

## RISK FACTORS ASSOCIATED WITH PERIPHERAL NEUROPATHY IN TYPE II DIABETIC PATIENTS

By

**Kamel Mahmoud Hewedi, Ahmad Farag El-Adawy, Amr Ahmed Rezk\*,  
Ahmed Gamal Ahmed Yassen**

Departments of Neurology and Clinical Pathology\*, Faculty of Medicine, Al-Azhar University, Cairo, Egypt

**Corresponding author:** Ahmed Gamal Ahmed yassen,

**E-Mail:** [ahmed.gamal312000@gmail.com](mailto:ahmed.gamal312000@gmail.com)

### ABSTRACT

**Background:** Diabetic peripheral neuropathy (DPN) is a progressive distal-to-proximal degeneration of the peripheral nerves which leads to a variety of neuropathic manifestations. The estimated prevalence of DPN is approximately 50% among type 2 diabetic patients. It accounts for hospitalization more frequently than other complications of diabetes, and also is the most frequent cause of non-traumatic amputation.

**Objectives:** The current study was performed to assess risk factors of peripheral neuropathy among a sample of Egyptian diabetics.

**Patients and Methods:** One hundreds type II diabetic patients, diagnosed according to the American Association of Diabetes criteria, were included in the current study. Patients were furtherly categorized based on the presence of DPN into DPN and non-DPN groups.

**Results:** Patients who fulfilled the eligibility criteria were enrolled in the current study. Of them 46(46%) patients had manifestations of DPN, whereby 54(54%) patients did not have DPN. The mean age of the included patients was  $55.74 \pm 7.48$  and  $45.96 \pm 7.26$  years among patients with DPN and those without DPN, respectively. Patients aged more than 60 years, illiterate people and patients who did not complete secondary school patients with family history of diabetes, patients with longer duration of disease (>10years), hypertensive patients, patients with high levels of triglycerides, and patients with uncontrolled glycaemic status were more susceptible to develop DPN.

**Conclusion:** The prevalence of DPN is relatively high among Egyptian patients with T2DM. Appropriate screening programs along with adequate treatment should be given for high risk patients in order to improve the quality of life and to reduce the tumbledown complications of DPN.

**Keywords:** Peripheral neuropathy, diabetes, risk factors.

### INTRODUCTION

Type II diabetes mellitus (T2DM) is one of the most common progressive disorders worldwide. In particular, the estimated burden of T2DM was nearly 382 million patients all over the world in 2013 and the number is expected to rise to 592 million in 2035 (*Atlas, 2013*). The International Diabetes Federation has

estimated Egypt as the ninth leading nation worldwide for the number of patients with T2DM. The prevalence of T2DM in Egypt is deemed to be approximately 15.6% with nearly 87,000 deaths related to diabetes anniversary. Subsequent to that, the economic impact of T2DM in Egypt was 1.29 billion dollars

in 2010 (Aguiree *et al.*, 2013 and Hegazi *et al.*, 2015).

Patients with T2DM are more susceptible to develop peripheral arterial disease, lower limb amputation, and peripheral neuropathy twice in contrast to non-diabetic patients (Thiruvoipati *et al.*, 2015). Diabetic peripheral neuropathy (DPN) is a progressive, distal-to-proximal degeneration of the peripheral nerves which leads to a variety of neuropathic manifestations. The estimated prevalence of DPN is approximately 50% among diabetic patients. Of them, more than 50% had silent peripheral neuropathy (Juster-Switlyk & Smith, 2016 and Watterworth & Wright, 2019).

Of note, DPN is a considerable risk factor of diabetic foot, which leads to foot ulceration and lower limb amputation (Iqbal *et al.*, 2018). The financial impact of treating DPN is significant; in particular, the total annual costs of treating DPN among patients with T2DM are estimated to be 10 billion dollars in the United States annually (Shah *et al.*, 2017).

Owed to the devastating sequels of peripheral neuropathy and its financial impact, early detection of such condition along with optimization of the appropriate therapy to control the glycemic status is mandatory to prevent such complications. The adequate treatment of DPN will minimize the risk of limb ulceration by 60% and limb amputation by 85% (Farhat and Yezback, 2016). To shed light on this issue, the current study was performed to assess the prevalence and potential risk factors of peripheral neuropathy among a sample of Egyptian diabetic patients.

## PATIENTS AND METHODS

This study was a prospective cross sectional randomized study which was conducted at neurology, diabetes outpatient clinics and patients admitted to Internal Medicine Department, at Al-Hussein and Sayed Galal University Hospitals, Faculty of Medicine, Al-Azhar University, Cairo, Egypt, from January 2018 to July 2019. Institutional research board approval had been gained, and all patients had assigned informed consents prior to study processing.

One hundred T2DM patients diagnosed according to the American Association of Diabetes criteria were categorized based on the presence of DPN into DPN and non-DPN groups (American Diabetes Association, 2013).

Patients having peripheral neuropathy due to diseases such as renal failure, liver failure, traumatic neuropathy, or central neurological disease were excluded. Patients received drugs causing neuropathy, with history of alcohol intake or those with peripheral vascular diseases were also rolled out.

All patient were subjected to Clinical assessment (full history, and general examination), and Neuropathy Symptom Score (NSS) and Neuropathy Disability Score (NDS) recorded by interview following the standard Guidelines (Kiani *et al.*, 2013). Laboratory evaluation (including blood profile, renal function tests, liver function tests, thyroid function tests, lipid profile, fasting and post prandial blood sugar and glycosylated hemoglobin). Electrophysiological studies using Nihon Kochden electromyography at 33°C room

temperature (sensory and motor nerve conduction studies, and electromyography).

**Statistical analysis** was performed using SPSS software version 23 for Windows (SPSS Inc., Chicago, IL, USA). Continuous normally distributed data were notified using mean, and standard deviation (SD), and were compared using student t-test. Continuous non-normally

distributed data were illustrated using median and range and were compared using Mann-Whitney U test. Categorical variables were reported using number and percentage and its related groups were compared using Pearson's chi-square test. The overall statistically significant difference was established when the two-sided p value of  $< 0.05$ .

## RESULTS

A total of 100 patients who fulfilled the eligibility criteria were enrolled in the current study. Clinical examination showed that 46% of patients had manifestations of DPN, whereby 54% of patients did not have DPN, but there were 57% patients had neuropathy by nerve conduction study with 19.2 % patients had subclinical neuropathy of all cases of neuropathy. The mean age of the included patients was  $55.74 \pm 7.48$  and  $45.96 \pm 7.26$  years among patients with DPN and those without DPN, respectively. There were 53.7% females among patients with DPN and 58.7% females among patients without DPN. Elderly patients (more than 60 years) were

more susceptible to experience DPN in contrast to patients aged less than 60 years ( $p < 0.001$ ). Additionally, illiterate patients and people who did not complete secondary education were more vulnerable to experience DPN, relative to those completed secondary education ( $P = 0.02$ ). Patients with positive family history of T2DM were more susceptible to develop DPN ( $p = 0.004$ ). Besides that, patients suffering from T2DM more than 10 years were more vulnerable to develop DPN, in contrast to those with disease duration less than 10 years ( $p < 0.001$ ). There was a statistically significant higher rate of insulin treatment among patients developed DPN ( $p = 0.002$ ). **Table.1**

**Table (1): Demographic characteristics of the included patients.**

Diabetic peripheral neuropathy		Absent	Present	P-value
Parameters				
Age(years)	Mean $\pm$ SD	45.96 $\pm$ 7.26	55.74 $\pm$ 7.48	0.001
	Range	37 – 68	39 – 68	
	< 50 yrs	43 (79.6%)	9 (19.6%)	0.001
	50 - 60 yrs	9 (16.7%)	22 (47.8%)	0.001
	> 60 yrs	2 (3.7%)	15 (32.6%)	0.001
Gender	Female	29 (53.7%)	27 (58.7%)	0.616
	Male	25 (46.3%)	19 (41.3%)	
Education	Illiterate	14 (25.9%)	24 (52.2%)	0.020
	< secondary	24 (44.4%)	11 (23.9%)	
	> secondary	16 (29.6%)	11 (23.9%)	
F.H of DM	No	43 (79.6%)	24 (52.2%)	0.004
	Yes	11 (20.4%)	22 (47.8%)	
DM duration (years)	Median (IQR)	7 (3.5 – 9)	11 (7 – 14)	0.001
	Range	1 – 15	2 – 18	
	< 5 yrs	17 (31.5%)	4 (8.7%)	0.005
	5 - 10 yrs	29 (53.7%)	16 (34.8%)	0.058
	$\geq$ 10 yrs	8 (14.8%)	26 (56.5%)	0.001
Medication	OHD	34 (63.0%)	15 (32.6%)	0.002
	Insulin	20 (37.0%)	31 (67.4%)	
Smoking	No	42 (77.8%)	31 (67.4%)	0.243
	Current smoker	4 (7.4%)	5 (10.9%)	0.546
	Ex-smoker	8 (14.8%)	10 (21.7%)	0.369

\*: Chi-square test; •: Independent t-test

Patients with DPN had high means of systolic (147.35  $\pm$  19.4) and diastolic blood pressure (89.24  $\pm$  9.13), relative to those without peripheral neuropathy ( $p < 0.001$ ). DPN is higher in obese diabetics with BMI  $\geq$  30 ( $p < 0.158$ ) than in

those with a normal BMI 18.5-24.9 ( $p < 0.136$ ), but not statistically significant. Weight and height were not statistically significant in patients with DPN ( $p < 0.453$ ) and ( $p < 0.136$ ), respectively. **Table.2**

**Table (2): Clinical characteristics of the included patients**

Diabetic peripheral neuropathy		Absent	Present	P-value
Parameters				
SBP(mmHg)	Mean ± SD	125.19 ± 16.02	147.35 ± 19.4	0.001
	Range	100 – 160	100 – 180	
DBP(mmHg)	Mean ± SD	81.3 ± 7.15	89.24 ± 9.13	0.001
	Range	70 – 100	70 – 100	
Wt(kg)	Mean ± SD	79.23 ± 18.34	81.91 ± 17.02	0.453
	Range	53.5 – 122.5	51.5 – 114	
Ht(m)	Mean ± SD	1.75 ± 0.04	1.75 ± 0.06	0.980
	Range	1.67 – 1.83	1.65 – 1.85	
BMI(kg/m <sup>2</sup> )	Mean ± SD	25.87 ± 5.92	26.63 ± 4.8	0.485
	Range	18.5 – 40	18.16 – 35.25	
	18.5-24.9	23 (42.6%)	13 (28.3%)	0.136
	25-29.9	17 (31.5%)	15 (32.6%)	0.902
	≥ 30	14 (25.9%)	18 (39.1%)	0.158

SBP= Systolic blood pressure, DBP=Diastolic Blood Pressure, BMI=Body mass index  
 •: Independent t-test; ≠: Mann-Whitney test

There was a statistically significant difference between patients developed DPN and patients did not develop such condition regarding the levels of hemoglobin ( $p < 0.001$ ), white blood cells count ( $p = 0.002$ ). But there was no a

statistically significant difference between patients developed DPN and patients did not develop such condition regarding the levels of red blood cells ( $p < 0.380$ ), and platelet ( $p < 0.233$ ). **Table.3**

**Table (3): Blood picture, liver function test and renal function test**

Parameters		Diabetic peripheral neuropathy		P-value
		Absent	Present	
HB (g/dL)	Mean $\pm$ SD	13.13 $\pm$ 1.45	11.32 $\pm$ 1.71	0.001
	Range	9.8 – 15.4	8.5 – 15.4	
WBC ( $10^3$ / uL)	Mean $\pm$ SD	6.42 $\pm$ 1.52	7.53 $\pm$ 2.02	0.002
	Range	4.5 – 10.6	4.5 – 11.3	
RBC ( $10^6$ / uL)	Mean $\pm$ SD	4.99 $\pm$ 0.46	4.91 $\pm$ 0.51	0.380
	Range	4.14 – 6	4.12 – 6	
Platelet( $10^3$ / uL)	Mean $\pm$ SD	278.67 $\pm$ 75.79	296.5 $\pm$ 71.99	0.233
	Range	162 – 425	197 – 450	
ESR(mm/hr)	Mean $\pm$ SD	14.81 $\pm$ 5.31	12.54 $\pm$ 4.57	0.025
	Range	5 – 25	5 – 21	
ALT(U/L)	Mean $\pm$ SD	20.07 $\pm$ 7.31	17.11 $\pm$ 6.38	0.035
	Range	9 – 30	10 – 33	
AST(U/L)	Mean $\pm$ SD	28.74 $\pm$ 7.55	22.89 $\pm$ 7.45	0.001
	Range	12 – 42	9 – 36	
Serum creatinine (mg/dL)	Mean $\pm$ SD	1 $\pm$ 0.18	0.92 $\pm$ 0.23	0.054
	Range	0.65 – 1.28	0.55 – 1.3	

HB=Hemoglobin, WBC= White blood cell count, RBC= red blood cell count, ESR= erythrocyte sedimentation rate, ALT= Alanine Transaminase, AST=Aspartate Transaminase

•: Independent t-test

The mean levels of T4 (1.09 $\pm$ 0.16), TSH (3.18 $\pm$ 0.78), triglyceride (214.44 $\pm$ 36.14), low density lipoproteins (109.48 $\pm$ 33.31), glycosylated hemoglobin (8.32  $\pm$ 1.43) and fasting blood glucose were significantly high among patients had DPN with P values of <0.001, <0.003, <0.002, <0.046, <0.001, and <0.001, respectively. But there was no

a statistically significant difference between patients developed DPN and patients did not develop such condition regarding the levels of free T3, total cholesterol, high density lipoproteins and 2 hours post prandial blood sugar with p value of <0.115, <0.210, < 0.148 and <0.072, respectively .**Table.5**

**Table (5): Thyroid hormones, lipid profile and blood sugar file**

Diabetic peripheral neuropathy		Absent	Present	P-value
Parameters				
Free T3(pg/ml)	Mean ± SD	3.23 ± 0.45	3.1 ± 0.35	0.115
	Range	2.18 – 3.9	2.2 – 4	
Free T4(ng/dl)	Mean ± SD	1.24 ± 0.19	1.09 ± 0.16	0.001
	Range	0.93 – 1.7	0.85 – 1.7	
TSH(uIU/ml)	Mean ± SD	2.7 ± 0.8	3.18 ± 0.78	0.003
	Range	1.6 – 4.28	1.61 – 4.3	
TC(mg/dl)	Mean ± SD	196.22 ± 47.67	207.96 ± 44.66	0.210
	Range	115 – 302	133 – 300	
TG(mg/dl)	Mean ± SD	191.41 ± 36.94	214.44 ± 36.14	0.002
	Range	106 – 252	104 – 269	
HDL(mg/dl)	Mean ± SD	55.02 ± 11.81	51.22 ± 14.24	0.148
	Range	30 – 85	28 – 88	
LDL(mg/dl)	Mean ± SD	96.96 ± 28.51	109.48 ± 33.31	0.046
	Range	47 – 156	55 – 180	
HbA1C (%)	Mean ± SD	6.89 ± 1.25	8.32 ± 1.43	0.001
	Range	5.2 – 10.1	5.9 – 10.4	
FBS(mg/dl)	Mean ± SD	138.98 ± 20.12	158.96 ± 25.08	0.001
	Range	110 – 204	119 – 207	
2HPP(mg/dl)	Mean ± SD	224.85 ± 38.84	238.41 ± 35.04	0.072
	Range	176 – 380	190 – 380	

TSH= thyroid stimulating hormone, TC= Total Cholesterol, TG=Triglyceride, HDL=High density Lipoprotein, LDL=Low density Lipoprotein, HbA1C= Glycosylated Hemoglobin, FBS= Fasting Blood Sugar. 2HPP=two hours post prandial

•: Independent t-test

## DISCUSSION

DPN is a considerable cause of morbidity among patients with T2DM. Despite that, DPN has not been investigated extensively as nephropathy, retinopathy, and macro-vascular complications. Moreover, the prevalence of DPN varied substantially between countries owing to the diversity in the diagnostic criteria and sampling methods (*Pop-Busui et al., 2017*).

In the current study prevalence of DPN was 46%. This proportion was 36.96% and 63.04% among males and females, respectively. This result brought to light that every two individuals in the population have T2DM, a patient has a chance of experiencing DPN. The estimated prevalence of DPN in the Middle East varied substantially. Apart from this, the burden of DPN was 45%, 39.5%, and 25.6% in Saudi Arabia,

Jordan, and the United Arab Emirates, respectively (*Al-Geffari, 2012; Al-Sarihin et al., 2013* and *Al-Kaabi et al., 2014*).

In our study 19.2% have subclinical neuropathy, they are the same as *Shereen. (2015)*, patients aged more than 60 years, patients with family history of diabetes, patients with longer duration of disease (>10 years), hypertensive patients, patients with impaired lipid profile, and patients with uncontrolled glycaemic status were more susceptible to develop DPN *Liu et al. (2019)* conducted a meta-analysis that comprehended 12,116 patients and revealed that; the duration of diabetes, age, and glycosylated hemoglobin are associated with significantly increased risks of DPN among diabetic patients. In this concern, *Khawaja et al. (2018)* notified that patients age, family history of diabetes, duration of diabetes, hypertension, dyslipidemia, insulin treatment, and glycosylated hemoglobin influenced dramatically the chances of developing DPN.

In the current investigation, the duration of the disease appeared to enhance the occurrence of DPN, whereby patients with disease duration of more than 10 years were more susceptible to develop DPN. This finding was compatible with *Bansal et al. (2014)* who found that health care providers should employ comprehensive screening programs for early diagnosis of diabetic patients in order to avoid the devastating sequels of peripheral neuropathy.

Patients with dyslipidemia appeared to be more vulnerable to develop DPN. The possible explanation of nerve damage in such cases might be attributed to fat

deposition, oxidative stress, activation of counter regulatory signaling pathways, and mitochondrial dysfunction, which ultimately lead to progressive inflammation and damage of the peripheral nerves (*Aguiar et al., 2016*). Elevated triglycerides may serve as a potential marker for the impairment of the Schwann cells lipid metabolism and the underlying pathological alterations of the myelin structure among DPN patients (*Al-Ani et al., 2011*). Based on this, diabetic patients should be subjected to optimal dietary control coupled with lipid lowering agents in order to prevent or to delay the occurrence of DPN.

In the present study, the glycemic status influenced significantly the chances to develop DPN. Intensive glycemic control should be implemented to reduce the risk of DPN. Apart from this, the type of diabetes treatment affected noticeably the occurrence of peripheral neuropathy. In this concern, patients received insulin therapy was more susceptible to develop DPN relative to those receiving oral hypoglycemic drugs. This finding might be attributed to the confounding effect of duration of diabetes, whereby patients received insulin was more likely to suffer from T2DM for a long duration. Subsequent to that, exogenous insulin in T2DM might reflect an advanced stage which could be associated with other comorbidities such as obesity, dyslipidemia, hypertension, and fluid retention (*Katulanda et al., 2012* and *Won et al., 2012*). In accordance with our findings, *Kostev et al. (2014)* reported that insulin use was one of the strongest risk factors for DPN among newly diagnosed diabetics in Germany and the United Kingdom.



## CONCLUSION

The prevalence of DPN is relatively high among Egyptian patients with T2DM. Patients aged more than 60 years, patients with family history of diabetes, patients with long standing diabetes mellitus, hypertensive patients, patients with impaired lipid profile, and patients with uncontrolled glycaemic status were more susceptible to develop DPN. Appropriate screening programs along with adequate treatment should be given for high risk patients in order to enhance their quality of life and to reduce the tumbledown complications of DPN.

## REFERENCES

1. **Aguiree F, Brown A, Cho NH, Dahlquist G, Dodd S, Dunning T and Patterson C (2013):** IDF diabetes atlas. International Diabetes Federation, 48-67.
2. **Aguiar PCM, Coletta M and de Souza JJS (2016):** The association of dyslipidemia and peripheral diabetic neuropathy: the influence of urea. *Diabetes Case Rep*, 1(109): 1-3.
3. **Al-Ani FS, Al-Nimer MS and Ali FS (2011):** Dyslipidemia as a contributory factor in etiopathogenesis of diabetic neuropathy. *Indian Journal of Endocrinology and Metabolism*, 15(2): 110-114.
4. **Al-Geffari M (2012):** Comparison of different screening tests for diagnosis of diabetic peripheral neuropathy in Primary Health Care setting. *International Journal of Health Sciences*, 6(2): 127-134.
5. **Al-Kaabi JM, Al Maskari F, Zoubeidi T, Abdulle A, Shah SM, Cragg P and Souid A (2014):** Prevalence and determinants of peripheral neuropathy in patients with type 2 diabetes attending a tertiary care center in the United Arab Emirates. *J Diabetes Metab*; 5(346):1-7.
6. **Al-Sarihin K, Althwabia I, Khaled MB and Haddad F (2013):** Prevalence of peripheral neuropathy among patients with diabetes mellitus at King Hussein Hospital, Amman, Jordan. *Rawal Medical Journal*, 38(2):92-96.
7. **American Diabetes Association (2013):** Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 36 (Suppl1): S67–S74.
8. **Atlas D (2013):** International diabetes federation. *IDF Diabetes Atlas*, 6th edn. Brussels, Belgium: International Diabetes Federation. 1-166.
9. **Bansal D, Gudala K, Muthyala H, Esam HP, Nayakallu R and Bhansali A (2014):** Prevalence and risk factors of development of peripheral diabetic neuropathy in type 2 diabetes mellitus in a tertiary care setting. *Journal of Diabetes Investigation*, 5(6): 714-721.
10. **Farhat NM and Yezback KL (2016):** Treatment of diabetic peripheral neuropathy. *The Journal for Nurse Practitioners*, 12(10): 660-666.
11. **Hegazi R, El-Gamal M, Abdel-Hady N and Hamdy O (2015):** Epidemiology of and risk factors for type 2 diabetes in Egypt. *Annals of Global Health*, 81(6): 814-820.
12. **Iqbal Z, Azmi S, Yadav R, Ferdousi M, Kumar M, Cuthbertson DJ and Alam U (2018):** Diabetic peripheral neuropathy: epidemiology, diagnosis, and pharmacotherapy. *Clinical Therapeutics*, 40(6): 828-849.
13. **Juster-Switlyk K and Smith AG (2016):** Updates in diabetic peripheral neuropathy. *F1000 Research*, 5792-778.
14. **Katulanda P, Ranasinghe P, Jayawardena R, Constantine GR, Sheriff MR, Matthews DR (2012):** The prevalence, patterns and predictors of diabetic peripheral neuropathy in a developing country. *Diabetol Metab Syndr*, 4(1): 1-21.
15. **Khawaja N, Abu-Shennar J, Saleh M, Dahbour SS, Khader YS and Ajlouni KM (2018):** The prevalence and risk factors of peripheral neuropathy among patients with type 2 diabetes mellitus; the case of Jordan. *Diabetol Metab Syndr*, 10(1): 1-8.

16. **Kiani J, Moghimbeigi A, Azizkhani H and Kosarifard S (2013):** The prevalence and associated risk factors of peripheral diabetic neuropathy in Hamedan, Iran. *Archives of Iranian Medicine*. 16(1):17-19.
17. **Kostev K, Jockwig A, Hallwachs A and Rathmann W (2014):** Prevalence and risk factors of neuropathy in newly diagnosed type 2 diabetes in primary care practices: a retrospective database analysis in Germany and UK. *Primary Care Diabetes*, 8(3): 250-255.
18. **Liu X, Xu Y, An M and Zeng Q (2019):** The risk factors for diabetic peripheral neuropathy: A meta-analysis. *PLoS One*, 14(2): 1-16.
19. **Pop-Busui R, Boulton AJ, Feldman EL, Bril V, Freeman R, Malik RA and Ziegler D (2017):** Diabetic neuropathy: a position statement by the American Diabetes Association. *Diabetes Care*, 40(1): 136-154.
20. **Shah P, Shamon F, Bikkina M and Kohl HW (2017):** Medical cost of type 2 diabetes attributable to physical inactivity in the United States in 2012. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, 11(1): 13-17.
21. **Shereen KR, Hamdy M, Omar HA, Kamal A, Ali LH and Elkarim AH (2015):** Clinical diagnosis of distal diabetic polyneuropathy using neurological examination scores: correlation with nerve conduction studies. *Egyptian Rheumatology and Rehabilitation*. 42(3):128-136.
22. **Thiruvoipati T, Kielhorn CE and Armstrong EJ (2015):** Peripheral artery disease in patients with diabetes: Epidemiology, mechanisms, and outcomes. *World Journal of Diabetes*, 6(7): 961-969.
23. **Watterworth B and Wright TB (2019):** Diabetic Peripheral Neuropathy. Ch. Pain. Springer. pp. 911-913.
24. **Won J, Kwon H, Kim C, Lee J, Park T, Ko K and Cha B (2012):** Prevalence and clinical characteristics of diabetic peripheral neuropathy in hospital patients with type 2 diabetes in Korea. *Diabetic Medicine*, 29(9): 290-296.

## عوامل الخطورة المقترنة بالتهابات الأعصاب الطرفية لدي عينة من مرضى السكري من النوع الثاني

كامل محمود هويدي، أحمد فرج العدوي، عمرو أحمد رزق\*، أحمد جمال أحمد يسن

قسمي طب المخ والأعصاب والباثولوجيا الاكلينيكية\*، كلية الطب، جامعة الأزهر

**خلفية البحث:** الاعتلال العصبي الطرفي السكري هو تدهور تدريجي في وظيفة الأعصاب الطرفية التي تؤدي إلى مجموعة متنوعة من مظاهر الاعتلال العصبي. ويقدر معدل إنتشار الاعتلال العصبي الطرفي السكري بحوالي ٥٠٪ بين مرضى السكري. كما يعد من أكثر مضاعفات السكري التي تحتاج للعلاج داخل المستشفيات، وأيضا يعد من أكثر الأسباب المتكررة للبتيرالغير ناتجة عن الحوادث.

**الهدف من البحث:** تقييم مدى إنتشار الاعتلال العصبي الطرفي السكري والعوامل المسببة له بين عينة من مرضى السكري المصريين.

**المرضى وطرق البحث:** تضمن البحث المرضى الذين يعانون من داء السكري من النوع الثاني، و الذين تم تشخيصهم وفقا لمعايير الجمعية الأمريكية لمرض السكري، وقد تم تقسيم المرضى بناء على وجود أعراض الاعتلال العصبي الطرفي السكري إلى مجموعتين: مجموعة تعاني من الاعتلال العصبي ومجموعة لا تعاني من ذلك.

**النتائج:** تم تسجيل ما مجموعه ١٠٠ مريض الذين إستوفوا معايير الأهلية في الدراسة الحالية، وجد أن منهم ٤٦٪ من المرضى لديهم مظاهر الاعتلال العصبي الطرفي السكري، بينما لم يكن ٥٤٪ من المرضى يعانون من الاعتلال العصبي الطرفي السكري. وكان متوسط العمر للمرضى  $55,74 \pm 7,48$  و  $45,96 \pm 7,26$  سنة بين المرضى الذين يعانون من الاعتلال العصبي الطرفي السكري والذين لا يعانون من الاعتلال العصبي الطرفي السكري، على الترتيب. كما وجد أن المرضى الذين تزيد أعمارهم عن ٦٠ عامًا والأميون الذين لم يكملوا المرحلة الثانوية، والمرضى الذين لديهم تاريخ عائلي لمرض السكري، والمرضى ذوي الفترات المرضية الطويلة من المرض (أكثر من ١٠ سنوات)، والمرضى الذين

يعانون من ارتفاع ضغط الدم، والمرضى الذين يعانون من مستويات عالية من الدهون الثلاثية، والمرضى الذين يعانون من حالة سكر الدم غير المنضبط أكثر عرضة لمرض الاعتلال العصبي الطرفي السكري.

**الاستنتاج:** إنتشار الاعتلال العصبي الطرفي السكري ذو إرتفاع نسبي بين المرضى المصريين الذين يعانون من مرض السكري من النوع الثاني. لذلك يجب تنفيذ برامج فحص شاملة للكشف المبكر عن الاعتلال العصبي الطرفي السكري إلى جانب العلاج المناسب للمرضى المعرضين لمخاطر الإصابة بهذا المرض من أجل تحسين جودة حياتهم، وتقليل مضاعفات الاعتلال العصبي الطرفي السكري.