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STUDY OF PHYSIOLOGICAL CHANGES IN PATIENTS PRESENTED WITH DIABETIC PERIPHERAL NEUROPATHY IN KARBALA CITY

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ABSTRACT

This study conducted to know changes that occur for some physiological parameters in patients with diabetes disease related to peripheral neuropathy to important these changes in identification, determine, and know some factors and response of patient to treatment and total samples are 200 sample divided into 100 sample patient and divided into 50 sample infected to diabetes disease related to peripheral neuropathy and 50 sample infected to diabetes disease without peripheral neuropathy and 100 sample as control from female only about 40-65 year old and each patient present in diabetes center in alhussein hospital in Karbala. This study included measured glucose level in blood and hemoglobin level and this study present significant increase ($P \leq 0.05$) in glucose level in blood and hemoglobin level compared with control group

Keywords: Diabetes Disease, Peripheral Neuropathy, Glucose in Blood and Hemoglobin

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INTRODUCTION

Diabetes Mellitus (DM) is a clinical syndrome characterized by hyperglycemia due to absolute or relative insulin deficiency (1). There are 2 types of complications of diabetes mellitus: - macrovascular and microvascular complications. The macrovascular complications include: - cerebrovascular, cardiovascular, and peripheral vascular diseases. The microvascular complications include nephropathy, retinopathy and neuropathy and it result from chronic hyperglycemia (2). Diabetic neuropathy is the most common chronic complication affecting both type 1 and type 2 diabetic patients (3). The signs and symptoms of DPN vary depending on fiber type involved. With large fiber disease impairing proprioception and light touch. Small fiber disease impairs pain and temperature perception, leading to paresthesias, dysesthesias, and/or neuropathic pain. Distal weakness occurs only in the most severe cases. Diabetic peripheral neuropathy might be presented early with diminished or absent deep-tendon reflexes, particularly the Achilles tendon reflex often indicates mild and otherwise asymptomatic DPN. More advanced neuropathy may be firstly presented with late complications such as ulceration or neuroarthropathy (Charcot's joints) of the foot (4). The duration of diabetes and degree of metabolic control are the two major risk factors of the development of neuropathy and determinant of its severity. Other factors, such as patient's age, sex, type of DM, height, lipid profile abnormalities, and presence of proliferative retinopathy, nephropathy, and cardiovascular diseases, also have been implicated (5). Diabetic peripheral neuropathy can be diagnosed by variety of ways including: - full history and neurological examination, nerve biopsy and

electrophysiological study (nerve conduction study) which shows a pattern of abnormality that reflects the pathological process of DPN. The electrophysiological changes include prolongation of latency (sensory and motor), decrease amplitude & decrease conduction velocity. These changes are elicited first in the sensory nerves of lower limbs (6)

MATERIALS AND METHODS

study groups and blood samples collection

this study included 200 sample divided into 100 sample patient and divided into 50 sample infected to diabetes disease related to peripheral neuropathy and 50 sample infected to diabetes disease without peripheral neuropathy and 100 sample as control about 40–65-year-old and each patient present in diabetes center in alhussein hospital in Karbala. taken 5ml of blood sample, 2 ml put in anticoagulant tube uses for hemoglobin test and 3 ml placed in centrifuge for 10 minutes after waiting for 45 minutes to separate serum from whole blood. Serum samples stored in refrigerator (-20c°). Serum samples were used for measurement of blood sugar (7)

Glycosylated hemoglobin measurement: -

Whole blood preparation was mixed with a weakly binding cation-exchange resin; the non-glycosylated hemoglobin was bound to the resin, leaving HbA1c. The percent of HbA1c was determined by measuring the absorbance values at 415nm of the HbA1c fraction to the total Hb fraction, according to the procedure explained by the company (Stanbio lab., USA) (8).

Serum Glucose measurement: -

Glucose is oxidized by glucose-oxidase to gluconate and hydrogene peroxide according to the following equation.

$$\text{Glucose} + O_2 + H_2O \xrightarrow{\text{GOD}} H_2O_2 + \text{Gluconate}.$$

$$2H_2O_2 + \text{Phenol} + 4\ \text{Amino-antipyrine} \xrightarrow{\text{POD}} H_2O + \text{Quinonimine}$$

The absorbance of standard & samples are measured against reagent blank at 546nm according to the procedure recommended by the company (Human, Germany) (9).

Statistical analysis

The data were presented as mean ±SE and subjected to analysis of variance by using one way ANOVA Post hoc test was used LSD to specify the significant difference among means the software package IBM SPSS Program version 20 was used for the analysis of data **(10).**

RESULTS

Table) effect of diabetes disease related to peripheral neuropathy on blood glucose and hemoglobin (Means ± SE)

Parameters / Groups	Fasting blood sugar (mmol/L)	Glycosylated hemoglobin %
Patient persons (Diabetic peripheral neuropathy patients)	A 13.2±1	A 10.5±1
Patient persons Diabetics without neuropathy	B 8±2	B 5±1
Healthy persons	C 5.9±2	C 5±0.7

N=100 Different letters represent a significant difference at (p≤0. 05)

DISCUSSION

The study shows higher prevalence fasting blood sugar in DPN in patients DM than control groups with statistically significant difference This result agrees with results of other studies (11)(5) showed that in that hyperglycemia is the most important risk factor for DPN. The explanation of this result is that DM is associated with absolute deficiency of insulin and C-peptide. C-peptide has much insulin like and neuroprotective properties and so its deficiency will result in severer and rapid progression of DPN (3). Chronic hyperglycemia represents the main causative factor involved in the pathogenesis of diabetic neuropathy. Nerve damage may be directly induced by the accumulation of intracellular glucose (the consequences of which include the generation of glycating sugars and advanced glycation end-products (AGE), enhanced oxidative damage and protein kinase C activation). Apart from direct hyperglycemia-induced damage, ischemia caused indirectly from decrease in neurovascular flow almost certainly plays a role (12). The results of HbA1c of DPN patients are higher than those of control groups with statistically significant differences. These results agree with results of other studies (11) (12) (13) (14)(15) showed that higher HbA1c values is reported in DPN patients. High HbA1c values indicate elevated mean blood sugar value over the last 2 months.

REFERENCES

1. 1-Davidsons, (2006). Principles & practice of medicine. 18th edition. Churchil Livingston: 420-520.
2. 2-Aaberg, M.L.; Burch, D.M.; Hud, Z.R. and Zacharias, M.P. (2008). Gender differences in the onset of diabetic neuropathy. Journal of Diabetes and Its Complications. 22:83– 87.
3. 3- Sima, A. A. F. (2004). Diabetic neuropathy in type 1 & type 2 diabetes & the effect of C-peptide. Journal of clinical sciences, 220:133-136.
4. 4-Yagihashi, S.; Yamagishi, S. & Wada, R. (2007). Pathology and pathogenetic mechanisms of diabetic neuropathy: Correlation with clinical signs and symptoms. Diabetes Research and Clinical Practice, 77:184–S189.
5. 5-Harati, Y. (2007). Diabetic Neuropathies: Unanswered Questions. Neurol Clin, 25:303–317.
6. 6-Kimura, J. (2001) Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice, 3rd ed. Oxford University Press.
7. 7-Lewis, S. M.; Bain, B. J. & Bates, I. (2006). Dacei & Lewis practical hematology. 10th ed., Churchil Livingstone Elsevier. Germany.
8. 8-Frank P (1988). Monitoring the diabetic patient with glycosylated hemoglobin measurments. Clin. Prod., 4:112.
9. 9-Burits, C. A. & Ashwood, E. R. (1999). Teitis textbook of clinical chemistry. 4th. Ed. W. B., Saunders comp. USA, 2 (1500-1503).
10. 10-SPSS Statistical Packages for the Social Sciences. (2001): Statistical software for windows version 13.0 Micrisoft. SPSS, Chicago, IL, USA.
11. 11-Coppini, D.V.; Wellmer, A.;.Weng, C.; Young, P.J.; and, Sönksen, H. (2001). The natural history of diabetic peripheral neuropathy determined by a 12 year prospective study using vibration perception thresholds Journal of Clinical Neuroscience. 8:520–524.
12. 12-Boucek, 2006. Advanced Diabetic Neuropathy: A Point of no Return? The Review of Diabetic Studies, 3(3):144-152.
13. 13-Sandberg, G.E. & Wikblade, K.F. (2003). Oral dryness & peripheral neuropathy in subjects with type 2 DM. Journal of Diabetes and Its Complications, 17:192–198.

14. 14-Booya, F.; Bandarian, F.; Larijani, B.; Pajouhi, M.; Nooraei, M. & Lotfi, J. (2005). Potential risk factors for diabetic neuropathy: a case control study. BMC neurology, 5:24.
15. 15-Shiraiwa, T.; Kaneto, H.; Miyatsuka, T.; Kato, K.; Yamamoto, K.; Kawashima, A.; Kanda, T.; Suzuki, M.; Imano, E.; Matsuhisa, M.; Hori, M. & Yamasaki,Y. (2005). Post-prandial hyperglycemia is an important predictor of the incidence of diabetic microangiopathy in Japanese type 2 diabetic patients. Biochemical & biophysical research communication, 366:339-345.
16. 16-Weintrob, N.; Amitaya, I.; Lilos, P.; Shalitina, S.; Lazar, L. & Josefsberg, Z. (2007). Bedside neuropathy disability score compared to quantitative sensory testing for measurement of diabetic neuropathy in children, adolescents, and young adults with type 1 diabetes. Journal of Diabetes and Its Complications 21:13– 19.