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Research article

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Clinical and electro Physiological pattern of peripheral neuropathy in patients with diabetes

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ABSTRACT

Background

Diabetes mellitus is most prevalent non communicable disease across all social strata in India and is debilitating, with complications that produce severe morbidity and mortality in people. Diabetic neuropathy is common and very severe complication deserving due prompt recognition and care. The study was undertaken to find out positive correlation of peripheral neuropathy severity and duration of diabetes.

Objectives

To evaluate the positive correlation of peripheral neuropathy severity and duration of diabetes

Methodology

A prospective study of diabetic peripheral neuropathy patients admitted in OP and IP of SMCH between January 2019 to February 2019

- ✓ To study the clinical pattern of peripheral neuropathy in diabetic patients
- ✓ To study the CMAP and SNAP amplitudes and latency of peripheral nerves in lower limbs
- ✓ To study the minimum latency of peripheral nerves in lower limbs
- ✓ To correlate the electrophysiological outcome with duration of diabetes

Results

This study shows that the neuropathic involvement increases with age of the subjects. Sensory axonopathy occurs earlier by motor and sensory axonopathy. There is no significant sex predilection for pattern of neuropathy. So in our study, there is obvious involvement of sensory neuropathy in the form of axonopathy with decreased amplitude and NC.

Conclusion

The most common pattern of involvement of neuropathy in our study subjects were sensory or sensorimotor axonal neuropathy. The severity of neuropathy increased with the age of the patient.

INTRODUCTION

Neuropathy is among the most frequent and disabling complication of diabetes mellitus. As

with other major diabetic complications, pathogenesis remains enigmatic and its therapy controversial. The prevalence of neuropathy

appears to correlate with duration and severity of hypoglycaemic in both type-1 and type-2 DM. A pathogenic. A pathogenic role for metabolic alterations of insulin deficiency is well established for diabetic nerve that are consequences of elevated ambient glucose concentration have been established.

They are

1. Increased polyol pathway activity
2. Decreased myo-inositol content
3. Decreased sodium- potassium ATPase action
4. Increased non enzymatic glycosylation of proteins
5. Hexosamine pathway
6. Protein kinase C (PKC) pathway
7. Poly-ADP Ribose polymerase (PARP) pathway
8. Inflammation

Both vascular and metabolic factors have been involved in pathogenesis of diabetic neuropathy. Nerve damage caused by diabetes can also lead to

problems with internal organs like the digestive tract, heart and sexual organs, causing indigestion, or diarrhoea, constipation, dizziness, bladder infections and impotence. Numbness and tingling in feet are often the first sign.

METHODOLOGY

The patient were screened for diabetes based on the American Diabetes association criteria for diabetes mellitus. Then patients under went detailed general examination and neurological examination. All the patients were subjected to nerve conduction study (NCS) of lower limb by selecting two sensory (sural nerve and superficial nerve) and two motor nerves (Tibial nerve and common peroneal nerve) with Allengers Medicare Pvt Ltd Scorpio model for EMG, EP and NCS under the guidance of neurophysiologist. The findings were recorded.

DETAIL OF NCS RECORDING

Nerve	Point of stimulation	Point of recording
Peroneal	Ankle	Extensor digitorum brevis
Tibial	Medial malleolus	Extensor digitorum brevis
Sural	At the junction of middle and lower 1/3rd of the leg	Ankle
Superficial peroneal	Upper edge of the lateral malleolus	Lateral 1/3th of the line connecting the malleoli

All studies were performed with surface electrodes on physio-pac single channel polygraph with NCV, using the standardized technique. The nerves were stimulated using 0.1 ms electrical pulses with an intensity sufficient to elicit maximal amplitude of compound muscle action potential and sensory nerve action potential. Onset latency, conduction velocity, amplitude and F minimal responses were measured. For the F response, 10 stimuli is given at frequency of 1/s. A F wave is defined as an action potential of amplitude $\geq 20\mu V$

The latency to onset of the first deflection from the baseline was marked for each trace and the shortest latency (minimal F – wave latency) was determined. In addition, F- wave persistence (number of stimuli eliciting F-waves) was determined. For each nerve study was conducted on both the sides of subjects would be assessed based on the pattern of involvement of neuropathy, motor sensory axonopathy and sensory axonopathy.

RESULTS

Age

	Motor and sensory axonopathy N = 12	Sensory axonopathy N = 14	Normal NCS N = 4
No of patterns			
Mean	61.2	57.3	48.5
SD	7.2	12.3	9.2

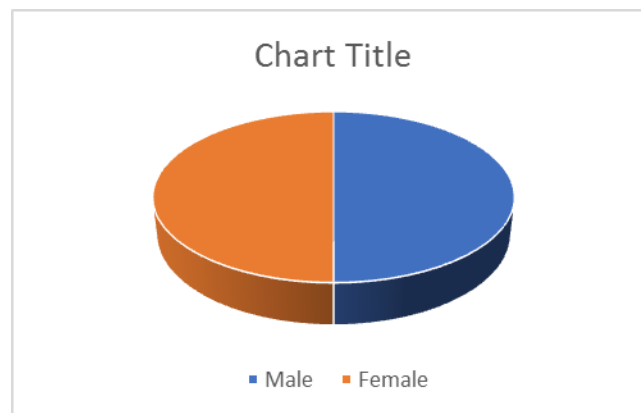
The Mean age of the study group of motor and sensory axonopathy (N=12) was 61.2 ± 7.2 . 65 years, mean age of the study group with sensory

axonopathy (N=14) was 57.3 ± 12.369 years and mean age of the study group with normal NCS (N=4) was 48.5 ± 9.2 . 88years.

Gender

Distribution of motor and sensory axonopathy

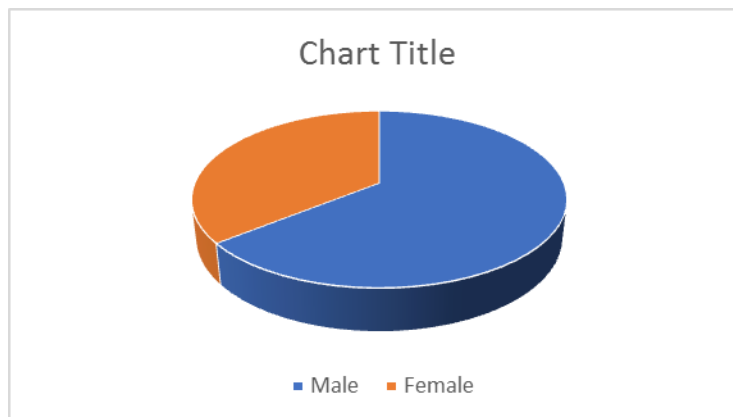
Number of patients in motor and sensory axonopathy		
Gender		Percentage
Male	6	50
Female	6	50
Total	12	100



In the present study group of motor and sensory axonopathy (N=12), 50% were males and 50% were females.

Distribution of sensory axonopathy

Number of patients with sensory axonopathy		
Gender		Percentage
Male	9	64.240
Female	5	35.760
Total	14	100



In the present study group, of sensory axonopathy (N=14) 64.24% were males and 35.76% were females.

Comparison of motor NCS between motor and sensory axonopathy (N=12) and normal (N=4)

Comparison of peroneal nerve with normal NCS subject

Peroneal	Subject	N	Mean	SD	t- value	P- value
D_LL	Normal	4	3.802	.3586	-1.254	.233
	Study	12	4.17	.8203		
P_LL	Normal	4	11.09	.6373	2.159	.045
	Study	12	13.1	3.163		
D_RL	Normal	4	4.505	.5919	1.087	.297
	Study	12	3.941	1.474		
P_RL	Normal	4	11.927	.651	.154	.880
	Study	12	11.743	3.986		
D_AL	Normal	4	5.425	1.703	3.478	.014
	Study	12	1.908	1.887		
P_AL	Normal	4	4.425	1.857	2.853	.041
	Study	12	1.491	1.528		
D_AR	Normal	4	3.900	.7615	3.622	.004
	Study	12	1.683	1.659		
P_AR	Normal	4	2.9000	.5597	3.125	.008
	Study	12	1.3667	1.396		
ncv_1	Normal	4	43.190	1.2153	3.504	.004
	Study	12	35.987	6.801		
ncv_R	Normal	4	42.3750	2.507	2.394	.032
	Study	12	33.798	11.626		
FMIN_L	Normal	4	48.997	5.091	.415	.684
	Study	12	46.796	16.11		
FMIN_R	Normal	4	49.6200	3.127	.769	.457
	Study	12	44.771	21.165		

D_LL: distal left latency; **P_LL:** proximal right latency; **D_RL:** distal right Latency; **P_RL:** proximal right latency; **D_AL:** distal amplitude left

P_AL: proximal amplitude left ,**D_AR:** distal amplitude right ,**P_AR:** proximal amplitude right ,**NCV_L:** nerve conduction velocity left ,**NCV_R:** nerve conduction velocity right ,**FMIN_L:** F

minimal left ,**FMIN_R:** F minimal right shows significant difference in amplitude of peroneal nerves on both sides compared, to diabetic with normal NCS parameter by P value 0.014 the difference is significant for both proximal and distal amplitude. Comparison of Tibial nerves with Normal NCS subject

Tibial	Subject	N	Mean	SD	t- value	P - value
D_LL	Normal	4	4.1425	.656	1.92	.107
	Study	12	4.897	.761		
P_LL	Normal	4	13.4900	1.578	1.97	.083
	Study	12	15.625	2.689		
D_RL	Normal	4	4.582	.5810	.65	.53
	Study	12	4.8342	.8255		
P_RL	Normal	4	13.647	1.892	1.79	.123
	Study	12	15.686	2.129		
D_AL	Normal	4	11.525	5.662	1.64	.17
	Study	12	6.325	5.189		
P_AL	Normal	4	8.3750	5.579	1.32	.25
	Study	12	4.3667	4.248		
D_AR	Normal	4	11.000	3.732		

P_AR	Study	12	6.9750	5.177	1.69	.13
	Normal	4	8.200	1.775		
ncv_1	Study	12	4.983	4.521	2.02	.046
	Normal	4	41.41	.8095		
ncv_R	Study	12	35.1792	5.780	3.69	.003
	Normal	4	41.1475	2.646		
FMIN_L	Study	12	34.1792	4.819	3.65	.005
	Normal	4	50.0025	4.470		
FMIN_R	Study	12	45.6533	15.8189	.86	.408
	Normal	4	53.0650	5.674		
	Study	12	48.4150	15.74	.89	.396

D_LL: distal left latency; **P_LL:** proximal right latency; **D_RL:** distal right Latency; **P_RL:** proximal right latency; **D_AL:** distal amplitude left

P_AL: proximal amplitude Left, **D_AR:** distal amplitude Right, **P_AR:** proximal amplitude right, **NCV_L:** nerve conduction velocity left, **NCV_R:**

nerve conduction velocity right, **FMIN_L:** F minimal left, **FMIN_R:** F minimal right shows no significant difference in latency and amplitude of tibial nerves on both sides between normal NCV and tibial axonopathy subjects.

Comparsion of sensory NCS between Sensory axonopathy {N=12} and normal NCS {N=4}

Comparison of Superficial peroneal nerves with Normal NCS subjects

	Subject	N	Mean	SD	t-value	p-value
SP_LL	Normal	4	4.1700	.6237		
	Study	12	1.4333	1.859	4.58	.001
SP_LR	Normal	4	3.77700	.9351		
	Study	12	.59333	1.589	4.78	.001
SP_AL	Normal	4	10.733	4.456		
	Study	12	1.06	1.867	3.9765	.0234
SP_AR	Normal	4	7.750	2.234		
	Study	12	.3567	.7890	6.567	.006
SPNCV_L	Normal	4	43.08	4.789		
	Study	12	20.11	25.675	2.987	.0123
SPNCV_R	Normal	4	47.98	8.89		
	Study	12	8.168	21.078	5.289	.000

SP_LL: superficial peroneal latency left, **SP_LR:** superficial peroneal, **SP_AL:** superficial peroneal amplitude left, **SP_AR:** superficial

peroneal amplitude right, **SPNCV_L:** superficial peroneal nerve conduction study left, **SPNCV_R:** superficial peroneal nerve conduction study right

Comparison of Sural nerves with Normal NCS subject

	Subject	N	Mean	SD	t-value	p-value
SP_LL	Normal	4	3.5567	.3678		
	Study	12	1.4832	1.8543	3.59	.005
SP_LR	Normal	4	3.1765	.2543		
	Study	12	1.4673	1.847	3.167	.009
SP_AL	Normal	4	13.045	4.945		
	Study	12	1.0876	1.674	4.87	.015
SP_AR	Normal	4	11.987	3.452		

	Study	12	.6000	.9683	6.57	.007
SPNCV_L	Normal	4	46.567	3.753		
	Study	12	16.432	20.564	4.83	.000
SPNCV_R	Normal	4	47.654	5.126		
	Study	12	15.987	19.432	5.18	.000

SP_LL: superficial peroneal latency left, **SP_LR:** superficial peroneal, **SP_AL:** superficial peroneal amplitude left, **SP_AR:** superficial peroneal amplitude right, **SPNCV_L:** superficial peroneal nerve conduction study left, **SPNCV_R:** superficial peroneal nerve conduction study right

DISCUSSION

Nerve conduction study was carried out on patients of Diabetes mellitus in Saveetha Medical Hospital of duration from January 2019 to February 2019

In this Study, the patient's with diabetes were assessed with nerve conduction. Nerve conduction changes associated with diabetic Neuropathy include decrease in amplitude, conduction velocity and prolongation in latency. Axonal loss reflects smaller amplitude changes and slowing of conduction velocity could be the result of a combination of segmental demyelination, loss of fastest conduction axons, and metabolic alterations.

Statistical Analysis was applied to study the difference between motor and sensory nerves parameters within the subgroups. The subgroups were motor and sensory axonopathy, sensory axonopathy and those with normal NCS parameters.

In present study, a significant difference was found between the NCS parameters of study group. A longer latency with smaller amplitude and smaller conduction velocity was found in all the nerves in both motor and sensory NCS of diabetic patients.

AGE The Mean age of the study group with motor and sensory axonopathy (N=12) is 59.08 ± 6.05 years. The Mean age of the study group with sensory axonopathy (N=14) is 56.57 ± 13.69 years. The Mean age of the normal group with diabetes (N=4) is 49.5 ± 9.88 years. So, the neuropathic involvement increases with age of the subjects. Sensory axonopathy occurs earlier by motor and sensory axonopathy.

SEX In the present study group of motor and sensory axonopathy (N=12) 50% (6) were males

and 50% (6) were females. In the present study group of sensory axonopathy (N=14) 57% (8) were males and 42.9% (6) were females. In the present study group of diabetic patients with normal conduction (N=4) 50% (2) were males and 50% (2) were females. There is no significant sex predilection for pattern of neuropathy.

In our study, there is significant difference in peroneal motor amplitude ($P = 0.014$) on both sides compared to normal NCS parameter diabetic subjects. This indicates significant peroneal nerve involvement compared to the tibial nerve.

In our study, there is significant difference in amplitude, latency and NCV of both sural and superficial peroneal amplitude on both sides compared with normal subjects. This indicates significant involvement of both sensory nerves.

In our study, involving comparison of pure sensory axonopathy with normal NCS parameter subjects, there is significant difference in peroneal motor amplitude. This indicates early involvement of peroneal nerves with sensory neuropathy.

So in our study, there is obvious involvement of sensory neuropathy in the form of axonopathy with decreased amplitude and NC.

CONCLUSION

We studied patient with diabetes from January 2019 to February 2019 conducted neurophysiological assessment with nerve conduction studies. They were appropriately analysed statistically.

The most common pattern of involvement of neuropathy in our study subjects were sensory or sensorimotor axonal neuropathy. Though demyelinating neuropathies are reported in various studies, there was no demyelinating pattern in our study.

The severity of neuropathy increased with the age of the patient. Sensory neuropathy occurred at earlier age followed by sensorimotor neuropathy. There was earlier involvement of peroneal motor neuropathy in patients with pure sensory axonal damage. However further studies are required to

assess the significance of duration of diabetes with the pattern of involvement of neuropathy.

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