Effect of Vitamin D supplementation on Lipid profile in Vitamin



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Background

It was suggested that vitamin D (VD) has both direct and indirect effects on modifying the lipid profile in patients with diabetes through its regulatory action that increases the activity of lipoprotein lipase in adiposity (**Wang et al., 2009**). There is no general agreement on the effects of 25OHD on serum levels of TG in interventional studies with supplementation of VD. Although a positive association was observed in some studies, other studies showed an inverse relationship between serum levels of 25OHD and TG (**Jorde and Grimnes, 2011**).

Objectives

To detect the relationship between serum 25OHD and lipid profiles in patients with T1D and dyslipidemia and to study the effect of VD supplementation on lipid profiles of VD deficient T1D patients with dyslipidemia.



This prospective cohort study included 50 patients with T1D and dyslipidemia with history of diabetes more than 2 years (after taking informed consents from their legal guardians). Patients with hypothyroidism, chronic liver or kidney disorders, those who used drugs affecting the lipid profile or calcium and bone metabolism were excluded. Patients included in the study were subjected to full history taking including age of the patient, onset and duration of diabetes, insulin regimen (type and dose), glycemic control as judged by frequency of DKA per year, mean HbA1c, symptoms suggestive of hypertension (e.g, headache) and family history of dyslipidemia, hypertension or premature coronary heart disease. Thorough general examination was performed with special emphasis on anthropometric measures (weight, height and BMI) and BP assessment. Biochemical assessment for HbA1c, thyroid functions (free T4 and TSH), liver enzymes (ALT) and kidney functions (creatinine), fasting (14 hours) lipid profile (TC, TG, LDL-C, HDL-C) and Vitamin D level were done initially. According to results of 250HD, patients were divided into 2 groups; vitamin D deficiency (VDD) and vitamin D sufficiency. Those with VDD received vitamin D3 in the form of 4000 IU/day for a period of 4 months. Then, lipid profile was re-evaluated for both groups.

Results

This cohort study included 50 patients (25 males and 25 females) with mean age of 12.56 ±3.53 years and mean age at the onset of diabetes of 5.06 ±2.35 years. Family history was positive for T2D in 17 patients (34%), for hypertension in 12 patients (24%), for coronary heart diseases in 8 patients (16%) and for dyslipidemia in 4 patients (8%). Blood pressure was within normal range in all patients according to their ages and sex according to Egyptian blood pressure curves (between 25th and 75th centiles). Pubertal assessment of the study group showed that 52% were pubertal and 48% were pre-pubertal. Mean values for lipid profile were 128.37 ± 36.12 mg/dl for LDL, 52.02 ± 14.42 mg/dl for HDL, 211.46 ± 41.36 mg/dl for TC and 117.74 ±60.98 mg/dl for TG.

Serum 25(OH) D levels ranged from 0.1 to 62 ng/ml with a mean of 25.95 ng/ml. There was no statistically significant correlation between vitamin D level and different studied parameters (age, diabetes duration, hypoglycemia frequency, DKA frequency, insulin dose, HbA1c, thyroid functions, ALT, creatinine and lipid profile within the study group (p>0.05).

The patients were then divided into two groups according to their vitamin D level; group (A) with vitamin D deficiency (< 20 ng/ml) or insufficiency (20-29ng/ml) and included 30 patients and group (B) with sufficient vitamin D levels (\geq 30 ng/ml) including 20 patients. There was statistically significant difference among the two studied groups regarding family history of coronary heart disease (p= 0.036). Regarding biochemical assessment, no significant difference was found between the 2 groups except in free T4 (p = 0.035). No statistically significant difference was found as well among the two studied groups regarding basal LDL, basal HDL, basal cholesterol and basal triglycerides (p>0.05).

Table (3): Clinical and biochemical parameter	s in	n both	groups
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			Group (A)		Group (B)		p value	
Age (years)		12.7	'8 ± 3.66	12.45 ± 3.4		0.747		
Diabetes onse	Diabetes onset (yrs)		4.6	5 ± 2.53 5.68 ±		2.17	0.132	
DKA frequency (/yr)		0.7	3 ± 1.57	0.5 ± 1		0.56		
Hypoglycemia frequency /yr		1.97 ± 4.3		1.75 ± 3.73		0.86		
Insulin requirements (IU/kg/d)		1.12 ± 0.55		1.22 ± 0.66		0.55		
TSH (μlU/ml)	TSH (μIU/ml)		1.66±0.89		1.8±0.57		0.52	
Free T4 (ng/dl)		1.13±0.23		1.26±0.14		0.035*		
ALT (U/L)		21.17±8.58		23.1±7.85		0.42		
Creatinine (mg/dl)		0.63±0.17		0.59±0.16		0.48		
HbA1c (%)	Ba	sal	8.93±1.85		8.71±0.74		0.61	
	Ро	st VD	8.86±1.6		8.5±1.32		0.05*	
LDL (mg/dl)	Basal		126.9±35.14		130.6±38.36		0.73	
	Post VD		117.13±31.9		128.55±31.1		0.56	
HDL (mg/dl) Bas Pos		sal	50.67 <u>+</u> 13.31		54.05 <u>+</u> 16.07		0.42	
		st VD	54.23±16.07		50.95±14.17		0.46	
Cholesterol (mg/dl)	Basal		208.8±43.96		215.4±37.9		0.59	
	Post VD		201±54.26		199.3±32.4		0.43	
Triglycerides	Basal		111.6±47.55		127±77.38		0.39	
(mg/dl)	Ро	st VD	111.4±43.84		131.5±65.51		0.36	
		Group	A Grou		up B	p value		
HbA1c (%)	0.07 (0.4		0.21		(0.34)	(0.04*	
LDL (mg/dl)	9.78 (8.6		57)	2 (11	L.04)	0.02*		
HDL (mg/dl)	-3.56 (3.8		31)	3.46 ((4.79)	0.47		
TC (mg/dl)		7.83 (12.7	75) 16.1 (11.15)		0.99	
TG (mg/dl)	(mg/dl) 0.17 (11.		81)	-4.5 (22.67)		0.2		



Figure(1): Pattern of dyslipidemia within study group

 Table (1): Correlation between VD and study paranmeters.

	r (correlation	p value
	coefficient)	
Age (yrs)	-0.179	0.21
Duration of diabetes (yrs)	0.028	0.85
Hypoglycemia (no./yr)	-0.133	0.36
DKA (no./yr)	-0.076	0.56
Insulin dose (IU/kg/d)	0.125	0.387
HbA1c (%)	-0.005	0.97
ALT (U/L)	0.233	0.104
Creatinine (mg/dl)	-0.120	0.408
Free T4 (ng/dl)	0.188	0.192
TSH (μU/ml)	0.074	0.61
LDL (mg/dl)	0.156	0.278
HDL (mg/dl)	0.111	0.443
Total cholesterol (mg/dl)	0.187	0.194
Triglycerides (mg/dl)	0.160	0.266

Table (2): Family history of diseases in both groups.

		Group (A)		Group (B)		р
		No.	%	No.	%	value
Sex	Male	15	50	10	50	0.613
	Female	15	50	10	50	
Family history of	yes	2	6.7	6	30	
Coronary heart disease	no	28	93.3	14	70	0.036*
Family history of	yes	3	10	1	5	
dyslipidemia	no	27	90	19	95	0.472
Family history of	yes	6	20	7	35	
hypertension	no	24	80	13	65	0.196

Patients within group (A) were supplemented with VD (4000 IU daily) for 4 months, then lipid profile and HbA1c were reassessed in both groups. Results (after 4 mo) showed a significant difference between both groups in HbA1c (p= 0.05). There was improvement in HbA1c in both groups (at 0 and 4 mo). However, this improvement was not statistically significant (p > 0.05). There was no statistically significant difference between the two studied groups regarding LDL, HDL, TC and TG (basal and after 4 months) and also there was no significant difference in these parameters at 0 and at 4 mo within each group. However, when the mean difference at 0 and 4 mo was calculated, a statistically significant difference was detected between both groups in LDL (p = 0.02).

LDL (basal and at 4 mo) in both groups



Figure(2): LDL (basal and at 4 mo) in both groups.

Conclusion

VDD was highly prevalent in patients with T1D.There was no significant correlation between 250HD levels and lipid profile. VD supplementation for 4 months had a significant lowering effect on LDL.

Disclosure: Authors have no confict of interest to disclose

Bibliography

*Wang JH, Keisala T, Solakivi T, Minasyan A, Kalueff AV and Tuohimaa P (2009): Serum cholesterol and expression of ApoAI, LXR [beta] and SREBP2 in vitamin D receptor knock-out mice. The J Steroid Biochem ; 13(3–5):222–226. *Jorde R and Grimnes G (2011): Vitamin D and metabolic health with special reference to the effect of vitamin D on serum lipids. Prog Lipid Res.; 13(4):303–312.

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 Diabetes

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