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Effect of Cholecalciferol Supplementation on Glycemic Control, Beta Cell Function and Insulin Resistance among Type 2 Diabetics Attending the Family Medicine Outpatient Clinic Affiliated to Suez Canal University Hospital, Ismailia City, Egypt

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Abstract

Background: Diabetes is a complex chronic illness that adversely affects patients' quality of life. Approximately 425 million adults (20-79 years) were living with diabetes in 2017. Vitamin D levels had been shown to alter insulin synthesis and secretion suggesting its role in the pathogenesis of type 2 diabetes mellitus.

Aim of Study: To promote the quality of care provided to type 2 diabetic patients in the family practice setting.

Patients and Methods: This is a randomized controlled trial conducted on 60 uncontrolled type 2 diabetics. Patients were randomly allocated to Vitamin D_3 group; received oral daily 2000IU of Cholecalciferol plus their usual care and a control group; received only their usual care for 3 months. Baseline anthropometrics, blood pressure, FBS, HBA_1C, fasting insulin, HOMA indices and lipid profile were measured and repeated after 3 months.

Results: No statistically significant sociodemographic differences were found between both groups. The majority were younger than 60 years, hypertension was found in 76.7% of them. There were post intervention statistically significant differences ($p<0.05$) between both groups in blood pressure, FBS and HOMA-β. The pre-post relation in the intervention group shows statistically significant differences ($p<0.05$) in blood pressure, FBS, fasting insulin, HOMA-β and HOMA-IR. There is a statistically significant positive correlation between HBA_1C, blood pressure, and FBS.

Conclusion: Adding a daily dose of 2000IU of oral Vitamin D_3 for type 2 diabetic patients may be beneficial through improving blood pressure, fasting blood glucose and HOMA-β.

Key Words: *Diabetes – Cholecalciferol – HBA_1C – HOMA-β – HOMA-IR.*

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Introduction

DIABETES is a complex chronic illness that is aggressively becoming a major burden all over the world **[1,2]**. Approximately 425 million adults (20-79 years) were living with diabetes in 2017 world-wide; by 2045 this will rise to 629 million. IDF (International Diabetes Federation) estimates that about 39 million persons were diagnosed as type 2 diabetics in the Middle East and North Africa in 2017, a number that will almost double to 82 million by 2045 **[3]**.

Vitamin D levels had been shown to alter insulin synthesis and secretion suggesting its role in the pathogenesis of type 2 diabetes mellitus **[4]**. The effect of Vitamin D is thought to be through a direct action on beta-cell function, Vitamin D Receptors (VDR) and Vitamin D-binding Proteins (DBP) in the tissue of pancreas **[4,5]**. Vitamin D at the same time may affect glucose homeostasis by regulation of plasma calcium levels which affects insulin synthesis and secretion **[6]**.

There was a negative correlation between Vitamin D serum levels, metabolic control and insulin resistance among diabetic patients **[7-9]**. Many intervention trials had documented the positive impact of different concentrations of cholecalciferol supplementation on type 2 glycemic control; on the contrary other studies had proved the lack of association between Vitamin D supplementation and improving glycemic control **[10]**.

Type 2 diabetes mellitus is a growing problem in Egypt and it's accompanied by short and long term complications that adversely affect patients' health and quality of life which is associated with

enormous related costs. So, in this study we are going to improve the quality of care provided to type 2 diabetics in Family Medicine practice through detection whether adding Vitamin D_3 to their routine plan of management will be beneficial or not.

Patients and Methods

This is a nine months randomized single blinded clinical trial (from September 2016 to May 2017) with a pre-post assessment of the effect of adding oral Cholecalciferol to the treatment regimen for type 2 diabetics attending the Family Medicine clinic, Suez Canal University Hospital, Ismailia City, Egypt. Our inclusion criteria was uncontrolled type 2 diabetics aged 18 years and elder while according to patients' medical records; our exclusion criteria were type 1 diabetes, recent macrovascular complications, decompensated liver disease, chronic kidney disease, known malignancies, Vitamin D supplements within previous 3 months, hypervitaminosis D symptoms, pregnancy and lactation. Ethical clearance from the Institutional Ethical Committee and informed consent from patients were taken. A total of randomly selected 60 type 2 diabetics (26 males and 36 females) were included in the study. Our sample size was 60 patients according to this equation:

$$n = \frac{[(Z\alpha/2 + Z\beta)2 \text{ X } \{2(Ó)2\}]}{(\propto 1 - \mu 2)2} \quad \textbf{[11]}$$

Sampling and random allocation:

Multistage sampling was used; uncontrolled diabetic patients were selected and then listed by names, 60 uncontrolled diabetic patients had been randomly selected using simple random sampling then they were randomly allocated into a 1:1 allocation ratio. Every patient had an equal chance to be selected either group.

Stages of the study:

1- *Pre-intervention stage:* For both groups; an Interview questionnaire **[12]** was used which covered personal, socio-demographic, medical and drug histories. Body Mass Index (BMI) was calculated by dividing weight (kg) to height (m^2) **[13]**. Blood Pressure (BP) was recorded after the subjects had rested for at least 5min. All subjects were asked to submit overnight fasting blood samples to analyze the different metabolic parameters. Sera were separated by centrifuging blood at 3,000rpm for 15 minutes and stored at –20ºC until analysis then it was used for estimation of HBA_1C, FBS, and fasting insulin which was measured using the Cobas® electrochemiluminescence **[14]** immunoassay (Elecsys Insulin Assay) with absence of cross-reactivity of exogenous insulin **[15]**. Sera for lipids were centrifuged for 10 minutes at 3,000rpm, for collection of the aqueous phase. TC, HDL-C, and TG were measured by BioMerieux Laboratory, Marcy l'Etoile, France while LDL-C was calculated according to the Friedewald formula **[16]**.

HOMA indices were calculated through the following equations:

$$\text{HOMA-IR} = \frac{\text{Glucose X Insulin}}{405} \quad \textbf{[17]}$$

IR is insulin resistance.

$$\text{HOMA-}\beta = \frac{360 \text{ X Insulin}}{\text{Glucose} - 63} \quad \textbf{[17]}$$

β is the β-cell function.

2- *Intervention stage:* The intervention group had received Vitamin D_3 supplementation in the form of Cholecalciferol 2000IU daily for 12 weeks. Oral liquid Vitamin D_3 was purchased from the Medical Union pharmaceutical. Each 1ml of the used drug contains 2800IU of Cholecalciferol (each drop is equivalent to 100IU of Vitamin D_3), so patients were asked to consume a daily dose of 20 drops of the medicine. Patients were instructed to store the medicine in a temperature below 25ºC. The oral route of administration had been selected as it results in less inter-individual variability in achieved serum 25(OH) D concentrations compared to the intramuscular route **[18,19]**, it was also used by other investigators in previous studies and showed safety of the dosage regimen that had been used in our trial **[20]**. The control group didn't receive oral Vitamin D_3 supplementation. Both groups had followed their medications for diabetic control and both were advised on maintaining their usual dietary and physical activity habits at the time of intervention. Instructions to report any kind of therapy other than prescribed were given. Patients were asked to return bottles at every follow-up visit to assess adherence.

Safety: Using 2000IU of Cholecalciferol is a recommended daily dosage by the Institute of Medicine on 2011 **[21]**.

Follow-up and outcome measures:

On the 12th week of the study; both groups had undergone laboratory investigations for HBA_1C, FBS, fasting insulin and lipid profile. HOMA-IR, HOMA-β were assessed according to the previously discussed equations. All investigations were held at Suez Canal University Hospital Laboratory. Laboratory was blind to groups of therapy. Our

safety outcomes were according to the reported adverse events; patients developed symptoms similar to that of hypervitaminosis D had been excluded from the study.

Results

There are statistically significant differences (p-value <0.05) in four outcome variables; systolic blood pressure, diastolic blood pressure, fasting blood sugar and HOMA-β after 12 weeks, while the rest of variables didn't show any significance.

The post intervention decrease in systolic, diastolic blood pressure and fasting blood sugar (post-test minus pre-test) is highly statistically significant (p<0.05), on the contrary fasting Insulin, HOMA-IR and HOMA β had been increased on the 12th week of intervention compared to base line values with a statistically significant difference (p<0.05). HOMA-IR is a means to assess the peripheral insulin resistance with a normal value of healthy human ranges from 0.5-1.4, more than 1.9 indicates early insulin resistance while more than 2.9 indicate significant insulin resistance [22].

There is a statistically significant difference noted between both groups' post intervention HBA-IC (p-value <0.05).

There are statistically significant positive correlations between the post intervention HBA1C, systolic blood pressure and fasting blood sugar, while both fasting Insulin and HOMA-β had statistically significant negative correlations.

There is significant linear association between HbA1C with FBS and fasting Insulin.

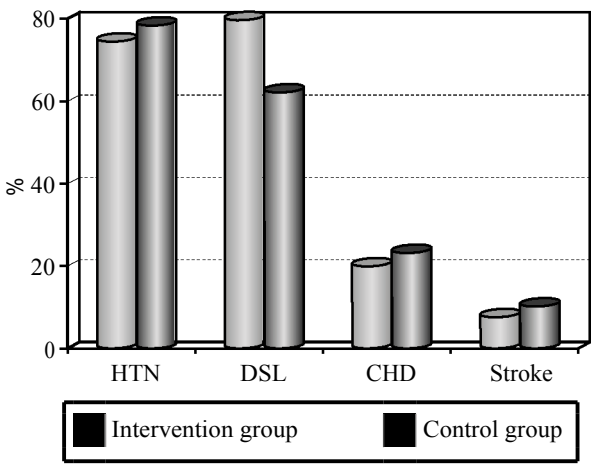

Fig. (1): Distribution of study groups according to their associated comorbidities.

Table (1): Sociodemographic characteristics of the two study groups.

Patient variables	Cholecalciferol group (n=30) %	Control group (n=30) %	p-value
Gender:			
• Male	11 (36.7%)	13 (43.3%)	0.598
• Female	19 (63.3%)	17 (56.7%)	
Socio-economic status:			
• Very low	3 (10%)	4 (13.3%)	0.400
• Low	14 (46.7%)	18 (60%)	
• Middle	13 (43.3%)	8 (26.7%)	
Usual source of health care:			
• More than one source	7 (23.3%)	2 (6.7%)	0.129
• Free governmental health services	23 (76.7%)	27 (90%)	
• Health insurance	0 (0%)	1 (3.3%)	
Treatment:			
• Oral drugs	16 (53.3%)	17 (56.7%)	0.795
• Mixed Insulin either alone or combined with Metformin	14 (46.7%)	13 (43.3%)	

Table (2): Baseline clinical characteristics of the study groups.

Clinical characteristics	Cholecalciferol group (n=30)		Control group (n=30)		t-value	p-value
	Mean	S.D	Mean	S.D		
Age	54.57	6.01	54.40	6.08	0.11	0.915
Weight	85.1	11.09	85.20	12.03	0.03	0.973
BMI	31.47	3.42	30.54	4.17	0.94	0.353
Systolic BP	130.50	12.41	131.00	15.89	0.136	0.892
Diastolic Bp	82.00	6.51	82.17	7.6	0.091	0.928

S.D : Standard Deviation. BMI : Body Mass Index.
SES : Socioeconomic Status. B.P : Blood Pressure.

Table (3): Comparison between study groups regarding clinical and biochemical outcome variables in the 12th week of follow-up.

Out-come variables (12th week)	Intervention group (n=29)		Control group (n=30)		t-value	p-value
	Mean	(SD)	Mean	(SD)		
Systolic BP	118.97	13.91	131.67	17.68	3.059	0.003*
Diastolic BP	73.28	5.39	81.17	7.85	4.49	0.000*
Weight	85.34	10.27	86.27	10.92	0.334	0.740
BMI	31.53	3.1	30.54	3.73	1.108	0.273
FBS	156.48	45.39	193.27	37.48	3.401	0.001*
HbA1 C	8.07	1.17	8.59	1.23	1.67	0.1
Fasting Insulin	23.29	13.58	17.31	9.45	1.97	.05
HOMA-IR	8.31	4.09	7.91	4.14	0.371	0.712
HOMA-β	153.9	248.3	55.73	43.7	2.13	0.037*
LDL	113.8	39.43	116.47	24.94	0.308	0.759
HDL	43.31	10.77	40.4	10.77	1.04	0.304
TG	148.8	78.46	155.37	74.13	0.322	0.748

**: n=59 because of one patient withdrawal in the intervention group.
HOMA-IR: Hemostatic Model Assessment for Insulin Resistance.
HOMA-β: Hemostatic Model Assessment for Insulin Beta cell function.
*: Statistically significant (p-value <0.05).
S.D : Standard Deviation.
B.P : Blood Pressure.
F.B.S : Fasting Blood Sugar.
HbA1 C : Glycosylated Hemoglobin.
LDL : Low Density Lipoprotein.
HDL : High Density Lipoprotein.
TG : Triglycerides.

Table (4): Comparison between pre intervention (baseline) and post intervention (12th week) follow-up regarding the outcome variables of the intervention group (Cholecalciferol group; n=29**).

Outcome variables	Pre intervention (*t* 0)		Post intervention (*t* 2)		Mean difference *t* 0-*t* 2		*t*-value	*p*-value
	Mean	S.D	Mean	S.D	Mean	S.D		
Systolic BP	130.52	12.63	118.97	13.91	11.55	13.24	0.000	0.000*
Diastolic BP	81.9	6.6	73.28	5.8	8.62	8.65	0.137	0.000*
Weight	85.34	11.21	85.34	10.27	0.000	2.44	4.70	1
BMI	31.56	3.44	31.53	3.1	0.03	1.22	5.37	0.892
FBS	174.52	43.57	156.48	45.39	18.04	38.11	2.55	0.017*
HBA1C	8.41	1.01	8.07	1.17	0.34	1.042	1.77	0.89
F. Insulin	17.26	13.06	23.29	13.58	–6.024	11.57	2.8	0.009*
HOMA-IR	7	4.67	8.3	4.01	–1.34	3.45	2.09	0.05*
HOMA-P	73.82	75.47	153.94	248.38	–80.12	205.43	2.1	0.045*
LDL	123.1	38.73	113.8	39.43	9.28	27.25	1.8	0.077
HDL	42.6	9.14	43.3	10.78	–0.66	8.84	0.4	0.693
TG	149	69.13	149	78.46	0	58.51	0	1

** : n=29 because of one patient withdrawal in the intervention group.
HOMA-IR : Hemostatic Model Assessment for Insulin Resistance.
HOMA-P : Hemostatic Model Assessment for Insulin Beta cell function.
*t*0 : Pre-test.
*t*2 : Post-test.
S.D : Standard Deviation.
B.P : Blood Pressure.
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Table (5): Relation between patients' adherence to their medications and the 12th week FBS and HBA_1C.

Outcome variables	Adherent (n=27)		Non-adherent (n=2)		*t*-value	*p*-value
	Mean	S.D	Mean	S.D		
12th week FBS	154.3	46.1	185.5	24.7	0.935	0.358
12th week HBA1C	7.95	1.12	9.7	0.42	2.17	0.039*

**: n=29 because of one patient withdrawal.

Table (6): Correlation between HBA_1C and patient's continuous variables on (n=59**).

Variables	*r*	*p*
Age	0.106	0.426
BMI	–0.116	0.382
Systolic blood BP	0.445	0.000*
Diastolic BP	0.237	0.071
FBS	0.829	0.000*
Fasting insulin	–0.509	0.000*
HOMA-IR	–0.202	0.124
HOMA-P	–0.445	0.000*
LDL	0.247	0.06
HDL	–0.067	0.612
Triglycerides	0.212	0.108

Dependent variable: Post intervention HbA1 C.
r : Pearson's correlation coefficient.
** : n=59 because of one patient withdrawal in the intervention group.
F.B.S : Fasting Blood Sugar.
HbA1C : Glycosylated Hemoglobin.
HOMA-IR : Hemostatic Model Assessment for Insulin Resistance.
HOMA-P : Hemostatic Model Assessment for Insulin Beta Cell Function.
* : Statistically significant (*p*-value <0.05).

Table (7): Multiple regression (analysis of variance, ANOVA): Predictors of post intervention HBA_1C in the 12th week of follow-up in both study group.

	Un-standardized coefficients		Beta	*t*	*p*
	B	Std. Error			
(Constant)	4.202	0.789		5.33	0.000
FBS	0.021	0.002	0.767	8.36	0.000*
Systolic BP	0.007	0.006	0.094	1.177	0.244
Fasting Insulin	–0.023	0.011	–0.221	–2.06	0.044*
HOMA-P	0.001	0.001	0.156	1.409	0.165

Dependent Variable: HbA1C on the 12th week of follow-up.
F.B.S
BP
HbA1C
HOMA-P ell function.
* : Statistically significant (*p*-value <0.05).

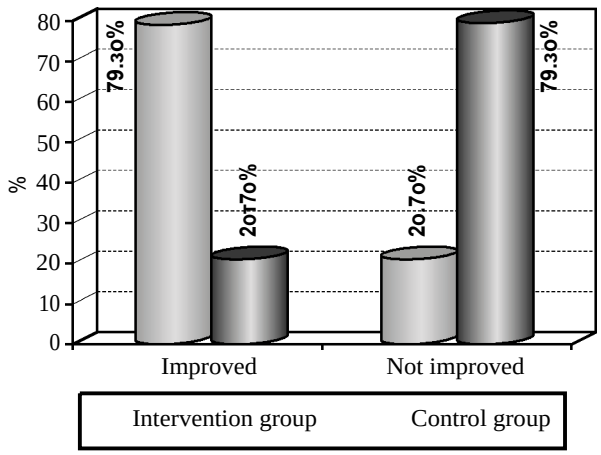

Fig. (2): Distribution of the study groups regarding their glycemic improvement on 12th week follow-up (n=59).

Discussion

Socio-demographic characteristics:

The majority of our patients in both groups were younger than 60 years. This finding is in congruence with Al-Daghri et al., **[23]**, Al-Sofiani et al., **[24]** and Al-Shahwan et al., **[10]**. It was confirmed that age is one of the important risk factors for T_2DM as aging induces decreased insulin sensitivity and insufficient compensation of beta cell function in the face of increased insulin resistance **[25]**. In our study; female gender was slightly higher among patients of both groups in a percentage of 60 similarly; Heshmat et al., had 64% female subjects **[26]**. Female gender was high in our study population as it reflects the frequency of gender distribution among the attendants of the Family Medicine Clinic. More than half of our study population was of the low socioeconomic level in a percentage of 53.3 which was harmonious with other studies like Veghari et al., **[27]** and Hwang et al., **[28]** that were all carried out in developing low income countries under similar circumstances. Simultaneously our study setting is considered a governmental institution whose customers are mainly of the low socioeconomic level searching for getting the benefit of the free governmental services that had been used by more than three fourths (83.3%) of the studied population. This is in agreement with the DeVoe et al., **[29]** who found that more than 87% of the diabetic individuals in the U.S. had full-year coverage with Usual Source of Care, whereas Zhang et al., **[30]** had estimated 16.0% of known diabetic adults were uninsured.

Associated comorbidities and antihyperglycemic medications:

Hypertension and dyslipidemia were the most prevalent chronic illnesses among the study population (76.7% and 68.3% respectively). Jelinek et al., **[17]** had the same declaration as he had found that hypertension (83.40%) and dyslipidemia (93.43%) were the most common associated comorbidities to type 2 diabetes that is in congruence to the findings of the Indian study by Borah et al., **[31]**. Whiledisagreed with Reddy et al., **[32]** who found only 33.3% of their diabetic participants had hypertension.

In this study more than half of our patients used Oral Glucose Lowering Drugs (OGLD) for the control of their T_2DM (53.3% and 56.7% in intervention and control groups respectively) without significant difference between both groups. This is in agreement with the results of Chadli et al., **[33]** who estimated 61% were treated with an OGLD alone while 23% received an OGLD plus insulin and 13% received insulin only. On the other hand Hanne et al., **[34]** found OGLD were used as monotherapy in only 39.4% while insulin was used in 54.5%.

Baseline clinical and biochemical characteristics:

Our distributions of the baseline clinical and biochemical characteristics were identical across the treatment groups and this is in congruence to the findings by Forouhi et al., **[35]**. Most of our patients in both groups were obese in agreement with Al-Shahwan et al., and Nada et al., **[10,36]**. However the majority of the current participants had controlled blood pressure which is consistent with Forouhi et al., and Nada et al., **[35,36]**.

The baseline HBA_1C among our participants showed a non significantly slightly higher value in the Cholecalciferol group than that of the control group which is consistent with Hanne et al., **[34]** and was higher than what was reported in SUNNY trial by Yvonne et al., **[38]**. Most of patients in both groups were dyslipidemic; the mean LDL was 123.1mg/dl and 115mg/dl in the intervention and control groups respectively which was conflicting with the results of Yvonne et al., **[37]**, Al-Daghri et al., **[23]** and Nada et al., **[36]** whose participants had within normal serum lipids, this difference may be related to recruitment methods and sample size variation. Nevertheless, the present study unexpectedly corresponded with the previously mentioned studies **[23,36]** in the basal levels of fasting insulin, HOMA-IR and HOMA-β cell function.

The post intervention effects:

There were decreases in systolic, diastolic blood pressure and fasting blood sugar which were highly statistically significant, on the contrary fasting Insulin, HOMA-IR and HOMA β had been increased on the 12th week of the intervention compared to baseline values with statistically significant differences too. This is in agreement with recent systematic reviews that had suggested beneficial effects of Vitamin D supplementation in poorly controlled diabetics **[38,39]**. Antihypertensive activity of Vitamin D could be related to negative regulatory effect of Vitamin D on renin production **[40]**. Despite the lack of significance of HBAIC reduction between current both groups that agreed with Calvo et al., and other studies **[34,37,41]**; our study reported statistically significant differences between intervention and controls regarding fasting blood glucose (156.48±45.39 versus 193.27±37.48 respectively) that agreed with Hanne et al., and Yvonne et al., **[34,37]**.

In a non corresponding comparison between pre and post biochemical parameters of their participants; Nada et al., [36] found a significant reduction of HbA$_1$c (7.9±1.7 versus 7.4±1.2) and FPG (9.1±4.3 versus 7.9±2.4, p=0.034), this agreed with the study of Soric et al., [42] in which patients had a significantly greater reduction in HbA$_1$c on receiving Vitamin D for 12 weeks. This reduction was only significant when baseline HbA$_1$c was >9%.

The significant differences in β-cell activity within the intervention group and between both our study groups were associated with a non-statistically significant superiority in fasting serum insulin levels in the intervention group which disagreed with Calvo et al., [41] that had found a non significant reduction in fasting insulin and disagreed with Borissova et al., and Inomata et al., [43,44] which concluded that three to four weeks of Vitamin D supplementations were sufficient to improve insulin secretion. Similar improvements in HOMA-β and insulin secretion were noted in Arab participants after daily supplementations with the same dose of 2000IU of Cholecalciferol [10] despite lack of statistically significant difference in HbA$_1$c between groups after 18 months of Vitamin D intake.

Coinciding our realization of the improvement of HOMA-β and fasting insulin; Vaidya et al., [45] had found that HOMA-β had increased significantly by 35.9% and insulin had increased by 1.82 ∝U/mL compared with baseline. However HbA$_1$c levels didn't show such improvement [45].

Contradiction with our non statistically but clinically significant improvement in the serum lipids (LDL, HDL and TG) in the intervention group, Al-Daghri et al., [23] had reported a significant improvement in the lipid profile as evidenced by the decrease in LDL-cholesterol which was in agreement with Jafari et al., [46] that had found significantly improved only serum TC and LDL-C in patients with T2DM through Vitamin D intervention. Baseline Vitamin D3, its dosage, intervention duration, and the method of its intake influence the effect on lipid markers.

Several possible explanations exist for the lack of beneficial effect of Vitamin D on some metabolic outcomes related to glucose; the appropriate dose for non-skeletal benefits of Vitamin D still remains unclear, baseline Vitamin D status is a potential confounder on glycemic status, genetic factors related to Vitamin D metabolism might play a role and individual variability may also be partly explained by Vitamin D receptor polymorphisms. Moreover, the lack of significant association might have occurred due to our power calculations that may have been too optimistic, and it is possible that a larger sample size and longer follow-up would be needed to observe an effect. Diabetes related reasons likely responsible for not finding a beneficial effect with Vitamin D treatment include degree of hyperglycemia and duration of diabetes which were two limitations in our study.

Relation between different outcomes and patient variables:

We found a statistically significant difference noted between the adherent and non adherent groups in 12th week HBA$_1$C. Correspondingly; Gordon et al., [47] concluded that increasing levels of medication adherence were typically associated with greater 1-year HbA$_1$c reductions across all lines of oral antihyperglycemic therapy that was consistent with McClintock et al., [48].

In the current study there was statistically significant positive correlation between the post intervention HBA$_1$C and fasting blood sugar which is in agreement with Zhou J et al., [49] and Behary et al., [50], while both fasting Insulin and HOMA-β had statistically significant negative correlation with our participants' 12th week HBA$_1$C; corresponding to the findings of Al-Hakeim et al., and Bower et al., [51,52] and opposing Dahlqvist et al., [53] who reported Positive correlation between HBA$_1$C and fasting insulin. Unlike our negative results; other studies proved significant correlations between HBA$_1$C and other patients' variables; Hammad et al., [54] had documented that patients income was negatively correlated with HBA$_1$C, while Borah et al., [31] found that BMI showed positive correlation with HbA$_1$C. There was positive correlation between HbA$_1$c and serum lipids (TG, LDL) among the Afghani diabetics through Husain et al study [55].

The current results showed significant linear association between HbA$_1$C with FBS and fasting Insulin that agreed with Zhou et al., [49], whereas age, duration of illness and income significantly predicted A1C by Hammad and his colleagues [54]. Al-Hakeim et al., [51] had confirmed significant linear association between HBA$_1$C with HOMA-β.

Our study had detected a highly statistically significant difference between both groups in the post intervention glycemic improvement through HBA$_1$C reduction (79.3% versus 20.7% in the intervention and control groups respectively) which

was compatible with Anyanwu et al., **[56]** who found the proportion of participants with poor glycemic control (HBA_1c >6.5%) who converted to good control after Vitamin D supplementation was significantly higher in the treatment arm compared to control (33.3% in the intervention group versus –9.1% in the control). Our findings are in agreement with the results of many studies **[42,57-59]**. The strengths of the present study were its prospective, randomized design, stable treatment regimen throughout the study, objective assessment of endpoints and compliance to medication.

Conclusion:

Type 2 diabetic patients may benefit from adding a daily dose of 2000IU of oral Vitamin D_3 supplementation to their management plan through improving some cardiovascular parameters like systolic and diastolic blood pressure, fasting blood glucose and HOMA-β.

Recommendations:

Further studies for better understanding of diabetic patients' needs, knowledge and expectations about Vitamin D deficiency and its supplementation to improve their quality of life as well as a survey team to assess prevalence of Vitamin D deficiency among them are needed.

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تأثير العلاج التكميلى الكوليكالسيفيرول فى التحكم بمستوى سكر الدم، وظيفة خلايا بيتا بالبنكرياس ومقاومة الإنسولين لمرضى السكرى من النوع الثانى المترددين على العيادة الخارجية لطب الأسرة - بمستشفى جامعة قناة السويس - مدينة الإسماعيلية - مصر

المقدمة: مرض السكرى هو مرض مزمن معقد يؤثر سلبا على جودة حياة المرضى، يوجد ما يقرب من ٤٢٥ مليون بالغ (٢٠-٧٩ سنة) مصاب بمرض السكرى فى عام ٢٠١٧. وجد أن لفيتامين (د) دور فى تنشيط خلايا بيتا البنكرياسية لإفراز الأنسولين وذلك عن طريق حث المستقبلات عليها والخاصة بفيتامين (د). مما يوحى بدوره فى ضبط نسبة السكر بالدم.

الهدف: رفع مستوى العناية المقدمة إلى مرضى السكر وذلك عن طريق الوصول لأفضل مستوى للتحكم بنسبة السكر فى الدم.

الموضوعات والطرق: تم تنفيذ الدراسة فى العيادة الخارجية لطب الأسرة بمستشفى جامعة قناة السويس بمحافظة الإسماعيلية على ٦٠ مريضا (٣٠ لكل مجموعة) تم إختيارهم عشوائيا ثم تم توزيعهم عشوائيا على مجموعة فيتامين د التى تلقت يوميا ٢٠٠٠ وحدة دولية من الكوليكاليسفيرول عن طريق الفم بالإضافة إلى رعايتهم المعتادة ومجموعة المراقبة التى تلقت الرعاية المعتادة فقط لمدة ثلاثة أشهر. تم قياس معدل كتلة الجسم، ضغط الدم، قياس نسبة السكر الصائم، الهيموجلوبين السكرى، نسبة الأنسولين، مستوى الدهون فى الدم وتم حساب معدلات إفراز ومقاومة الأنسولين وتم تكرارها بعد ثلاثة أشهر.

النتائج: التدخل بإستخدام فيتامين د أظهر فروق ذات دلالة إحصائية بين مجموعة التدخل ومجموعة المراقبة فى ضغط الدم والسكر الصائم وإفراز الأنسولين ووظيفة خلايا بيتا. التحسينات فى مجموعة التدخل بإستخدام فيتامين د أظهرت فروق ذات دلالة إحصائية فى ضغط الدم والسكر الصائم ونسبة الأنسولين ومقاومة الأنسولين ووظيفة خلايا بيتا. يوجد علاقة ترابط إيجابية ذات دلالة إحصائية بين الهيموجلوبين السكرى وضغط الدم والسكر الصائم. بينما يوجد علاقة ترابط سلبية ذات دلالة إحصائية بين الهيموجلوبين السكرى ونسبة الأنسولين ووظيفة خلايا بيتا.

الخلاصة: إضافة جرعة يومية ٢٠٠٠ وحدة دولية من فيتامين د عن طريق الفم لمرضى السكرى من النوع الثانى قد يكون مفيدا من خلال تحسين ضغط الدم، معدل السكر الصائم ووظيفة خلايا بيتا.