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Diabetic Neuropathy: Discordance between Symptoms and Electrophysiological Testing in Saudi Diabetics

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Objective: To determine the prevalence of nerve conduction velocity alteration in symptomatic diabetic neuropathy and to report the associated risk factors.

Method: A cross sectional study of Saudi diabetics followed up in the medical outpatient clinic at King Abdulaziz University Hospital between January 1998 to May 1999. Diabetic neuropathy was diagnosed using the Michigan Neuropathy Program. Patients included in the study were those who had symptomatic neuropathy and scored >2 on simple clinical examination. Nerve conduction studies were done, detailed information of each patients age, sex, Body Mass Index (BMI), type and duration of diabetes, mode of treatment, degree of glycemic control, presence of hypertension, hyperlipidemia and smoking were recorded.

Results: A total of 136 patients were included in the study with mean age of 56.2 years and mean duration of diabetes 12.49 years. Normal nerve conduction studies were found in 36% of the patients. Patients using insulin , prolonged and poorly controlled diabetes were the most important risk factors associated with diabetic neuropathy ($p<0.001$).

Conclusion: The discordance between symptoms and nerve conduction studies means that we need nerve conduction studies for the proper diagnosis of diabetic neuropathy. Prolonged, poorly controlled diabetes and insulin use were risk factors associated with diabetic neuropathy. Aggressive/strict control of blood glucose is the key in the ultimate prevention of diabetic neuropathy.

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Diabetic neuropathy (DN) is a common complication of diabetes mellitus (DM) that eventually affect the majority of diabetic patients and is associated with significant morbidity and disability[1-3]. Nerve conduction velocity (NCV) alteration may not be concordant with signs and symptoms of DN[4,5].

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The aim of our study is to determine the prevalence of NCV alteration in symptomatic DN and to report on the risk factors associated with it.

METHOD

This is a cross sectional study in which Saudi diabetic patients being followed in the medical outpatient clinic of King Abdulaziz University Hospital (KAUH) Jeddah, from January 1998 till May 1999.

All patients had established DM classified as either type I or II using WHO criteria[6]. Neuropathy was diagnosed using the Michigan Neuropathy Program[7] which is a two-step program, Diabetic Neuropathy Index (DNI) and Diabetic Neuropathy Score (DNS). Patients were initially screened for DN using 15 yes or no questionnaire and a simple clinical examination (which consist of foot inspection, an assessment of vibration sensation on the great toe and the presence of ankle reflex) known as DNI. Patients who were symptomatic and scored more than 2 on simple clinical examination were selected for the study and referred to a neurologist for the second component of the program, which is the DNS. This component consists of a more complete neurological examination of sensation in the feet, distal strength and reflexes. It is followed by nerve conduction studies (NCS) (motor and sensory conduction velocities of common peroneal and median nerve) performed with the temperature maintained at 22^0 C using standard protocols[8]. A nerve is considered abnormal if the calculated conduction velocity is less than average conduction velocity which is defined as values between the 1^{st} and 99^{th} percentile[8]. Nerve conduction abnormalities were classified into normal and abnormal (mild, moderate and severe abnormalities) according to common peroneal (CPN) and median nerve (MN) conduction. The values for CPN are: Normal > 44.4 m/s, mild 40-44.3 m/s, moderate 36-39.9 m/s and severe <36 m/s. Whereas the values for MN are: normal > 52.8 m/s, mild 48-52.7 m/s, moderate 40-47.9 m/s and severe <40 m/s. The following information were recorded: age, sex as well as their body mass index (BMI) (weight in kilograms divided by square height in meters), type and duration of DM and type of treatment (diet, oral hypoglycemic agents, insulin or combined). Poorly controlled patients were diagnosed by their non-compliance to diet and medications, symptoms of hyperglycemia and HbA1c > 7%. The presence of hypertension, hyperlipidemia and smoking were recorded. Statistical analysis was done using the Statistical Package for Social Sciences (SPSS 7.5). T- test and Chi-square were used appropriately. Results were considered significant if the P value is less than 0.05.

RESULTS

A total of 136 patients fulfilled the criteria and were included in the study. The mean age was 56.20 years (17-80 years) with male to female ratio 1:1.6 (53:83) and mean BMI 28.62 ± 4.85. Most of the patients were type II, 120 (88.2%) while 16 (11.8%) were type I. The mean duration of DM was 12.49 ± 6.81 years. Oral hypoglycemic agents (OHG) were the treatment used by 81 patients (59.6%), followed by insulin 46 (33.8%), diet 5 (3.7%) and combined OHG and insulin in 4 (2.9%). Poor glycemic control was found in 87 patients (64%) while 49 (36%) were well controlled. Forty-eight (35.3%) patients

were hypertensive while hyperlipidemia was found in 36 (26.5%) and a history of smoking in 29 (21.3%). Normal NCS were found in 49 patients (36%). Abnormal NCS were found in 87 patients (64%) of which most of them had mild conduction defects 37 (42.5%), while moderate defects were found in 26 (29.8%) and severe in 24 (27.5%).

Nerve conduction abnormalities in symptomatic patients were significantly related to poor glycemic control. Seventy-one (81.6%) poorly controlled patients had abnormal NCS as compared to 16 (18.4%) well controlled patients (P <0.001). Long duration of DM was also strongly related to abnormalities in NCS, the mean duration of DM in patients with NCS abnormalities was 14.4 years as compared to 9.1 years in those with normal NCS (P <0.001). Abnormal NCS were also significantly associated with insulin use, 32 (69.6%) of those on insulin showed abnormal NCS compared to 14 (30.4%) who showed normal NCS. There was no significant relation between abnormal NCS and patients age (p0.4) sex (p0.7) BMI (p0.36), type of DM (p0.1), hypertension (p0. 5), hyperlipidemia (p0.23) or smoking (p0.13).

DISCUSSION

DN is a common complication of DM and it is encountered in more than one third of diabetic patients[9]. Pirar et al[10] had found a five fold increase in the incidence of DN after 25 years of follow up. Discordance between nerve conduction velocity and symptoms and signs of DN has been reported before[4,5]. We found that 36% of our patients with symptomatic DN had normal NCS, which is higher than that reported by Sangiorgio et al[4] and Fedele et al[5]. This discordance between symptoms and NCS means that we can not rely on patient's symptoms for the diagnosis of DN and we need NCS for better assessment and diagnosis of DN.

Prolonged and poorly controlled DM were the most significant factors associated with DN and this has been reported by others[4,5,11,12]. Cheng et al[13] has also reported a significant relation between insulin use and DN and this is similar to our findings. As reported by Hillson et al[14] and Maser et al[15] no significant relation has been found between DN and sex, BMI, hypertension and hyperlipidemia which is in agreement with our findings.

The relation between smoking and DN varies, some reports showed significant relation[11] while others[16] didn't find any relation. We found no significant relation between DN and smoking. No significant relation has been found between age and abnormal NCS, while on the other hand a strong relation was found with poor glycemic control, this means that even young patients can develop alteration in NCS if they are not well controlled. As the pathogenic mechanisms of DN are not fully understood, there is no satisfactory and fundamental therapy for DN. Therefore, further researches are needed especially into pathogenic mechanisms in order that satisfactory treatment is achieved. Good glycemic control is essential if the risk of diabetic complications is to be minimized[17]. There was a strong relation between baseline glycated hemoglobin and the loss of tactile sensation and temperature sensation[18]. Intensive diabetic control had been shown to reduce the occurrence of clinical neuropathy by 60%[19,20]. Several prospective randomized clinical

trials have shown the beneficial effect of tight glycemic control on the progression of chronic microvascular complications of DM[21-23]. This means that strenuous control of blood glucose is the key in the ultimate prevention of DN.

CONCLUSION

The discordance between symptoms and nerve conduction studies means that we need nerve conduction studies for the proper diagnosis of diabetic neuropathy. Prolonged poorly controlled diabetes and insulin use were risk factors associated with diabetic neuropathy. Aggressive/strict control of blood glucose is the key in the ultimate prevention of diabetic neuropathy.

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