

Prevalence of Peripheral Neuropathy in Type 2 Diabetic Patients

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مدى انتشار التهاب الأعصاب المحيطية عند مرضى السكري نوع ٢

الخلاصة:

- من أجل تقييم مدى انتشار اعتلال الأعصاب المحيطية لدى مرضى السكري، فحص ١٠٥ من مرضى السكري الذين كانت أعمارهم (52 ± 7.8) سنة، ٥٤ من الذكور و ٥١ من الإناث.
- دراستنا اعتبرت إمكانية تشخيص الاعتلال العصبي المحيطي عند وجود اثنين أو أكثر من شذوذ ثلاثة: أعراض الاعتلال العصبي السكري، وفحص الأعصاب سريريا ودراسات التوصيل العصبي.
- كان انتشار الاعتلال العصبي المحيطي في مرضى السكري ٧٦.١٩ %.
- وجد أن العمر، نسبة الهيموغلوبين السكري وأمد الإصابة بالسكري له أثر في انتشار الاعتلال العصبي المحيطي ($P < 0.05$) في حين الجنس، مؤشر كتلة الجسم، نوع العلاج، لمحة الدهون (الكوليسترول الكلي في الدم، دهون عالية الكثافة، دهون منخفضة الكثافة، الدهون الثلاثية عند الصيام) لم تؤثر على انتشار الاعتلال العصبي المحيطي ($P > 0.05$).

Abstract:

- In order to assess the prevalence of diabetic peripheral neuropathy in diabetic patients 105 type 2 diabetic patients aged (52 ± 7.8) years, 54 males and 51 females were investigated.
- This study considered the diagnosis of peripheral neuropathy and it was made when two or more of the following three abnormalities: diabetic neuropathy symptom, diabetic neuropathy examination and nerve conduction studies were present.
- Prevalence of peripheral neuropathy in diabetic patients was 76.19 %
- It has been found that age, HbA1c % and duration of diabetes mellitus (DM) significantly effect the prevalence of peripheral neuropathy ($P < 0.05$), while gender, body mass index, therapeutic regimen of DM and lipid profile (total serum cholesterol, high density lipoprotein, low density lipoprotein and fasting triglyceride) not effect the prevalence of peripheral neuropathy ($P > 0.05$).

Keywords: Type 2 diabetes mellitus, Peripheral neuropathy, Nerve conduction study

Introduction:

Diabetes mellitus is a chronic disease that occurs when the pancreas does not produce enough insulin, or when the body cannot effectively use the insulin it produces. Hyperglycaemia is a common effect of uncontrolled diabetes and over time leads to serious damage to many of the body's systems, especially the nerves and blood vessels.⁽¹⁾

More than 220 million people worldwide have diabetes. Type 2 diabetes comprises 90% of people with diabetes around the world, and is largely the result of excess body weight and physical inactivity.⁽²⁾

Diabetes mellitus (DM) is a common cause of peripheral neuropathy worldwide. In different studies, the incidence of neuropathy in diabetic patients varies from 10 to 50 percent; a wide variability, is related to the lack of consistent criteria for the definition of peripheral neuropathy.⁽³⁾

Peripheral neuropathy is a common microvascular complication of diabetes. Diabetic neuropathy is recognized by the American Diabetes Association (ADA) as "the presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes after the exclusion of other causes".⁽⁴⁾

As with other microvascular complications, the risk of developing diabetic neuropathy is proportional to both the magnitude and duration of hyperglycemia, and some individuals may possess genetic attributes that affect their predisposition to developing such complications.⁽⁵⁾

The precise nature of injury to the peripheral nerves from hyperglycemia is not known but is likely related to mechanisms such as polyol accumulation, injury from advanced glycation end products (AGEs), and oxidative stress.⁽⁶⁾

Peripheral neuropathy in diabetes may manifest in several different forms, including sensory, focal/multifocal, and autonomic neuropathies.

Clinical manifestations of peripheral neuropathy:

Diabetic polyneuropathy is primarily a symmetrical sensory neuropathy, initially affecting the distal lower extremities. With disease progression, sensory loss ascends and, when reaching approximately mid-calf, appears in the hands. This gradual evolution causes the typical "stocking-glove" sensory loss. This pattern reflects preferential damage according to axon length; the longest axons are affected first. Motor involvement with frank weakness occurs in the same pattern, but only later and in more severe cases.⁽⁷⁾

The earliest signs of diabetic neuropathy are loss of vibratory sensation and altered proprioception reflect large-fiber loss, and impairment of pain, light touch and temperature secondary to loss of small fibers.

Diagnosis of diabetic peripheral neuropathy:

The assessment of patients with diabetic neuropathy begins with a detailed neurologic and medical, history and examination and continues with electrodiagnostic studies. Up to this date nerve conduction studies (NCS) remain the most reliable, accurate and sensitive measure of peripheral nerve function in diabetic neuropathy.⁽⁸⁾

Nerve conduction studies (NCS) are the most sensitive and specific DPN detection method.⁽⁹⁾ The information is extremely detailed to the extent that the cellular pathology of a patient's neuropathy is usually defined best by physiological testing rather than by biopsy.

Nerve conduction velocity (NCV) alteration may not be concordant with signs and symptoms of DN.⁽¹⁰⁾ It is generally agreed that DN should not be diagnosed on the basis of one symptom, sign, or test alone: a minimum of two abnormalities (symptoms, signs, nerve conduction abnormalities, quantitative sensory tests, or quantitative autonomic tests) is recommended.⁽¹¹⁾

Nerve conduction can be measured orthodromically (physiological direction of nerve conduction) or antidromically (opposite to the physiological direction). Motor and sensory nerve action potentials can be measured through skin electrodes if the nerve is sufficiently superficial. It is more common (and technically easier) to measure motor nerves by picking up electrical activity from the muscle it innervates.

Electrodiagnostical findings in peripheral neuropathy may include:

1. Decreased conduction velocity in demyelinating neuropathies (decreased motor conduction velocity in motor demyelinating neuropathies and decreased sensory conduction velocity in sensory demyelinating neuropathies).
2. Decreased amplitude of the CMAP or SNAP in axonal neuropathies (decreased CMAP amplitude in motor axonal neuropathies and decreased SNAP amplitudes in sensory axonal neuropathies).⁽¹²⁾

Patients and Methods:

Study population:

This study involved 105 type 2 diabetic patients, 54 males and 51 females, aged 35–60 years with a mean of (52 ± 7.8) years.

The patients were randomly selected from diabetic clinic for research and treatment of diabetes mellitus in the Al-Sader Medical City in Al – Najaf city.

All patients were informed about the aim of the study and the technique of nerve conduction test and they were convinced to participate in this study then the electrophysiological tests of peripheral nerves were done at the unit of neurophysiology in Al-Sader Medical City in Al – Najaf city.

In this study we evaluate the effect of many variables (age, gender, BMI, duration and control of DM, therapeutic regimen of DM, and lipid profiles) on the development of peripheral neuropathy.

The diagnosis of diabetic peripheral neuropathy was made when two or more of the following three abnormalities were present; symptoms of diabetic neuropathy, signs of diabetic neuropathy and positive nerve conduction studies.

It was difficult to find uniform normal values of NCS for selected nerves because of different distances and different sites of stimulation were used in different studies and the resulting normal values were different also accordingly. Also in this study we did search for symmetrical polyneuropathy and cannot depend on adjacent nerve or contralateral nerve as standard value.

For reference normal values, this study depends on ⁽¹³⁻¹⁶⁾ because their technique is more similar to the technique used in this study for measurement of the selected parameters.

Materials:

Micromed System-plus EMG machine was used for electrophysiological analysis of sensory and motor nerve fibers conduction studies. This system includes eight channels preamplifiers and built-in two isolated stimulators with separate jacks.

The stimulus intensity can be manually adjusted (0-99 mA), and the evoked responses can be displayed on the monitor, on which four channels can be displayed at the same time. The machine also contains an audio-amplifier which helps to localize the site of stimulation of the nerve in case of the NCS.

For sensory nerve conduction study (SNCS), a pair of finger ring electrodes was used to record the antidromic responses from the skin of the finger innervated by the nerve to be tested. Each electrode is formed of a stainless spring with a small rubber fixator to keep them tight fitted on the finger size. They were connected to the amplifier socket by an electrode cable. For Sural nerve recording, surface electrode just like that of the motor recording were used.

For motor nerve conduction study (MNCS), a pair of surface cup electrodes was used to pick up the responses from the muscle enervated by the nerve to be tested. Each electrode is formed of a cup disc with 7 mm diameter. These surface electrodes were connected to the amplifier by an electrode cable.

Methods:

Neuropathy was evaluated using a questionnaire on neuropathic symptoms and Neuropathy Symptoms Score (NSS) like presence of neuropathic pain, parasthesia and anesthesia. The calculation was done as present (1) or absent (0). Neuropathic pain was defined as pain in the limbs in the absence of a history of trauma or other external cause.

Paraesthesia was defined as a sensation which is characteristically perceived as tingling, numbness, sharpness or burning while anesthesia defined as a loss of sensation.⁽¹⁷⁾

Three clinical tests were carried out including **ankle reflexes** bilaterally (most sensitive to early diabetic peripheral neuropathy (DPN)), **joint position** sense (of the interphalangeal joint of the big toe and thumb) and sensation to **pinprick** over the centre of the palm and sole. The latter was compared to pinprick sensation at the midsternal area as reference. The relevant physical sign was defined as absence of joint position sensation, an absent ankle reflex and reduced pinprick sensation in the tested area compared to the reference area over the sternum. ⁽¹⁸⁾

All patients were investigated for serum creatinine, Lipid profiles (Total cholesterol, HDL, LDL and Triglyceride), and HbA1c.

Nerve Conduction Study:

Nerve Conduction Studies were done for all diabetic patients using Micromed System-plus EMG machine. The simplified nerve conduction studies (NCS) protocol was followed as below:

- 1) Sural sensory and peroneal motor NCS were performed in one lower extremity. If both studies were normal, NCS was discontinued.
- 2) If sural sensory or peroneal motor NCS were abnormal, NCS of at least the ulnar sensory, median sensory, and ulnar motor nerves in one upper extremity were done.
- 3) If a response was absent for any of the nerves (sensory or motor), NCS of the contra lateral nerve was performed.
- 4) If a peroneal motor response was absent, an ipsilateral tibial motor NCS was performed. ⁽¹⁹⁾

Polyneuropathy affects multiple nerves and may affect more than one extremity, often on both sides of the body (symmetric). ⁽²⁰⁾

Measuring the variables of NCS:

1. Sensory nerve fibers measurements including: latency, conduction velocity and amplitude for (median, ulnar, and sural nerves).
2. Motor nerve fibers measurements including: distal motor latency, motor conduction velocity and amplitude for (median, ulnar, posterior tibial and common peroneal nerves).
3. F-wave measurements including the latency.

Neurophysiological tests were done in a quiet environment in the examining room at temperature 25 to 28 °C.⁽¹⁵⁾

The antidromic method was used for the determination of the sensory conduction parameter as it is less time consuming than the orthodromic method and is more easier to the subject and more informative because the nerve is more superficial distally at the fingers.⁽¹⁵⁾

Square wave electrical shocks of 0.1 msec. duration and a frequency of 1 stimulus/ sec were used for stimulation. This stimulus intensity was adjusted by gradually increasing the stimulation current until maximum potentials were evoked on the screen.⁽²¹⁾

For Motor nerve conduction study, Square wave electrical shocks of 0.1 msec is used, and the duration and a frequency of 1 stimulus / sec were used for stimulation. This stimulus intensity was gradually increased until maximum response evoked on the screen. Then 20 to 30 % supramaximal stimulation was used to guarantees the activation of all the axons innervated the recording muscle. ⁽¹⁵⁾

Statistical analysis are presented as numbers with percentages for discontinuous categorical qualitative variables or means \pm standard deviation for continuous quantitative variables. Data were collected, arranged and classified using SPSS Statistics 17.0 and Microsoft Excel computerized programs.

Results:

This cross-sectional study involved 105 diabetic patients aged (52 ± 7.8) years, 54 males and 51 females. All patients were exposed to nerve conduction study.

1) Conduction velocity: In all patients sural nerves (sensory) and some of other nerves (sensory and motor) show a decrease in conduction velocity, so sural nerve in all diabetic patients with peripheral neuropathy is affected (Table 1).

Table 1: The results of conduction velocity according to different nerves.

Nerve	Conduction velocity (m/sec)		
	Diabetic without Peripheral neuropathy Mean \pm SD (n = 25)	Normal range Mean (lower limit)	Diabetic with Peripheral neuropathy Mean \pm SD (n = 80)
Median (motor)	52.9 \pm 4.4	57.7 (48)	47.8 \pm 6.6
Median (sensory)	47.7 \pm 3.4	56.2 (44)	41.2 \pm 7.5
Ulnar (motor)	54.7 \pm 5.9	58.7 (49)	52.4 \pm 6.97
Ulnar (sensory)	53 \pm 3	54.8 (44)	50.1 \pm 10
Common Peroneal	45.6 \pm 5.5	48.3 (40)	41.1 \pm 7.3
Posterior Tibial	47.1 \pm 5.8	48.5 (41)	39.4 \pm 8.9
Sural	50.6 \pm 3.7	Mean \pm 1 SD 52.5 \pm 5.6	39 \pm 7.8

Lower Limit = mean + 2SD

2) Amplitude: Some of the sensory nerve fibers of upper and lower limbs and some motor nerve fibers of the lower limbs show a decrease in the amplitude while the amplitude of the motor nerve fibers of upper limbs were not affected (Table 2).

Table 2: The results of amplitude according to different nerves.

Nerve	Amplitude (mV or μ V)		
	Diabetic without Peripheral neuropathy Mean \pm SD (n = 25)	Normal range Mean (lower limit)	Diabetic with Peripheral neuropathy Mean \pm SD (n = 80)
Median (motor)	11.6 \pm 4.1	> 5mV	9.8 \pm 3.9
Median (sensory)	20.3 \pm 8.2	30.93 (10) μV	11.2 \pm 7.3
Ulnar (motor)	10.4 \pm 3.2	> 5mV	9.1 \pm 3.2
Ulnar (sensory)	15.7 \pm 3.9	22.74 (8.0) μV	12.1 \pm 6.6
Common Peroneal	7.4 \pm 1.1	>4 mV	2.8 \pm 2.3
Posterior Tibial	8.6 \pm 3.3	> 5mV	5.98 \pm 4.7
Sural	16 \pm 3.3	Mean \pm 1 SD 18.12 \pm 6 μV	11.97 \pm 6.7

Lower Limit = mean + 2SD

3) Latency: The latency of the sural nerve exceed the upper normal limit while in other nerves are controversial i.e. exceed in some nerves and within normal limits in others (Table 3).

Table 3: The results of latency according to different nerves.

Nerve	Latency (ms)		
	Diabetic without Peripheral neuropathy Mean \pm SD (n = 25)	Normal range Mean (upper limit)	Diabetic with Peripheral neuropathy Mean \pm SD (n = 80)
Median (motor)	3.4 \pm 0.4	3.49 (4.2)	3.6 \pm 0.4
Median (sensory)	2.4 \pm 0.4	2.84 (3.5)	3.2 \pm 0.8
Ulnar (motor)	2.8 \pm 0.5	2.59 (3.4)	2.8 \pm 0.5
Ulnar (sensory)	2.2 \pm 0.3	2.54 (3.1)	2.5 \pm 0.7
Common Peroneal	4.3 \pm 0.7	3.77 (5.5)	4.4 \pm 0.7
Posterior Tibial	4.8 \pm 0.98	3.96 (6.0)	5 \pm 0.8
Sural	2.2 \pm 0.3	Mean \pm 1 SD 2.7 \pm 0.3	3.4 \pm 0.3

Upper Limit = mean + 2SD

4) F – wave latency: Most of the examined nerves show no prolongation of the F – wave latency except in some nerves and this is due to the affect of central root by diabetic neuropathy (Table 4).

Table 4: The results of F-wave latency according to different nerves.

Nerve	F-wave latency (ms)		
	Diabetic without Peripheral neuropathy Mean \pm SD (n = 25)	Normal range Mean	Diabetic with Peripheral neuropathy Mean \pm SD (n = 80)
Median (motor)	22.5 \pm 1.9	25	24.1 \pm 1.3
Ulnar (motor)	22 \pm 2	25	24.1 \pm 1.4
Common Peroneal	39.6 \pm 6.1	47	35.5 \pm 9.1
Posterior Tibial	37.1 \pm 7.1	46	41.8 \pm 4.2

The prevalence of peripheral neuropathy in diabetic patients:

Prevalence of PN in diabetic patients = $80 / 105 \times 100 = 76.19\%$ (Figure 1)

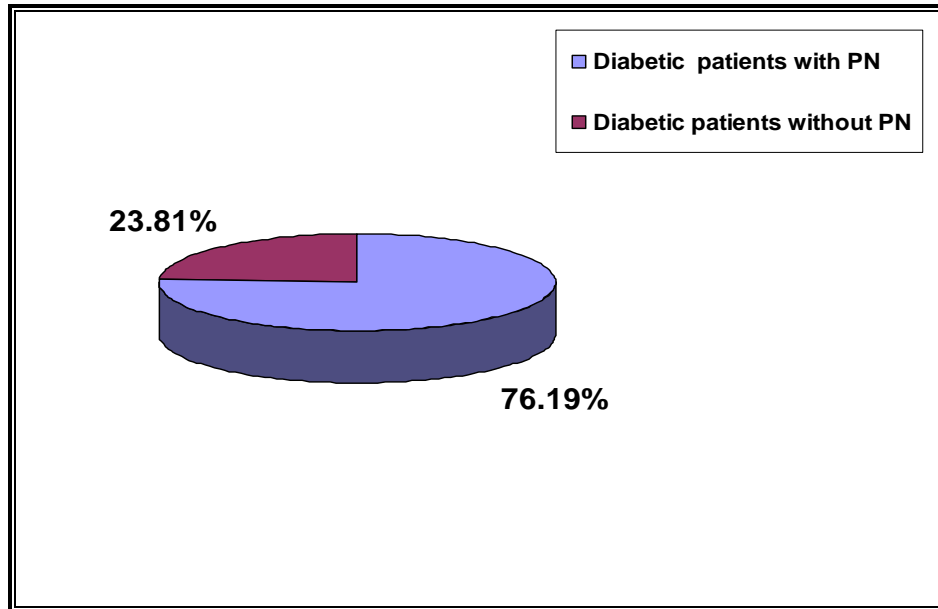


Figure 1: Prevalence of peripheral neuropathy in diabetic patients.

Effect of age, gender and body mass index (BMI) on the development of peripheral neuropathy in diabetic patients.

There was statistically significant effect of age of diabetic patients on the development of peripheral neuropathy ($P < 0.05$) (Table 5) but there was no significant effect of gender and BMI on prevalence of peripheral neuropathy in diabetic patients ($P > 0.05$) (Table 5).

Table 5: Effect of age, gender and BMI on development of peripheral neuropathy in diabetic patients.

Variables	With Neuropathy (n = 80)	Without neuropathy (n = 25)	P value
Age (year)			
30 – 45	41.9 ± 2.99	38.7 ± 4.1	$P < 0.05$
46 – 60	56.5 ± 4.2	52.5 ± 3.9	
Mean \pm SD			
Gender			
male	41 (51.25)	13 (52)	$P > 0.05$
female	39(48.75)	12 (48)	
No. (%)			
BMI (Kg/m^2)			
Mean \pm SD	28.96 ± 5.59	26.92 ± 5.06	$P > 0.05$

Effect of duration, control and therapeutic regimen of diabetes mellitus on the development of peripheral neuropathy.

In this study, there was a statistically significant effect of duration and control of diabetes mellitus on the development of peripheral neuropathy ($P < 0.05$) (Table 6). However was no significant effect of therapeutic regimen of diabetes mellitus on the development of peripheral neuropathy ($P > 0.05$) (Table 6).

Table 6: Effect of duration, control and therapeutic regimen of diabetes mellitus on the development of peripheral neuropathy.

Variables	With neuropathy (n = 80)	Without neuropathy (n = 25)	P value
Duration of diabetes (year) Mean \pm SD	8.9 \pm 6.04	5.5 \pm 4.7	$P < 0.05$
Control (HbA1c) Mean \pm SD	8.4 \pm 2	6.4 \pm 1.1	$P < 0.001$
Therapeutic regimen OHG Insulin No. (%)	77 (96.25) 3 (3.75)	20 (80) 5 (20)	$P > 0.05$

OHG = oral hypoglycemic

Effect of total serum cholesterol, HDL, LDL and triglyceride on the development of peripheral neuropathy.

There was no statistically significant effect of lipid profile (Total serum Cholesterol, HDL, LDL and triglyceride) on the development of peripheral neuropathy ($P > 0.05$) (Table 7).

Table 7: Effect of total serum cholesterol, HDL, LDL and triglyceride on the development of peripheral neuropathy.

Variables	With neuropathy Mean \pm SD (n = 80)	Without neuropathy Mean \pm SD (n = 25)	P value
Total cholesterol (mg/dl)	212.11 \pm 60.37	204.08 \pm 76.78	$P > 0.05$
HDL (mg/dl)	39.88 \pm 11.95	40.12 \pm 12.02	$P > 0.05$
LDL (mg/dl)	126.89 \pm 49.96	127.63 \pm 61.24	$P > 0.05$
Triglyceride(mg/dl)	226.75 \pm 130.18	181.64 \pm 86.41	$P > 0.05$

Discussion:

Sural nerves (sensory) show a decrease in conduction velocity (demyelinated neuropathy) in all diabetic patients with peripheral neuropathy, this indicates that the sensory nerves in lower limbs are more affected by diabetic peripheral neuropathy than those in upper limbs and more than motor nerves and this is consist with. (22)

The amplitude was low (axonal neuropathy) in the sensory nerves of lower and upper limbs and motor nerves of lower limbs in diabetic patients with peripheral neuropathy. This indicate that the diabetic neuropathy began at sensory nerves then motor nerves and in lower limbs then in upper limbs.(22)

The distal latency of the sural nerves exceeds the upper normal limit in diabetic patients with peripheral neuropathy because in sensory nerves, there is only one distance was measured and since the conduction velocity was measured by:

$C.V = \text{Distance} / \text{Latency} \dots\dots\dots(23)$

So it is logically when C.V decrease, the sensory latency are prolonged and vice versa.

F – wave latency was not prolonged in most tested nerves and this indicates no root entrapment (compression) except some nerve in diabetic patients with peripheral neuropathy. After the exclusion of root compression, they appear due to central involvement of diabetic neuropathy.

Diabetes is the leading cause of neuropathy, and neuropathy is the most common complication and greatest source of morbidity and mortality in diabetes patients. (22)

In this study of 105 diabetes patients, it has been found that an overall prevalence of peripheral neuropathy (PN) is 76.19 % (80 patients) and this prevalence seems high for patients with type 2 diabetes.

Prevalence rates in various studies around the world show considerable variation⁽²⁴⁾ as a result of variations in study design, the method of ascertainment (i.e. symptoms only, signs and symptoms, electromyography / nerve conduction studies (EMG/NCS), or composite parameters), the examination of patients at different stages in the natural history of diabetes, or in the definition of PN, and the study of selected populations, from as low as 1.5% to as high as 100%⁽²⁵⁾, making comparisons between studies of limited value.

Some authors considered findings on physical examination indicative of neuropathy while others regarded minor parasthesia as neuropathic manifestations in yet asymptomatic patients. (26)

The prevalence of diabetic PN in type 2 diabetic outpatients in Chinese city hospitals is 17.2% ⁽²⁷⁾, PN was seen in 34.7% of diabetics in Al Ain, United Arab Emirates ⁽²⁸⁾, 13.7% to 35.9%, in Saudi Arabia ⁽²⁹⁾, in a Turkish study the overall prevalence of neuropathy in patients with type 2 diabetes was 60% ⁽³⁰⁾, the prevalence of PN among Iranian (in the Isfahan area) patients with type 2 diabetes was (75.1%).⁽²⁶⁾

The present study confirms the well established association between the prevalence of PN and age in a diabetic population ($P < 0.05$). This finding agrees with other observations ⁽³¹⁾ who find that diabetic neuropathy can occur at any age but is more common with increasing age of diabetic patients. However, this result disagrees with others such as ⁽³²⁾ who found no significant relation between the age and abnormal NCS.

The factor of age in the development of distal diabetic neuropathy could be due to:

- 1) Increase in duration of diabetes as the age advanced.
- 2) Higher incidence of concomitant atherosclerosis leading to PN.
- 3) Lowering of the resistance of the peripheral nerve.

In this study, there was no statistically significant effect of gender on the development of PN ($P > 0.05$) and this finding agrees with other observation⁽³²⁾, but not with other observation⁽³³⁾ which show that male diabetic patients had more risk to develop diabetic neuropathy than female diabetic patients. Other observations find that a percentage of women with severe clinical neuropathy was significantly higher than that of men.⁽³⁴⁾

This study observed that prevalence of diabetic neuropathy in obese diabetics is more than in normal BMI but not significant statistically. This is corroborated with the finding of Akbar, (2002).⁽³⁵⁾ Other studies have shown associations of neuropathy with BMI. ⁽³⁶⁾

There was significant association between peripheral neuropathy and duration of diabetes and poor glycemic control. Other studies showed significant correlation between the presence of diabetic peripheral neuropathy with duration of diabetes.⁽³⁷⁾ A study observed in a cohort of 4400 subjects that the prevalence of DPN increased from 7% within 1 year of diagnosis to 50% for those with diabetes for more than 25 years. ⁽³⁸⁾

The results from the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS) convincingly demonstrated the importance of glycemic control for the prevention of microvascular complications of diabetes and there is a strong association between hyperglycemia and the incidence and progression of microvascular and macrovascular complications. ⁽³⁹⁾

In this study, there was no significant effect of therapeutic regimen of DM on the development of PN ($P > 0.05$). In univariate analysis, we found that the prevalence of PN was higher in patients treated with insulin than in noninsulin treated patients. ⁽³⁵⁾

A higher prevalence of PN among insulin-treated patients could be attributable to their longer duration of diabetes, delay in insulin treatment and possible insulin neuritis at the time of neurologic examination than in non-insulin-treated type 2 diabetes. After adjustment for other covariates in the multivariate analysis, the type of treatment was non-significant, indicating that insulin treatment is at least partially confounded by duration of diabetes. ⁽²⁶⁾

The present study was not able to find associations with PN for lipid status (Total serum Cholesterol, HDL, LDL and fasting Triglyceride) ($P > 0.05$).

This is corroborated with the finding of Lehtinen *et al.*, (1993)⁽⁴⁰⁾; Akbar, (2002)⁽³⁵⁾; and Janghorbani *et al.*, (2006)⁽²⁶⁾. Other studies have shown associations with PN for high-density lipoprotein cholesterol, an increase in the levels of fasting triglyceride. ^(24,41)

Conclusions:

From the results of this study, it is concluded that:

- 1- Nerve conduction study is important method to evaluate peripheral nerve function. They help in the diagnosis, extent and distribution of peripheral neuropathy.
- 2- In diabetic patient the sensory nerves are more affected than motor nerves and lower limbs are more affected than upper limbs.

- 3- There is a high prevalence of peripheral neuropathy in diabetic patients (76.19 %).
- 4- Age, duration of diabetes and level of glycated Hb significantly effect the prevalence of diabetic peripheral neuropathy while the gender, BMI, therapeutic regimen of diabetes and lipid profiles not effect the prevalence of diabetic peripheral neuropathy.

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