EFFECT OF VITAMIN D SUPPLEMENTATION ON LIPID PROFILE AND GLYCEMIC CONTROL IN TYPE 2 DIABETICS WITH UNTREATED DYSLIPIDEMIA

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Introduction

Vitamin D ,a secosteroid hormone , is well known for its roles in calcium metabolism, bone health, and immune system modulation. Emerging evidence suggests that vitamin D may modulate metabolic processes, especially insulin sensitivity and lipid metabolism, through its nuclear receptor (VDR) found in pancreatic β-cells, adipocytes, vascular endothelium, and skeletal muscle. Type 2 Diabetes Mellitus (T2DM) persists as a global health problem. Often associated with dyslipidaemia, this raises the risk of cardiovascular disease substantially. Vitamin D deficiency is prevalent among Egyptian adults, owing to limited sun exposure, sedentary lifestyles, and inadequate dietary intake. This study aims to determine whether recommended replacement doses of vitamin D with the aim of correcting vitamin D deficiency in patients with uncontrolled T2DM and untreated dyslipidaemia could improve their lipid profiles and glycaemic control. Understanding these interactions is critical to optimise therapeutic strategies and reduce cardiovascular morbidity and mortality in this high-risk population .

Aim of the Work

The aim of this study is to evaluate the effect of vitamin D supplementation on:
Plasma lipid profile and glycemic control (Fasting blood glucose, HbA1C) in patients with type 2 diabetes mellitus with associated untreated dyslipidemia and vitamin D deficiency.

Patients and Methods

Inclusion criteria : 80 Patients on oral antidiabetic agents. Vitamin D <20 nanograms/milliliter.
Plasma lipid profile: Total Cholesterol > 200 mg/dl, LDL-C > 100 mg/dl, HDL-C <40 mg/dl, Triglycerides > 150 mg/dl. **Exclusion criteria** Regular intake of vitamin D or drugs affecting plasma lipid profile . Underlying diseases affecting vitamin D metabolism or plasma lipid profile .
Methods : 80 cases were divided into 2 groups A and B according to their therapeutic intervention, each consisting of 40 cases of uncontrolled type 2 diabetes (HbA1C >7% and fasting blood glucose >130 mg/dl) with untreated dyslipidemia : Group A received standard of care of dyslipidemia and diabetes , while group B received standard of care plus oral vitamin D3 supplementation; 50.000 IU/week for 3 months . All cases were subjected to: Physical examination , anthropometric parameters. Laboratory investigations : Fasting blood glucose, Glycated Haemoglobin (HbA1C) , Plasma Lipid profile, 25(OH) vitamin D level. Laboratory investigations were done initially for both groups of the study and 3 months afterwards.

Table (1) : Comparison between the two studied groups according to 25(OH) vitamin-D

25(OH) vitamin-D	Group A (n = 40)	Group B (n = 40)	t	p
Initial (ng/ml)				
Min. – Max.	6.80 – 17.0	6.0 – 16.90		
Mean ± SD.	11.35 ± 2.17	10.18 ± 2.21	2.393*	0.019*
Median (IQR)	11.10(10.1 – 12.4)	9.55(8.85 – 11.30)		
Follow up (ng/ml)				
Min. – Max.	7.0 – 16.0	31.0 – 48.0		
Mean ± SD.	12.19 ± 2.32	38.36 ± 4.49	32.753*	<0.001*
Median (IQR)	12.45(10.90 – 14.10)	38.0(35.0 – 41.3)		
p1	<0.001*	<0.001*		

*: Statistically significant at $p \leq 0.05$

Results

Table (2) : Comparison between the two studied groups according to glycemic profile

	Glycemic profile	Group A (n = 40)	Group B (n = 40)	U	p
HbA1C %	Initial				
	Min. – Max.	7.30 – 14.10	7.10 – 11.90		
	Mean ± SD.	8.13 ± 1.40	8.36 ± 1.13	644.00	0.132
	Median (IQR)	7.50 (7.40 – 8.35)	8.05(7.5 – 9.0)		
	Follow up				
	Min. – Max.	6.10 – 12.50	6.89 – 11.11		
	Mean ± SD.	7.20 ± 1.28	8.52 ± 1.13	242.00*	<0.001*
	Median (IQR)	6.80(6.5 – 7.4)	8.33(7.6 – 9.4)		
	p_1	<0.001*	0.667		
	% Reduction				
	Min. – Max.	0.0 – 18.75	-33.06 – 9.09		
	Mean ± SD.	11.39 ± 4.06	-2.38 ± 10.24	89.50*	<0.001*
	Median (IQR)	12.25 (9.21 – 13.70)	-1.75 (-3.98 – 5.29)		

*: Statistically significant at $p \leq 0.05$

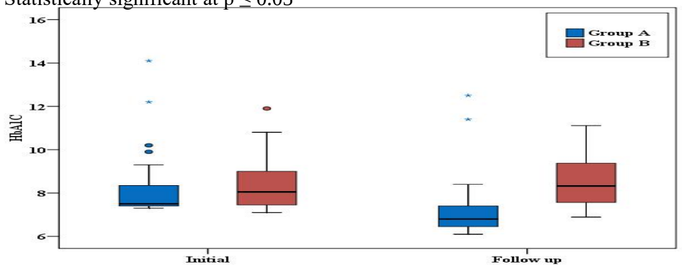

Figure 1: Comparison between the two studied groups according to HbA1C

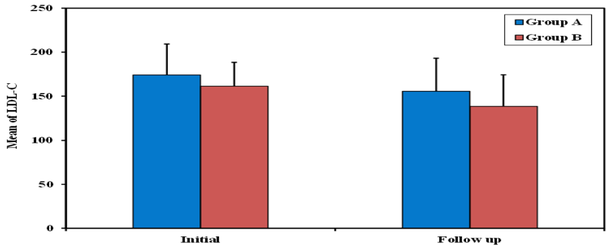

Figure 2: Comparison between the two studied groups according to LDL-C

Table (3) : Comparison between the two studied groups according to lipid profile

	Lipid profile	Group A (n = 40)	Group B (n = 40)	Test of Sig.	p
Total cholesterol mg/dl	Initial				
	Min. – Max.	201.0 – 321.0	201.0 – 308.0		
	Mean ± SD.	244.8 ± 35.64	232.2 ± 29.47	U= 659.00	0.175
	Median (IQR)	244.0 (209.0 – 279.0)	224.0 (209.5 – 248.0)		
	Follow up				
	Min. – Max.	181.0 – 344.0	149.0 – 345.0		
	Mean ± SD.	224.1 ± 37.52	203.9 ± 37.67	U= 535.00*	0.011*
	Median (IQR)	225.0 (188.0 – 254.5)	196.0 (176.0 – 232.5)		
	$^z p_1$	<0.001*	<0.001*		
	% of reduction				
	Min. – Max.	-20.11 – 18.68	-33.20 – 26.60		
	Mean ± SD.	8.52 ± 6.20	12.07 ± 12.51	U= 435.00*	<0.001*
	Median (IQR)	9.86 (9.09 – 10.13)	15.49 (5.78 – 19.95)		
LDL-C mg/dl	Initial				
	Min. – Max.	106.8 – 252.8	118.0 – 236.4		
	Mean ± SD.	174.0 ± 35.20	161.2 ± 27.15	t= 1.814	0.074
	Median (IQR)	173.2 (141.9 – 197.9)	162.1 (142.8 – 172.7)		
	Follow up				
	Min. – Max.	90.10 – 278.6	78.40 – 270.2		
	Mean ± SD.	155.5 ± 37.61	138.3 ± 35.97	t= 2.089*	0.040*
	Median (IQR)	154.7 (123.7 – 178.1)	131.8 (113.8 – 162.0)		
	$^t p_1$	<0.001*	<0.001*		
	% of reduction				
	Min. – Max.	-27.80 – 23.92	-39.42 – 37.97		
	Mean ± SD.	10.90 ± 8.58	14.15 ± 17.23	U= 556.0*	0.019*
	Median (IQR)	12.80 (11.56 – 13.61)	19.41 (3.36 – 24.98)		

*: Statistically significant at $p \leq 0.05$

Conclusion

In T2DM patients with untreated dyslipidaemia and vitamin D deficiency, high-dose vitamin D supplementation to sufficiency significantly improved total cholesterol and LDL-C. Contrary to expectations, supplementation worsened fasting blood glucose and HbA1C. In chronic T2DM, vitamin D's metabolic effects seem to affect lipid metabolism more than glucose homeostasis.
Variability in glycaemic outcomes may be due to MENA populations' elevated baseline BMI, β-cell reserve, and VDR polymorphisms. Vitamin D supplementation may improve lipid profiles in diabetic dyslipidaemia, but it should not be relied on solely to improve glycaemic control. Future research should examine how genetic, phenotypic, and dosing variables interact to personalise vitamin D therapy for cardiometabolic benefits.