

EARLY DIAGNOSIS OF NEUROPATHY IN DIABETIC PATIENTS USING NERVE CONDUCTION STUDIES

Background: Neuropathy is one of the common complications of diabetes, in which the patient's quality of life is compromised. Nerve conduction studies (NCS) are not commonly employed to detect the neuropathy.

Aims & Objective: To find out the utility of Nerve conduction studies (NCS) as early indicator of neuropathy in diabetic patients.

Materials and Methods: 50 diabetes mellitus patients with normal HbA1c levels and 50 diabetes mellitus patients with elevated HbA1c levels were selected, making it a total of 100 diabetes mellitus patients. 50 non-diabetic, healthy subjects were chosen as a control group. The nerve conduction velocity was tested in all the diabetic subjects and the healthy controls.

Results: The analysis showed that the nerve conduction velocity progressively decreased from the controls (49.0 ± 3.9) to the diabetics with a good glycaemia control (47.2 ± 2.8), to the diabetics with a poor glycaemic control (45.3 ± 3.1).

Conclusion: There is a progressive neuronal involvement in the diabetic process which is accelerated by poor glycaemic control. Therefore, nerve conduction studies can be employed for testing and for the early indication of neuropathy in diabetic patients.

Key Words: Nerve Conduction Velocity; HbA1c; Diabetes Mellitus; Diagnosis

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Received Date: 25.05.2013

Accepted Date: 04.12.2013

DOI:
10.5455/njppp.2014.4.041220132

INTRODUCTION

Diabetes mellitus, the most common endocrine disorder is characterized by metabolic abnormalities and in the long run with micro and macro vascular complications that cause significant morbidity and mortality.^[1] Diabetic neuropathy (DN) is one of the most commonly occurring microvascular complications accounting for 28% of all the complications in diabetics.^[2] It is a progressive process that has a long asymptomatic stage.^[3] It is important to identify neuropathy in the asymptomatic stages as the disease process progresses to the diabetic foot, a highly morbid condition that arises from the infection and the ulceration of the foot, finally leading to amputation.^[4] Early identification and glycaemic control are the key factors for preventing DN. The American Academy of Neurology recommends at least one of the five criteria for diagnosing DN: Symptoms, Signs, Electrodiagnostic tests, Quantitative sensory tests and Autonomic testing.^[5] Practically, electrodiagnostic tests are less utilized for the diagnosis or for the follow-up of DN. Nerve conduction studies (NCS) are electrodiagnostic tests which are used to evaluate the ability of the electrical conduction of the motor and the sensory nerves. Also, it is known that poor glycaemic control is responsible for microvascular complications.^[6] Glycated haemoglobin (HbA1c) has not only been established as a marker of glycaemic control but it also indicates the risk of developing small vessel complications.^[7] Therefore, we intended to correlate NCS with HbA1c to establish the role of NCS in diabetes

mellitus and spread of neuropathy, so that it can help in identifying the asymptomatic stage of DN and so that preventive measures can be instituted.

MATERIALS AND METHODS

This study was undertaken in the Department of Neurophysiology, King Abdullah Hospital Bisha Saudi Arabia, after the following approval from the research and ethical committees. Type 2 diabetic patients attending the OPD of the Hospital were selected after obtaining their consent. Male, right handed patients with established type 2 diabetes of 5 to 10 years duration and those who were on treatment were tested for glycated haemoglobin (HbA1c) levels. Based on the HbA1c levels, 50 diabetic patients with HbA1c levels of <7.0 and 50 patients with HbA1c levels of >7.0 were selected and grouped into group 1 and group 2 categories respectively. 50 age matched male, right handed volunteers, who were non diabetics and healthy were selected as controls and were grouped as group 3. The nerve conduction velocity was studied for the ulnar nerve of the right hand in all the 150 individuals. The neurophysiological measurements were performed in a warm room with the participants in a sitting position, with their forearms partially flexed. Nerve conduction velocity measurements were made by using Medtronic Keypoint® 2 EMG EP software. The surface metal plated, stimulating electrodes were placed at 5 cm below the medial epicondyle and at 5 cm above the medial epicondyle and the recording electrodes were placed over

the abductor digiti minimi (ADM) muscle on the ulnar side of the hand i.e. between the fifth metacarpophalangeal joint and the pisiform bone. The compound muscle action potentials (CMAPs) were evoked by the electrical stimulation (0.1 ms duration, constant current pulse) of the ulnar nerve, starting with a minimum and progressing to the maximum intensity of the stimuli. Latency, amplitude and nerve conduction velocity were assessed. The data was summarized to test the difference in the mean values between the groups 1, 2 and 3 by using the Students' t test; p values < 0.05 were taken as the level of significance. Further, Pearson's correlation was used to correlate between the different parameters. The HbA1c levels were determined by the borate affinity assay (Nycocard, AXIS-SHIELD PoC AS, Norway).

RESULTS

The study included 50 controls and 100 diabetics. Both the cases and the controls were aged between 40 to 60 years. The results are shown in the Tables 1 and 2. Table-1 shows the Mean \pm SD of HbA1c, nerve conduction velocity (NCV), total latency and amplitude in groups 1, 2 and 3. The nerve conduction velocity showed a progressive decrease with the HbA1c levels (49.0 \pm 3.9, 47.2 \pm 2.8 and 45.3 \pm 3.1 in controls, in diabetics with normal HbA1c levels and in diabetics with raised HbA1c levels respectively). Total latency showed a significant increase with poor glycaemic control in the diabetics. Table 2 shows the Pearson's correlation between the parameters. There was an inverse correlation between HbA1c and the nerve conduction velocity and a significant positive correlation with latency.

Table-1: Mean \pm SD of HbA1c, nerve conduction velocity (NCV), total latency and amplitude in groups 1, 2 and 3

Parameters	Groups			p - value		
	1	2	3	3 Vs 1	3 Vs 2	1 Vs 2
HbA1c (%)	5.3 \pm 0.8	8.2 \pm 1.6	4.5 \pm 0.5	< 0.55	< 0.001**	< 0.01*
NCV (m/s)	47.2 \pm 2.8	45.3 \pm 3.1	49.0 \pm 3.9	< 0.001**	< 0.001**	0.01*
Total Latency (ms)	4.8 \pm 2.1	4.9 \pm 2.2	4.6 \pm 1.9	< 0.001**	< 0.001**	< 0.01*
Amplitude (mV)	5.1 \pm 1.1	5.0 \pm 1.3	5.3 \pm 0.8	< 0.01*	< 0.001**	< 0.05*
DM Duration (years)	7.3 \pm 2.4	7.9 \pm 2.1	-	-	-	< 0.68

The values are expressed as their Mean \pm SD. Group 1: Diabetics with Normal HbA1c (n =50); Group 2: Diabetics with High HbA1c (n =50); Group 3: Controls (n =50); * Significant (p<0.05); ** Highly significant (p<0.001); Not significant (p>0.05); NCV: Nerve Conduction Velocity

Table-2: Pearson's correlation between the parameters

Relationship Between	r - Values	p - Value	Significance
HbA1c vs. NCV	-0.43	< 0.01	S
HbA1c vs. TL	+ 0.33	0.07	NS
HbA1c vs. Amplitude	- 0.14	0.01	S

r = Pearson's correlation co-efficient; HS = highly significant (p<0.001); S = Significant (p<0.05); NS = Not significant (p>0.05)

DISCUSSION

Diabetic neuropathy is a common complication of diabetes mellitus with severe morbidity, compromising the quality of life. An intensive treatment of neuropathy at the sub clinical level decreases the risk of neuropathy.^[8] Therefore, there is a need of methods to identify the at-risk diabetic patients for neuropathy. Nerve conduction studies are one of the important methods for assessing nerve functions in DN. In this study, it was observed that the nerve conduction velocity progressively decreased from the controls (49.0 \pm 3.9) to the diabetics with good glycaemic control (47.2 \pm 2.8), to the diabetics with poor glycaemic control (45.3 \pm 3.1). These findings are in accordance with those of previous researchers.^[9] Bansal et al (2006) have suggested that the slowing of NCV indicates the ongoing damage to the myelin sheaths and they are also of the opinion that the amplitude decreases with the rising HbA1c levels, thus suggesting the onset of axonopathy.^[10] Therefore, the monitoring of diabetic patients with NCS may help in predicting the onset of DN. Since this was a cross sectional study, follow-up studies and interventional studies are required to emphasize the importance of the NCV estimation. Since the underlying inflammation was the cause for establishing the diabetes and its complications, this study would have been strengthened if it had correlated with one of the inflammatory markers like hsCRP. In conclusion, the estimation of both NCV and the HbA1c levels in diabetics is helpful in identifying the risk category for DN, which is one of the main causes for severe morbidity among the diabetes mellitus patients.

CONCLUSION

There is a progressive neuronal involvement in the diabetic process which is accelerated by poor glycaemic control. Therefore, nerve conduction studies can be employed for testing and for the early indication of neuropathy in diabetic patients.

ACKNOWLEDGEMENT

The authors are deeply indebted to the subjects of the study without whose co-operation this endeavor would not have been possible and to the authorities of the hospital, our sincere thanks to Dr. Morris AL Dayoub, consultant neurologist King Abdullah Hospital, Bisha Saudi Arabia for his guidance and support.

REFERENCES

1. Zargar AH, Wani AI, Masoodi SR, Laway BA, Bashir MI. Mortality in diabetes mellitus – data from a developing region of the world. *Diabetes Res Clin Pract.* 1999; 43: 67-74.
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. Perkins BA, Bril V. Diagnosis and management of diabetic neuropathy. *Curr Diab Rep.* 2002; 2: 495-500.
4. Partanen J, Niskanen L, Lehtinen J, Mervaala E, Siitonen O, Uusitupa M. Natural history of peripheral neuropathy in patients with non-insulindependent diabetes mellitus. *NEJM.* 1995; 333: 89 -94.
5. Consensus statement. Report and recommendations of the San Antonio conference on diabetic neuropathy. American Diabetic Association, American Academy of Neurology. *Diabetes Care.* 1988; 11: 592-7.
6. Mayurasakorn K, Somthip N, Caengow S, Chulkarat N, Wanichsuwan M. Glycemic control and microvascular complications among type 2 diabetes at primary care units. *J Med Assoc Thai.* 2009; 92: 1094-101.
7. Chandalia HB, Krishnaswamy PR. Glycated hemoglobin. *Current Science.* 2002; 83: 1522-1532.
8. Dahl-Jorgensen K, Brinchmann-Hansen O, Hanssen KF, Ganes T, Kierulf P, Smeland E, et al. Effect of near normoglycaemia for two years on progression of early diabetic retinopathy, nephropathy, and neuropathy: The Oslo Study. *Br Med J.* 1986; 293: 1195-1199
9. The DCCT Research Group. Factors in the development of diabetic neuropathy in feasibility phase of Diabetes Control and Complications Trial (DCCT). *Diabetes.* 1988; 37: 476-481.
10. Bansal V, Kalita J, Misra UK. Diabetic neuropathy. *Postgrad Med J.* 2006; 82: 95-100.

Cite this article as: Parkhad SB, Palve SB. Early diagnosis of neuropathy in diabetic patients using nerve conduction studies. *Natl J Physiol Pharm Pharmacol* 2014; 4:158-160.

Source of Support: Nil

Conflict of interest: None declared