Original Research Article

A study of peripheral neuropathy in cases of type-II diabetes mellitus patients with or without hypothyroidism

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ABSTRACT

Background: Type 2 diabetic patients have a higher prevalence of thyroid disorders, particularly hypothyroidism. Peripheral neuropathy is a common and disabling complication of diabetes mellitus. Peripheral nervous system involvement in hypothyroidism is also a well-documented fact. Nerve conduction studies are generally considered to be the most sensitive and reproducible in the assessment of peripheral neuropathies. This study helped to determine the prevalence of peripheral neuropathy in diabetic hypothyroid patients as well as to compare it in diabetic patients with or without hypothyroidism. It compared the onset latency, amplitude, conduction velocity and F-wave latency of some nerves in type 2 diabetes mellitus patients with or without hypothyroidism.

Methods: With RMS, EMG, EP MARK-II, nerve conduction studies are done in 30 cases (type 2 diabetes mellitus patients with hypothyroidism) and 30 controls (type 2 diabetes mellitus patients of diabetes without hypothyroidism) respectively, attending the Diabetic Clinic and Biochemistry Laboratory of North Bengal Medical College.

Results: Data were treated with Unpaired t-Test. The study reveals that type 2 diabetes mellitus patients with hypothyroidism have higher prevalence of peripheral neuropathy. There is statistically significant (p<0.00) decrease in motor nerve conduction study in both right and left median nerves in diabetic patients with hypothyroidism than in diabetic patients without hypothyroidism.

Conclusions: All diabetic patients should be screened for early detection of hypothyroidism as type 2 diabetic patients with hypothyroidism have higher prevalence of peripheral neuropathy. The nerve conduction study remains the most reliable, accurate, and sensitive method to evaluate peripheral nerve function.

Keywords: Diabetic mellitus, Hypothyroidism, Peripheral neuropathy, Nerve conduction

INTRODUCTION

Diabetes mellitus and thyroid diseases are the most common endocrine disorders encountered in clinical practices. Diabetes and thyroid disorders have been shown to mutually influence each other and association between both conditions have long been reported.¹ There is a higher occurrence of thyroid diseases, particularly hypothyroidism, among people with Type 2 diabetes mellitus. The complications of the diabetes are reported to be more in diabetic patients with thyroid disorder in various studies. The prevalence of thyroid disorder in diabetic population was reported to be 13.4%.² An unrecognised hypothyroidism may adversely affect the metabolic control and hence aggravate the complications of diabetes mellitus. Diabetic peripheral neuropathy (DPN) is a common, disabling, and costly complication of diabetes mellitus.³ The disease course in type 2 diabetes
mellitus is present very early and nerve conduction velocities are reduced before the diagnosis of the disease. The nerve conduction study performed by Singh et al showed the distal motor latency (DML) is the most frequent abnormal parameter in studied nerves of upper limbs, while mean F, and MNCV is the most frequent abnormality in lower limb nerves in diabetic patients and the most frequent abnormal parameter of the sensory nerve conduction study was the onset latency. The Peripheral nervous system involvement in hypothyroidism is a well-documented fact. Asia and Warker performed a cross sectional analytical study in 26 adult patients, recently diagnosed with thyroid dysfunction and found slowing of conduction velocity in 71% patients for median and sural sensory nerves. Distiller et al, randomly surveyed the records of 922 people with Type 2 diabetes (576 men and 342 women) to identify diagnoses of hypothyroidism and showed close association between diabetes mellitus and hypothyroidism. Peripheral nerve abnormalities known to be associated with hypothyroidism are entrapment neuropathy or sensorimotor polyneuropathy. Up to 50% of diabetic peripheral polyneuropathy are asymptomatic, non-diabetic neuropathies may be present in diabetic patients. Patient Wright et al have shown a positive association of diabetic neuropathy with hypothyroidism. Patients with asymptomatic DPN are at risk of insensate injury to foot resulting in foot ulceration and consequently amputation. Nerve conduction studies are generally considered to be the most sensitive, easy, non-invasive, confirmatory and reproducible in the assessment of peripheral neuropathies.

Aims

In this study, we have assessed the degree of impairment of peripheral nerve functions in Type 2 Diabetes Mellitus patients with or without Hypothyroidism. We have compared the latency, amplitude, conduction velocity of Median nerves, tibial nerves and sural nerve in Type 2 Diabetes Mellitus patients with or without Hypothyroidism. Moreover, the frequency of nerve conduction abnormalities in these patients has been calculated.

METHODS

Type of study

The study was case control study.

Study design

It was a cross sectional study.

Target population

After obtaining the written clearance from the Institutional Ethical Committee, data collection was started with prior informed consent of each of the participants. All the participants were interviewed for collection of baseline informations.

Case - Patients suffering from type 2 diabetes mellitus for more than 5 years (fulfilling the criteria for DM by the American Diabetic Association 2015) with hypothyroidism.

Control - Patients suffering from type 2 diabetes mellitus for more than 5 years without hypothyroidism.

Patients less than 18 years of age, and taking drugs, affecting thyroid profile were excluded.

Study population

The study population included thirty cases and thirty controls.

Study settings

The study was conducted in the Department of physiology, North Bengal Medical College, Darjeeling.

Duration

Duration of the study was one year (from April 2016 to March 2017).

Sampling

Blood for FBS, PPBS, HbA1c and thyroid hormones (TSH, FT4) were tested in biochemistry laboratory and nerve conduction study was done in the neurophysiology laboratory.

Methods and tools

Nerve conduction study was done in the neurophysiology laboratory with RMS, EMG, EP MARK-II. Motor nerve conduction was done in Median nerve and Tibial nerve and sensory nerve conduction was done in median and sural nerve.

Skin temperature and room temperature were maintained between 32-34°C and 21-23°C respectively. Skin was adequately prepared by cleaning the skin with spirit and then drying, before application of stimulating and recording electrodes. This ensures good contact between the electrodes and skin and avoid any shock artifacts. The electrodes were also prepared for good conduction and to decrease skin impedance. Soaking of the ground electrodes, sensory recording electrodes were done and felt tips of the stimulating electrodes were soaked in normal saline. Electrode gel was applied over the recording electrodes before being attached to the skin. After all the electrodes are in place, the instrument is set to deliver repetitive stimuli, usually at 1 Hz. The stimulus voltage is initially set to zero, then gradually increased with successive stimuli. Nerve Conduction Studies were done in both upper and lower limbs on both side of the selected nerves.
Table 1: The normal values of these parameters: (normal value and range of parameters).13,14

| Motor nerve      | Onset latency (DML) ms | Amplitude of AP (mv) | MNCV (m/s) | F-Wave Mean Lat.(ms) |
|------------------|------------------------|-----------------------|------------|---------------------|
| Median nerve     | <4 (3.77±0.40)         | >5 (8.10±2.62)        | <50 58.52±3.76 | <31                 |
| Tibial nerve     | <6 3.49±0.34           | >5 (7.0±3.0)          | >40 48.3±4.5  | <61                 |

| Sensory nerve    | Onset latency (ms)     | Amplitude of AP (µV) | SNCV (m/s) |
|------------------|------------------------|----------------------|------------|
| Median nerve     | <3 3.06±0.41           | >10 8.91±4.48        | >52 45.45±9.40 |
| Sural nerve      | <2.75                  | >10 18.0±10.5        | >42 (50.9±5.4) |

Nerve conduction was performed according to the principles described by Mishra et al and Ma.13,14

Parameters of motor nerve conduction study

Onset latency, amplitude of compound muscle action potential, nerve conduction velocity, F-wave.

Parameters of sensory conduction study

Onset latency, amplitude of sensory nerve action potential, nerve conduction velocity.

Normal values of these parameters: (Normal value and range of parameters).13,14

Data analysis

Collected data were analyzed using Statistical package for social sciences (SPSS) software 22 version. The data presented as mean value with standard deviation and evaluated to determine whether any difference exists between cases and controls. Unpaired t- Test was used to draw inferences. P value of less than 0.05 was considered statistically significant at 5% precision and 95% confidence interval (CI).

RESULTS

Data collected from all 30 type 2 diabetes mellitus (T2DM) patients with hypothyroidism as well as 30 type 2 diabetic patients without hypothyroidism (comparison group) were analysed without any disintegrity in the data set.

Analysis of thyroid profile of the participants revealed that diabetic patients with hypothyroidism (cases) have higher TSH level than diabetic patients without hypothyroidism (controls) which is statistically significant (p value 0.000). The FT4 level is lower in cases than controls which is statistically significant (p value 0.000).

Table 2: Comparison of thyroid profile (TSH and FT4 levels) between cases and controls.

|               | Controls (n=30) Mean±SD | Cases (n=30) Mean±SD | Unpair ed ‘t’ | P value |
|---------------|-------------------------|----------------------|---------------|---------|
| TSH (µIU/ml)  | 2.96±1.15               | 21.36±5.82           | -16.98        | 0.00    |
| FT4 (ng/dl)   | 1.29±0.26               | 0.37±0.11            | 17.77         | 0.00    |

Figure 1: Comparison of onset latency (DML) between controls and cases (motor nerve conduction study).

RM, LM= Right and left Median Nerve RT, LT= Right and left Tibial Nerve.

In our study we have seen that in the upper limbs, the Median nerve distal motor latency (DML) and distal sensory latency (DSL) were significantly prolonged in cases as compared to controls on both the right and left sides.
The median nerve motor conduction velocity (MNCV) and sensory conduction velocity (SNCV) were reduced significantly in cases as compared to controls on both right and left sides.

The motor and sensory amplitude of the median nerve were reduced in cases than controls on both right and left sides which were not statistically significant.

The mean values of the DML of the Lt. Tibial Nerve in cases was 5.38 + 1.42 ms and in controls was 4.65+0.70 ms and the levels were increased in cases than controls which was statistically significant (p<0.05).

The analysis revealed that in diabetic patients with hypothyroidism (cases) latency has increased significantly in Rt. & Lt. Median Nerve and Lt. Tibial Nerve.

Our study showed that in lower limbs, the Tibial nerve distal latency was prolonged, amplitude and motor conduction velocity were reduced in cases as compared to controls on right side, but were not statistically significant.

On left side, the Tibial nerve motor latency was significantly prolonged and amplitude was significantly reduced, motor conduction velocity was reduced which was not significant in cases as compared to controls.
The sural nerve sensory latency was increased, sensory amplitude and conduction velocity were reduced in cases in comparison to controls on both right and left sides, but these were not statistically significant. In our study we have seen that, all the parameters are more abnormal in diabetic patients with hypothyroidism (cases) compared to control. Moreover, upper limb parameters are much abnormal compared to lower limb parameters for both motor and sensory nerves.

Table 3: Comparison of nerve conduction parameters between cases and controls in upper limbs.

| Parameters       | Controls (n=30) Mean±SD | Cases (n=30) Mean±SD | Unpaired ‘t’ | P value |
|------------------|-------------------------|----------------------|--------------|---------|
| **Rt. Median Nerve** |                         |                      |              |         |
| DML (ms)         | 4.16±1.30               | 5.44±1.77            | -3.205       | 0.002   |
| CMAP (mV)        | 8.39±3.65               | 7.98±3.27            | 0.470        | 0.640   |
| MNCV (m/s)       | 60.18±7.06              | 54.10±7.04           | 3.339        | 0.001   |
| Mean F Latency (ms) | 26.70±2.86             | 28.29±4.51           | -1.626       | 0.109   |
| DSL (ms)         | 3.04±0.61 (n=29)*       | 3.69±0.92 (n=27)**   | -3.088       | 0.003   |
| SNAP (µV)        | 38.04±15.94             | 31.62±14.63          | 1.625        | 0.110   |
| SNCV (m/s)       | 42.51±12.58             | 33.08±14.65          | 2.674        | 0.010   |

*Some patients where the latency was not recordable were excluded from analysis. **Some patients where the latency was not recordable were excluded from analysis.

Table 4: Comparison of nerve conduction parameters between cases and controls in lower limbs.

| Parameters       | Controls (n=30) Mean±SD | Cases (n=30) Mean±SD | Unpaired ‘t’ | P value |
|------------------|-------------------------|----------------------|--------------|---------|
| **Rt. Tibial Nerve** |                         |                      |              |         |
| DML (ms)         | 4.72±0.61               | 5.26±1.49            | -1.834       | 0.072   |
| CMAP (mV)        | 10.38±2.63              | 9.96±2.19            | 0.668        | 0.507   |
| MNCV (m/s)       | 44.48±4.22              | 43.02±5.10           | 1.205        | 0.233   |
| Mean F Latency (ms) | 51.13±4.40             | 53.05±7.78           | -1.179       | 0.243   |
| **Lt. Tibial Nerve** |                         |                      |              |         |
| DML (ms)         | 4.65±0.70               | 5.38±1.42            | -2.552       | 0.013   |
| CMAP (mV)        | 11.04±3.27              | 9.31±1.56            | 2.624        | 0.011   |
| MNCV (m/s)       | 44.74±4.11              | 43.27±5.63           | 1.153        | 0.254   |
| Mean F Latency (ms) | 50.85±3.61             | 52.64±7.45           | -1.184       | 0.241   |
| **Rt. Sural Nerve** |                         |                      |              |         |
| DSL              | 2.39±0.71 (n=23)*       | 2.58±0.96 (n=25)**   | -0.768       | 0.446   |
| SNAP             | 14.08±12.55             | 12.69±9.72           | 0.480        | 0.633   |
| SNCV             | 32.44±20.51             | 31.91±18.52          | 0.105        | 0.917   |
| **Lt. Sural Nerve** |                         |                      |              |         |
| DSL              | 2.40±0.74 (n=23)*       | 2.39±1.02 (n=25)**   | 0.032        | 0.975   |
| SNAP             | 14.42±13.48             | 13.35±12.59          | 0.319        | 0.751   |
| SNCV             | 31.15±20.07             | 30.85±20.12          | 0.058        | 0.954   |
Table 5: Percentage of abnormalities in nerve conduction parameters of the studied nerves.

| Nerves       | Parameters | Diabetics without Hypothyroidism (Controls) With abnormal parameters | Diabetics with Hypothyroidism (Cases) With abnormal parameters |
|--------------|------------|---------------------------------------------------------------------|-----------------------------------------------------------------|
|              | N         | %                     | N                  | %                     |
| Rt. median nerve | DML       | 12                    | 40                 | 20                   | 66.7                   |
|              | CMAP      | 2                     | 6.7                | 2                    | 6.2                    |
|              | MNCV      | 3                     | 10                 | 9                    | 30                     |
|              | Mean F    | 3                     | 10                 | 9                    | 30                     |
|              | DSL       | 25                    | 83.3               | 28                   | 93                     |
|              | SNAP      | 1                     | 3.3                | 3                    | 10                     |
|              | SNCV      | 24                    | 80                 | 28                   | 93                     |
| Lt. median nerve | DML       | 8                     | 26.7               | 17                   | 56.7                   |
|              | CMAP      | 2                     | 6.7                | 0                    | 0                      |
|              | MNCV      | 3                     | 3.3                | 10                   | 33.3                   |
|              | Mean F    | 0                     | 0                  | 8                    | 26.7                   |
|              | DSL       | 24                    | 80                 | 27                   | 90                     |
|              | SNAP      | 1                     | 3.3                | 3                    | 10                     |
|              | SNCV      | 23                    | 76.7               | 27                   | 90                     |
| Rt. tibial nerve | DML       | 0                     | 0                  | 8                    | 26.7                   |
|              | CMAP      | 0                     | 0                  | 0                    | 0                      |
|              | MNCV      | 5                     | 16.7               | 11                   | 36.7                   |
|              | Mean F    | 2                     | 6.7                | 7                    | 23.3                   |
| Lt. tibial nerve | DML       | 0                     | 0                  | 7                    | 23.3                   |
|              | CMAP      | 0                     | 0                  | 0                    | 0                      |
|              | MNCV      | 5                     | 16.7               | 10                   | 33.3                   |
|              | Mean F    | 2                     | 6.7                | 7                    | 23.3                   |
| Rt. sural nerve | DSL       | 12                    | 40                 | 13                   | 43.3                   |
|              | SNAP      | 10                    | 33.3               | 10                   | 33.3                   |
|              | SNCV      | 17                    | 56.7               | 21                   | 70                     |
| Lt. sural nerve | DSL       | 12                    | 40                 | 11                   | 36.7                   |
|              | SNAP      | 12                    | 40                 | 12                   | 40                     |
|              | SNCV      | 17                    | 56.7               | 22                   | 73.3                   |

DISCUSSION

The association of diabetes mellitus and thyroid dysfunction have long been reported. The occurrence of hypothyroidism is more among people with Type 2 diabetes mellitus. The involvement of the peripheral nervous system is well known in both of these diseases. Our study comprises of different parameters of motor and sensory nerve conductions, such as latency (DML and DSL), amplitudes (CMAP and SNAP), conduction velocity (MNCV and SNCV) and F latency. In both motor and sensory nerve conduction studies, we have observed that latency (DML and DSL) increased and velocity (MNCV and SNCV) decreased in the median nerve of both sides (right and left) significantly in cases than controls. The changes in amplitudes (CMAP and SNAP) and F latency were statistically insignificant. These findings favour a demyelinating rather than an axonal process. In our study, the frequency of nerve involvement was more in diabetic patients with hypothyroidism than diabetic patients without hypothyroidism and nerves of the upper extremity were more affected than lower extremity.

Peripheral neuropathy is an important complication of diabetes mellitus. It may present with clinical symptoms and signs that are nonspecific and with slow progression. It may be silent and go undetected. Diabetes and hypothyroidism have been shown to mutually influence each other and association between both conditions have long been reported. The complications of diabetes are reported to be more in diabetic patients with thyroid disorder in various studies. Distal sensory or sensory-motor polyneuropathy encompasses almost three-fourth of all diabetic neuropathy. Peripheral nerve abnormalities are also known to be associated with hypothyroidism. The detection of such cases is of great importance where hypothyroidism contributes to diabetic neuropathy and where it is the cause for poor control of the associated conditions. In this study, we have tried to determine the prevalence of peripheral neuropathy in Type 2 diabetic hypothyroid patients as well as to compare it with diabetic patients without hypothyroidism.

We have chosen peripheral nerves of both sides, because, it has been seen that distal symmetrical polyneuropathy is
the commonest form of diabetic neuropathy. Median (motor as well as sensory) nerves of upper limbs were selected, as the involvement of median nerve is common in hypothyroidism and in lower limbs, sural (sensory) and tibial (motor) nerves were selected, as the involvement of lower limb nerves is more than upper limbs in diabetic neuropathy.

Table 3 and Figure 1 explain that the onset latency (DML) of motor nerve conduction study has increased in both right and left median Nerves in diabetic patients with hypothyroidism (than in diabetic patients without hypothyroidism, which are statistically significant (p<0.00). DML is increased in right tibial nerve in cases than controls but not significant (p value 0.072; p>0.05). Table 4 explains that the Left Tibial nerve, DML has increased significantly in cases than controls (p value 0.013; p<0.05). In Figure 1 it is also seen that DML has increased abnormally in all the studied nerves of both sides in cases and also in controls, except in the left median nerve. This increase in DML indicates demyelination of peripheral nerves more in cases than in controls.

Amplitude (CMAP) has decreased in all the studied nerves on both sides in cases in comparison to controls, but the changes are statistically insignificant (p>0.05).

Moreover, Table 3 explains that the motor nerve conduction velocity (MNCV) has significantly decreased in both right and left median nerves in cases than in controls (p<0.01). Table 4 and Figure 2 explain that the change of MNCV in right and left tibial nerves in cases in comparison controls are statistically insignificant (p>0.05).

In the motor nerve conduction study, thus we have observed that DML increased and MNCV decreased in the median nerve of both side (right and left) significantly in cases than controls. The changes in amplitudes (CMAP) were insignificant. These findings favour a demyelinating rather than an axonal process. In the right tibial nerve, change in DML, CMAP and MNCV was insignificant in cases other than controls. In the left tibial nerve, DML increased and CMAP decreased significantly in cases than controls (p<0.05). There were no significant changes in MNCV in cases other than controls. Thus, the left tibial nerve is more involved than the right tibial nerve.

In the F wave study, the Mean F latency was increased in all the four studied nerves in cases in comparison to controls but the differences were not statistically significant (p>0.05).

In Table 3, we have seen that the mean value of sensory latency (DSL) increased in both right and left median nerves in cases than in controls, which are statistically significant (p<0.00). As per Table 4, the mean value of DSL of both right and left sural nerves in cases were comparable with the controls (p>0.05). Figure 3 also explains that significant impairments were seen in both upper limbs (median) nerves than lower limb (sural) nerves in cases than controls.

However in our study, the sensory amplitude (SNAP) have decreased in all the studied nerves on both sides in cases than in controls, but the changes are statistically insignificant (p>0.05).

Table 3 and Figure 4 explain sensory conduction velocity (SNCV) have decreased in both right and left median nerves in cases than in controls which are statistically highly significant (p<0.01). As per Table 4 and Figure 4 the SNCV in both right and left sural nerves decreased in cases in comparison to controls but not statistically significant (p>0.05).

The prevalence of diabetic polyneuropathy is generally estimated to be 10% to 50% in patients with type 2 diabetes mellitus (T2DM), and the incidence increases with age and duration of diabetes mellitus. Singh et al observed that, the nerves of the lower extremity were most affected. The most affected nerve was the sural sensory nerve (77%). This is not in agreement with our findings (Table 5, Figure 5). However, they have included newly diagnosed diabetic patients in their study, in contrast to our study, where the duration of diabetes was more than five years.

Abdulsalam et al, studied the electrophysiological findings in patients with newly diagnosed T2DM and found the frequency of abnormalities in the studied peripheral nerves as 60% for the median, 63% ulnar, 33% peroneal, 16% tibial and 8% sural, which agree with our findings among the diabetic patients without hypothyroidism (controls). In their study, nerves of the upper limbs were most affected. This is in contrast to the findings of Sing et al, Kersidag et al, Dyck et al, who have found motor and sensory nerves of the lower limbs were most affected. In their study, diabetic patients were newly diagnosed. In contrast, the average duration of diabetes was more than 10 years in our study subjects, which might be responsible for more involvement of the nerves of the upper extremity with the progression of the disease. Wagghmore et al performed a nerve conduction study in patients of hypothyroidism and found that the latencies were significantly prolonged (p<0.05) and conduction velocity and amplitude were significantly reduced in Median and Peroneal nerves. Parkhad et al found significant polyneuropathy, mainly axonal in hypothyroid subjects. In our study, we found mainly demyelinating type of neuropathy in diabetic patients with hypothyroidism. In their study, nerves of the upper extremity were mostly affected which agrees with our observations. Han et al observed that T2DM with subclinical hypothyroidism was more likely to have DPN, which agrees with our observation.

After evaluating the results, it is reflected that peripheral neuropathy was more prevalent in diabetic patients with hypothyroidism. With the diagnosis of concomitant
hypothesis, there were significant changes in upper limbs (the right and left median nerves) in cases than controls which manifested by the features of Carpal Tunnel Syndrome. Advanced age, longer duration of diabetes with disease progression and concomitant hypothyroidism might be responsible for extensive neuropathy which is demyelinating in pattern in contrast to the axonal pattern seen in hypothyroid patients.

In both motor and sensory nerve conduction studies, we have observed that latency (DML and DSL) increased and velocity (MNCV and SNCV) decreased in the median nerve of both side (right and left) significantly in cases than controls. The changes in amplitudes (SNAP) were insignificant. These findings favour a demyelinating rather than an axonal process. Above data has been explained in Table 3 and 4.

CONCLUSION

The study thus reveals Type 2 diabetes mellitus patients with hypothyroidism have a higher prevalence of peripheral neuropathy. All diabetic patients should be screened for early detection of hypothyroidism who are likely to benefit from timely intervention to prevent severe and disabling neurological complications of diabetes. To improve the diagnosis of peripheral neuropathy in the early and asymptomatic stage, the routine clinical examination should be complemented by a nerve conduction study, which is the gold standard in the evaluation of nerve function.

Limitation

Study has been done with limited population. The study has been done better with more number of cases and controls.

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