocity(m/s)
Median	10(20%)	22(44%)	24(48%)
Ulnar	6(12%)	20(40%)	23(46%)
Sural	21(42%)	39 (78%)	37(74%)

The most common nerve affected being sural followed by median and ulnar. The most common parameters affected was SNAP amplitude followed conduction velocity and latency. (table 8)

Late Response Parameters.

F wave parameters of peroneal, tibial nerves were abnormal in 30(60%), and of median nerve in 17(34%), ulnar nerve in 16(32%).Early Diagnostic abnormalities are predominantly seen in sural nerve and peroneal nerves.

Pattern of Neuropathy

In 39 patients with uremic neuropathy 36(92%) have axonal type with reduced CMAP and SNAP amplitudes and remaining 3(8%) have primary axonal changes with secondary demyelination (Reduced SNAP, CMAP and CV, with mild prolongation of Distal latencies)

DISCUSSION

The incidence of uremic neuropathy has recently decreased because of the technical improvements in haemodialysis. However, there are still wide differences in the reported prevalence of polyneuropathy all over the world. Bolton et al. [17] showed that 80 percent of patients have electro physiologic evidence of impaired nerve function, one- half of these patients are symptomatic.

The prevalence of polyneuropathy in the current study was 78 %. This was similar to the study by Buzzi et al., [18] Krishnan et al., [19] Clinically evident neuropathy was present in 62% out of which symptomatic neuropathy in 42% and 20% were asymptomatic with only objective evidence of neuropathy. In 16% of patients, neuropathy was demonstrated only on electrophysiological examination.

The overall percentage of abnormal parameters for each studied nerve was estimated. Motor, sensory and late response parameters were included. The nerves with highest abnormal conductions were the sural (78%) and Peroneal (76%) nerves followed by tibial nerve (68%).The least affected nerves were the median and ulnar (48% and 46% respectively).

The nature of nerve affection in uremic peripheral neuropathy is not well established. It is thought that the primary lesion is axonal degeneration with secondary myelin degeneration. [20] Some early pathological studies suggested that the primary defect was at the level of Schwann cell. [21,22] Other investigators found that segmental demyelination was secondary to primary axonal degeneration. Said et al., [23] reported that chronic renal failure (CRF) may be associated with a variety of neuropathies, including acute axonal neuropathy, progressive axonal neuropathy, progressive axonal neuropathy with secondary segmental demyelination and predominately demyelinating neuropathy.

The current study showed that uremic neuropathy was predominantly axonopathic in type. None of the patients in our study did not meet the electrophysiological criteria for chronic demyelination. Conduction block and temporal dispersion which indicate acquired demyelination did not exist. Electrophysiological evidence of axonopathy was evident by marked decrease in amplitudes of both Compound Muscle Action Potential (CMAP) and Sensory Nerve Action Potentials (SNAP).

The present study revealed that neither the prevalence nor the severity of uremic neuropathy was related to the patient's age, nature of underlying kidney disease or duration of dialysis treatment. These findings were consistent with Mendoza. Guevara et al.,[24]

CONCLUSIONS

Nerve conduction studies are sensitive modalities for detecting neuropathy in uremic patients even if they are asymptomatic. Eighty percent of uremic patients showed evidence of neuropathy out of which 25% were asymptomatic. Axonopathic changes were predominantly seen, Sural Sensory Nerve Action Potentials (SNAP) and peroneal Compound Muscle Action Potential (CMAP) amplitudes were more sensitive parameters for detection of Peripheral Neuropathy.

Limitations of the Study

We had limitations as this was a single-centre study with limited sample size accessing hospital care limiting generalisability of study results.

Financial Support and Sponsorship

Nil.

Conflicts of Interest

There are no conflicts of interest.

Contributions of Authors

Venu Gopal Basam, collected data, interpreted the statistical values. He along with Gopi Srikanth Matta designed the study and contributed in drafting and editing the manuscript. Both the authors wrote final draft of manuscript, which was read and approved by both autho

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