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Glycemic Control and Clinico-Electrophysiological Severity of Diabetic Polyneuropathy

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Abstract

Background: Diabetic polyneuropathy (DPN) has a significant positive correlation with poor glycemic control (HbA1c ≥7%). The clinical, biochemical and electrophysiological parameters of DPN in Bangladeshi citizens have not yet been explored elaborately. **Objective:** The purpose of the study was to detect and categorize status of glycemic control of Bangladeshi people and to analyze its impact on clinical severity of DPN using Toronto Clinical Scoring System (TCSS) and electrophysiological severity by modified Michigan diabetic neuropathy score (MDNS). **Methodology:** This observational study was carried out on diabetic patients having probable DPN by purposive sampling, attending Neurology OPD of Dhaka Medical College Hospital, Dhaka, Bangladesh and Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine & Metabolic Disorder (BIRDEM) Hospital, Dhaka, Bangladesh from July 2014 to June 2016. Clinical parameters were recorded and DPN was graded as "no neuropathy", "mild", "moderate" and "severe" neuropathy by the Toronto Clinical Scoring System (TCSS). A standard nerve conduction study was performed on each patient and electrophysiological grading according to modified Michigan diabetic neuropathy score (MDNS) was done. Diabetic status of patients was classified into "controlled" (HbA1c <7.0%) and "uncontrolled" (HbA1c ≥7.0%) groups and HbA1c level and the clinical & electrophysiological severity scores were compared and were analyzed. **Results:** Mean age of the patients was 57.2±9.37 years. 51.0% cases were males and 49% cases were females. The mean HbA1c in the study population was 7.6±0.94% and 56.0% patients had HbA1c≥ 7% .Motor nerve conduction studies revealed that both CMAP amplitudes and MNCV in the ulnar, peroneal and tibial nerves were reduced significantly in patients of uncontrolled (HbA1c≥7%) DM ($p<0.001$). Sensory nerve conduction studies revealed significant reduction in SNAP amplitudes of median and ulnar sensory and sural nerves in the uncontrolled group ($p<0.001$). Electrophysiologically, 65.43% patients had mixed sensory-motor neuropathy ($p<0.00001$). Clinically severe DPN patients were higher (45.2%) within the uncontrolled (HbA1C ≥7%) group ($p<0.00001$). Similarly, severity in electrophysiological grading was more in patients with uncontrolled DM (48.8%) ($p<0.00001$). **Conclusions:** Neuropathic severity, either clinically or electrophysiologically, was associated with higher values of HbA1c. *[Journal of National Institute of Neurosciences Bangladesh, 2019;5(2): 177-184]*

Keywords: : Glycemic control; clinico-electrophysiological; severity; diabetic polyneuropathy

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Introductio

Neuropathy is considered the most common micro-vascular complications of both types 1 and 2 diabetes mellitus[1-3]. Diabetic polyneuropathy is defined

as the presence of clinical or subclinical symptoms and/or signs of peripheral nerve damage in patients with diabetes mellitus in the absence of the other causes of peripheral neuropathy (Report and recommendations of the San Antonio conference on diabetic neuropathy 1988). Distal peripheral neuropathy, also known as diabetic polyneuropathy (DPN), is by far the most common type of neuropathy seen in DM[3-4] which is distal, symmetric, sensori-motor predominantly sensory and mostly axonal. HbA1c as an index of long-term diabetes control has been shown to be related to the incidence and the prevalence of DPN in both cross-sectional and prospective epidemiological studies.

Evaluation of neuropathy is generally undertaken by electrophysiological measurement which is more sensitive than clinical examination as the former is quantitative. To observe the functional status of peripheral nerves in diabetic neuropathic patients, distal latencies (DL), compound muscle action potential (CMAP) and motor nerve conduction velocities (MNCV) are assessed.

According to previous studies, the prevalence of peripheral neuropathy is specific to the population tested & nerve conduction parameters vary in different racial groups taking environmental and nutritional heterogeneity into consideration[5]. Consensus statement 1992 suggested that all aspects of nerve conduction studies should be standardized and every laboratory should have population based reference values[6].

Diabetic polyneuropathy (DPN) can be assessed clinically and electrophysiological in different ways & different scoring system[7-8]. However, the newly developed validated Toronto Clinical Scoring System (TCSS) does correlate effectively with electrophysiological severity[9]. The Toronto CSS was based on classic neurological history and examination techniques and designed to be simple and relevant to the clinician. The MDNS electrophysiological portion deals with nerve conductions done on sural, peroneal motor, median sensory and motor, and ulnar sensory nerves where a nerve is considered abnormal if any attribute(amplitude, distal latency, or conduction velocity) was not within the normal values between the first and 99th percentiles[10]. It was a unique part of the study to match the clinical and electrophysiological severity grading against different levels of glycemic control in a notion to emphasize on early controlling of DM in a particular HbA1c group people.

In Bangladesh, Sultana et al[5] demonstrated early electrophysiological changes in motor nerves of patients of DM type 2, particularly in those who have a longer duration of illness. However, clinical characteristics, biochemical determinants like HbA1c & electrophysiological parameters of DPN in Bangladeshi citizens have not yet been explored elaborately. The present study aimed at detecting and categorizing various status of glycemic control of Bangladeshi people, described as “controlled & uncontrolled” on the basis of HbA1c, and analyzing its impact on clinical and electrophysiological severity of DPN.

Methodology

This observational study was carried out in the Neurology Department of Dhaka Medical College Hospital (DMCH) and samples were collected from outdoor of neurology department of DMCH & Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine & Metabolic Disorder (BIRDEM) Hospital, Dhaka, Bangladesh. Patients of diagnosed type I or II Diabetes Mellitus who have probable DPN, defined as a combination of any two or more of the neuropathic symptoms like decreased sensation, pain, paresthesia, tingling, numbness, burning, weakness in symmetrical lower limbs ± upper limbs within previous 06 months, decreased distal sensation and/or unequivocally decreased or absent ankle reflexes (criteria recommended by The Toronto Diabetic Neuropathy Expert Group), were included in the study by purposive sampling. Detailed socio-demographic data, family and medical history and thorough clinical examination findings were collected in a structured questionnaire. Clinical severity was expressed as the validated Toronto Clinical Scoring System (TCSS) which produces a score derived from the clinical assessment of 6 symptoms, 5 sensory tests, and lower limb reflexes, giving a maximal score of 19. The degree of neuropathy was based on the TCSS score as no neuropathy ≤5, mild neuropathy 6–8, moderate neuropathy 9–11 and severe neuropathy ≥12[3,9]. Light touch, pain sense, vibration sense and temperature sense were tested using a wisp of cotton, a pinprick, a 128 Hz tuning fork and the cold surface of a tuning fork respectively while tendon reflex by a conventional reflex hammer. Patient’s HbA_1c level was documented from last 03 (three) months’ record or if not found, blood sample was collected for measuring percentage of HbA_1c, to be measured in a standard laboratory. Investigations necessary to exclude other causes of polyneuropathy were done according to clinical clues, like protein electrophoresis if monoclonal gummopathy was suspected by severe bone pain and high ESR in an elderly patient. Each patient was classified according to

glycemia regulation as measured by HbA_1c into "controlled" (if HbA_1c <7.0%) and "uncontrolled" (if HbA_1c ≥7.0%) groups. Nerve conduction study was performed by an experienced neurologist in standard procedure; bilateral recordings of the median, ulnar, peroneal, tibial and sural nerve in at least three limbs were done. In the motor nerves, the compound muscle action potential amplitudes (CMAP), distal motor latencies (DML) and motor nerve conduction velocities (MNCV) were recorded; the amplitudes of the responses were measured from baseline to the negative peak of the CMAPs. In the sensory nerves, sensory nerve action potential amplitudes (SNAP) and distal sensory latencies (DSL) were recorded. Latencies and amplitude values were read from the equipment after ensuring accurate cursor placement. Electrophysiological grading was done according to the number of abnormal nerves involved which was defined as any attribute like amplitude, distal latency, or conduction velocity not within the normal limits. Overall score (grade) was assigned as mild, moderate or severe neuropathy according to modified Michigan diabetic neuropathy score. Abnormality involving two, three to four and five nerves was graded as mild, moderate & severe neuropathy, respectively. To maintain equal standard of the study parameters, the investigator was present during the NCS examination. Finally, HbA_1c level and the clinical & electrophysiological severity scores were compared and analyzed. The data were analyzed using SPSS version 20.

Results

Among 243 patients of diabetic polyneuropathy included in the study, 124 (51.0%) were males and 119 (49.0 %) were females. Both the controlled & uncontrolled HbA1c levels were higher in the males (45.1% & 50.4%). Mean HbA1c in the study population was 7.6±0.94 (Table 1).

Table 1: HbA1c Distribution in the Study Population

HbA1c (%)	Male	Female	Total
6.5 to 7.0	56(45.1%)	52(43.7%)	108(44.4%)
7.0 to 7.5	5(4.0%)	8(6.7%)	13(5.4%)
7.5 to 8.0	9(7.3%)	11(9.4%)	20(8.2%)
8.0 to 8.5	39(31.4%)	32(26.9%)	71(29.2%)
8.5 to 9.0	6(4.8%)	12(10.1%)	18(7.4%)
9.0 to 9.5	9(7.3%)	4(3.4%)	13(5.3%)
Total	**124**	**119**	**243**
Mean (%)	7.6±.085	7.5±0.82	7.6±0.94

In this study 56.0% patients had uncontrolled DM whereas 44.0% had controlled DM (Figure I).

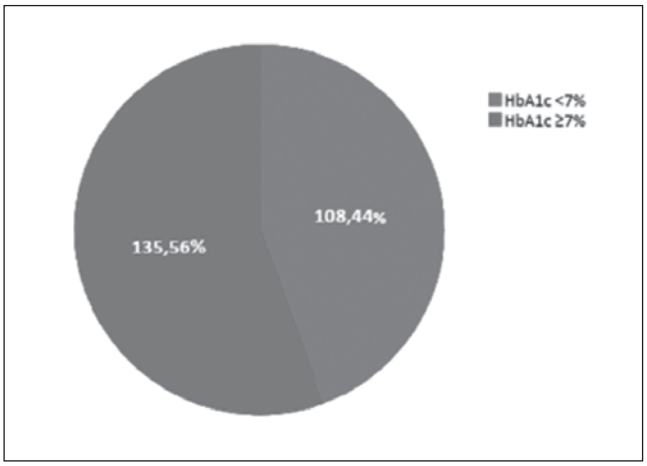

Figure I: Distribution of Study Subjects On the Basis of Glycemic Control (n=243)

Table 2: Differences in Presenting Symptoms & Clinical Findings between Patients with Uncontrolled and Controlled DM (n=243)

Presenting Symptoms & Clinical Findings	Uncontrolled DM	Controlled DM	P value
SYMPTOMS			
Tingling	132(97.7%)	100(92.6%)	0.0534
Numbness	126(93.3%)	96(88.9%)	0.218
Paraesthesia	102(75.6%)	63(58.3%)	0 .004
Pain	37(27.4%)	35(32.4%)	0.395
Weakness	30(22.2%)	15(13.9%)	0.097
CLINICAL FINDINGS			
Diminished tendon reflexes	94(69.6%)	26(24%)	<0.001
Absent tendon reflexes	81(60%)	21(19.45%)	<0.001
Diminished pain/temperature	95(70.4%)	23(21.3%)	<0.001
Diminished joint position/vibration	107(79.3%)	42(38.9%)	<0.001

*Z test of proportion (two sample); multiple responses

Of the presenting symptoms of the patients with DPN, tingling and numbness are more common in patients with uncontrolled DM which was 97.7% and 93.3%, respectively than uncontrolled DM. which was 92.6% and 88.9% respectively; though the difference was not statistically significant. Paraesthesia was the only symptom which was significantly (p=0.004) more common in patients with uncontrolled DM (75.5%) than those of controlled DM (58.3%) (Table 2). Of the clinical examination results, most frequent findings were diminished position sense & vibration sense (61.3%) followed by diminished tendon reflexes (49.4%) which were more common in the patient group with uncontrolled diabetes (p<0.001) (Table 2).

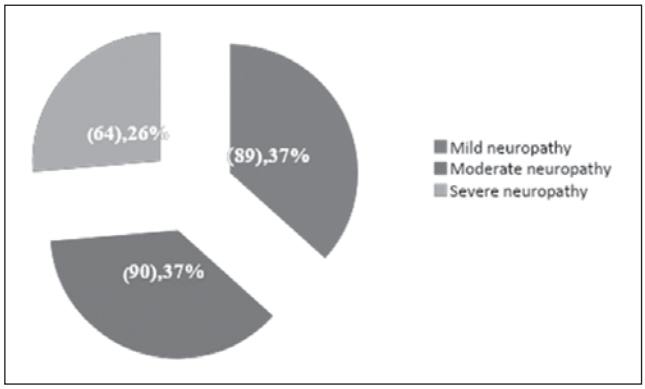

Figure II: Distribution of different grades of clinical DPN among study population (n=243)

According to clinical severity grading of DPN by TCSS,mild & moderate neuropathy constitute equal portions (37% each) and severe neuropathy constitutes 26% (Figure II) whereas according to electrophysiological severity grading of DPN, moderate neuropathy constitutes the majority (36%) followed by mild neuropathy (34%) (Figure III).

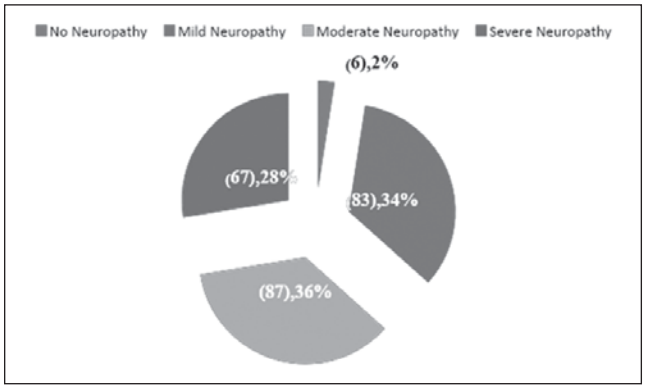

Figure III: Distribution of Different Grades of Electrophysiological Severity of DPN among Study Population (n=243)

CMAP amplitudes & MNCV in the ulnar, peroneal and tibial nerves were more reduced in patients with uncontrolled DM than the controlled group, which is statistically significant (p<0.05). Significant difference also exists in CMAP amplitude of median nerve between two groups (p <0.05) (Table 3).

There is also significant differences in SNAP amplitudes of median, ulnar & sural nerves between the two groups, patients with uncontrolled DM having the greater reduction of amplitudes (p <0.05) (Table 4).

Table 3: Electrophysiological Parameters (Motor Nerve Conduction Study) in the study population (n=243)

Nerves	Parameters	Uncontrolled DM	Controlled DM	P value
Upper Limbs				
Median	DL(msec)	4.0±0.29	3.8±0.27	<0.001
	CMAP(mV)	3.0±1.38	3.74±1.36	<0.001
	MNCV(m/sec)	50.3±4.75	49.1±4.80	0.052
Ulnar	DL(msec)	3.0±0.52	2.9±0.50	0.128
	CMAP(mV)	3.1±1.8	6.2±1.3	<0.001
	MNCV(m/sec)	46.8±2.1	51.5±2.2	<0.001
Lower Limbs				
Peroneal	DL(msec)	4.3±0.29	4.2±0.28	0.007
	CMAP(mV)	1.2±0.54	3.6±0.46	<0.001
	MNCV(m/sec)	32.5±1.5	39.2±1.7	<0.001
Tibial	DL(msec)	5.2±1.1	5.1±0.17	<0.001
	CMAP(mV)	2.4±1.3	2.8±1.6	0.035
	MNCV(m/sec)	36.5±2.1	40.2±2.3	<0.001

Values are expressed as mean ± SD; Two sample Z test

Table 4: Electrophysiological parameters (sensory nerve conduction study) in the study population (n=243)

Nerves	Parameters	Uncontrolled DM	Controlled DM	P value
Upper Limbs				
Median	DL(msec)	2.9±0.61	2.7±0.56	0.008
	SNAP(microvolt)	9.2±1.93	20±1.90	<0.001
Ulnar	DL(msec)	3.01±0.95	2.7±1.10	0.026
	SNAP(microvolt)	10.6±2.20	17.8±2.60	<0.001
Lower Limbs				
Sural	DL(msec)	2.46±0.25	2.31±0.33	<0.001
	SNAP (microvolt)	2.80±1.41	4.3±1.90	0.003

Values are expressed as mean ± SD;*Two sample t test

Mixed sensory-motor neuropathy pattern were higher in uncontrolled DM group and pure sensory patterns were higher in controlled group which is statistically significant (p<0.05) (Table 5).

Table 5: Differences in type of neuropathy (mixed/sensory) between patients with uncontrolled & controlled DM (n=237)

Neuropathy type	Diabetes Mellitus		p value
	Uncontrolled	Controlled	
Mixed sensory-motor	114 (84.4)	42 (41.17)	
Sensory	21 (15.56)	60 (58.82)	<0.00001
Total	**135(100.0)**	**102(100.0)**	

Percentage was mentioned within the parentheses;*Chi square test

Clinically severe neuropathy was more common in patients with uncontrolled DM whereas mild neuropathy predominated in controlled diabetic patients, (p-value <0.00001). Electrophysiologically, polyneuropathy was more severe in patients with uncontrolled group. Diabetic patients with good glycemic control predominantly had mild neuropathy electrophysiological (p-value <0.00001) (Table 6).

While comparing the distribution of different grades of clinical & electrophysiological severity of DPN in patients of controlled and uncontrolled DM, there was statistically no difference between the two grading scales (Table 7).

Table 6: Association of glycemic control and clinical & electrophysiological severity of DPN (n = 243)

Glycemic status	Clinical Severity			Electrophysiological Severity			
	Mild	Moderate	Severe	No	Mild	Moderate	Severe
HbA1C < 7%	62(57.4)	43(39.8)	3(2.8)	6(5.6)	63(58.3)	38(35.2)	1(0.9)
HbA1C: ≥7%	27(20.0)	47(34.8)	61(45.2)	0(0.0)	20(14.8)	49(36.3)	66(48.8)
P value		<0.00001*			<0.00001*		

*Chi square test; Percentage was mentioned within the parentheses

Table 7: Comparison between clinical and electrophysiological grading in patients of controlled & uncontrolled DM (n = 243)

Neuropathy	Controlled DM(n=108)		Uncontrolled DM(n=135)	
Grading	Clinical Grading	Electrophy Grading	Clinical Grading	Electrophy Grading
No Neuropathy	**0(0.0%)**	6(5.6%)	0(0.0%)	0(0.0%)
Mild Neuropathy	**62(57.4%)**	63(58.3%)	27(20%)	20(14.8%)
Moderate Neuropathy	**43(39.8%)**	38(35.2%)	47(34.8%)	49(36.3%)
Severe Neuropathy	**3(2.8%)**	1(0.92%)	61(45.2%)	66(48.9%)
P value	0.062*		0.527*	

*Chi Square test; Electrophy=Electrophysiological

Discussion

Two hundred & forty three patients of Diabetic Polyneuropathy (DPN) are included in this study. Majority of cases are aged in between 45 to 59 years (63.8%) in both sexes (62.1% and 65.5 % among males and females, respectively). Mean age is 57.2±9.37 years. Fifty one percent are male. Females are slightly higher in proportion than the male in the age group 45 to 59 years and above 75 years. The age distribution is consistent with most of the previous studies conducted in Bangladesh and Srilanka[3,9,11]. Sex-specific predisposition to DPN is observed with female preponderance in a study[12] while males being at higher risk in the Diabetes Control and Complications Trial (DCCT)[13], corresponding to the findings of present study. Majority of the cases are from urban areas (63%) with most having an average income of taka <5000 (37.9%) and taka 5000-10,000(48.6%). This observation matches with a Srilankan study[3] with the exception of maximum patients residing in urban areas, probably for samples here are taken from BIRDEM and DMCH, two core hospitals of Dhaka metropolitan city.

The mean HbA1c in the study population is 7.6±0.94 (%) with mean duration of DM 10.42±3.22 years. Maximum patients (56.8%) have a DM duration for >10 years. HbA_1c and longer duration of DM are strongly related with development of DPN which have been documented in a number of previous studies[9,14-17] which demonstrates the similar pattern of HbA_1c mean value and disease duration in patients of DPN. Maximum patients (56%) of the study population have HbA1c≥ 7%.

In this study, paraesthesia is the only symptom which is significantly more common in patients with uncontrolled DM (75.5%) than those of controlled DM (58.3%). This observation is supported by another study[15] whose cohort of patients with DPN show predominant symptoms in both sexes being paraesthesia followed by hypoaesthesia. On clinical examination, diminished lower limb tendon reflexes (49.4%) and diminished position and vibration sense (61.3%) are the commonest findings in both patients of uncontrolled and controlled DM, uncontrolled group (HbA1c≥7%) containing the higher proportions. The differences are statistically significant, consistent with the findings of several studies[12,18] showing only the absence of vibration sense correlates significantly with the higher values of HbA_1c in 88% of the patients with poorly regulated glycemia.

Nerve conduction studies of the motor nerves of the patients reveal that CMAP amplitudes in the median, ulnar, peroneal and tibial nerves are greatly reduced in patients of uncontrolled (HbA_1c≥7%) DM than those of controlled (HbA_1c<7%); the differences are statistically significant. Kovac et al[12] and Rodika et al[17] observe the similar type of result where the amplitude of motor response in median, tibial and peroneal motor nerves significantly correlates with glycaemic control, but the most important correlation is with CMAP in the median nerve. Motor nerve conduction velocity (MNCV) of ulnar, peroneal and tibial nerves are also significantly reduced in the uncontrolled glycemic status group, supported by the studies of Sultana et al[5] and Kovac et al[12]. In these studies, patients with higher levels of HbA_1c have longer distal latency of peroneal, ulnar and tibial nerves; but present study shows that although there is statistically significant difference between distal latencies of median, peroneal and tibial nerves, distal latencies are not actually prolonged in any group.

Sensory nerve conduction studies reveal significant differences in sensory nerve action potential (SNAP) amplitudes of median and ulnar sensory and sural nerves between the two groups, patients with uncontrolled DM having the greater reduction of amplitudes. Previous studies show the amplitude of sensory responses in all investigated nerves significantly correlate with glycaemic control; patients with higher levels of HbA_1c have lower conduction velocity of sensory fibers (SNCV) of ulnar and sural nerves compared to DM patients with lower HbA_1c[12]; however, the most important correlation is found between glycaemic control and SNAP in the sural nerve[14,17]. Electrophysiologically, most patients of this study have mixed sensory-motor polyneuropathy (65.43%) while pure sensory neuropathy constitute 34.57% of the study population. Other studies are consistent with this statistically significant observation.

Number of patients having clinically severe DPN are significantly higher (45.2%) within the uncontrolled group (HBA_1C: ≥7%) than that (2.8%) within the controlled (HbA_1c<7%) group. Mild clinical neuropathy predominates in controlled diabetic patients (57.4%) than the uncontrolled group (20%), the difference being statistically significant. Similarly, electrophysiological severity is more in uncontrolled DM (48.8%) and mild degree of electrophysiological changes in controlled DM (58.3%). Similar results are observed, though not using the exact clinical and electrophysiological scores used in this study, in the works of Kovac et al[12] as well as Tkac and Bril[19].

The clinical scoring system and electrophysiological

scoring system for grading severity of diabetic polyneuropathy which are Toronto Clinical Scoring System, TCSS & Michigan Diabetic Neuropathy Scoring System, MDNS, respectively are not applied simultaneously before this study. TCSS is an excellent scoring system which incorporates both the symptoms and signs of DPN in a simplified way. The MDNS scoring system also has a clinical part; however, it lacks the symptom analysis of DPN patients; the electrophysiological part includes involvement of maximum nerves. In the present study, a comparison is tried between the two scoring systems showing no significant difference between the number of patients among the neuropathy grades (mild neuropathy, moderate neuropathy & severe neuropathy) in both group of patients ($HbA_1c<7\%$ & $HbA_1c\geq7\%$). This indicates that TCSS scoring system is equivalent to the electrophysiological grading of MDNS, as almost similar proportion of patients in clinical grading have equivalent grading in their electrophysiology. Asad et al[14] similarly calculated the Diabetic Neuropathy Score (DNS) & evaluated their concordance with the NCS results; since exactly the same proportion of subjects who scored positively on the DNS showed electrophysiological alterations on the NCS, they proposed both ways seemed to be equally sensitive in detecting neuropathy.

The limitations of this study are that it is neither randomized nor prospective. Besides, late responses like F waves are not incorporated within the study parameters. Although Kovac et al[12] found a relationship of F latency with neuropathic severity; it is excluded to avoid the association of radiculopathies or plexopathies. Secondary demyelination may occur in severe diabetic polyneuropathy, the finding of demyelination cannot be ruled out in this study for presence of associated CIDP (Chronic Inflammatory Demyelinating Polyneuropathy).

Conclusion

In this study, glycemic control significantly affects both clinical and electrophysiological grading of diabetic polyneuropathy. However, in a country like Bangladesh, the observations of this study can be beneficial in the sense that only clinical scoring will be helpful to guide the physician to categorize the patients of DPN without opting for a costly investigation like NCS and thus emphasizing on early & vigorous management of glycemic control for better management of the patients. The Toronto Clinical Scoring System for grading of neuropathy is a simple but useful tool to categorize the patients of DPN. Further validation of this scoring system is required for practical utility for better management of patients.

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