Original Article

The Relationship Between the Duration of Type 2 Diabetes Mellitus and Obesity with Complications Neuropathy

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ABSTRACT

Objective: To observe the type 2 diabetes mellitus in combination with obesity levels affects diabetic neuropathy occurrence.

Study Design: Cross-sectional study

Place and Duration of Study: This study was conducted at the Department of Biochemistry of Al-Rafai Education Hospital in Dhi Qar Iraq from 31st December 2024 to 31st March 2025.

Methods: A total of 90 participants assigned to three separate groups consisting of healthy control patients and participants with type 2 diabetes mellitus without neuropathy as well as type 2 diabetes mellitus patients with confirmed diabetic neuropathy. Researchers took blood samples which they used to measure fasting blood sugar and haemoglobin A1c and hemoglobin and ferritin along with the lipid profile.

Results: Diabetic patients especially those with neuropathy experienced a marked increase in fasting blood sugar and haemoglobin A1c and body mass index levels when tested. The diabetic neuropathy patient group demonstrated elevated ferritin compared to other participant groups. A high relationship emerged between haemoglobin A1c measurements and both fasting blood sugar levels and body mass index values as well as ferritin levels.

Conclusion: Both type 2 diabetes mellitus duration extending in years and obesity condition increase the susceptibility to neuropathic complications.

Key Words: Type 2 diabetes mellitus, Diabetic neuropathy, Obesity, Ferritin

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INTRODUCTION

People with type 2 diabetes mellitus (T2DM) show persistent elevated blood glucose levels due to insulin resistance combined with inadequate insulin action. An impaired β-cell status prevents patients from increasing their insulin secretion levels successfully. Multiple factors including genetics and inflammation together with visceral obesity contribute to the exact pathogenesis of insulin resistance in T2DM.[1] The defective insulin-mediated receptor phosphorylation occurs at the receptor kinase domain tyrosine position.

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Research showed that Glut4 molecules on adipocytes play a significant role in downregulating PPARγ coactivator 1α (PGC1α) gene activity in patients with T2DM which contributes to insulin resistance. Endogenous free fatty acids (NEFA) affect glucotoxicity in pancreatic islet cells by creating higher reactive oxygen species (ROS) concentrations which generate increased oxidative stress. The reduction in number of β-cells becomes the final cause of developing T2DM.[2] Throughout its development T2DM results in declining insulin secretion from pancreatic cells because apoptosis is prevented in β-cells throughout the body. The numbers of people with T2DM have grown remarkably since the previous years. The population numbers of both adult and adolescent T2DM patients have demonstrated rising figures. By 2025 the level of T2DM diagnosis is expected to increase substantially for adults between 20 to 79 years of age.[3]

Death statistics show that diabetes complications now appear with greater frequency. The occurrence of diabetic peripheral neuropathy in diabetes exists from 30-50% of patients. Late detection of diabetes often results in amputation of feet requiring removal because of ulcers along with gangrene development. T2DM develops when genetic and immune and metabolic

dysfunctions together cause persistent high blood glucose levels. Tissues and functions of nerves undergo biochemical transformations that modify structure because of persistent high blood glucose levels. The length of time someone has diabetes stands alone as a critical risk factor in developing diabetic neuropathy.[4,5]

The initial pathological changes from glycating end products formation in diabetes affect synapses far from the cell body first. The long axons experience structural support alterations which produce Wallerian degeneration together with distal segment dying back while proximal neuron nerve function declines.[6] The conditions which define T2DM include different types of metabolic problems with fat compounds and proteins alongside carbohydrates. Over the period diabetes type 2 patients who are overweight have grown in numbers. The condition develops due to reduced insulin sensitivity in end organs and reflects weight problems and inactive lifestyles. Type 2 diabetes connected to obesity has produced increased adult diabetes cases which ultimately result in delayed complications. Patient diagnoses need to consider diabetic neuropathy because it ranks as the second manifestation that makes life worse for diabetic patients. Research currently fails to explain all of the factors which trigger neuropathy progression in diabetic patients.[7]

Millions of people worldwide experience substantial health problems because of diabetes which has become the speediest spreading long-term disease today. Global healthcare providers diagnosed 382 million people with diabetes during the count of 2013. The research indicates that diabetes will affect 591.9 million people worldwide during 2035. Researchers indicate that diabetes patients amount to 50% of the global population who live in five key countries. The five leading countries with diabetes cases are the United States of America alongside India, Brazil, China and Indonesia. The two countries with the most frequent occurrences of diabetes are China followed by India among the five nations examined. These countries face a realistic possibility of becoming known as "diabetes capitals" if they fail to implement effective diabetes control measures because of their worsening situation.[8]

The rate at which people suffer from diabetes differs between separate nations around the world. Diabetes prevalence throughout South and Southeast Asia ranked in the third position in 2013 with 72 million cases yet the highest case counts were detected in Western Pacific and North American regions. The population of Malaysian adults with diagnosed diabetes passed 2.4 million in 2013. Research data indicates that diabetes cases will increase by 123% to reach this number by 2035. The leading complication of neuropathy affects all body regions starting from autonomic and continuing to cranial and spinal nerves. High levels of glucose within the bloodstream of diabetes patients lead to nerve destruction and degeneration. The lengthy nerves belong to the group most susceptible to damage thus rendering them the first targets of this process. Researchers consider peripheral neuropathy to be among the most prevalent diabetes-related conditions.[9,10]

METHODS

This cross-sectional research was conducted at the Department of Biochemistry of Al-Rafai Education Hospital in Dhi Qar Iraq from 31st December 2024 to 31st March 2025 vide letter No. 43egr/QM/ Approval/rgEEUI3 dated September 24, 2024. A total of 90 participants were enrolled. The number of samples included 30 apparently healthy adults (C, aged 30-50 years of healthy volunteers), 30 patients with type 2 diabetes (DM, aged 38-65 years) and 30 patients with type 2 diabetes with neuropathy (DF, aged 40-80 years). All study participants underwent their annual medical examination at Al-Rifai Teaching Hospital. The subjects were those diagnosed with type 2 diabetes and its complications as defined by the World Health Organization diagnostic criteria. This study excluded patients who were found to have nephropathy, retinopathy, chronic kidney disease, chronic liver disease, tuberculosis, arthritis, systemic lupus erythematosus, mononucleosis, Bechet's disease, or cancer. Screening interviews were used where specific questions were asked to collect basic facts.

Venous blood samples of 5 ml each were collected in fasting state from the subjects using sterile method. 3 ml of blood sample was taken in gel tube for measurement of random blood sugar (RBS) in mg/dl and lipid profile including Triglyceride, Total Cholesterol HDL, LDL, VLDL in mg/dl unit and serum HB, firetine levels. We collected 2 ml of blood in EDTA tube for determination of hemoglobin A1c levels using automated biochemical analyzer. For measurement of hemoglobin A1c, a semi-automated biochemical analyzer known as HumaLyzer-3500 was used. However, the remaining parameters were analyzed using a spectrophotometer manufactured by Biotech Engineering. BMI is given as weight expressed in kilograms divided by square height measured in meters. The data was analyzed using SPSS-26. The Kolmogorov-Smirnov test was used to divide the variables across the research groups. The one-way ANOVA test was used to compute and compare the means and standard deviations of the homogeneously distributed variables. P values less than $P<0.05$ were used by Medcalc to calculate the ROC and statistical significance.

RESULTS

The DF patients who had diabetic neuropathy displayed considerably elevated FBS, HbA1c, and ferritin levels than participants in both control and T2DM groups. The

DF group presented with the greatest FBS mean values at 256.10±106.36 mg/dl whereas both results differed significantly from control patients and those with T2DM ($p < 0.001$). The mean HbA1c levels in DF patients stood at 8.88±1.72% above those measured in control and T2DM groups at $p<0.001$ significance. The DF group showed elevated ferritin levels which supported the notion of disease severity relationships.

Table No. 1: Comparison of HbA1c, HB, Firretin and BMI, among different groups (n=90)

Parameter	Controls	Diabetes mellitus	Diabetic nephropathy	P value	
Age (mg/dl)	41.40±11.23	49.23±7.62	58.03±9.064	Control _DM	0.001**
				Control*DF	0.001**
				Diabetic*DF	0.001**
Gender	1.46± 0.507	1.36±0.49013	1.500±0.5085	Control*DM	0.041*
				Control*DF	0.031*
				Diabetic*DF	0.051
BMI (Kg/m2)	27.36±3.60	34.61±5.76	30.82±5.186	Control*DM	0.9621
				Control*DF	0.065
				Diabetic*DF	0.031*
Duration		34.61±5.76	42.82±5.186	Diabetic*DF	0.031*

**p<0.01 is extremely significant. *p<0.05 is significant

Table No. 2: Comparison of FBS, HbA1c, HB, Firetin and BMI, among different groups (n=90)

Parameter	Controls	Diabetes mellitus	Diabetic nephropathy	P value	
FBS (mg/dl)	136.40±17.64	195.43±40.45	256.10±106.36	Control*DM	0.001**
				Control*DN	0.001**
				Diabetic*DN	0.001**
HbA1c %	4.87±0.51	7.79±1.43	8.88±1.72	Control*DM	0.001**
				Control*DN	0.001**
				Diabetic*DN	0.001**
HB	13.40±1.762	13.36 ± 1.53	11.75±2.31	Control*DM	0.9621
				Control*DN	0.065
				Diabetic*DN	0.031*
Ferritin	96.29±83.274	131.61 ± 89.71	162.76±180.98	Control*DM	0.9621
				Control*DN	0.065
				Diabetic*DN	0.031*

**p<0.01 is extremely significant. *p<0.05 is significant

Table No. 3: Comparison of lipid profile, Triglyceride, Cholesterol, HDL, LDL and VLDL among different groups (n=90)

Parameter	Controls	Diabetes mellitus	Diabetic nephropathy	P value	
Triglyceride	185.00±54.14	200.50±65.26	240.90±136.37	Control*DM	0.001**
				Control*DF	0.001**
				Diabetic*DF	0.001**
Total cholesterol	190.500±30.33	209.13±34.37	206.06±44.84	Control*DF	0.001**
				Conytrol*DF	0.001**
				Diabetic*DF	0.001**
HDL	47.200±13.324	54.7000±13.61	42.1667±15.66	Control*DM	0.001**
				Control*DF	0.001**
				Diabetic*DF	0.001**
LDL	108.93±27.48	111.63±29.109	106.73±39.82	Control*DM	0.030
				Control*DF	0.004
				Diabetic*DF	0.744
VLDL	35.03±9.21	43.40±10.47	49.96±29.76	Control*DM	0.030
				Control*DF	0.004
				Diabetic*DF	0.744

**p<0.01 is extremely significant. *p<0.05 is significant

Table No. 4: The Pearson correlation of haemoglobin A1c activity

Parameters	Hemoglobin AIc					
	Diabetes mellitus (n= 30)			Diabetic nephropathy (n=30)		
	r	p	Sig.	r	P	Sig.
FBS (mg/dl)	0.69538**	0.001	HS	0.7521**	0.001	HS
Hb	0.8601**	0.001	HS	0.876**	0.001	HS
Ferritin	0.4382**	0.058	S	0.4919**	0.0042	HS
BMI (Kg/m2)	0.46799**	.0071	HS	0.4528*	0.0450	S

Pearson coefficient (r), Significant (S) at p≤0.05, **Highly significant (HS) at p≤0.01

Table No. 5: The Pearson correlation of haemoglobin in males and females

Parameters	Hemoglobin					
	DM group (n= 30)			DF group (n=30)		
	r	p	Sig.	r	P	Sig.
FBS (mg/dl)	0.6991**	0.001	HS	0.636**	0.001	HS
HbA1c (%)	0.8601**	0.001	HS	0.876**	0.001	HS
BMI (Kg/m2)	0.6592**	0.001	HS	0.491**	0.004	S
Ferritin	0.3342	.0623	NS	0.3972*	0.0253	S

Pearson coefficient (r), Not significant (NS) p>0.05*, Significant (S) at p≤0.05, **Highly significant (HS) at p≤0.01

Table No. 6: The Pearson correlation of ferritin

Parameters	Ferritin					
	DM group (n= 30)			DF group (n=30)		
	r	p	Sig.	r	P	Sig.
FBS (mg/dl)	0.5863**	0.031	HS	0.5839**	0.001	HS
HbA1c (%)	0.4382**	0.058	S	0.4919**	0.0042	H
BMI (Kg/m2)	0.5863**	0.001	HS	0.3836*	0.0300	S
HB	0.4909**	0.0430	S	0.280	0.1182	No S

Pearson coefficient (r), Not significant (NS) p>0.05*, Significant (S) at p≤0.05, **Highly significant (HS) at p≤0.01

The DF group displayed temporary mild decreases in hemoglobin levels yet this change was not considered statistically important. Both HbA1c and FBS levels showed statistically significant correlations with BMI measurement in the DF group because obesity acts as an important risk factor. Diabetic patients with neuropathy show increasing metabolic problems (Tables 1-6, Figs. 1-2).

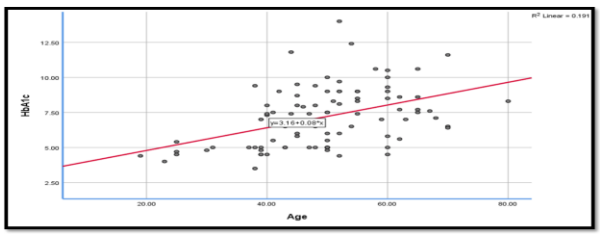

Figure No. 1: Correlation between Age and HbA1C

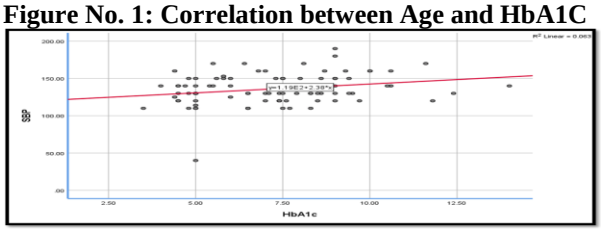

Figure No. 2: Correlation between SBP and HbA1C

DISCUSSION

The widespread chronic disease known as Type 2 diabetes mellitus causes several complications which diabetic neuropathy (DN) represents one of the major issues among patients.[11] Multiple research studies show poor diabetes management together with being overweight and having abnormal lipids play critical roles in developing diabetic neuropathy. The research evaluated fasting blood sugar, hemoglobin A1c, body mass index and ferritin levels together with hemoglobin and lipid profile results to establish their connection with diabetic neuropathy.[12]

Fasting Blood Sugar and HbA1c concentrations increased substantially in diabetic patients especially those with neuropathy compared to people in the control group according to this research. Extensive research has confirmed that prolonged high blood sugar levels play a central role in causing nerve damage for diabetic patients.[13] A Diabetes Care study established that a 1% rise in HbA1c increases diabetic neuropathy risk by 10%. Chronic high blood sugar levels generate oxidative stress with inflammation and damage the endothelium and these processes jointly harm nerve cells. The reduction of neuropathic complications in

diabetic patients is achievable when maintaining HbA1c levels at or below 7%.[14]

The study data shows obesity measured through BMI creates substantial connections between HbA1c and FBS levels mainly among patients who have neuropathy. The dual process of elevated insulin resistance and increased systemic inflammation intensifies diabetic complications because of obesity.[15] The study documenting how weight gain beyond safe limits leads to elevated inflammation and damage to nerve cells stands confirmed through research findings. The analysis uncovered evidence that weight reductions between 5 to 10 percent enhance insulin sensitivity and minimize the risk of diabetic neuropathy. The data demonstrates how effective weight control remains vital to stop diabetic neuropathy from appearing in patients with diabetes.[16]

The medical community recognizes dyslipidemia as a well-established diabetic complication risk factor that includes neuropathy as part of its effects. The research on diabetic neuropathy patients found that their blood TG and LDL cholesterol concentrations were higher and their HDL cholesterol levels were lower.[17] The DN group displayed significantly higher triglyceride level values which indicate that triglycerides could contribute to the damage of nerves. Scientific research shows that triglyceride (TG) levels exceeding 204 mg/dL lead to increased susceptibility for diabetic polyneuropathy.[18] The available evidence suggests high LDL levels may cause microvascular complications yet other researchers found no direct connection while healthcare providers should keep LDL levels under 100 mg/dL to reduce vascular and nerve complications. Reductions in HDL levels stand as a risk factor to develop DN.[19] Research based in Denmark proved that male participants with HDL below 39 mg/dL and female participants with HDL below 50 mg/dL showed increased rates of diabetic polyneuropathy.The connection between lipid management and diabetic patients became essential according to these results.[20]

The diabetic neuropathy participants had elevated ferritin levels when compared to other patient groups. When ferritin levels increase in the body they signal high iron stores yet these elevated levels frequently trigger oxidative stress together with chronic inflammation that damages nerves. Elevated ferritin levels in Egyptian diabetic patients with diabetic nephropathy support an expanded role of this factor in diabetes complications according to research findings.[21] Assessing ferritin levels in diabetic patients provides useful information to evaluate their neuropathy development risk.[22]

Studies revealed no meaningful distinctions in hemoglobin levels neither among the groups nor did they show an actual change in neuropathy participants. Researchers identify similar findings because diabetes causes impairment to red blood cell production through its impact on erythropoietin secretion.[23] Diabetic patients are more prone to developing mild anemia mainly because this condition makes neuropathy progression more likely. Veterinary practice involves recommending routine anemia testing for diabetic patients because it helps provide enough oxygen to peripheral nerves.[24]

CONCLUSION

Long-term type 2 diabetes combined with obesity together with lipid disturbances leads directly to diabetic neuropathy development. The levels of HbA1c, FBS, ferritin and triglycerides show that metabolic disorder plays an important role in causing nerve damage. Biochemical markers monitoring along with proper lifestyle adjustments can decrease the amount of diabetic complications patient's experience. Academics should pursue research to develop new ways to prevent neuropathy in diabetic patients.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Hussein Flayyih Hassan, Auday Abd Al-Razaq Al-Husseiny
Drafting or Revising Critically:	Sadeq Lafta Mousa AL-Zamili, Qayssar A. Obaid
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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