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Nerve conduction velocity in diabetes mellitus type II

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Abstract
Neuropathy is the most serious complications of diabetes mellitus. Neuropathy may impact sensory defects, neurological disabilities, limb ulcers, osteomyelitis, and amputation. The purpose of the study is to prevalence of neuropathy in asymptomatic diabetics. To study type of neuropathy in asymptomatic diabetics. The present study was done to determine the occurrence of sensorimotor neuropathy in patients having diabetes mellitus type 2 with no symptoms of neuropathy, to determine whether any co relation exits between duration, blood sugar levels and HbA1C to NCS. The study included total 80 asymptomatic diabetics who were divided into two groups. These patients were diagnosed cases of DM type 2 and were not having any symptoms of neuropathy. The results showed that there were significant relation between glycemic index and neuropathy. The prevalence of diabetic neuropathy is less in patients who are having good glycemic control.

Keywords: neuropathy, diabetes mellitus, nerve, asymptomatic, ncs, hba1c

Introduction
Diabetes Mellitus is the third most frequent source of morbidity and mortality related to coronary disease and malignancies. As per recent WHO estimates, India currently has 32 million diabetic subjects and is projected to increase to 100 million, i.e. by 25 per cent by 2035. This ensures that India will contributes one-fifth (more than 20 per cent) of overall diabetic population of the world [1, 2]. Diabetic neuropathy (DN) is the most popular and serious complication of D.M. It leads to high death and morbidity. This encompasses a broad variety of disorders affecting both peripheral and autonomic nerve activity [3].

Aim
Role of Nerve Conduction Velocity in Asymptomatic Diabetes Mellitus Type II.

Objectives
To study the prevalence of neuropathy in asymptomatic diabetics. To study type of neuropathy in asymptomatic diabetics.

Review of Literature
'Diabetes mellitus' is characterized by' chronic hyper glycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both' [4].
The worldwide prevalence of diabetes mellitus has risen dramatically over the past two decades. It is expected to rise further because of sedentary life style, increasing obesity, and increased life expectancy [5]. Roll is credited with having recorded this association in 1798, and until the middle of the 19th century, diabetes itself was attributed to a primary disorder of the central nervous system. It was Marchel de Calvi in 1864 who first suggested that diabetes might be the cause rather than the effect of neuropathy [6]. Pavy's description in 1885 of neuropathic symptoms is noteworthy for its completeness as we know today [7]. 'Diabetic neuropathy is a descriptive term meaning a demonstrable disorder, either clinically evident or sub-clinical, that occurs in the setting of diabetes mellitus without other causes for peripheral neuropathy. The neuropathy disorder includes manifestations in the somatic and / or autonomic parts of the peripheral nervous system' [8].
The classification by Thomas and Tomlinson in 1993 divided diabetic neuropathies into symmetrical, asymmetrical, and mixed forms [9]. The first group consisted of distal symmetrical polyneuropathies, the second of focal and multifocal neuropathies and the third of mixed varieties. One study found that in newly diagnosed diabetics the prevalence of neuropathy to be 11.6% [10]. Another study, in their current opinion, have stated that the average prevalence was 28% with a range of 15-60%. The diagnosis of DN was based on clinical abnormalities, QSE and autonomic testing. The independent risk factors stated were duration of diabetes, HbA1C, triglyceride levels, hypertension, severe DKA, and retinopathy [11]. In another study conducted in NIDDM patients, the prevalence of neuropathy was in the range of 35- 40 % [12]. One study showed that most patients present with predominantly sensory symptoms and that too positive symptoms like pain and paresthesia. They also concluded that these symptoms were present in both upper limbs and lower limbs at presentation and that too distally [13]. However another study stressed the predominance in the lower limbs [14]. Case reports on the presence of diabetic neuropathy at diagnosis were brought to attention by a study [15], but another study found clinical signs of neuropathy in 7.5% of diabetics at diagnosis of diabetes. In a study of 235 patients over 20 years, it

revealed that neuropathies appeared about a decade earlier and severer in symptoms and signs than in affluent countries, probably due to poorly controlled diabetic status. DN was the most common form of peripheral neuropathy they observed, again reflecting the common nature and the seriousness of this disorders [16]. The average interval between the onset of DM and neuropathy was approximately 4 years, which was similar to a study done, which showed that sustained hyperglycemia is related to at least functional changes in peripheral nerves on sensory and motor conduction at a duration of 4 years, with a slightly greater prevalence among males than in females [17]. The most common symptoms observed in one study were: sensory - pain and paraesthesia (67.2%) and the most common sign was loss of vibration sense. In 5% of patients, the neuropathic symptoms seemed to precede the symptoms of DM, while in approximately 34.5%, the two symptom categories were simultaneous. All types of presentations were seen, from simple mono-neuropathy (meralgia-paraesthetica) or cranial nerve involvement, to severe generalized poly-neuropathy. But they have not commented on the exact prevalence of this syndrome, especially the sensorimotor type, but have mentioned it as the most common. Metabolic factors were implicated as the cause, as the biopsy studies did not show any vasa nervora involvement [16].

One study found NCV abnormalities in more than 80% of asymptomatic recently diagnosed diabetics of one year duration [18]. Another study found that the peripheral nerve function in newly diagnosed diabetics treated with OHA's and insulin showed impairment of NCV present in them both and there was an improvement following treatment, in only the insulin treated patients [19]. A study reported reduced NCV in 20% of diabetic of 5 years duration3 while another found significant decrease in NCV and latency rates especially in newly diagnosed diabetics of one year duration with no correlation between the two or with fasting hyperglycemia and duration of diabetes [20].

Material and Methods

Patients, Male / Female, coming to OPD/IPD of Krishna hospital, who has been a diagnosed case of diabetes mellitus type 2 were taken for this study. The patients should be diagnosed as DM type II to for more than a year and should have no symptoms of neuropathy. Diagnosed case of diabetes mellitus Type II for more than 1 year. No symptoms suggestive of neuropathy. The patients would also be screened for glycosylated haemoglobin (HbA1C) performed in department of biochemistry on choral kit at Krishna hospital.

Observations

Table 1: Mean Age, Mean FBS, Mean PPBS, Mean HBA1C

	Group	N	Mean	SD	P-value
AGE	Group B	40	53.65	6.39	0.761
	Group A	40	53.20	6.81	
FBS mg/dl	Group B	40	169.45	48.92	0.016
	Group A	40	207.55	84.25	
PP mg/dl	Group B	40	209.63	75.78	0.022
	Group A	40	250.88	81.53	
HbA1C %	Group B	40	5.66	0.63	< 0.001
	Group A	40	10.36	2.23	

There were total 40 patients each in both the groups as shown in Table 1. Group A (HbA1C 7.1%) & Group B (HbA1C 7.0%). The mean age in group B and group A was 53.65, 6.39 and 53.20, 6.81 years respectively. The mean FBS in Group B and group A was 169.45, 48.92 mg % and 207.55, 84.25 mg % respectively. The mean PPBS in Group B and Group A was 209.63, 75.78 and 250.88, 81.53 respectively. The mean HbA1C in Group B and Group A was 5.66, 0.63 and 10.36, 2.23 respectively.

Table 2: Relation of Treatment to Abnormal NCS Report

Treatment	NCS		Total
	Normal	Abnormal	
Insulin	9	1	10
Oha	17	30	47
Oha + Insulin	15	8	23
Total	41	39	80

In our study total 41 patient were having normal NCS report out of which 9 (21.95%) were on insulin alone 17 (41.46%) were on OHA'S and 15 (36.58%) were on OHA's + insulin (p=0.001). There were 39 patients with abnormal NCS report out of which 1 (2.56%) was on insulin 30(76%) on OHA'S and 8 (20.51%) on 0HA+Isulin, showing in table 2.

Table 3: Distribution With Respect to Gender

Sex	Group B	Group A	Total	P-value
Male	24	19	43	0.37
Female	16	21	37	
Total	40	40	80	

In group A there were total 19 (47.5%) males and 21 (52.5%) females. In group B there were 24 (60%) males and 16 (40%) females.

Table 4: Relation between Duration of Diabetes and Abnormal NCS Report

Duration of DM in Yrs	Abnormal NCS Report			
	Group A		Group B	
	M	F	M	F
1-3	5	3	1	
3-5	7	7	3	1
5-7	1	3	2	1
More Than 7	1	0	1	0

As shown in Table no. 4, the maximum number of patients 14 (46.66%) in group A were between 3-5 years of duration of DM where as in group B 4 (44.44%) were in the same duration as seen in group A. Duration of more than 5 years shows less number of patients with abnormal NCS report as such patients becomes symptomatic and here they were excluded from the study.

Discussion

The present study was conducted to determine the occurrence of neuropathy in asymptomatic diabetics and also to determine whether any association is existing between the duration and glycemic index to the electrophysiological study done. The study was divided into two groups Group A: HbA1C more

than 7.1% and Group B: HBAIC less than 7.0%. No similar studies keeping this margin of duration has been so far determined from the extensive search of literature, although Pirart says that the prevalence is about 7.5% at the time of diagnosis of diabetes. Lehtinen *et al* from Finland have studied the prevalence of DN in newly diagnosed diabetes where it was diagnosed as within 4 weeks of diagnosis date[21]. Diabetic patients who are on OHA's are found to have more clinical symptoms of neuropathy and their electro physio logical studies done are highly abnormal. Those patients on insulin are found to be asymptomatic and have normal electrophysiological study. Hence insulin could be protective against diabetic neuropathy probably because of good glycemic control.

Conclusion

The prevalence of diabetic neuropathy in asymptomatic diabetics is very high in patients with poor glycemic control. Nearly 80% of the patients who were having abnormal NCS in both the group were in the age group of 51-60 years. Maximum number of patients having abnormal NCS were having DM type 2 in the duration of 3-5 years. This is because the patients who were in the duration of 5-7 years were mostly symptomatic and were excluded from this study. The prevalence of diabetic neuropathy is less in patients who are having good glycemic control. In our study total 41 patient were having normal NCS report out of which 9(21.95%) were on insulin alone, 17 (41.46%) were on OHA'S and 15 (36.58%) were on OHA's + insulin (p=0.001). The electrophysiological methods are more sensitive tools to detect diabetic neuropathy in asymptomatic diabetics. The neuropathy was mainly of axonal type in both the groups 18 (60%) patients were having axonal type of neuropathy 6 (20%) patients were having demyelinating type and 6 (20%) were having mixed neuropathy. In group B 9 (100%) patients were having axonal type of neuropathy. Hence the most common type of neuropathy seen in diabetes mellitus type 2 is axonal type of neuropathy.

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