Vitamin D assessment in elderly diabetic obese Egyptian patients in Zagazig University Hospitals

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Background
The relationship among vitamin D (VD) deficiency, metabolic syndrome, and type 2 diabetes mellitus (T2DM) is still doubtful, whether it is causal or jumbling.
Aim
To assess the link between VD deficiency and T2DM in old obese Egyptian patients and to evaluate the correlation between VD and some variables in T2DM.
Patients and methods
The study was conducted in Internal Medicine and Clinical Pathology Departments, Zagazig University Hospitals, from 02/2018 to 10/2018, involving 60 patients, aged more than or equal to 60 years, comprising 18 males and 42 females, who were divided into two groups: 30 patients with T2DM and 30 patients were proved to be without diabetes during the sample collection. Each group was subdivided according to their BMI into obese and nonobese.
Results
A highly significant difference was detected in VD level between nondiabetes and diabetes groups (23.85±12.1 vs. 16.2±7.9 ng/ml, respectively, P=0.001). VD deficiency was significantly higher in diabetes group (χ^2=10.38, P=0.006). Insignificant difference was detected regarding age (67.3±2.4 vs. 67.7±2.5, P=0.5) and BMI (30.7±5.1 vs. 32.7±5.3 kg/m^2, P=0.14) between nondiabetes and diabetes. There was a highly significant difference in VD level between obese nondiabetes and obese diabetes (27±17.5 vs. 13.3±8.2 ng/ml, respectively, P=0.001). A positive correlation was detected between VD level and fasting blood sugar (r=0.438, P=0.032), whereas a negative correlation between VD level and BMI in diabetes group only (r=–0.437, P=0.033) and in obese group only (r=–0.515, P=0.012).
Conclusion
Approximately 78.3% of patients had hypovitaminosis D (51.7% deficiency and 26.6% insufficiency). VD deficiency was significantly higher in diabetes group and in obese diabetics.

Keywords:
BMI, elderly, fasting blood sugar, type 2 diabetes mellitus, vitamin D

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Introduction

Vitamin D (VD) is an important nutrition element in metabolism and physiological effects compared with other vitamins [1]. VD deficiency is a global health problem. People who practice restraint from direct sun light are at higher danger of low VD levels [2]. Older people closely are at higher risk of low VD levels because of a decline in efficiency of VD synthesis and lower renal conversion to its active form [3]. Approximately 40 and 100% of the old US and European nonhospitalized people are either VD lacking or deficient [4].

Type 2 diabetes mellitus (T2DM) is one of the most widespread and devastating conditions in old individuals [5]. More than 40% of all diabetic patients are diagnosed late in older ages, and the number of old patients with diabetes is likely to extremely increase in the following years [6]. Numerous potential reversible/modifiable hazard factors for diabetes (overweight, inactive life, hypertension, and blood cholesterol levels) are basic factors for avoidance of diabetes [7].

VD deficiency is related with the metabolic disorder and T2DM in some epidemiological examinations. It is doubtful whether this relationship is causal or because of jumbling. The dynamic metabolite 1,25(OH)2D3 affects pancreatic β-cells and insulin discharge, and through different mechanisms, it might affect insulin sensitivity [8]. There is a potential link between obesity and VD deficiency among global populations [9]. Several studies reported low serum level of VD in obese individuals [10]. However, other studies

reported no link between hypovitaminosis D and T2DM, especially after adjusting for some potential confounders such as obesity [11].

The controversies regarding this issue were motivating for constructing this work. Therefore, the aim of this work is to assess the link between VD deficiency and T2DM in old obese Egyptian patients and to evaluate the correlation between VD and some variables in T2DM.

Patients and methods

Study design

The study was conducted in the Internal Medicine and Clinical Pathology Departments, Zagazig University Hospitals, in the period from February 2018 to October 2018. The study was a comparative cross-section design that involved two groups. The ages of the participants ranged from 60 to 75 years and comprised 18 (30%) males and 42 (70%) females, who were divided into two groups: diabetic group involved 30 elderly patients with T2DM as defined by American Diabetes Association, 2018 [12], and nondiabetic group involved 30 elderly patients who were proved not to have diabetes mellitus (DM) during sample collection. Each group was subdivided according to their BMI into obese (BMI≥25 kg/m^2) and nonobese patients (BMI<25 kg/m^2).

On the contrary, we excluded patients with chronic kidney diseases, liver diseases, dermatological disorders, malabsorption, history of/or current celiac disease or inflammatory bowel disease, patients who were taking prescriptions that may affect serum levels of 25-hydroxyvitamin D_3, calcium and VD therapy, smoking, and insulin injection.

Every patient had a case record structure in which the accompanying information was recorded: patient number, age, BMI, past medical and surgical history, and clinical examination, including general, abdominal, and pelvic examination. The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans. Ethical approval from the ethical committee for Medical Research in the Faculty of Medicine, Zagazig University, was obtained before the study, and a written informed consent was taken from all participants after explaining details and benefits before participating in the study.

There is not yet broad consensus on what constitutes VD deficiency. Different organizations have slightly different definitions, based on serum levels of 25-hydroxyvitamin D, or 25 (OH)D [13–16].

The Endocrine Society	The Institute of Medicine (Health and Medicine Division of the National Academies)	The Mayo Clinic	The American Association of Clinical Endocrinologists
Deficiency: ≤20 ng/ml	Deficiency: <12 ng/ml	Severe deficiency: <10 ng/ml	Deficiency: <30 ng/ml
Insufficiency: 21–29 ng/ml	Insufficiency: 12–20 ng/ml	Mild to moderatedeficiency: 10–24 ng/ml	
Optimal: ≥30 ng/ml	Optimal: ≥20 ng/ml	Optimal: 25–80 ng/ml	Optimal: 30–50 ng/ml

Method

All included patients were subjected to full history taking; complete clinical assessment; laboratory investigations [calcium, phosphorus, and intact parathormone (IPTH), which were determined in fasting blood sample]; anthropometric measurement including BMI; and measurement of serum vitamin D_3 (25-hydroxyvitamin D) level. Laboratory investigations were done to verify exclusion criteria, for example, liver function tests and renal function tests and to rule out DM [fasting blood sugar (FBS), 2-h postprandial blood sugar, and glycated hemoglobin (HbA1c)].

Measurement of 25 (OH) vitamin D and iPTH levels was carried out by electrochemiluminescence immunoassay (ECLIA) (Cobas e601; Roche diagostics, Mannheim, Germany), which is a compact automatic immunoassay system based on the enzyme-linked fluorescence assay principles, manufactured in France. The normal range of VD levels was 30–100 ng/ml.

Table D VD status by blood 25(OH) D concentration used in the current study

Status	Cut-off (ng/ml)
Deficiency	<20
Insufficiency	20–29
Sufficiency	≥30

VD status was judged according to the guidelines of Endocrine Society (Table D), which concluded that serum level of VD more than 30 ng/ml is required for optimal skeletal outcomes without any upper limits that would be concerning for safety which need more studies to withstand on level of toxicity [17].

Statistical analysis

Data were collected, coded, and entered using the Statistical Package for the Social Sciences. version

24. Quantitative data were expressed as mean and SD or median and range as appropriate. Comparison between two groups was done using independent sample *t* test or Mann–Whitney test. Qualitative data were expressed as frequency and percentage. χ^2 test and Fisher's exact test were used to examine the relation between qualitative variables. Spearman correlation coefficient (*r*) method was used to test correlation between numerical variables. *P* values less than 0.05 were considered as statistically significant, *P* value more than 0.05 insignificant, *P* value less than 0.05 significant, and *P* value less than 0.001 highly significant.

Results

A comparative cross-section study was conducted that involved 60 elderly patients with ages ranged from 60 to 75 years, comprising 18 (30%) males and 42 (70%) females, who were divided into two groups: 30 diabetic and 30 nondiabetic. Table 1 shows the demographic characteristics and laboratory investigations of two groups. There was a higher difference in diabetic group than nondiabetic group regarding patients' body weight (90.2±14.4 vs. 83.3 ±13.5, respectively, $P>0.05$) and BMI (32.7±5.3 vs. 30.7±5.1, respectively, $P>0.05$), though statistically insignificant. There was a highly significant difference between diabetic and nondiabetic group regarding FBS, 2h-pp, and HbA1c ($P<0.01$). Liver function tests, kidney function tests, serum calcium, serum phosphorus, and IPTH were within normal and statistically insignificant between both groups. Then, the included patients were subdivided into obese and nonobese and the frequency of distribution of obese patients in both groups is present in Table 2.

Table 2 shows that more than half of the included patients were obese [34/60 (56.7%)] compared with nonobese [26/60 (43.3%)]. The obese nondiabetic represented 16/30 (53.3%) and the obese diabetic represented 18/30 (60%). Thus, the number of obese diabetic patients was randomly more in this study but statistically insignificant ($\chi^2=0.27$, $P=0.32$).

Table 3 shows VD status in relation to obesity and revealed a highly significant difference of VD level on comparing nondiabetic obese versus diabetic obese (27.7±17.5 vs. 13.3±8.2, respectively; $P=0.001$), recording lower levels of VD in the diabetic obese patient (this means VD deficiency is linked with diabetes and obesity).

Table 1 Demographic characteristics and laboratory investigations of the two studied groups

Parameters	Non-DM group (*N*=30)	DM group (*N*=30)	*P* value
Age (years)	67.3±2.4	67.7±2.5	0.529
Males	9 (30)	9 (30)	1.00
Females	21 (70)	21 (70)	
Weight (kg)	83.3±13.5	90.2±14.4	0.061
Height (cm)	164±6	166±7	0.239
BMI (kg/m^2)	30.7±5.1	32.7±5.3	0.142*
Blood urea (mg/dl)	27.3±5.5	26.4±4.4	0.47
Serum Cr. (mg/dl)	0.9±0.2	0.93±0.21	0.57
ALT (IU/ml)	8.9±2.6	8.8±2.8	0.89
AST (IU/ml)	15.8±4.4	17.8±6.1	0.15
FBS (mg/dl)	83.5±9.0	97.6±11.0	<0.001***
2-h pp (mg/dl)	91.8±7.6	138.6±38.3	<0.0001***
HbA1c (%)	4.7±.6	8.4±1.1	<0.0001***
Serum calcium (mEq/dl)	9.1±0.6	9.2±0.6	0.52
Serum phosphorus (pg/dl)	3.7±0.5	3.8±0.5	0.44
IPTH (pg/ml)	31.2±14.7	32.3±14.4	0.76

Data are presented as *n* (%) and mean±SD. 2-h pp, 2-h postprandial; DM, diabetes mellitus; HbA1c, glycated hemoglobin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; Cr, serum creatinine; FBS, fasting blood sugar; IPTH, intact parathormone hormone. *Nonsignificant ($P>0.05$). ***Highly significant ($P<0.001$).

Table 2 Frequency of obesity in the two studied groups

	Non-DM group (*N*=30)	DM group (*N*=30)	χ^2	*P* value
Nonobese [26 (43.3%)]	14 (46.7)	12 (40)	0.27	0.32*
Obese [34 (56.7%)]	16 (53.3)	18 (60)		
60 (100%)	30 (100)	30 (100)		

Data are presented as *n* (%). χ^2, χ^2 test; DM, diabetes mellitus. *Nonsignificant ($P>0.05$).

Table 3 Vitamin D_3 status in relation to obesity in the two studied groups

	Obese [34 (56.7%)]		*P* value
	Non-DM obese (mean±SD)	DM obese (mean±SD)	
Vitamin D level (ng/ml)	27.7±17.5	13.3±8.2	0.001***

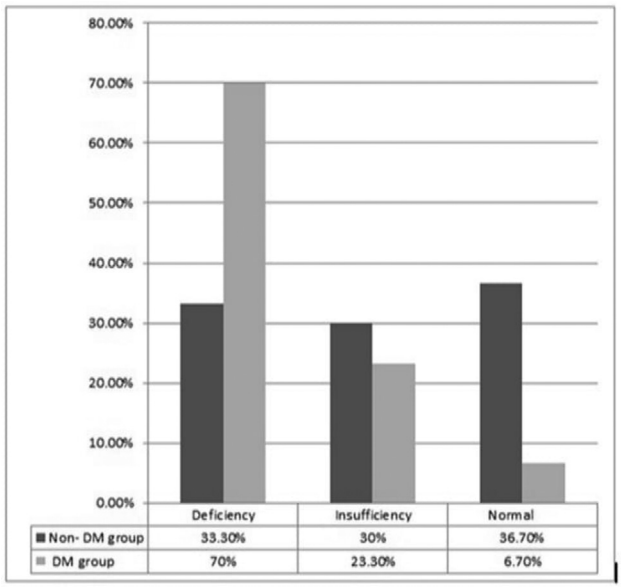
DM, diabetes mellitus. ***Highly significant (*P*=0.001).

Table 4 Vitamin D_3 Levels and status in two studied groups

Vitamin D_3 (ng/ml)	Non-DM group (*N*=30)	DM group (*N*=30)	χ^2	*P* value
Mean±SD	23.85±12.1 (9.5–39.2)	16.2±7.9 (8.2–30)		0.001***
Deficiency	10 (33.3)	21 (70)	10.38	0.006***
Insufficiency	9 (30)	7 (23.3)		
Normal	11 (36.7)	2 (6.7)		

Data are presented as *n* (%). χ^2, χ^2 test; DM, diabetes mellitus. ***Highly significant (*P* =0.001).

Figure 1



Vitamin D status in the two groups.

Table 4 reveals VD level in nondiabetic versus diabetic group, recording lower levels in diabetic group, with statistically highly significant difference (23.85±12.1 vs. 16.2±7.9, respectively, *P*=0.001).

Regarding VD status, Table 4 and Fig. 1 reveal that VD deficiency was significantly higher in diabetic patients than nondiabetic (70 vs. 33.3%, χ^2=10.38, *P*=0.006), whereas VD insufficiency is oppositely higher in nondiabetic than diabetic (30 vs.23.3%) (this means the insufficiency in VD is progressing from elderly nondiabetic to elderly diabetic till deficiency occurred).

Figure 1 shows that VD deficiency was significantly higher in diabetic patients than nondiabetic (70 vs. 33.3%), whereas VD insufficiency is higher in nondiabetic than diabetic (30 vs.23.3%).

Table 5 shows the number of patients according to VD levels in males and females in both diabetic and nondiabetic groups : there was an insignificant difference between males in both groups regarding VD level, whereas there was a significance difference between females in both groups regarding VD (*P*=0.015). Moreover, there was an insignificant difference between male and female regarding VD level within the same group.

Table 6 shows the correlation of VD with different variables in diabetic group only. A negative relation was detected between VD level and age but was statistically insignificant (*P*=0.06). VD_3 level was negatively correlated with BMI (*r*=-0.437, *P*=0.033) and was positively correlated with FBS (*r*=0.438, *P*=0.032), whereas there was a significant negative correlation between VD level and BMI in the diabetic group only (*r*=-0.437, *P*=0.033), as shown in Fig. 2, and a negative correlation between VD and BMI in obese group only (*r*=-0.515, *P*=0.012), as shown in Fig. 3.

Figure 2 shows a significant negative correlation between BMI and VD in diabetic group only (*r*=-0.437, *P*=0.033).

Figure 3 shows a significant negative correlation between BMI and VD in obese group only (*r*=-0.515, *P*=0.012).

Discussion

DM predominance generally is rising around the world [18] and is turning into a pandemic and endemic issue, with social and financial load [19]. Nevertheless, its prevalence and its comorbidities and mortality are higher in old people than in young [20]. As a rule, 20% of elderly individuals have DM, and a similar percentage has undiscovered DM [21]. Furthermore, 30% of elderly individuals have reduced glucose

Table 5 Shows the number of patients according to VD levels in males and females in both diabetic and nondiabetic groups

Vitamin D level	Nondiabetic group		Diabetic group	
	Male [9 (30%)]	Female [21 (70%)]	Male [9 (30%)]	Female [21 (70%)]
Vitamin D deficiency	4	6	6	15
Vitamin D insufficient	2	7	3	4
Normal	3	8	0	2
P value	0.68		0.49	
P value between males in both groups	0.16			
P value between females in both groups	0.015**			

**Significant ($P<0.05$).

Table 6 Correlation of vitamin D3 levels with the different variables in diabetic group (*N*=30)

Parameters	Correlation coefficient (*r*)	*P* value
Age	−0.378	0.069
BMI	−0.437	0.033**
Blood urea	−0.317	0.131
Serum creatinine	−0.268	0.206
ALT	0236	0.267
AST	−0.035	0870
FBS	0.438	0.032**
Random blood sugar	−0.051	0.812
HbA1c	−0.24	0.258
Serum calcium	0.108	0.615
Serum phosphorus	−0.073	0.734
IPTH	−0.317	0.131

ALT, alanine aminotransferase; AST, aspartate aminotransferase; FBS, fasting blood sugar; HbA1c, glycated hemoglobin; IPTH, intact parathormone. **Significant ($P<0.05$).

regulation, which means an expanded hazard for DM [22].

T2DM in old people is obviously because of numerous mechanisms among which one can refer to hereditary basis, prolonged lifetime leads to reduction in insulin release, and the alteration of some environmental elements accountable for increased central obesity. This obesity is responsible for insulin resistance, which is the primary driver of metabolic disorder and T2DM in adults and elderly persons [23].

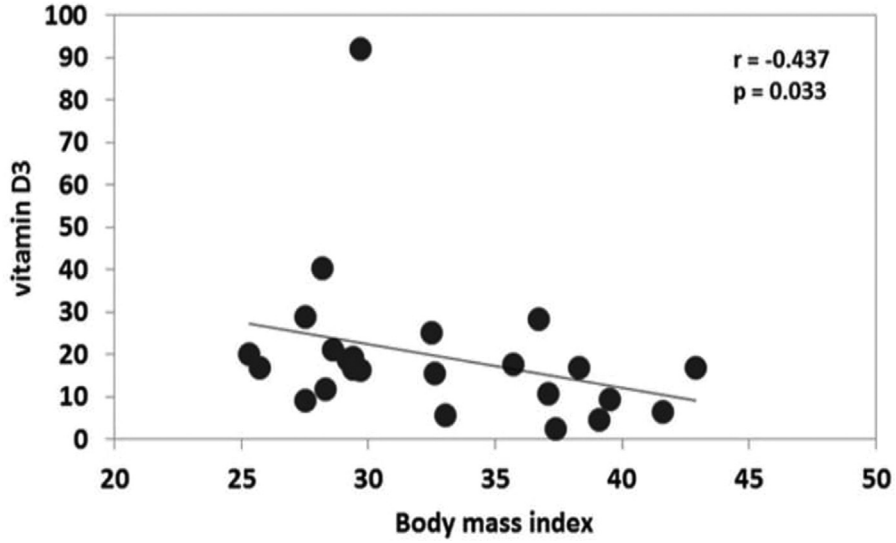
Several physiological effects of VD in both mineral metabolism and extra-skeletal effects have already been covered, with more controversies, reinforcing the need for further investigations. Among the list of widely debated areas in VD, the cut-off used for defining VD deficiency, the methods used for measuring VD status, and the right treatment for VD deficiency are included [24].

Our study involved 60 patients. Their ages ranged from 60 to 75 years. They were divided into two main groups: 30 diabetics and 30 nondiabetics. Each group comprised a subgroup with and without obesity. In our study, VD status was judged according to the Endocrine Society, that is, a blood level of 25(OH)D more than 30 ng/ml is interesting for ideal skeletal results without upper border that would be regarding safety. Blood levels les than 20 ng/ml was considered deficiency and the area between 20 and 29 ng/ml was judged as VD insufficiency. Therefore, levels below 30 ng/ml are collectively considered hypovitaminosis D. We found that VD deficiency in both groups was seen in 31 (51.7%) and VD insufficiency in 16 (26.6%). This means that ~78.3% of the studied sample had hypovitaminosis D.

Our results showed a negative relation between VD_3 levels and age but was statistically insignificant ($r=-0.37$, $P=0.06$). Many previous studies reported high prevalence rates of hypovitaminosis D among elderly people admitted to rehabilitation centers in Singapore, where 85.5% of patients had hypovitaminosis D, of whom 44.0% were VD deficient [25]. Shinchuk *et al.* [26] reported prevalence rates ranging from 68.4 to 94%. On the contrary, other studies reported much lower rates like Kiebzak *et al.* [27], who reported a rate of 11.0%, and Pellicane *et al.* [28], who reported a rate of 8.1%. However, definition of VD deficiency is usually the cause of this marked difference. The first two studies used levels of less than 20 ng/ml to define deficiency and from 21 to 29 ng/ml to define insufficiency like the current study.

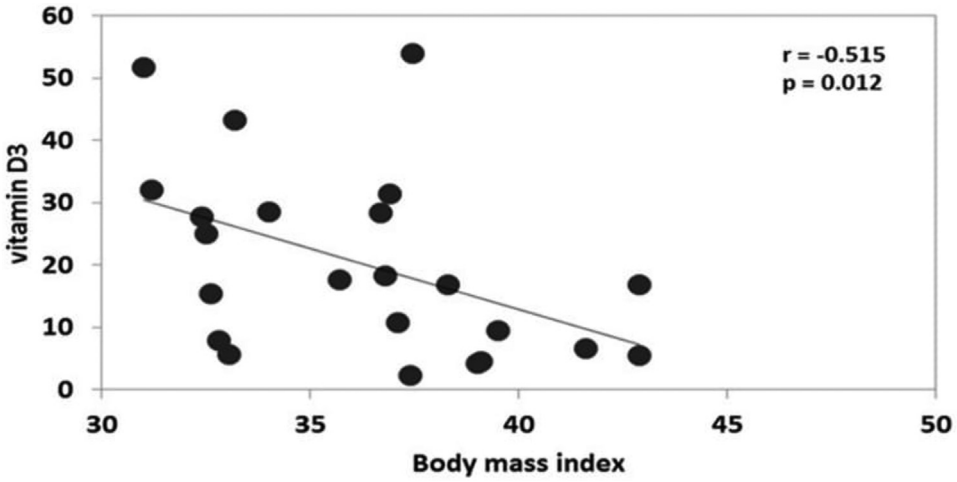
In elderly persons, VD lack may assume a role of T2DM, as certain authors might suspect VD insufficiency is a connection between osteoporosis, insulin resistance [29], overweight, DM [30], and Alzheimer disorder [31]. Our results showed that diabetes was associated with higher percentage of VD deficiency compared with nondiabetics (70% vs. 33.3%). In fact, serum levels of VD were significantly different between diabetic and nondiabetic groups ($P=0.001$), as shown in Table 4, and VD deficiency was significantly higher in diabetic patients ($P=0.006$), as shown in Table 4, and diabetic obese patients ($P=0.001$), as shown in Table 3.

Figure 2



Correlation between BMI and vitamin D in diabetic group only.

Figure 3



Correlation between BMI and vitamin D in obese group only.

Our result showed that there is no significant difference between the two groups regarding VD comparing male and female. However, comparing VD levels between females of two groups showed a significant difference (Table 5) (P=0.015), and insignificant difference between males in two studied groups regarding VD levels. This may be because of the large number of females (70%) compared with number of male (30%). This result agreed with the study done by Hidayat *et al*. [32], where most patients were females (66.7%) and obese (44.0%).

Various cross-sectional and case–control studies in older adults have detailed that no significant association had been detected between hypovitaminosis D and existence of diabetes, especially after controlling of some probable confusing matters like obesity [11]. Additionally, two randomized control trials have considered VD supplementation in older people at higher risk of diabetes. However, these studies distinguished that oral VD supplementation has no effect on glucose parameters in those people at higher risk of diabetes [33].

Another study in India found that the assessed insulin resistance by HOMAIR was significantly higher in patients with 25(OH)D) less than or equal to 20 ng/ml compared with those with 25(OH)D more than 20 ng/ml. The authors decided that VD affects glucose metabolism, and its insufficiency can lead to insulin resistance and diabetes [34]. The link between Low VD level and insulin resistance could be associated with the inflammatory process, as VD inadequacy is related

with expanded inflammatory markers. What is more, the varieties of VD-related genes may prompt impeded glycemic control and T2DM [35].

In animal studies utilizing a high-fat regimen to encourage overweight model of pre-T2DM, an increased supplementation of VD deferred advancement of T2DM and adiposity and was related with improved blood markers of diabetes and VD dietary and hormonal status [35]. What is more, supplementation with 500 mg of calcium and 700 IU of VD averted an ascent in fasting glucose and eased back the advancement of insulin resistance over a 3-year time frame in patients with impaired fasting glucose [36].

Nonetheless, others considered the deficiency in VD might be an outcome of overweight and chronic diseases, for example, general medical diseases [37]. Cândido and Bressan [37] found experimentally that VD suppresses fat buildup, keeps pancreatic islet cells, increases insulin production, diminishes insulin resistance, and decreases appetite. Accordingly, VD replacement should prevent and control general medical diseases. However, there are no enough logical approvals to help VD use in prevention and treatment of DM and obesity. Our results showed a highly significant difference detected between two groups regarding FBS, 2-h postprandial blood sugar, and HbA1c (P=0.001). On the contrary, no remarkable difference was identified between both groups in the levels of serum calcium (P=0.52), phosphorus (P=0.44), and IPTH (P=0.76), as shown in Table 1. There was no statistical significant correlation between VD with other laboratory parameters of kidney and liver function tests as well as level of IPTH, as shown in Table 6.

In the current study, we found an obvious association between VD deficiency and obesity in both studied groups. There was a significant difference between obese diabetic and obese nondiabetic in the VD_3 level (P=0.001), as shown in Table 3. Moreover, the obesity was more frequent in diabetic group compared with nondiabetic (60 vs. 53.3%). This was in concurrence with a study done by Linnebur *et al.* [38] which found that obese patients had significantly lower 25(OH)D concentrations than nonobese patients. Meanwhile, other studies discovered that a remarkable connection distinguished between poor VD status and diabetes vanished subsequent to controlling for BMI or other adiposity processes [39,40].

There was a significant negative correlation between VD level and BMI (r=–0.43, P=0.03), as shown in Table 6. If we consider the obese group only, a significant negative correlation is found between the two parameters (r=–0.515, P=0.012), as shown in

Fig. 3. Similarly, this negative correlation is found in the diabetic group separately (r=–0.437, P=0.03), as shown in Fig. 2.

On the contrary, Neo and Kong [41] did not demonstrate a correlation between high BMI and VD deficiency among elderly patients. However, Portela *et al.* [42] recommended reversal of VD deficiency to maintain a stability in bone turnover and to guarantee the endocrine and paracrine actions of VD for general health.

Renzaho *et al.* [43] stated that findings on the relationship of VD status and adiposity are conflicting and the reverse association was just remarkable in female patients. They suggested that ethnicity, sex, and age may assume a role in intervening the connection of low concentrations of serum 25(OH)D and overweight/weight status.

Several factors have been proposed to explain the link between VD deficiency and obesity, such as fat people may not get enough sun exposure because of restricted movement or clothing trends [44]; their body can hardly release VD as it is deposited in the body fat stores; and they have an expanded need of VD for stronger bones to support their overweight however, unfit to meet such requirements because of diminished bioavailability of 25(OH)D [45].

We believe that elderly people were more prone to lack of adequate sun exposure. Being obese adds to this problem. This may explain the negative correlation found in obese patients of the current study regarding VD level and BMI. In any case, all of these findings about VD in obese or diabetic population are intended to find a target for better management of these two morbidities especially in the vulnerable group of the elderly. However, there is not enough evidence to support VD supplementation to enhance the treatment of obesity or diabetes.

An important limitation in the study is that the effect of some confusing factors, for example, diet consumption, physical action, instructive level, and season of the year, were not included in the study. Moreover, the cross-sectional design of the study makes it progressively hard to inspect the

relationship of causality between obesity and VD inadequacy.

Conclusion

Our study showed ~78.3% of the studied sample experienced hypovitaminosis D. VD deficiency was recorded in 51.7% and VD insufficiency in 26.6% in total included patients. Hypovitaminosis D in elderly was connected with DM because of imperfections in pancreatic β cell work and/or insulin production, and associated with obesity, which may play a major role in developing DM owing to increase insulin resistance. In general, assessment of VD status should be routinely done to detect those with vitamin deficiency for adequate supplementation. Elderly patients should be encouraged for adequate sun exposure to enhance VD levels. Empirical VD supplementation is recommended for elderly diabetic patients if they have vitamin deficiency. Further follow-up study on a larger number of patients is advised to assess the effect of VD on patients with DM and obesity and also the effect of weight reduction on VD level. Moreover, further follow-up study on the effect of winter versus summer on the level of VD is advised to be done.

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Conflicts of interest

There are no conflicts of interest.

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