Original Communication

Serum vitamin D status does not correlate with the severity of obstructive sleep apnea in male adults: A controlled study design with minimized factors influencing serum vitamin D levels

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Abstract: Observational studies performed in homogeneous groups to objectively investigate the cause and effect relationship between vitamin D deficiency and sleep disorders are scarce. In this study, it was aimed to analyze the relationship between the severity of OSAS and vitamin-D levels among the participants whose features affecting serum vit-D levels were minimised. Serum 25-OH vitamin-D levels in 121 OSAS Male patients diagnosed by polysomnography without any systemic disease or vitamin-D supplement that may effect the vitamin-D metabolism were measured. The study was conducted in winter (latitude: 41°). Anthropometric measures and biochemical tests were also performed. The distribution of vitamin-D levels was determined as severe deficiency, deficiency, insufficiency and sufficiency. Apnea-hypopne index (AHI) < 5 was considered as a control group. Patients were categorized into four groups according to AHI as control, mild, moderate and severe. The groups were similar in terms of age, BMI, lipid profile, serum calcium, anthropometric measures and smoking. There was no significant difference in the distribution of vitamin-D levels between the patient and control groups and also within OSAS subgroups ($p = 0.57$, $p = 0.86$, respectively). Odds ratio to have OSAS in patients with vitamin-D deficiency was found as 0.745 (95 %CI: 0.33–1.7). Multinominal regression analysis showed no significant relationship between the OSAS severity and the extent of vitamin-D status. Correlation analysis showed no significant relationship between vitamin-D and AHI ($r = 0.017$, $p = 0.877$). Vitamin-D status does not alter the severity of OSAS. Vitamin-D deficiency might be the result of lifestyle changes due to OSAS rather than a cause.

Keywords: Anthropometric measurements, obstructive sleep apnea syndrome, polysomnography, sleep-disordered breathing, snoring, vitamin D, vitamin D deficiency, waist circumference

Introduction

Obstructive Sleep Apnea Syndrome (OSAS) represents a major public health problem and is associated with significant morbidity [1]. HypnoLaus prevelance study showed that the prevalence of moderate-to-severe sleep-disordered breathing was 23.4 % in women and 49.7 % in men over 40 years of age [2]. The median apnea-hypopnea index (AHI) that indicates the severity of sleep apnea was found to be mild in general population with 6.9 and 14.9 in women and men, respectively.

OSAS is a common syndrome characterized by intermittant hypoxia and episodic and repetative upper airway collapses [3]. Oxygen desaturation, hypercapnia and sleep fregmantation caused by OSAS leads to metabolic, neurocognitive and cardiovascular morbidities [3].

The potential role and wide range of use of the vitamin D beyond bone health has received increasing attention

[4]. It is well known that vitamin D metabolism effects the immunregulation, cardiovascular disorders, tonsiller hypertrophy, allergic rhinitis, pain, myopathy and depression [5]. Those conditions may cause sleep disruption and increase the risk for obstructive sleep apnea. Vitamin D as an endogen steroid prohormone has been suggested to have receptors in places that were considered to have role in the initiation and maintenance of sleep such as anterior and posterior hypothalamus, substantia nigra, midbrain central gray, raphe nuclei, and the nucleus reticularis pontis oralis and caudalis [6]. A relationship between the vitamin D deficiency and excessive daytime sleepiness and hypersomnia, which are major components of sleep disturbances, has been proposed previously [7, 8].

More evidence is needed to clear the association between vitamin D deficiency and OSAS whether it is due to a pathophysiologic pathway or due to confounders [9]. The studies investigating the association between vitamin D metabolism and sleep disorders were limited in the literature [10–14]. However, the majority of those studies hold potential selection bias in their patient cohorts. Residual confounding factors caused by inadequate adjustment for the social and environmental reasons have been proposed as the reason for the disparity between the observational and trial findings conducted in vitamin research [15]. The observational nature of confounded studies precludes assessing the relationship between the cause and effect [16]. Therefore, there was a need to design a study that included participants whose demographic and clinical characteristics that could affect vitamin D levels were minimized.

It was hypothesized in this study that vitamin D status may be lower in patients with OSAS when compared to control group. Secondly, the severity of OSAS may be inversely correlated with vitamin D levels. To test these hypotheses, the association between serum 25-OH vitamin D level and AHI was assessed in a well-designed cohort.

Materials and Methods

This study was conducted in the Sleep Disorders Diagnosis and Treatment Center of a tertiary center specialized in neurology and psychiatry (latitude 41°). The study protocol was approved by the Institutional Ethics Committee (No: 16/0026). Written informed consent was obtained from all participants prior to the study.

Population

Male patients aged between 18 and 50 years diagnosed as Obstructive Sleep Apnea Syndrome (OSAS) with polysomnography (PSG) (*Neuron-Spectrum-5, Neurosoft, Russia*) between October 2016 and February 2017 who fulfilled the inclusion criteria were recruited for the study.

Patients who reported to have multivitamin, calcium, vitamin D supplement or medical treatment that modulate vitamin D serum levels (*e.g.* steroids, antiepileptycs *etc.*) for the last one year and those with disorders known to influence vitamin D metabolism/absorption were excluded fom the study. Patients with obesity (BMI $\geq$ 30 kg/m^2) and any systemic diseases such as hypertension, diabetes mellitus, hyperlipidemia, renal disorders, thyroid and parathyroid disorders that are known to influence vitamin D metabolism were excluded. Patients were selected among Male gender and the study was undertaken in winter season to vigorously minimize the factors that could alter the 25-OH vitamin D serum levels.

Patients were categorized according to the Apnea-Hypopnea Index (AHI) into four groups as control (AHI < 5), mild (AHI 5–14.9), moderate (AHI 15–29.9) and severe (AHI $\geq$ 30) [17]. Control group consisted of whom PSG revealed no OSAS but only simple snoring (AHI < 5).

Sleep Measurements

The diagnosis of OSAS was established by full polysomnography *(Neuron-Spectrum-5, Neurosoft, Russia*). This polysomnogram consisted of overnight recording of 6-channel electroencephalography (EEG) (C3A2 or C4A1), left and right electrooculography (EOG), submental electromyography (EMG), left and right tibialis anterior EMG, electrocardiography (ECG), airflow measurement, respiratory effort and pulse oximetry. Measurements included the recording of oronasal flow, thoracoabdominal movements, body position, oxygen saturation with pulse oximeter and snoring measurements.

Apnea was defined as the absence of airflow for more than 10 seconds. Hypopnea was defined as any airflow reduction that lasted more than 10 seconds and resulted in arousal or oxygen desaturation. A decrease in SaO2 greater than 3 % was considered to represent an oxygen desaturation (AASM 2014). The apnea-hypopnea index (AHI) was defined as the sum of the number of apneas plus hypopneas per hour of sleep. Excessive daytime sleepiness was quantified subjectively by the validated version of the Epworth sleepiness scale [18].

Anthropometric measurements

Body mass index (BMI) was calculated from measured height and weight. Neck circumference was measured at the level of superior border of the cricothyroid membrane with the patient in the upright position by the same clinician

(O.Y.Y.). The minimal waist circumference was measured at the site of midpoint between the iliac crest and the lowest rib over the umbilicus and paralelly to the ground. Body fat (BF), visceral fat (VF) and body muscle (BM) proportions were measured with bioelectrical impedance analysis (BIA) using the OMRON Body Composition Monitor BF511 (*Omron Healthcare Co., Kyoto, Japan*). All the patients were explained in detail about the instuctions prior to the BIA. Instructions included no heavy physical activity and alcohol use 24–48 hours prior to the BIA and no having meal in the last 2 hours.

Vitamin D analysis and biochemical tests

After fasting 12 hours, venous blood samples were collected, centrifuged, aliquoted, and frozen to −80 °C in a dark setting between 8 and 10 a.m. Circulating levels of total 25(OH)D, calcium, phosphorus and magnesium level were analysed using competitive chemiluminescence immunoassays by *Architect Abbott i2000, IL, USA* device.

Vitamin D levels was stratified into 4 categories: severe vitamin D deficiency (< 10 ng/ml), vitamin D deficiency (10–19.9 ng/ml), vitamin D insufficiency (20–29.9 ng/ml) and sufficient vitamin D levels ($\geq$ 30 ng/ml) [19]. Patients with serum vitamin D levels at a toxicity level (> over 150 ng/ml) were excluded.

Statistical analysis

All analyses were performed using NCSS (Number Cruncher Statistical System) 2007 (*Kaysville, Utah, USA*) program. Besides the desciptive statistical methods, student's t test and Mann Whitney U test were used for normally and not normally distributed variables, respectively. One-way ANOVA test was used for normally distributed three or more groups and Tukey HSD test to detect differences. Kruskal Wallis test was used for not normally distributed three or more groupsand Mann Whitney U test to detect differences. Pearson Chi-Square, Fisher's Exact Test, Continuity Correction and multinominal regression tests were used for comparisons of qualitative data. Spearman test was used as the correlation test. All p values reported are 2-tailed with statistical significance set at < 0.05.

Results

Patients were recruited until the participant number of minimum 30 was reached for each group. A total of 121 patients were included. All participants agreed to participate without any lost. Table 1 shows all demographic features, Epworth Scale scores, neck and waist

Table 1. Demographic and physiologic characteristics of subjects by OSAS severity.

		Control (n = 31)	Mild (n = 30)	Moderate (n = 30)	Severe (n = 30)	P
Age (year)	Mean ± SD	34.8 ± 7.17	38.9 ± 6.76	39.0 ± 8.08	38.5 ± 8.17	[a]0.100
ESS	Mean ± SD	5.97 ± 4.36	5.70 ± 4.28	6.83 ± 4.73	6.83 ± 5.53	[b]0.812
Neck c. (cm)	Mean ± SD	39.7 ± 2.01	41.2 ± 1.78	40.5 ± 1.91	41.33 ± 1.71	[a]0.002**
Waist c. (cm)	Mean ± SD	97.9 ± 7.91	101 ± 7.66	99.9 ± 7.44	104 ± 6.04	[a]0.018*
BMI (kg/m^2)	Mean ± SD	26.8 ± 2.34	26.9 ± 2.04	27.1 ± 2.5	27.9 ± 1.91	[a]0.193
Body fat	Mean ± SD	24.1 ± 4.05	25.5 ± 3.89	24.5 ± 4.07	26.1 ± 4.14	[a]0.220
Body Muscle mass	Mean ± SD	36.6 ± 2.69	35.3 ± 2.45	35.8 ± 2.13	35.2 ± 2.46	[a]0.094
Visceral fat	Mean ± SD	10.2 ± 3.93	10.5 ± 2.5	10.4 ± 2.71	11 ± 3.19	[a]0.772
Calcium	Mean ± SD	9.60 ± 0.29	9.56 ± 0.29	9.62 ± 0.35	9.6 ± 0.24	[a]0.911
Phosphorus	Mean ± SD	3.63 ± 0.48	3.53 ± 0.41	3.47 ± 0.58	3.6 ± 0.59	[a]0.619
Magnesium	Mean ± SD	1.99 ± 0.15	1.98 ± 0.17	1.92 ± 0.13	2.01 ± 0.14	[a]0.107
Basal O_2	Mean ± SD	94.7 ± 4.27	94.6 ± 2.03	95.7 ± 1.30	95.2 ± 1.83	[a]0.369
SaO_2 < 90 time (sec)	Mean ± SD	283 ± 1131	2130 ± 3204	752 ± 1531	3217 ± 3609	[b]0.001**
Min O_2	Mean ± SD	89.93 ± 3.34	80.1 ± 15.4	83.23 ± 4.18	77.5 ± 9.91	[a]0.001**
Desaturation Index	Mean ± SD	2.69 ± 2.50	14.0 ± 7.57	20.7 ± 11.3	37.9 ± 19.7	[b]0.001**
Mean nocturnal heart rate	Mean ± SD	69.0 ± 20.8	66.5 ± 5.12	67.4 ± 7.55	70.4 ± 12.0	[a]0.727
Snoring Index	Mean ± SD	22.1 ± 61.6	66.1 ± 112	21.5 ± 65.0	99.4 ± 181	[b]0.135
		n (%)	n (%)	n (%)	n (%)	
Smoking, n (%)	Yes	6 (19.4)	15 (50.0)	13 (43.3)	10 (33.3)	[c]0.071

[a]One-WayAnova Test, [b]Kruskall Wallis Test, [c]PearsonChi-SquareTests, *p < 0,05, **p < 0.01.
ESS: Epworth sleepiness scale; BMI: Body-mass index; SaO2: Saturation; Min O2: Minimum oxygen.

Table 2. Vitamin D levels among patients.

Vitamin D (ng/ml)	Control (n = 31)	OSAS (n = 90)		P
Mean ± SD	19.0 ± 5.93	18.8 ± 8.83		0.567
Min-Max (Median)	5.50–30.6 (19.7)	2.40–44.1 (17.3)		
	Mild (n = 30)	Moderate (n = 30)	Severe (n = 30)	P
Mean ± SD	18.1 ± 7.96	19.7 ± 9.61	18.6 ± 9.07	0.862
Min-Max (Median)	2.40–34.9 (17.6)	6.10–36.3 (17.3)	8–44.1 (17.3)	

Kruskall Wallis Test.
SD: Standard deviation; OSAS: Obstructive sleep apnea syndrome.

circumferences, anthropometric measurements and biochemical test results. The age, BMI, anthropometric measurements (Body fat, visceral fat and muscle mass), smoking status and serum levels of calcium, phosphorus and magnesium were similar between patients with mild, moderate and severe OSAS and control groups.

The blood lipid profile was similar between the control and OSAS groups. HDL, total cholesterol and triglyceride levels were 44.43 ± 11.87 *versus* 39.9 ± 9.34 mg/dl, 194.13 ± 37.29 *versus* 208.41 ± 41.03 mg/dl and 191.16 ± 145.18 *versus* 240.85 ± 162.49 mg/dl, respectively ($p < 0.05$).

Neck and waist circumferences showed statistical significance between the groups with a p value of 0.002 and 0.018, respectively. Tukey HSD test to detect the differences in within groups exhibited neck enlargement in mild and severe OSAS groups when compared to controls (p = 0.009; p = 0.003; p < 0.01). Similarly, patients with severe OSAS were with larger waist circumference when compared to controls (p = 0.010; p < 0.05).

Ages of the patients with OSAS and without OSAS showed statistically significant difference with mean values of 34.84 ± 7.17 (Range: 18–48, median: 25) and 38.80 ± 7.61 (Range:19–49, median: 40), respectively. This significance disappeared in subgroup comparisons of patients between normal, mild, moderate and severe OSAS ($p = 0.1$). Similarly, muscle mass proportions differed between patients with and without OSAS (36.6 ± 2.7 and 35.4 ± 2.3, respectively; $p = 0.022$), however, this significance was absent between subgroup comparisons of OSAS severity ($p = 0.094$).

Respiratory functions including duration of oxygen saturation below 90, minimum oxygen and desaturation index were significantly different between groups ($p = 0.001$). Within groups comparisons showed that duration of $SaO_2 < 90$, Min O_2 levels and desaturation index were different in favour of control group when compared to patients with mild, moderate and severe OSAS ($p \leq 0.001$).

Serum 25-OH vitamin D levels did not reach a statistically significance between patients with and without OSAS ($p = 0.567$) and between patients between mild, moderate and severe OSAS ($p = 0.862$) as shown in Table 2.

The extent of vitamin D deficiency did not show a significant risk to have OSAS (Table 3). Nominal regression also showed no significant link between the OSAS severity and the degree of vitamin D deficiency (Table 4).

Table 3. Vitamin D status between patients with and without OSAS.

Vitamin D status (ng/ml)	AHI < 5	AHI ≥ 5	P	OR	95% CI
	n (%)	n (%)			
Severe vitamin D deficiency	1 (3.2)	16 (17.8)	0.87	6.486	0.82–51.1
Vitamin D deficiency	15 (48.4)	37 (41.1)	0.62	0.745	0.33–1.69
Vitamin D insufficiency	14 (45.2)	24 (26.7)	0.09	0.442	0.19–1.03
Sufficient vitamin D level	1 (3.2)	13 (14.4)	0.17	5.06	0.63–40.4

*Fisher's Exact Test, Continuity Correction.
AHI: Apnea-Hypopnea Index; OR: Odds ratio; CI: Confidence interval.

Correlation analysis showed no statistically significant relationship between vitamin D and AHI (r = 0.017, p = 0.877, p < 0.05) as shown in Table 5. However, a statistically significant but very weak correlation in the negative direction between the snoring index and vitamin D was found ($r = -0.211$, $p = 0.024$).

Discussion

This study showed no association between serum vitamin D levels and the severity of OSAS in a controlled design. The degree of vitamin D deficiency was found to be similar between the patients with and without OSAS. Anthropometric measures of the patients did not significantly differ among patients with mild, moderate or severe OSAS and the control group.

Unknown confounders are known as the main source of bias in observational studies. Studies adjusted for factors which are previously shown to alter vitamin D levels are considered to be with the lowest risk of bias [20]. Those factors include age, gender, use of hormonal therapy, demographics, anthropometry, chronic disease, body mass index, geographical location, season and physical activity [16, 20–22]. A study investigating modifiable predictors of vitamin D in 2621 healthy individuals found that being female, being obese (BMI ≥ 30 kg/m2), not being physically active, having low dietary vitamin D intake and supplement

Table 4. Multinominal regression analysis for OSAS severity by vitamin D status.

Severity of OSAS*		B	SE	Wald	df	Sig.	Exp(B)	95 % CI for Exp (B)	
								Lower	Upper
No OSAS	Intercept	−1.39	1.12	1.54	1.00	0.22			
	[Severe Deficiency]	−0.22	1.57	0.02	1.00	0.89	0.80	0.04	17.2
	[Deficiency]	1.39	1.18	1.39	1.00	0.24	4.00	0.40	40.1
	[Insufficiency]	2.23	1.22	3.35	1.00	0.07	9.33	0.85	102
	[Normal]	0.22	1.57	0.02	1.00	0.89	1.25	0.06	26.9
Mild OSAS	Intercept	−0.29	0.76	0.14	1.00	0.71			
	[Severe Deficiency]	0.29	0.99	0.08	1.00	0.77	1.33	0.19	9.31
	[Deficiency]	0.07	0.86	0.01	1.00	0.94	1.07	0.20	5.71
	[Insufficiency]	0.80	0.92	0.75	1.00	0.39	2.22	0.37	13.5
	[Normal]	−0.29	0.99	0.08	1.00	0.77	0.75	0.11	5.24
Moderate OSAS	Intercept	0.41	0.65	0.40	1.00	0.53			
	[Severe Deficiency]	−0.22	0.89	0.06	1.00	0.80	0.80	0.14	4.53
	[Deficiency]	−0.81	0.76	1.13	1.00	0.29	0.44	0.10	1.99
	[Insufficiency]	−0.12	0.84	0.02	1.00	0.89	0.89	0.17	4.63
	[Normal]	0.22	0.89	0.06	1.00	0.80	1.25	0.22	7.08

*The reference category is: Severe OSAS. OSAS: Obstructive sleep apnea syndrome; CI: Confidence interval.
B, SE, Wald, df, Sig and Exp refer to the coefficients of the regression analysis.

Table 5. Correlation between vitamin D levels and anthropometric and polysomnographic indices.

	Vitamin D	
	Spearman rho (r)	p
BMI (kg/m^2)	0.03	0.79
Body fat	0.17	0.1
Neck circumference	0.01	0.9
Waist circumference	−0.07	0.5
Visceral fat	0.03	0.78
AHI	0.02	0.88
Epworth Scale Scores	0.11	0.29
Desaturation Index	0.18	0.09
Mean nocturnal hearth rate	0.02	0.89
Snoring index	−0.24	0.03
Basal O_2	0.02	0.84
Duration of $SaO_2 < 90$	0.01	0.96

*$p < 0.05$.
BMI: Body-mass index; AHI: Apnea-Hypopnea Index; Basal O^2: Basal oxygen; SaO2: Oxygen saturation. Spearmen rho refers to the correlation coefficient.

use were associated with low vitamin D status [22]. The BMI is inversely related to the serum vitamin D [16]. Older age reduces the capacity of ultraviolet B-induced cutaneous synthesis of vitamin D and is known to regulate the outcome of the supplementation [23]. A keystone meta-analysis investigating the best therapeutic dose of vitamin D has used 50 years of age as the threshold consistently with the current study [24]. Besides, total serum, ionized and urine calcium, PTH and dihydroxy-vitamin D are known to be varied significantly over the menstrual cycle [25]. Hence, studies including age- and sex-matched controls without any systematic disease nor vitamin D supplementation came to the fore with an effort to minimize the confounding factors [26]. Assessing the vitamin D levels among a confined group was the main strength of the current study. Mainly, this design holds the substantial advantage of limiting the confounding factors that are the major cause of conflicting results about the vitamin D in the literature.

It was previously reported that vitamin D deficiency was related to the excessive daytime sleepiness [27]. Gominal and Stumpf observed an improvement in neurologic symptoms and sleep after vitamin D supplementation and therefore hypothesised that a narrow range of vitamin D serum levels was needed to produce a proper sleep [6].

However, it is still not clear that the clinical conditions which were previously linked with vitamin D deficiency are contributed by low vitamin D concentrations or the life style changes that may lead to decreased concentrations. It was argued that the disorders itself such as being obese, having diabetes, depression or dementia may actually cause less physical activity and sun exposure and therefore result in with vitamin D deficiency [4]. Supportingly, Mesci *et al* found that vitamin D deficiency was associated with fatigue and daytime sleepiness but also was related with a reduced level of physical activity [28].

In this study, the groups were homogeneously constituted in winter season by younger than 50 years healthy male adults whose BMI was below 30 kg/m^2. Patients who did not have any systemic disease and any medication that might have modulated vitamin D serum levels were included only. Tufik *et al* diagnosed moderate to severe obstructive sleep apnea in 62.6 % of all obese male patients in their epidemiological study [29]. It was an interesting

observation of our study that the lipid profile and anthropometric measures were found to be similar among the groups when the variables such as obesity, hypertension, diabetes, thyroid and parathyroid diseases were controlled prior to the study.

Salepci *et al* evaluated the correlation between vitamin D levels and AHI and BMI. Vitamin D levels were found as similar in patients with and without OSAS and also among patients with mild, moderate and severe OSAS [14]. Their results were consistent with ours although the size of their control group was relatively small (10.5 %). Massa *et al* compared the vitamin D levels with nightly total sleep time, sleep efficiency, and wake time after sleep onset by wrist actigraphy in patients older than 68 years [13]. Low serum vitamin D levels were found to be associated with poorer sleep including short sleep duration and lower sleep efficiency in the older Male cohort. In that study, statistically significance was lost for wake time after sleep onset in the multivariable model adjusted for age, clinic, season, BMI, SF-12, congestive heart failure, marital status. In addition, the majority of patients with sufficient vitamin D levels (65.1 %–76.5 %) were reported as on vitamin D supplementation. The current study found no significant relation with vitamin D with a design of initially controlled variables of age, supplementation and BMI. Barcelo *et al* also showed no causative nature of the association between the grade of vitamin D deficiency and OSAS severity. They showed high prevalence of low vitamin D levels among patients with OSAS, however, no statistically difference was found between the AHI and serum vitamin D levels consistently with the results of the current study [10]. Furthermore, vitamin D deficiency was found to be associated with diabetes and metabolic syndrome.

In contrary to our results, Kerley *et al* found a significant, independent and inverse relationship between serum vitamin D levels with AHI and nocturnal hearth rate [12). Vitamin D supplementation and systemic diseases were also excluded in their study. The possible explanation of this inconsistency in results of their and our studies might be the Nocturnal hearth rate. Occult cardiovascular disorders related to nocturnal hearth rate might be the underlying cause of the conflicting results. In another study investigated the association between OSAS and serum Bisphenol A, 25-OH-D, and parathormon levels [11]. They found that vitamin D levels were significantly lower in patients with moderate and severe OSAS when compared to the healthy subjects. They observed no significant difference in vitamin D levels among patients with OSAS. However, age, gender and BMI indices were statistically unmatched between the groups, that might explain the controversy with the results of the current study.

Individuals who use vitamin supplements may have other health habits that correlate with better sleep [13]. More controlled studies with better homogenized cohorts may overcome the controversy about whether vitamin D leads to OSAS or vitamin D causes from the condition [4]. It was suggested that individuals with depression or any chronic disorders are often with vitamin D deficiency due to the lifestyle changes as a consequence of the mental and physical limitations by their clinical conditions [30]. Thus, vitamin D deficiency should not be initially seen as the cause of the disorder.

This study has some limitations. We included a relatively mall sample size and did not assess physical activity. Secondly, patient numbers in the groups of "severe deficiency" and "sufficient" level of vitamin D were very small. This may have affected the multinominal regression analysis and the sub-analysis for the vitamin D status between patients with and without OSAS. Finally, control group included patients who have admitted to the clinic with any symptoms. However, we do not think that it is clinically relevant because all patients including control group underwent PSG and OSAS or any other sleep disorders were excluded. Further researches should focus on genetical basis of polymorphisms in the vitamin D to prove the absence of causality effect. Longitudinal studies with multiple repetative measurements of vitamin D are needed to assess the effect of vitamin D use.

Conclusion

Vitamin D status did not alter the severity of OSAS in healthy male adults aged between 18–50. Vitamin D deficiency might be the result of lifestyle changes due to OSAS rather than a cause of sleep disturbances.

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Authors Contribution
Ozge Yagcioglu Yassa: Study conception and design, Acquisition of data, Analysis and interpretation of data, Writing manuscript, Critical revision.
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Authorship Credits
The authors declare that are qualified for authorship and have participated sufficiently in the work to take public responsibility for appropriate portions of the content. The authors declare that; the authors made a substantial contribution to the concept or design of the work; or acquisition, analysis or interpretation of data, drafted article or revised it critically for important intellectual content, approved the version to be published and each author have participated sufficiently in the work and take public responsibility for appropriate portions of the content.

Conflict of Interest
No conflicts of interest exist.

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