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Electrophysiological evaluation of peripheral neuropathy in chronic kidney disease patients: A study from tertiary care centre, Maharashtra

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ABSTRACT

Introduction: CKD has turned a major cause of morbidity and mortality. The prevalence of peripheral neuropathy is directly proportional to duration and severity of CKD. Peripheral neuropathy becomes evident after the patient reaches stage 4 CKD, but electrophysiological evidences occurs earlier. NCS is an important means of evaluating the functional integrity of peripheral nerves and has implications regarding clinical course and prognosis.

Objectives: To study electrophysiological findings of peripheral neuropathy in CKD patients

Methodology: The present study was a cross sectional; descriptive study was conducted in October 2016 to October 2018. Data analysed by using SPSS 23.0 version. Clinical and neurological examinations were done and blood investigations were performed following which NCS was done. Results: Out of 90 subjects, majority were from 45-54 years age group (26). 70% were male and 30% were females. Total 10 (11.11%) patients showed pure sensory type of PN. Total 47 (52.22%) patients showed sensory-motor type of PN. sensory-motor type of PN was the predominant type (52.22%) found in study followed by pure sensory type of PN (11.11%). Pure axonal sensory motor pattern of PN found in 15 (25%) patients in pre-HD group, 11 (36.66%) patients in HD group. The difference between the pre-HD and HD groups were statistically significant for the median nerve amplitude, common peroneal nerve CV, posterior tibial nerve CV, posterior tibial nerve distal latency and sural nerve distal latency ($p < 0.05$).

Conclusion: Peripheral neuropathy is very common in CKD, more common in dialysis patients as compared to predialysis patients. It's frequency and severity increase as the duration of disease and stage of CKD increases. Sensory motor type of neuropathy is more common than pure sensory type of neuropathy.Pure axonal sensory motor and mixed (axonal + demyelinating) sensory motor neuropathy are common patterns of PN in CKD.

Key words: CKD, peripheral neuropathy, Nerve conduction study

Introduction

Chronic kidney disease (CKD) includes range of pathophysiological processes that are associated with abnormal kidney function. Also, there is gradual reduction in glomerular filtration rate (GFR).[1] CKD has accounted as one of the major causes of morbidity and mortality in the world.Global Burden of Disease Study states that kidney disease was the 12th leading cause of death whereas CKD ranked as the 17th leading cause of morbidity worldwide.[2] Worldwide prevalence of CKD is 13.4% and that of stages of 3 to 5 is 10.6%.[3] In India, the prevalence of CKD is 17.2%. Individual stage of CKD prevalence was 7% (stage 1), 4.3%(stage 2), 4.3%(stage 3), 0.8%(stage 4) and 0.8%(stage 5).[4] Recently estimates in India revealed that the age-adjusted incidence rate of End stage renal disease (ESRD) to be 229 per million population, of which more than 1,00,000 new patients need renal replacement therapy each year.[5]

The Kidney Disease: Improving Global Outcomes (KDIGO) defines CKD as abnormalities of kidney structure or function, present for more than 3 months, with implications for health.[6]Based on GFR, CKD is categorized into 5 stages whereas based onalbuminuria, it is classified into 3 stages. Etiological basis of CKD is diverse and includes diabetic nephropathy, hypertensive nephrosclerosis, glomerulonephritis, chronic interstitial nephritis, obstructive uropathy, renovascular, genetically mediated. In western countries, diabetes and hypertension are accountable for two-third of CKD cases.[7] Diabetes and hypertension are also at epidemic threshold in India.8, 9In India, diabetes and hypertension are responsible for 40-60% cases.

Neuropathy in CKD is distal, symmetrical, mixed sensory motor polyneuropathy. It mainly affects lower limbs greater than upper limbs. Prevalence of peripheral neuropathy is directly proportional to duration and severity of CKD. Peripheral neuropathy becomes evident once the patient attains stage 4 of CKD, but electrophysiological evidences occurs quite earlier. At the initial phase, sensory nerves are involved more than motor. If patient did not receive dialysis

soon after onset of sensory abnormalities, motor involvement follows including musclular weakness. Evidence of peripheral neuropathy without any other cause (e.g. diabetes mellitus) is an indication of renal replacement therapy.[1]

Electrophysiological testing for peripheral nerves can be performed by nerve conduction study (NCS) as well as by electromyography (EMG).[11] Electrodiagnostic studies confirms the site of lesion, assessment of fiber type involvement (motor, large sensory, small fiber: sensory and autonomic), distribution of nerve involvement (distal symmetric, polyradiculo neuropathy, multiple mononeuropathies or mononeuropathy multiplex, upper/lower extremity predominant), identifying the underlying pathophysiologic process (axon loss, demyelination, mixed, channelopathy) and also determining the severity of fiber involvement i.e. mild, moderate, severe involvement along with monitoring recovery or treatment effect. [12]

In Electromyography (EMG) motorlesions of both nerves and muscles can be detected whereas in Nerve conduction study (NCS) only lesions of nerves can be detected of both nerves (motor and sensory). So, NCS plays vital role in evaluating the functional integrity of peripheral nerves and thus it has implications regarding clinical course and prognosis.

Also, nerve conduction study (NCS) when supplemented with meticulous neurological examination would definitely provide invaluable input. Therefore, the present study was conducted for evaluation of peripheral neuropathy, both by clinical and electrophysiological assessment in CKD patients at our tertiary care cenre.

Objectives: To study electrophysiological findings of peripheral neuropathy in CKD patients

Materials and Methods:

The present cross sectional observational study was undertaken to evaluate electrophysiological findings of peripheral neuropathy in CKD patients. The study was conducted in October 2016 to October 2018.

All the patients visiting to our tertiary health care centre in OPD, wards, haemodialysis (HD) centre, during the time frame of study and fulfilling the following study criteria of CKD were included in our study. During the study period our study included total 90 cases of which 60 patients who were receiving conservative management without HD included in pre-HD group and 30 patients who were on HD included in HD group.

Inclusion criteria:

- All the diagnosed CKD patients (as per to KDIGO guidelines) and willing to give voluntarily and informed consent.
- Subjects with serum creatinine more than 2 mg %.
- eGFR < 45 ml/min/1.73m2 (stage G3b, G4, G5 of CKD) which is calculated by MDRD (Modification of Diet in Renal Disease) formula1 as:

  GFR (mL/min/1.73 m2) = 1.86 × (S. Creatinine)-1.154 × (Age)-0.203 × (0.742 if female)
- Abnormalities on renal imaging (e.g. Ultrasound abdomen-kidney size < 9 cm with loss of corticomedullary differentiation.)

Exclusion criteria:

- Patients with preexisting peripheral neuropathy before the diagnosis of CKD or with other recognizable risk factors for peripheral neuropathy were excluded from the study (e.g. Diabetes mellitus, Alcoholism, Drug induced peripheral neuropathy, Hansen's disease)
- Patients with collagen vascular disorders, amyloidosis, or any primary neurologic disorder.
- Patients on peritoneal dialysis and kidney transplant recipients.
- Patients on immunosuppressants and steroids.

Study proforma was filled after a written informed consent. The proforma included socio-demographic details like name, age, sex, address, occupation, detailed history of symptoms, ongoing treatment, general physical and neurological examination, biochemical investigations including blood urea, serum creatinine and serum electrolytes were measured in all the patients as per the standard methods used in the department of biochemistry, radiological investigations and nerve conduction study. For HD group, 2 days after HD cycle, clinical, neurological examinations were done and blood investigations were performed following which NCS was done.

All 90 cases were subjected to the standard protocols of nerve conduction studies (NCS) using NCS machine: Octopus 2 CH – NCS/EMG/EP. The room temperature was kept at 25-28°C. The filters were set at 2-5 kHz for the motor studies and at 20-2kHz for the sensory studies. The sweep speed was set at 5ms/division for the motor studies and at 2 ms/division for the sensory studies. A stimulus duration of 50 ¼s to 1000 ¼s and a current of 0–100 mA is required for an effective nerve stimulation. The supramaximal stimuli were delivered in order to get adequate responses.[13]

NCS procedure was done for both motor conductions and sensory conductions. For motor conductions median nerve, ulnar nerve, common peroneal nerve and posterior tibial nerve were assessed, in which distal latency, conduction velocity, amplitude and F wave were studied. For sensory conductions median nerve, ulnar nerve and sural nerve were assessed in which distal latency, conduction velocity and amplitude were studied. A standardized technique was used to obtain and to record the action potentials for the motor and sensory studies. [14].

Statistical analysis plan:

Data was collected by using a structure proforma. Data entered in MS excel sheet and analysed by using SPSS 23.0 version IBM USA. Qualitative data was expressed in terms of proportions. Quantitative data was expressed in terms of Mean and Standard deviation. Association between two qualitative variables was seen by using Chi square. Comparison of mean and SD between two groups was done by using unpaired t test to assess whether the mean difference between groups is significant or not. Descriptive statistics of each variable was presented in terms of Mean, standard deviation, standard error of mean. A p value of <0.05 was considered as statistically significant whereas a p value <0.001 was considered as highly significant.

Results:

Out of 90 subjects, majority were from 45-54 years age group i.e. 26 followed by 16 each from 25-34- and 35-44-years age group. Thirteen patients were from 55-64 years age group. Least number i.e. 9 were from 65-74 years age group **(Figure 1).**

In our study, 70% were male and 30% were females. We observed male predominance with male to female ratio as 2.33:1. **(Figure 2)**.

In our study there were total 90 CKD patients, of which 60 patients were not on HD and 30 were on HD. Out of 60 pre-HD patients, 33 (55%) showed peripheral neuropathy. Out of 30 HD patients, 24 (80%) showed peripheral neuropathy. Out of total 90 patients, 57 (63.33%) showed peripheral neuropathy. The difference in pre-HD and HD was statistically significant (p<0.05). **(Table 1)**

Pure sensory type of peripheral neuropathy (PN) found in 6 (10%) patients in pre-HD group, 4 (13.33%) patients in HD group. Total 10 (11.11%) patients showed pure sensory type of PN. Pure motor type of PN was not present in any patient. Sensory-motor type of PN found in 27 (45%) patients in pre-HD group, 20 (66.66%) patients in HD group. Total 47 (52.22%) patients showed sensory-motor type of PN. In this study sensory-motor type of PN was the predominant type (52.22%) found in study followed by pure sensory type of PN (11.11%). **(Table 2)**

Pure axonal sensory motor pattern of PN found in 15 (25%) patients in pre-HD group, 11 (36.66%) patients in HD group. Total 26 (28.88%) patients showed pure axonal sensory motor PN. Mixed (axonal + demyelinating) sensory motor pattern of PN found in 12 (20%) patients in pre-HD group, 9 (30%) patients in HD group. Total 21 (23.33%) patients showed mixed sensory motor PN. In this study pure axonal sensory motor neuropathy (28.88%) was most common pattern followed by mixed (axonal + demyelinating) sensory motor (23.33%). **(Table 3)**

For this study each nerve was tested to examine amplitude (amp), conduction velocity (CV) and distal latency (dL) and F wave. The frequency of abnormality of each parameter for individual nerve is shown in**(table 4)**. Most common affected nerves were sural nerve, ulnar sensory nerve, median nerve followed by common peroneal and posterior tibial nerve. The total F wave abnormality in individual nerve as, for median nerve 48 (53.33%), for ulnar nerve 43 (47.77%), for common peroneal nerve 39 (43.33%), for posterior tibial nerve 43 (47.77%).

The mean and standard deviation values for these parameters in pre-HD and HD group are mentioned in the **(table 5).** The difference between the pre-HD and HD groups were statistically significant for the median nerve amplitude, common peroneal nerve CV, posterior tibial nerve CV, posterior tibial nerve distal latency and sural nerve distal latency (p < 0.05).

List of tables and figures

Fig.1: Distribution according to age group

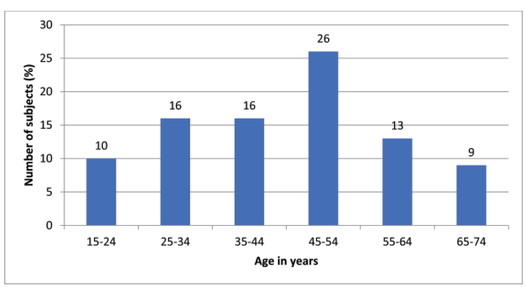

Fig.2: Distribution according to gender

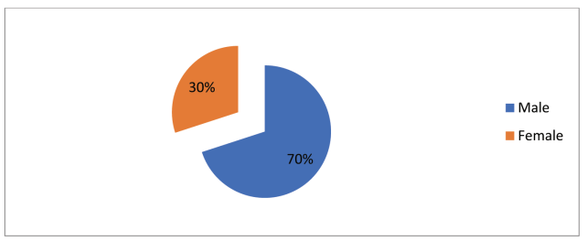

Table 1: Prevalence of peripheral neuropathy in pre hemodialysis and Hemodialysis group

Line of management	Number of patients examined	Patients with peripheral neuropathy	P
Pre-Hemodialysis (pre-HD)	60	33 (55%)	Chi sq-5.38, p-0.02, Significant
On hemodialysis (HD)	30	24 (80%)	
Total	90	57(63.33%)	

Table 2: Type of neuropathy in pre-HD and HD patients

Type of neuropathy	Pre HD	HD	Total
Pure sensory	6(10%)	4(13.33%)	10/90(11.11%)
Pure motor	0	0	0
Sensory-motor	27(45%)	20(66.66%)	47/90(52.22%)
Total	33/60(55%)	24/30(80%)	57/90(63.33%)

Table 3: Pattern of peripheral neuropathy in pre-HD and HD patients

	Pre HD	HD	Total
Pattern of peripheral neuropathy	(n = 60)	(n = 30)	(n = 90)
Pure axonal sensory motor	15 (25%)	11 (36.66%)	26 (28.88%)
Mixed sensory motor (axonal + demyelinating)	12 (20%)	9 (30%)	21 (23.33%)

Table 4: Frequency of nerve conduction abnormalities in CKD patients

Nerve Conduction Parameters	Pre HD (n=60)	HD (n=30)	Total Pts. (n=90)
1) Median Nerve			
Reduced CMAP	18 (30%)	15 (50%)	33 (36.66%)
Reduced MCV	17 (28.33%)	11 (36.66%)	28 (31.11%)
Prolonged mdL	10 (16.66%)	6 (20%)	16 (17.77%)
F wave: Prolonged / Absent	30 (50%)	18 (60%)	48 (53.33%)
2) Ulnar Nerve			
Reduced CMAP	16 (26.66%)	12 (40%)	28 (31.11%)
Reduced MCV	15 (25%)	11 (36.66%)	26 (28.88%)
Prolonged mdL	9 (15%)	9 (30%)	18 (20%)
F wave: Prolonged / Absent	24 (40%)	19 (63.3%)	43 (47.77%)

3) Common Peroneal Nerve			
Reduced CMAP	20 (33.33%)	19 (63.33%)	39 (43.33%)
Reduced MCV	20 (33.33%)	18 (60%)	38 (42.22%)
Prolonged mdL	15 (25%)	9 (30%)	24 (26.66%)
F wave: Prolonged / Absent	22 (36.66%)	17 (56.66%)	39 (43.33%)
4)Posterior Tibial Nerve			
Reduced CMAP	19 (31.66%)	17 (56.66%)	36 (40%)
Reduced MCV	18 (30%)	16 (53.33%)	34 (37.77%)
Prolonged mdL	15 (25%)	10 (33.33%)	25 (27.77%)
F wave: Prolonged / Absent	28 (46.66%)	15 (50%)	43 (47.77%)
5) Median Nerve (sensory)			
Reduced SNAP	28 (46.66%)	17 (56.66%)	45 (50%)
Reduced SCV	26 (43.33%)	17 (56.66%)	43 (47.77%)
Prolonged mdL	15 (25%)	12 (40%)	27 (30%)
6) Ulnar Nerve (sensory)			
Reduced SNAP	30 (50%)	20 (66.66%)	50 (55.55%)
Reduced SCV	28 (46.66%)	19 (63.33%)	47 (52.22%)
Prolonged mdL	18 (30%)	15 (50%)	33 (36.66%)
7) Sural Nerve (sensory)			
Reduced SNAP	31 (51.66%)	23 (76.66%)	54 (60%)
Reduced SCV	32 (53.33%)	20 (66.66%)	52 (57.77%)
Prolonged mdL	27 (45%)	18 (60%)	45 (50%)

Table 5: Comparison of nerve conduction parameters in pre HD and HD patients

		Group	N	Mean	Std. Deviation	T	P	Inference
MOTOR	Median nerve Amp (mV)	Predialysis	60	6.53	1.72	2.367	0.020	Significant
		Dialysis	30	5.66	1.47		(<0.05)	
	Median nerve CV (m/s)	Predialysis	60	49.44	7.91	1.126	0.063	Not Significant
		Dialysis	30	47.38	8.78		(>0.05)	
	Median nerve dL (ms)	Predialysis	60	4.08	0.82	-0.373	0.710	Not Significant
		Dialysis	30	4.16	1.09		(>0.05)	

	Ulnar Nerve Amp (mV)	Predialysis	60	6.55	1.76	1.351	0.180	Not Significant
		Dialysis	30	6.05	1.45		(>0.05)	
	Ulnar Nerve CV (m/s)	Predialysis	60	49.36	7.84	1.072	0.287	Not Significant
		Dialysis	30	47.47	8.01		(>0.05)	
	Ulnar Nerve dL (ms)	Predialysis	60	2.86	0.73	-1.619	0.109	Not Significant
		Dialysis	30	3.12	0.76		(>0.05)	
	Ulnar Nerve dL (ms)	Predialysis	60	2.86	0.73	-1.619	0.109	Not Significant
		Dialysis	30	3.12	0.76		(>0.05)	
	Common Peroneal Nerve Amp (mV)	Predialysis	60	4.71	1.60	1.490	0.140	Not Significant
		Dialysis	30	4.17	1.73		(>0.05)	
	Common Peroneal Nerve CV (m/s)	Predialysis	60	43.32	7.97	2.554	0.012	Significant
		Dialysis	30	38.60	8.84		(<0.05)	
	Common Peroneal Nerve dL (ms)	Predialysis	60	4.11	1.04	-1.428	0.157	Not Significant
		Dialysis	30	4.46	1.20		(>0.05)	
	Posterior Tibial Nerve Amp (mV)	Predialysis	60	5.77	1.93	1.751	0.083	Not Significant
		Dialysis	30	4.98	2.13		(>0.05)	
	Posterior Tibial Nerve CV (m/s)	Predialysis	60	41.01	7.44	2.031	0.045	Significant
		Dialysis	30	37.41	8.78		(<0.05)	
	Posterior Tibial Nerve dL (ms)	Predialysis	60	4.16	0.92	-2.001	0.049	Significant
		Dialysis	30	4.68	1.52		(<0.05)	
SENSORY	Median nerve Amp (μV)	Predialysis	60	10.51	3.39	1.081	0.283	Not Significant
		Dialysis	30	9.68	3.53		(>0.05)	
	Median nerve CV (m/s)	Predialysis	59	45.96	8.86	0.846	0.400	Not Significant
		Dialysis	30	44.23	9.75		(>0.05)	
	Median nerve dL (ms)	Predialysis	60	3.45	0.98	-1.632	0.106	Not Significant
		Dialysis	30	3.81	0.95		(>0.05)	

	Ulnar Nerve Amp (μV)	Predialysis	60	10.32	3.64	1.504	0.136	Not Significant
		Dialysis	30	9.13	3.33		(>0.05)	
	Ulnar Nerve CV (m/s)	Predialysis	60	46.49	9.15	1.521	0.131	Not Significant
		Dialysis	30	43.35	9.43		(>0.05)	
	Ulnar Nerve dL (ms)	Predialysis	60	2.56	1.17	-1.629	0.107	Not Significant
		Dialysis	30	2.97	1.05		(>0.05)	
	Sural Nerve Amp (μV)	Predialysis	60	10.00	3.59	1.718	0.089	Not Significant
		Dialysis	30	8.64	3.50		(>0.05)	
	Sural Nerve CV (m/s)	Predialysis	60	41.79	10.67	0.822	0.413	Not Significant
		Dialysis	30	39.84	10.59		(>0.05)	
	Sural Nerve dL (ms)	Predialysis	60	2.89	1.31	-2.613	0.011	Significant
		Dialysis	30	3.73	1.70		(<0.05)	

Discussion :

In our study the pre-HD group out of 60 there were 39 (65%) males and 21 (35%) females. In the HD group out of 30there were 21 (70%) males and 9 (30%) females. **Sultan LI et al**[15] studied 20 patients in pre-HD group, 10 (50%) males and 10 (50%) females and 20 patients in HD group, 11 (55%) males and 9 (45%) females. **Jasti DB et al**[16]studied 135 (67.5%) males and 65 (32.5%) females. **Deniz et al**[17] studied 23 (60.52%) males and 15 (39.47%) females. **Alagesan et al**[18] studied 71 (63.96%) males and 40 (36.04%) females. **Ogura T et al**[19] studied 31 (44.28%) males and 39 (55.71%) females. **Janda K et al**[20] studied 46 (67.64%) males and 22 (32.35%) females. **Aggarwal HK et al**[21] studied 62% males and 38% females. Sex predilection in our study was almost similar to that of **Jasti DB et al**[16]**, Deniz et al**[17]**,** Alagesan et al18, **Janda K et al**[20]**,** i.e. number of malepatients were more than female patients.

In our study, sensory-motor type of PN was the predominant type present in 47 (52.22%) patients followed by pure sensory type of PN which was present in 10 (11.11%) of total patients. Pure motor type of PN was not present in any patient **(Table no.2)**.**Alagesan et al**[22] study revealed that 111 ckd patients out of which 72 showed PN in which sensory motor neuropathy was seen in 38 i.e. 34.23%, sensory neuropathy was in 18 i.e.16.21% and motor neuropathy was in 16 i.e. 20.51. Deniz et al23observed sensory motor neuropathy in 76%, followed by pure sensory neuropathy in 20% and pure motor neuropathy in 4%. Sensory-motor type of PN remained predominant not only in our study but also in that carried out by Alagesan et al22 and **Deniz et al**[23]. Pure motor neuropathy was absent in our study while it accounted for 4% in the study by **Deniz et al**[23] and 20.51% in **Alagesan et al**[22]**.** In all the studies, sensory-motor was the predominant type of PN followed by sensory type, similar results were found in our study.

In our study, in total 90 patients, pure axonal sensory motor pattern of neuropathy was present in 26 (28.88%) patients which was most common pattern followed by mixed sensory motor present in 21 (23.33%) **(Table no.3). Jasti DB et al**[24] found pure axonal sensory motor neuropathy in 33% and mixed sensory motor neuropathy in 30% patients of predialysis group. In haemodialysis group, 42% patients had mixed sensory motor neuropathy and 18% patients had pure axonal sensory motor neuropathy. **Sultan LI et al**[25]study showed pattern of uremic neuropathy was axonopathic affecting the sensory fibers more than the motor fibers, distal more than proximal portions of peripheral nerves. As shown by these studies axonal sensory-motor is common type followed by mixed sensory-motor neuropathy, similar results were found in our study.

NCS parameters: For comparison the amp (amplitude), CV (conduction velocity) and dL (distal latency) were expressed in mean ± SD in each group.

Comparison of NCS parameters in Pre HD group with other studies:

Our NCS results of pre-HD group were compared with **Jasti DB et al**[24]**, Sultan LI et al**[25]**,Aggarwal HK et al**[26] studies and most of the parameters were showing similar results as shown in table given below **(Table no.6)**.

Comparison of NCS parameters in HD group with other studies:

Our NCS results of HD group were compared with **Jasti DB et al**[24]**, Sultan LI et al**[25]**, Deniz et al**[23] studies and most of the parameters were showing similar results as shown in table given below **(Table no.6).**

Conclusion:

From the study results, we can conclude that peripheral neuropathy is very common in CKD. It is more common in dialysis patients as compared to predialysis patients. It's frequency and severity increase as the duration of disease and stage of CKD increases. Sensory motor type of neuropathy is more common than pure sensory type of neuropathy. Distal symmetrical sensory motor neuropathy is common type of neuropathy, which is more in lower limbs than upper limbs. Pure axonal sensory motor and mixed (axonal and demyelinating) sensory motor neuropathy are common patterns of PN in CKD. Electrophysiological changes occur in early stages of CKD as compared to clinical presentation, so serial monitoring should be done to assess progression of neuropathy. It is advised that newer treatment modalities are required to treat neuropathy in early stages as well as to stop its progression, that will help to improve quality of life in CKD patients.

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