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Research Article

A PROSPECTIVE STUDY FOR THE EVALUATION OF NERVE CONDUCTION ABNORMALITIES IN NEWLY DIAGNOSED TYPE 2 DIABETIC PATIENTS

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ABSTRACT

Objectives: Peripheral neuropathy is the one of the frequently encountered complication of type 2 diabetes mellitus (T2DM). Although, the prevalence of diabetic peripheral neuropathy is associated with the diabetes duration, in some cases, the state of neuropathy is evident at the time of diagnosis. In this backdrop, the present study was carried out to evaluate the nerve conduction abnormalities in newly diagnosed T2DM.

Methods: This was a prospective study carried out on 30 newly diagnosed T2DM within a time range of 1 month. The patient symptoms such as weakness, burning and tingling senzation, hyperesthesia, and foot ulcer and gait abnormalities were recorded. Nerve conduction analysis of upper limb and lower limb of non-dominant hand side was done using neuro pack S1 machine.

Results: Thirty newly diagnosed T2DM patients were enrolled in the present study. The mean age of the patients was found to be 58.12±15.28 years. Distal motor latencies were elevated in T2DM patients as compared to the controls ($p<0.05$). Further, there was significant prolongation of F-wave latencies in the upper and Lower limbs of the patients as compared to the controls ($p<0.05$). In addition, there was a significant decrease in sensory conduction velocities in T2DM patients as that of the controls ($p<0.05$).

Conclusion: The study concludes that newly diagnosed T2DM is susceptible to DPN with high incidence rates. Hence, it is essential to perform the nerve conduction studies in newly diagnosed T2DM patients for the early detection and better management and also to prevent the complications.

Keywords: Type 2 diabetes mellitus, Peripheral neuropathy, Newly diagnosed diabetes, Nerve conduction studies.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic condition reflected by dysregulated insulin secretion or its action, along with elevated blood sugar level with a state of hyperglycemia [1]. In recent years, the prevalence of T2DM is increasing in developed as well as in developing countries. Usually, the T2DM is under the stage of undiagnosed and the elapsed time before the diagnosis will be approximately 10 years [2]. Hyperglycemia elicits structural and functional damage to wide range of tissue and organs and, thus, imposes micro- and macrovascular injury and in majority of the cases these compactions are unrecognized. Wide range of reports display that in majority of the newly diagnosed T2DM patients, there is a presence of chronic diabetic complications [3-5].

Diabetic peripheral neuropathy (DPN) is the frequently encountered microvascular diabetic complications and imposes marked morbidity and mortality in diabetic subjects. Mounting studies showed the prevalence of DPN in the range between 8% and 59% [6,7]. For the clinical evaluation of DPN, many scores such as diabetic neuropathy symptom score, diabetic neuropathy examination, and neuropathy disability scores have been used. However, nerve conduction velocity test is a gold standard method for the detection of DPN with reliable diagnostic yield [8]. The nerve conduction studies (NCSs) assessed the following parameters such as the median, ulnar, perineal, and tibial nerves motor and sensory functions by measuring the onset latency, amplitude, and conduction velocity. In addition, during DPN, the motor response as described as F-wave in NCS is due to the activation of antidromic motor neurons mediated by motor axons peripheral stimulation [9]. Earlier reports display that measuring the upper limb sensory conductions are more reliable with good accuracy rate in the evaluation of DPN in newly diagnosed diabetes patients [10]. The previous reports show that motor conduction velocity dysfunction is observed in the diabetics a compared to healthy subjects [11].

Hence, the present study was carried out to evaluate the nerve conduction deficits in newly diagnosed T2DM patients.

METHODS

This was a prospective study conducted during the period from June 2019 to December 2019 among the 30 newly diagnosed T2DM patients attending the General Medicine, Outpatient Department of Government Medical College and ESI Hospital, Coimbatore. The 30 healthy non-diabetic patients were taken as control. The diabetes was diagnosed based on the criteria entitled in American Diabetes Association and the patients were recruited within 1 month after diagnosis after obtaining informed consent.

Inclusion criteria

Newly diagnosed T2DM patients, of both sexes and between 40 and 70 years of age were included in the present study.

Exclusion criteria

Patients with various comorbidities such as hypertension, anemia, and thyroid were excluded from the study.

Patients with current neuropathy disorders as a result of various pathologies and radiculopathy were excluded from the study.

The patients were subjected to complete neurological examination for the clinical evaluation of peripheral neuropathy. In addition, the routine laboratory investigations such as fasting and post prandial blood glucose levels and HbA1C levels were also estimated.

NCS was performed in the median, tibial, sural, and medial plantar nerves on the non-dominant pointer side using a Neuropack S1 (Nihon Kohden, Tokyo, Japan) with the bandpass filter set at 5 Hz–5 kHz. Compound muscle action potential (CMAP) was recorded from a pair of surface cup electrodes positioned over the target muscle (abductor pollicis brevis for the median nerve and abductor hallucis for the tibial nerve) using the belly-tendon system. Square pulse supramaximal electrical stimuli with a duration of 0.5 ms were delivered at the wrist and elbow to the median nerve and at the ankle and popliteal fossa to the tibial nerve. Twenty consecutive F-waves were also recorded in each nerve. Sensory nerve action potential (SNAP) was antidromically recorded from a pair of ring electrodes located over the distal and proximal interphalangeal joints of the index finger for the median nerve, and from a pair of surface cup electrodes placed at the points posterior to the lateral malleolus and 3 cm distal to it for the sural nerve. Square pulse supramaximal electrical stimuli with a duration of 0.2 ms were delivered at the wrist and elbow for the median nerve and at the midcalf, 12 cm proximal to the footage electrode, for the sural nerve. In the medial plantar nerve, compound nerve action potential was orthodromically recorded from a pair of surface cup electrodes placed over the tibial nerve at the ankle, posterior to the medial malleolus. Stimulation was carried out on the sole, placing the anode just lateral to the first metatarsal head, and the cathode 2.5 cm proximal to it. Skin temperature was maintained above 32°C in the upper limbs and above 31°C in the lower limbs.

Statistical analysis

Data were analysed using SPSS v 24. The comparison of NCS variables between cases (newly diagnosed diabetic patients) and controls were evaluated using student t test. $p<0.05$ was considered as statistically significant.

RESULTS

In this study out of 30 newly diagnosed diabetic patients, 20 were male and ten were female. The mean age of the patients was found to be 60.12±15.28 years. The time range between the diagnosis of diabetes and inclusion to the study was 1-30 days with a median of 7 days.

There was a significant elevation of FBS and PPBS in DPN patients as compared to the controls (FBS: 232.80±42.43 vs. 148.26±32.26; p=0.002; and PPBS: 312.12±46.35 vs. 246.12± 30.12; p=0.004). The mean HbA1C level was significantly higher in DPN patients as compared to the controls (12.76±2.43 vs. 5.65±1.65 %; p=0.02).

In newly diagnosed T2DM patients, the NCSs were normal in 10 patients (33.33%) and abnormal in 20 patients (66.67%).

In the present study, distal motor latencies of the median and the common peroneal nerves were significantly ($p<0.05$) higher in diabetic patients as compared to the controls (Table 1).

Further, CAMP amplitude of the common peroneal nerve was reduced in diabetics as that of the controls and it was significant (p=0.01). Meanwhile, CMAP amplitude in median and ulnar nerves was not significantly changed in diabetics as compared to controls and it was not significant ($p>0.05$) (Table 2).

In the present study, there was a significant ($p<0.05$) reduction in the median, ulnar, and common peroneal nerves motor conduction velocities in diabetic subjects as that of the controls (Table 3).

Table 4 showed the F-wave latencies of median, ulnar, and common peroneal nerves. In our study, the F-wave latencies showed marked prolongation in diabetics as that of the controls.

In our study, there was a significant decrease in sensory conduction velocities and SNAP amplitudes in median, superficial peroneal, and sural nerves of diabetic patient as that of the controls. The results are shown in Table 5.

DISCUSSION

In newly diagnosed diabetic subjects, there is a marked impairment of autonomic nerve function which is evident by symptomatic peripheral neuropathy diagnosed using NCS [12]. The pathology of DPN is mediated by increased vascular resistance and low blood flow. In addition, decreased nerve myoinositol content activation of during polyol is also an important mediator for the development of DPN [13].

We have observed a marked prolongation of distal motor latencies in diabetic patients. In concordance to the present study, the previous study done by Rota *et al.* displayed that distal median motor neuropathy in 42% of newly diagnosed diabetic subjects based on the nerved conduction evaluation [10].

Earlier studies show that reduction in motor conduction velocity and amplitude of SNAP is one of the earlier clinical features of DPN in newly diagnosed diabetic patients. These alterations may lead to sensory latencies prolongation and reduction of sensory velocity and amplitudes of CMAP [9]. In line with the previous reports, we have showed that SNAP amplitude and sensory conduction velocity are

Table 1: Comparison of distal motor latency between the diabetics and controls

Distal motor latency (ms)	Diabetics (n=30)	Control (n=30)	p-value
Median	3.45±0.45	2.76±0.38	0.006
Ulnar	2.83±0.42	2.47±0.27	0.75^{NS}
Common peroneal	4.67±0.75	3.54±0.62	0.03

p<0.05 - Significant; NS: Non-significant

Table 2: Comparison of compound muscle action potential between diabetics and controls

Compound muscle action potential (mV)	Diabetics (n=30)	Control (n=30)	p-value
Median	12.76±4.65	12.12±5.87	0.43^{NS}
Ulnar	9.12±3.76	8.98±2.18	0.72^{NS}
Common peroneal	5.98±2.12	7.43±3.65	0.03

p<0.05 - Significant; NS: Non-significant

Table 3: Comparison of motor conduction velocities between diabetics and controls

Motor conduction velocity (m/s)	Diabetics (n=20)	Control (n=20)	p-value
Median	51.76±6.76	66.12±6.12	0.001
Ulnar	53.34±5.89	68.45±7.65	0.007
Common peroneal	42.12±3.45	58.87±5.28	0.02

p<0.05 - Significant; NS: Non-significant

Table 4: Comparison of F-wave latency between diabetics and controls

F-wave latency (ms)	Diabetics (n=30)	Control (n=30)	p-value
Median	30.12±5.76	23.65±2.96	0.006
Ulnar	29.76±4.28	22.43±2.54	0.002
Common peroneal	53.62±7.92	42.28±5.12	0.005

p<0.05 - Significant; NS: Non-significant

Table 5: Comparison of SNAP amplitude and sensory conduction velocity between diabetics and controls

SNAP amplitude (μV)	Median	Ulnar	Superficial peroneal	Sural
Diabetics (n=30)	12.36±3.76	7.12±2.98	8.36±3.12	12.98±4.12
Control (n=30)	17.54±5.65	13.64±3.21	15.45±5.87	18.65±5.76
p-value	0.004	0.002	0.01	0.04
Sensory conduction velocity (m/s)	**Median**	**Ulnar**	**Superficial peroneal**	**Sural**
Diabetics (n=30)	48.32±12.43	47.98±8.78	44.12±6.76	45.87±7.29
Control (n=30)	58.42±5.12	59.65±11.34	58.41±8.36	55.42±5.65
p-value	0.001	0.03	0.000	0.001

p<0.05 - Significant; NS: Non-significant, SNAP: Sensory nerve action potential

decreased in superficial peroneal and sural nerves. In our study, there is a marked decrease in amplitudes of CMAP in common peroneal which is in line with the previous studies [9].

The excitability of complete motor length unit was measured in terms of F-waves. In the NCS analysis, addition of F-waves latencies increases the sensitivity for the detection of nerve conduction dysfunction in diabetic subjects to 3–40%, respectively [14]. Likewise, in our study, the F-wave latencies were significantly prolonged in diabetics as that of the controls.

CONCLUSION

Nerve conduction dysfunction is one of the prominent clinical features in the newly diagnosed diabetic subjects. Hence, performing a NCS is most important in these subset of diabetic patients for the early detection of DPN and effective treatment.

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AUTHORS' CONTRIBUTIONS

L.K.G., K.V, and V.P.V conception and design of research; L.K.G., and V.P.V. performed experiments; L.K.G., K.V, P.T and V.P.V. analysed data; L.K.G., K.V, P.T and V.P.V. interpreted results of experiments; L.K.G., K.V, P.T and V.P.V. drafted manuscript; L.K.G., K.V, P.T, R.K.T, V.D and V.P.V. edited and revised manuscript; L.K.G., K.V, P.T, R.K.T, V.D and V.P.V. approved final version of manuscript.

CONFLICTS OF INTEREST

Nil.

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