

Nerve Conduction Study of Newly Diagnosed Type 2 Diabetic Patients and Its Correlation with Glycosylated Hemoglobin (HbA1c)

AKTER S¹, AHMED KGU², ARIFUZZAMAN M³, ISLAM MR⁴, CHOWDHURY AH⁵, SAHA K⁶,
SINA H⁷, ALAM I⁸, MAHMUD R⁹, SARKER HK¹⁰, ULLAH M¹¹

Abstract:

Background: Peripheral neuropathy is one of the common complications of DM and may proceed by a long period of clinically silent impaired glucose tolerance, altering the nerve function by the time diabetes is diagnosed. Nerve conduction (NCS) remain the most reliable, accurate and sensitive measure for diagnosis of peripheral neuropathy.

Objectives: Evaluation of nerve conduction study of newly diagnosed type -2 diabetic patients and to find out its correlation with glycosylated hemoglobin (HbA1c). **Method:** This is a case control study. A total 50 newly diagnosed type-2 diabetic patients within 1 month of both sex and another 50 healthy controls were studied prospectively during January 2018 to December 2019 from outpatient department of Endocrinology and advised to attend the department of Neurology of Dhaka medical college hospital. Nerve conduction study of cross limbs was performed. Blood sugar and glycosylated hemoglobin (HbA1C) were determined **Results:** Motor nerve conduction parameters were more affected. Median nerve impairment in both motor and sensory nerve conduction study was statistically significant ($p<0.05$) between case and control group. Most abnormal parameter is CMAP of peroneal nerve 42% followed by DL of tibial (20%) and median(18%) in motor nerve conduction study whose majority of them HbA1C was $>10\%$. Combination tibial and peroneal nerve involvements were more common 11 (22.0%), followed. In multivariate regression analysis, HbA1C significantly ($p<0.05$) associated with tibial F-Lat but others were not significantly associated. There were significant positive correlations with median DL ($r=0.829$; $p=0.002$), median F-Lat ($r=0.814$; $p=0.001$), Tibial DL ($r=0.805$; $p=0.004$), tibial F-Lat ($r=0.832$; $p=0.001$), median sensory DL ($r=0.824$; $p=0.002$) **Conclusion:** Increased HbA1c level indicative of a state of hyperglycemia is a risk factor for polyneuropathy in diabetic patients. NCS parameters are valuable for identification, future prediction of diabetic neuropathy as well as quantitative measure of its severity.

Keywords: Diabetes mellitus, HbA1c, NCS.

Introduction:

Diabetes mellitus is one of the most common chronic diseases globally. It was estimated that in

2017 there were 451 million (age 18–99 years) people with diabetes worldwide who are expected to increase to 693 million by 2045. The high

1. Dr. Sanzida Akter, Phase B Student, Department of Neurology, Dhaka Medical College
2. Dr. Kazi Gias Uddin Ahmed, Associate Professor & Head, Department of Neurology, Dhaka Medical College
3. Dr. Md. Arifuzzaman, Assistant Professor, Department of Neurology, Dhaka Medical College
4. Prof. Md. Rafiqul Islam, Professor, Neurology, Bangabandhu Sheikh Mujib Medical University
5. Dr. Ahmed Hossain Chowdhury, Associate Professor, Department of Neurology, Dhaka Medical College
6. Dr. Kanol Saha, Associate Professor, Department of Neurology, Dhaka Medical College
7. Dr. Hashmi Sina, Assistant Professor, Department of Neurology, Dhaka Medical College
8. Dr. Iftekher Alam, Assistant Professor, Department of Neurology, Dhaka Medical College
9. Dr. Reaz Mahmud, Assistant Professor, Department of Neurology, Dhaka Medical College
10. Dr. Humayun Kabir Sarker, Phase B Student, Department of Neurology, Dhaka Medical College
11. Dr. Mohammad Ullah, Phase B Student, Department of Neurology, Dhaka Medical College

prevalence of diabetes has important social, financial and development implications especially in low and middle - income countries¹.

Diabetic neuropathy (DN) is one of the most commonly occurring microvascular complications accounting for 28% of all the complications in diabetics².

Prevalence of diabetic peripheral neuropathy in Bangladesh was 19.7% in male and 18.7% in female which increased with age and duration of diabetes^{3,4}. Diabetic peripheral neuropathy affecting approximately 8% of newly diagnosed patients and >50% of patients with long-term DM⁵.

It is a progressive process that has a long asymptomatic stage⁶. Approximately 15% of patients with DM develop foot ulcer during their lifetime, of which 70% have a neuropathic origin⁷.

The signs, symptoms, and neurologic deficits in DN vary depending on the classes of nerve fibers involved. Diabetic sensorimotor polyneuropathy is the most common clinical subtype seen in clinical practice⁸. Common symptoms are symmetrical paresthesia and burning pain that predominantly occurs distally in the legs according to length dependency⁹. 15% of patients with NIDDM have both symptoms and signs of neuropathy but nearly 50 percent have either neuropathic symptoms or slowing of Nerve Conduction Velocity, before patient develops any sign¹⁰. Peripheral neuropathy is the frequent, globally disabling and earliest complication among major three complications of diabetes mellitus. Its prevention and early detection is of utmost importance. However, because not all sensory and motor symptoms in the extremities correspond to an electrophysiological abnormality, screening for diabetic peripheral neuropathy in DM patients poses a challenge. Patient with recently diagnosed or poorly controlled diabetes frequently show reduced nerve conduction velocity that improves rapidly with establishment of euglycaemia¹¹. Therefore, a quantified, reliable screening tool is required for early detection of diabetic peripheral polyneuropathy.

Nerve conduction studies (NCS) are considered to be the most sensitive, reliable, non-invasive, and objective means of investigating diabetic

neuropathy (Claus et al. 1993) Routine NCS include evaluation of motor function of the median, ulnar, peroneal and tibial nerves, and sensory function of median, ulnar and sural nerves in terms of onset latency, amplitude, and conduction velocity. F waves are motor responses produced by antidiromic activation of motor neurons following stimulation of motor axons peripherally. Various F-wave parameters such as minimal F-wave latency and persistence have been reported to have a very high diagnostic reliability in diabetic patients. Plasma glycosylated hemoglobin (HbA1C) is an index of average glycemic control over the previous 2–3 months and indicates poor diabetic control; furthermore, increased HbA1C concentration is the most important risk factor for predicting DM complications. Maintaining an HbA1C level below 6.5% is critical to decreasing the incidence of diabetic complications⁵. The HbA1C level could be a screening tool for early detection of diabetic peripheral polyneuropathy and an indicator of the optimal time for performing NCS in patients with long-standing diabetes. Moreover, the quantified composite score of NCS may be associated with disease severity¹³.

Many investigators have studied the correlation between DM complications and HbA1C. However, very little research has considered the correlation between NCS parameters, HbA1C. The objectives of the present study were to evaluate various nerve conduction parameters in newly diagnosed (type 2) diabetic patients and any correlation with HbA1C.

Materials and methods:

This case-control study was conducted in the department of Neurology, Dhaka medical college hospital, from July 2018 to June 2020 after obtaining ethical clearance. The study sample consisted of newly diagnosed type 2 diabetic patients within 1 month of the diagnosis of as per criteria laid down by American Diabetes Association criteria¹⁴. The cases were both sexes, aged ≥18 years attending outdoor department of endocrinology advised to attend in department of neurology. The control group comprises an equal number of age-matched non diabetic healthy individuals. Written informed consent was obtained from all the participants. The patients type 1 DM, chronic diseases such as chronic renal failure,

hypothyroidism, hepatic dysfunction, inflammatory neuropathy, vasculitis, CVD, family history of neuropathy, chronic smokers, history of chronic alcohol abuse, toxin, metals or drugs were excluded.

All patients underwent detailed neurological examination and their BMI was calculated.

Neurophysiological examination includes measurement of nerve conduction study of cross limbs. Motor NCS and elicitation of F-waves were performed on median, ulnar, common peroneal and tibial nerves. Sensory conduction study was performed on median, ulnar and sural nerves.. With the help of stimulating electrodes supramaximal stimulation was given at two different sites (distal and proximal site) to obtain compound muscle action potential. Ground electrode was placed between stimulating electrode and recording electrode. Nerve conduction velocity was measured by a standard NCV-EMG machine (NIHON KOHDEN), model – MEB9400, software- NeuropackS1 in a room temperature. Nerve conduction parameter included according to the protocol recommended by Antonio Conference on Diabetic neuropathy. Nerves were stimulated using 1 ms electrical pulse at a repetition rate of 1 per second with intensity sufficient to elicit maximum amplitude of compound muscle action potential (CMAP) and sensory nerve conduction potential (SNAP). Nerve function was considered abnormal if any parameter [distal latency, compound muscle action potential (CMAP), sensory nerve action potential(SNAP), conduction velocity(CV)] of four motor and three sensory nerve modalities assessed was not the limits of normality.

Data was processed and analyzed using SPSS (Statistical Package for Social Sciences). To

observe the difference between the case and control test statistics like Chi-square (χ^2) was done to compare the qualitative variables and independent t-Test for quantitative variables. To measure the outcome between the case and control, multivariate logistic regression was done for qualitative variables. To observe the correlation HbA1C between the nerve conduction parameters of the patients, scatter diagram was formulated for quantitative variables and linear logistic regression test for the qualitative variables. P value less than 0.05 was considered statistically significant.

Results and observations: Out of total 100 patients, 50 consecutive newly diagnosed type-2 diabetic patients (group 1) 28 (56%) males and 22(44%) females. Mean age was 45.62 ± 11.92 years ranged from 25 to 79 years .Age ,sex, religion, occupation, educational status, socioeconomic status in diabetic patients did not differ significantly(P value <0.05)with control group(group 2). Table 1 shows the mean value of HbA1C level was significantly higher in diabetic group was 9.23 ± 2.57 (%) n group I and 5.63 ± 0.61 (%) in group II(P value was <.001).

Table 1 shows the mean value of HbA1C level was significantly higher in diabetic group was 9.23 ± 2.57 (%) n group I and 5.63 ± 0.61 (%) in group II(P value was <.001).

On comparison of motor nerve conduction parameters between two groups was revealed in table II. P value reached from unpaired t-test. The mean distal latency, compound muscle action potential, conduction velocity, F-Latency of median nerve between cases and controls were significantly deferent($P < 0.05$). The mean values are 3.75 ± 0.87 (ms), 9.0 ± 3.63 (mv) 45.15 ± 6.51 (m/s) 29.61 ± 1.75 (ms) respectively for diabetic group & 3.47 ± 0.39 (ms), 10.39 ± 3.12 (mv), 56.02 ± 7.19 (m/s),

Table-I
Distribution of the study patients by LAB test (n=100)

LAB test	Group I (n=50)		Group II(n=50)		P value
	Mean \pm SD	Range (min,max)	Mean \pm SD	Range (min,max)	
Fasting Blood Glucose (mmol/L)	10.81 ± 4.84	5.08,27.61	4.6 ± 0.8	3.3,6.1	0.001 ^s
2 Hours after 75g Glucose drink (mmol/L)	15.57 ± 5.3	8.03,31.86	5.65 ± 1.29	3.3,7.8	0.001 ^s
HbA1C (%)	9.23 ± 2.57	5.04,13.6	5.63 ± 0.61	4.4,6.6	0.001 ^s

26.53±2.41(ms) respectively for control group. The compound muscle action potential was 7.75±1.92 in group I and 9.29±2.07 in group II differ significantly ($P<0.05$). There were no significant difference in mean value of DL, MNCV, F-latency of ulnar nerve. The mean value of distal latency and F-latency for studied tibial nerve was significant difference ($P<.035$, .013 respectively), whereas mean value of CMAP, MNCV of tibial nerve was not significant different from control group. The mean value of CMAP of peroneal nerve 3.45±1.63 in diabetic group & 4.18±1.72 in control group was differ significantly ($P<.032$). DL, MNCV were comparable but not significant between two group.

While comparison of sensory nerve conduction parameters in diabetic and control group in table III. There was significant difference in mean value of distal sensory latency, SNAP, SNCV for studied median nerve the values were 2.97 ± 0.47 , 28.53 ± 16.96 , 45.16 ± 7.34 respectively in diabetic

group and 2.73 ± 0.4 , 35.2 ± 11.55 , 48.7 ± 3.68 in control group respectively. The mean value of SNCV of ulnar nerve was differ significantly from diabetic to control group ($P<0.028$). The mean value of SNAP of sural nerve was significantly differ from control group ($P<0.043$).

For median nerve most frequent abnormal parameters were distal latency(DL) and compound muscle action potential(CMAP(21%) whose majority of patients HbA1C was $>10\%$. For ulnar nerve, most frequent abnormal parameter was CMAP(28%) whose majority HbA1C $>8\%$. For tibial nerve, DL (30%)whose majority of patients HbA1C was 6.5-8%. For peroneal nerve ,most frequent abnormal parameter was CMAP (57%)whose majority HbA1C was $>8\%$

For median sensory nerve, most frequent abnormal parameter were DL and SNAP whose majority HbA1C was 6.5-8% & $>10\%$. For ulnar nerve ,most frequent abnormal parameter was SNAP whose

Table-II
Distribution of the study patients by motor nerve conduction study (n=100)

Motor nerve conduction study	Group I (n=50) Mean±SD	Group II (n=50) Mean±SD	P value
Median nerve			
DL (ms)	3.75±0.87	3.47±0.39	0.041 ^s
CMAP (mV)	9.0±3.63	10.39±3.12	0.042 ^s
CV (m/s)	45.15±6.51	56.02±7.19	0.001 ^s
F-Lat (ms)	29.61±1.75	26.53±2.41	0.001 ^s
Ulnar nerve			
DL (ms)	2.61±0.45	2.5±0.34	0.171 ^{ns}
CMAP (mV)	7.75±1.92	9.29±2.07	0.001 ^s
CV (m/s)	57.39±6.06	55.4±6.85	0.127 ^{ns}
F-Lat (ms)	26.47±2.65	25.8±2.38	0.186 ^{ns}
Tibial nerve			
DL (ms)	4.99±1.22	4.52±0.96	0.035 ^s
CMAP (mV)	10.48±4.09	9.19±3.29	0.085 ^{ns}
CV (m/s)	44.05±5.59	45.36±13.24	0.521 ^{ns}
F-Lat (ms)	47±4.26	45.12±3.12	0.013 ^s
Peroneal nerve			
DL (ms)	3.88±0.71	3.65±0.49	0.062 ^{ns}
CMAP (mV)	3.45±1.63	4.18±1.72	0.032 ^s
CV (m/s)	45.39±8.59	45.62±5.46	0.873 ^{ns}

Table-III
Distribution of the study patients by sensory nerve conduction (n=100)

Sensory nerve conduction	Group I (n=50) Mean±SD	Group II (n=50) Mean±SD	P value
Median nerve			
DL (ms)	2.97±0.47	2.73±0.4	0.007 ^s
SNAP (mV)	28.53±16.96	35.2±11.55	0.024 ^s
CV (m/s)	45.16±7.34	48.7±3.68	0.003 ^s
Ulnar nerve			
DL (ms)	2.25±0.37	2.32±0.33	0.321 ^{ns}
SNAP (mV)	36.03±16.15	32.5±15.28	0.264 ^{ns}
CV (m/s)	46.94±6.02	49.88±7.59	0.028 ^s
Sural nerve			
DL (ms)	2.22±0.36	2.24±0.33	0.772 ^{ns}
SNAP (mV)	17.88±10.06	14.34±6.94	0.043 ^s
CV (m/s)	47.61±7.31	47.67±1.33	0.955 ^{ns}

Table-IV
Abnormal findings of motor nerve conduction study subjects (n=50) with different level of HbA1C

Motor nerve conduction study	6.5-8 % (n=20)		>8-10 % (n=7)		>10 % (n=23)	
	n	%	n	%	n	%
Median nerve						
DL (≥ 4.4 ms)	4	20.0	0	0.0	5	21.7
CMAP (≤ 4.0 mV)	1	5.0	0	0.0	2	8.7
CV (<30 m/s)	0	0.0	0	0.0	0	0.0
F-Lat (≥ 31 ms)	1	5.0	0	0.0	0	0.0
Ulnar nerve						
DL (≥ 3.5 ms)	2	10.0	0	0.0	0	0.0
CMAP (≤ 6.0 mV)	2	10.0	2	28.6	5	21.7
CV (m/s)	0	0.0	0	0.0	0	0.0
F-Lat (≥ 32 ms)	1	5.0	0	0.0	0	0.0
Tibial nerve						
DL (≥ 6.0 ms)	6	30.0	0	0.0	4	17.4
CMAP (≤ 4.0 mV)	1	5.0	0	0.0	0	0.0
CV (m/s)	0	0.0	0	0.0	0	0.0
F-Lat (≥ 56 ms)	1	5.0	0	0.0	0	0.0
Peroneal nerve						
DL (≥ 5.5 ms)	0	0.0	1	14.3	0	0.0
CMAP (≤ 3.0 mV)	9	45.0	4	57.1	8	34.8
CV (m/s)	1	5.0	0	0.0	0	0.0

Table-V*Abnormal findings of sensory nerve conduction study subjects (n=50) in different level of HbA1C*

Sensory nerve conduction	6.5-8 % (n=10)		>8-10 % (n=7)		>10 % (n=23)	
	n	%	n	%	n	%
Median nerve						
DL (≥ 3.3 ms)	4	20.0	1	14.3	3	13.0
SNAP (≤ 20 mV)	6	30.0	1	14.3	7	30.4
CV (< 30 m/s)	0	0.0	0	0.0	0	0.0
Ulnar nerve						
DL (≥ 3.1 ms)	0	0.0	0	0.0	2	8.7
SNAP (≤ 17 mV)	3	15.0	1	14.3	3	13.0
CV (< 30 m/s)	0	0.0	0	0.0	0	0.0
Sural nerve						
DL (≥ 4.5 ms)	0	0.0	0	0.0	0	0.0
SNAP (≤ 6.0 mV)	1	5.0	0	0.0	1	4.3
CV (< 30 m/s)	0	0.0	0	0.0	0	0.0

majority HbA1C was 6.5-8%. For sural nerve, SNAP is mostly affected, whose HbA1C was 6.5-8%. Conduction abnormality was not found in the present study except peroneal nerve in motor conduction study. Combination nerve involvement with various parameter are Tibial + peroneal (22%) tibial+peroneal+sural(6%), isolated median nerve involvement(12%). Multivariate regression analysis showed there were no association between HbA1C and different nerve conduction parameters except tibial F-latency ($P<0.05$). Observations of correlation between HbA1C between different

parameters of NCS revealed, there were highly significant positive correlation ($P<0.05$) found in DL and F-latency of median nerve, DL an F-latency of tibial nerve, sensory DL of median nerve. Amplitude and conduction velocity showed inverse but nonsignificant correlation. (Fig. 1 & 2). Figure 1: Scatter diagram showing significant positive correlation ($r=0.829; p=0.002$) between the HbA1C and median DL. Figure 2: Scatter diagram showing not significant negative correlation ($r=-0.185; p=0.967$) between the HbA1C and peroneal CMAP

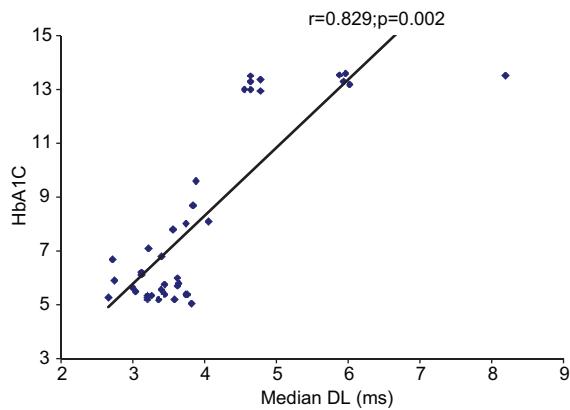


Fig.-1: Scatter diagram showing significant positive correlation ($r=0.829; p=0.002$) between the HbA1C and median DL

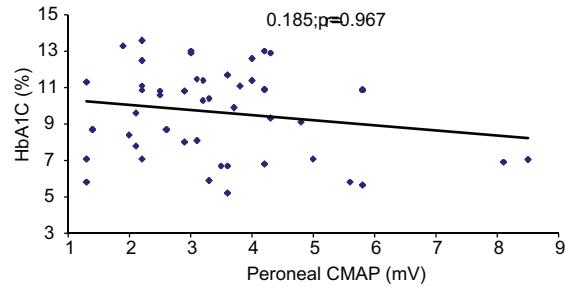


Fig.-2: Scatter diagram showing not significant negative correlation ($r=-0.185; p=0.967$) between the HbA1C and peroneal CMAP

Discussion:

The present study revealed abnormalities in the nerve conduction parameters in patients with newly diagnosed type 2 diabetic patients which correlated HbA1C values. Electrophysiological incidence of peripheral neuropathy has generally about 30%. In this study longer latency, smaller amplitude, declining velocity was found in median nerve both motor and sensory conduction study. F-wave latency was also prolonged⁸. found the same result in study of asymptomatic type 2 diabetes patients. The present studies also revealed ulnar prolonged conduction velocity and reduced amplitude, prolonged distal latency and F-latency of tibial nerve, reduced amplitude of peroneal nerve in motor conduction study. Whereas reduced conduction velocity in ulnar nerve and amplitude in sural nerve¹⁵. found longer latency, smaller amplitude and slower conduction velocity were found in all nerves they also found these parameters were inferior in those with higher HbA1C. In this study, motor nerves conduction were more affected than sensory nerves conduction¹⁶. found thirteen patients (54%) had abnormal sensory conduction and nine patients had abnormal motor conduction¹⁷. found significant motor function deterioration in the diabetic groups with shorter and longer duration comparison to healthy group. This finding is consistent with this study. Peroneal nerve was most affected followed by tibial, median motor and sensory, ulnar, sural in descending order¹⁸. found peroneal motor nerve had highest abnormality¹⁹ found that most of the study subjects showed two to five nerve combination, also high percentage of subjects with altered median nerve conduction that correspond to this study. the correlation observation of this study there was positive correlation with distal latency, F-latency of median and tibial nerve^{20,21}. found inverse correlation with conduction velocity. Agarwal et al. showed a weak to moderate statistical significant relationship with HbA1C. Smaller amplitude reflects axonal loss and slowing of conduction velocity could be the result of combination of segmental demyelination, loss of fastest conducting axons. Conduction abnormalities are more frequent in large myelinated

fibers in early stage of diabetes but also involvement of small myelinated and unmyelinated fibres especially in lower extremity. Thus nerve length might be an important factor in early dysfunction of nerve¹⁹.

Conclusion:

The present study showed that abnormalities of NCS parameters in early stage of diabetes at time of diagnosis, thus it is important to evaluate all newly detected diabetics for subclinical impairment of nerve function and incorporation of electrophysiological study as routine test. Occurrence of electrophysiological changes in nerve function correlates with HbA1C.

References:

1. Cho N., Shaw J.E., Karuranga S., Huang, Y., da Rocha Fernandes J.D., Ohlrogge, A.W. and Malanda, B., IDF Diabetes Atlas: Global estimates of diabetes prevalence for 2017 and projections for 2045. *Diabetes research and clinical practice*. 2018; 138: 271-281.
2. Tesfaye S, Stevens LK, Stephenson JM, Fuller JH, Plater M, Ionescu-Tirgoviste C et al., Prevalence of diabetic peripheral neuropathy and its relation to glycemic control and potential risk factors. *The Euro Diab IDDM complications study. Diabetologia* ; 1996; 39:1377-1384
3. Almuhanadi, H., Ponirakis, G., Khan, A. and Malik, R.A. Diabetic neuropathy and painful diabetic neuropathy: Cinderella complications in South East Asia. *J Pak Med Assoc* 2018; 68:85-89
4. Morkrid, K., Ali, L. and Hussain, A., 2010. Risk factors and prevalence of diabetic peripheral neuropathy: A study of type 2 diabetic outpatients in Bangladesh. *International journal of diabetes in developing countries*, 30(1), p.11.
5. Deli, G., Bosnyak, E., Pusch, G., Komoly, S. and Feher, G., Diabetic neuropathies: diagnosis and management. *Neuroendocrinology*. 2013; 98(4):267-280.
6. Perkins, B.A. and Bril, V., Diagnosis and management of diabetic neuropathy. *Current diabetes reports*. 2002 2(6):495-500.

7. Boulton, A.J., The diabetic foot: a global view. *Diabetes/metabolism research and reviews*. 2000; 16(1):2-5.
8. Owolabi L.F., Adebisi S., Danborno B. and Buraimoh, A.A., Electrodiagnostic evaluation of median nerve conduction in Type II diabetes mellitus patients that were asymptomatic for peripheral neuropathy: a case control study. *International Journal of Medicine and Biomedical Research*, 2015; 4(3):154-161.
9. Feng, Y., Schlösser, F.J. and Sumpio, B.E., 2011. The Semmes Weinstein monofilament examination is a significant predictor of the risk of foot ulceration and amputation in patients with diabetes mellitus. *Journal of vascular surgery*. 2011; 53(1):220-226.
10. Odusote K., Ohwovorole A. and Roberts O., Electrophysiologic quantification of distal polyneuropathy in diabetes. *Neurology*. 1985; 35(10):1432-1437.
11. Gregersen G., Variations in motor conduction velocity produced by acute changes of the metabolic state in diabetic patients. *Diabetologia* 1968; 4(5):273-277.
12. Claus, D., Mustafa, C., Vogel, W., Herz, M. and Neundörfer, B.. Assessment of diabetic neuropathy: Definition of normal and discrimination of abnormal nerve function. *Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine*, 1993;16(7):757-768.
13. Lee, W.J., Jang, S., Lee, S.H. and Lee, H.S., Correlation between the severity of diabetic peripheral polyneuropathy and glycosylated hemoglobin levels: a quantitative study. *Annals of rehabilitation medicine* 2016; 40(2):263-270
14. American Diabetes Association,. Standards of Medical Care in Diabetes (position statement). *Diabetes Care* 2020; 43: S1.
15. Agarwal S., Lukhmana S., Kahlon N., Malik, P. and Nandini, H., . Nerve conduction study in neurologically asymptomatic diabetic patients and correlation with glycosylated hemoglobin and duration of diabetes. *National Journal of Physiology, Pharmacy and Pharmacology* 2018; 8(11):1533-8.
16. Kulkarni, A.P., Saroja, A.O., Naik, K.R., Ghatnatti, V. and Hesarur, N., . Nerve conduction abnormalities in patients with newly diagnosed diabetes mellitus. *Journal of the Scientific Society* 2018 45(1):30.
17. Sultana, S., Begum, N., Ali, L., Hossain, M.M., Bhowmik, N.B. and Parveen, Z.,. Electrophysiological changes of motor nerves in patients with type 2 diabetes mellitus. *Journal of Armed Forces Medical College, Bangladesh* 2009; 5(2):14-17.
18. Dyck, P.J. and O'Brien, P.C., Meaningful degrees of prevention or improvement of nerve conduction in controlled clinical trials of diabetic neuropathy. *Diabetes Care* 1989; 12(9):649-652
19. Singh R.B., Chandel K., Kumar S. and Singh R.B. Nerve conduction study findings of subclinical diabetic neuropathy in newly diagnosed diabetic patients. *Indian Journal of Neurosciences* 2015 1(1):1-7.
20. Farheen A., Malipatil B.S. and Arif G., Nerve conduction in type 2 diabetics and its correlation with glycosylated haemoglobin. *Journal of Evolution of Medical and Dental Sciences* 2015 4(6):1023-1035.
21. Sayeed, M.A., Epidemiological study of diabetes mellitus in rural areas of Bangladesh. [Dissertation]. Dhaka: Bangladesh College of Physicians and Surgeons;1985.