Al-Azhar Med. J. .(Medicine) *Vol. 52 (1), January, 2023, 2023, 317-330*
DOI: 10.21608/amj.2023.276357
https://amj.journals.ekb.eg/article_276357.html

THE ASSOCIATION OF VITAMIN D STATUS AND THE COMPONENTS OF THE METABOLIC SYNDROME IN PREMENOPAUSAL WOMEN

By

[1]Hanan Balkhyoor, [2]Hanan A. Al-Kadi, [2,3]Gehan I. El-Salamony, [4]Mohammed-Salleh M. Ardawi

Affiliations:

[1]Department of Allied Health Diploma, King Abdullah Medical City, Makkah, Saudi Arabia

[2]Department of Physiology, Faculty of Medicine, King Abdulaziz University, Jeddah, Saudi Arabia

[3]Department of Physiology, Faculty of Medicine, Ain Shams University, Cairo, Egypt

[4]Department of Pathological Sciences, Fakeeh College for Medical Sciences, Jeddah, Saudi Arabia

Corresponding author: Dr. Gehan El-Salamony

Associate professor, Physiology Department, Faculty of Medicine, Ain Shams University, Cairo, Egypt

E-mail: gsalamony2009@hotmail.com

ABSTRACT

Background: Vitamin D deficiency, a widespread problem that is increasing worldwide, has been implicated in a diversity of diseases including metabolic syndrome. The metabolic syndrome is a cluster of risk factors that collectively increases predisposition to major chronic diseases, including diabetes mellitus and cardiovascular diseases.

Objectives: The aim of the present study was to determine the vitamin D status in a group of healthy premenopausal Saudi women, and to assess its correlation with the components of the metabolic syndrome.

Subjects and Methods: A cross-sectional study of 205 premenopausal Saudi women, aged 20 to 45 years, was carried out in the Center of Excellence for Osteoporosis Research at King Abdulaziz University, Jeddah, Saudi Arabia. Blood pressure and anthropometrics were assessed and the body mass index was calculated. Fasting blood samples were collected for measurements of 25-hydroxyvitamin D, fasting blood glucose, triglycerides, and high density lipoprotein-cholesterol. A modified "National Cholesterol Education Program-Adult Treatment Panel criteria" definition was used for the diagnosis of metabolic syndrome.

Results: Vitamin D deficiency was extensive, with 92.2% of women having 25-hydroxyvitamin D levels <50 nmol/L. Metabolic syndrome was prevalent in 7.8% of cases. An inverse association was demonstrated between vitamin D levels and all components of metabolic syndrome except high density lipoprotein-cholesterol, which was positively associated with vitamin D levels, although these associations were statistically insignificant. Severely vitamin D-deficient group (<12.5 nmol/L) revealed higher prevalence of metabolic syndrome and all of its components (except elevated blood pressure) when compared to the group of mild to moderate deficiency (12.5-49.99 nmol/L). However, only elevated plasma triglycerides and reduced high density lipoprotein-cholesterol achieved statistical significance.

Conclusion: The high prevalence of vitamin D deficiency in apparently healthy premenopausal Saudi women is quite alarming. An inverse association of vitamin D levels with almost all the components of the metabolic syndrome was determined, although statistically insignificant. Significance of such relationship was possibly obscured by the relatively young age of the studied population. Therefore, prospective studies are needed to confirm these findings and to assess vitamin D deficiency as a predictor for the development of metabolic syndrome.

Key words: Vitamin D deficiency, Metabolic syndrome, Premenopausal.

INTRODUCTION

Vitamin D (VitD) has attracted increasing attention due to its multiple extra-skeletal benefits including those for cardiometabolic health *(de la Guía-Galipienso et al., 2021)*. VitD deficiency is a widespread problem that is increasing worldwide *(Sarma, 2019)*. It has been linked to many adverse health consequences such as cardiovascular diseases, hypertension, diabetes mellitus, cancer, and multiple sclerosis *(Solanki and Gohil, 2014)*. According to previous studies, the prevalence of VitD deficiency among Saudi women is very high *(Alzaheb & Al-Amer, 2017* and *AlFaris et al., 2019)*.

Metabolic syndrome describes a group of cardiometabolic risk factors including glucose intolerance, abnormal lipid profile, central obesity and hypertension. It has become a global problem and its prevalence is often higher in the urban population of some developing countries *(Saklayen, 2018)*.

The association between VitD status and metabolic syndrome was previously investigated *(Ganji et al., 2020)*. However, such relationship in young individuals was not fully explored. Therefore, the aim of the current study was to assess VitD status among apparently healthy premenopausal Saudi women, and to correlate this status with the different components of the metabolic syndrome.

SUBJECTS AND METHODS

This cross sectional study was carried out in the Center of Excellence for Osteoporosis Research (CEOR) at King Abdulaziz University (KAU). The ethical committee at CEOR approved the study. A total of 205 premenopausal Saudi women living in Jeddah (age range: 20-45 years old) participated in the study.

Women diagnosed with thyroid disorders, chronic renal or liver diseases, diabetes or hypertension were excluded from the study. Those taking any regular medications that affect VitD metabolism (including steroids, antiepileptics, antibiotics, or weight-lowering drugs) were also excluded. A written informed consent was obtained from every participant.

Modified "National Cholesterol Education Program-Adult Treatment Panel (NCEP: ATPIII) criteria" (Grundy et al., 2004) were used for the diagnosis of metabolic syndrome, details of which have been described previously *(Balkhyoor and Al Kadi, 2014)*.

Anthropometric measurements were taken and body mass index (BMI) was calculated. A validated *(Myers, 2006)* BP monitor, Bp TRU (VSM Med Tech Ltd, Coquitlam, Canada), was used for standardized BP measurements while the

subject was sitting. Three readings were recorded after a period of 5 minutes rest, and the mean of the last 2 was used for analysis.

Fasting venous blood samples (10-12 hours of fasting) were obtained from each subject for the determination of different biochemical tests. High density lipoprotein-cholesterol (HDL-C), triglyceride (TG) and fasting blood glucose (FBG) were estimated using "VITROS 250 Clinical Chemistry Auto analyzer" (Ortho-Clinical Diagnostics Inc., Rochester, NY, USA).

25-hydroxyvitamin D [25(OH)D] was quantitatively measured by a direct competitive immunoassay (chemiluminescence) using a LIASON auto analyzer (DiaSorin Inc, Stillwater, MN, USA). The level of 25(OH)D was classified as; desirable VitD level: 100-250 nmol/L, sufficient: 75-99.9 nmol/L, insufficient: 50-74.9 nmol/L, mild VitD deficiency: 25-49.9 nmol/L, moderate deficiency: 12.5-24.9 nmol/L, or severe VitD deficiency <12.5 nmol/L *(Ardawi et al., 2011)*. In addition, follicle stimulating hormone was assayed for confirmation of premenopausal status.

The Statistical Package for the Social Sciences (SPSS program version 16, Chicago, IL, USA) was used for data analysis. The sample size was estimated using the Epi-Info statistical package (version 6) [USD, West Park Place, Stone Mountain, GA, USA]. Data were checked for normality using the Kolmogorov-Smirnov statistic. Continuous variables were presented as the Mean±Standard deviation, and categorical variables as frequencies and percentages. Depending on the variable distribution, Pearson's or Spearman's tests were used to study the correlation between different variables. Student's t-test was used to test for significant differences between continuous variables among VitD deficiency groups. Chi-square test was used to examine the association between different categorical variables (VitD status and the presence or absence of metabolic syndrome and any of its components). A p value < 0.05 was considered significant.

RESULTS

Anthropometric, blood pressure and biochemical characteristics of the study population were presented in **Table (1)**.

Table (1): Anthropometric, blood pressure, and biochemical characteristics of the study population

Variables	Mean (±SD)
Age (years)	32 ±8
Weight (kg)	67.7 ±15.3
Height (m)	1.58 ±0.06
WC (cm)	78.59 ±12.33
BMI (kg/m^2)	27.26 ±6.08
SBP (mmHg)	103 ±11
DBP (mmHg)	66 ±8
TG (mmol/L)	1.02 ±0.45
HDL-C (mmol/L)	1.45 ±0.37
FBG (mmol/L)	4.63 ±1.15
Serum 25(OH)D (nmol/L)	23.62 ±17.45

WC, waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglyceride; HDL-C, High density lipoprotein-cholesterol; FBG, fasting blood glucose; 25(OH)D, 25- hydroxyvitamin D; SD, standard deviation.

Mean value of VitD in the study population was 23.62 nmol/L± 17.45 **(Table 1)**. The majority of the study population was VitD-deficient (92.2%) with levels < 50 nmol/L, while 22.93% had severe VitD deficiency with levels< 12.5 nmol/L. Only 3.4% had sufficient VitD levels ≥ 75 nmol/L. Other categories of the VitD status are illustrated in the following graph **(Figure 1)**.

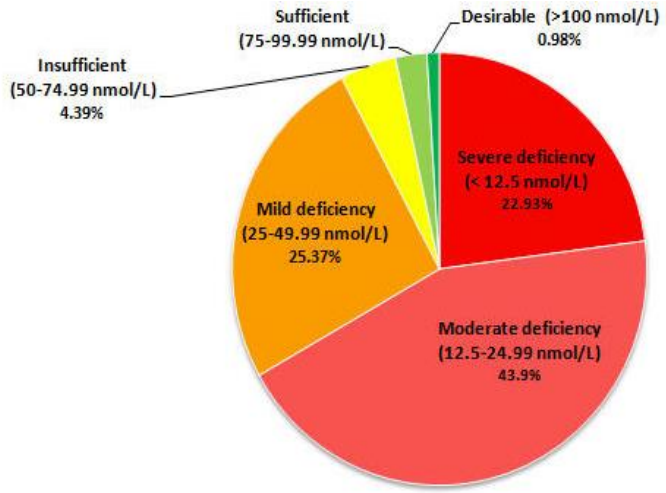


Figure (1): Vitamin D status in the study population

Sixteen women (7.8%) had metabolic syndrome according to the NCEP: ATPIII definition. Since 92.2% of the study population was VitD-deficient (<50 nmol/L), women were stratified into two groups according to the severity of VitD deficiency: severe VitD deficiency (<12.5 nmol/L) and mild to moderate VitD deficiency (12.5-49.99 nmol/L). Severely VitD-deficient group revealed higher prevalence of metabolic syndrome and all of its components (except elevated BP) as compared to the group of mild to moderate deficiency. However, only elevated plasma TG and reduced HDL-C achieved statistical significance **(Table 2** and **Figure 2)**.

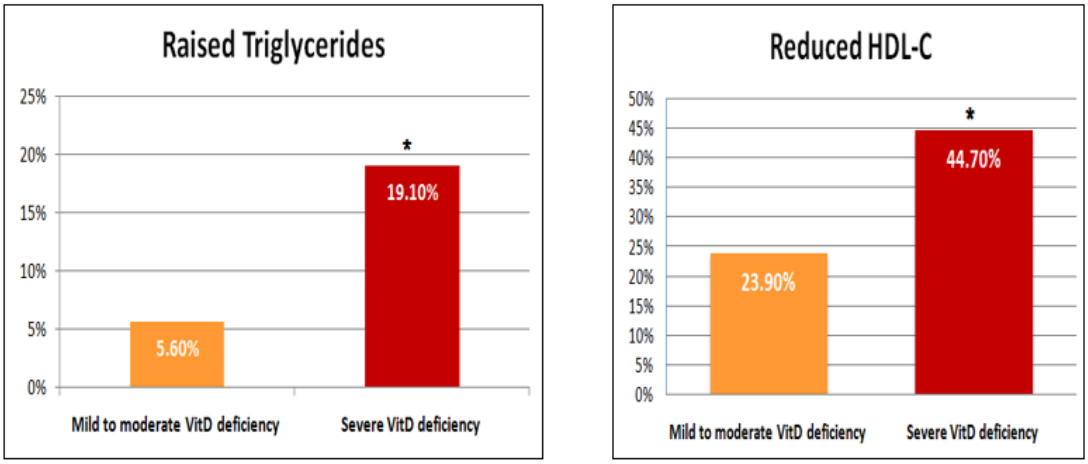
Table (2): Prevalence of metabolic syndrome and its components among the studied population and according to vitamin D status

Parameters \ Population	Mild to moderate VitD deficiency (n = 142) n (%)	Severe VitD deficiency (n = 47) n (%)	P value
Metabolic Syndrome	8 (5.6%)	5 (10.6%)	0.240
Central obesity	63 (44.4%)	24 (51.1%)	0.529
Reduced HDL-C	34 (23.9%)	21 (44.7%)	0.007*
Raised TG	8 (5.6%)	9 (19.1%)	0.005*
Impaired FBG	5 (3.5%)	4 (8.5%)	0.164
High BP	5 (3.5%)	0 (0.0%)	0.193

HDL-C, high density lipoprotein-cholesterol; TG, triglyceride; FBG, fasting blood glucose; BP, blood pressure; n, number of women

Mild to moderate VitD deficiency: (12.5-49.99 nmol/L); severe VitD deficiency: <12.5 nmol/L.

*Significance of difference of severe VitD-deficient compared to mild to moderate VitD-deficient group



Mild to moderate VitD deficiency: (12.5-49.99 nmol/L); severe VitD deficiency: <12.5 nmol/L.

*Significance of difference of severe VitD-deficient compared to mild to moderate VitD-deficient groups

Figure (2): Prevalence of raised triglycerides and reduced HDL-C when women were stratified into two groups according to the severity of vitamin D deficiency

Upon correlating 25(OH) D levels with the components of metabolic syndrome, a negative association was demonstrated with all of its components except HDL-C, which was positively correlated with 25(OH) D levels **(Table 3)**. However, none of these associations reached statistical significance.

Table (3): Correlation studies between 25(OH) D levels and each component of the metabolic syndrome

	WC	TG	HDL-C	FBG	SBP	DBP
25(OH)D (r)	-0.092	-0.037	0.045	-0.090	-0.132	-0.075
(P)	0.189	0.598	0.526	0.199	0.060	0.287

25(OH)D, 25-hydroxivitamin D; r, correlation coefficient; P, significant value; WC, waist circumference; TG, triglycerides; HDL-C, high density lipoprotein-cholesterol; FBG, fasting blood glucose; SBP, systolic blood pressure; DBP, diastolic blood pressure.

DISCUSSION

This study examined the association between vitamin D status and the individual components of metabolic syndrome in a group of healthy premenopausal Saudi women.

A high prevalence of VitD deficiency (92% had 25(OH) D levels < 50 nmol/L) was found among this group. Almost a quarter of these women had severe VitD deficiency (23% had 25(OH)D levels <12.5 nmol/L). Although apparently healthy young women with no known history of any previous medical illnesses were recruited, metabolic syndrome was prevalent among 7.8% of the studied subjects.

The current study supported other Saudi studies *(Alzaheb & Al-Amer, 2017* and *Altowijri et al., 2018)*, and confirmed the fact that VitD deficiency is common among young healthy Saudi women. Our results were similar to those reported by *Ardawi and Co-workers (2011)*, involving healthy women (aged 20-79 years old) living in Jeddah. VitD deficiency [25(OH)D levels <50 nmol/L] was found in 80%, and severe VitD deficiency [25(OH)D levels <12.5 nmol/L] in 10.5% of the studied women. They reported that the high prevalence of VitD deficiency was ascribed to obesity, limited sunlight exposure, inadequate dietary VitD supplementation and old age.

Possible causes of VitD deficiency in Jeddah area include limited exposure to sunlight due to nearly year-round hot weather. Consequently, people avoid direct sun exposure in such a hot climate. In addition, the traditional clothing style covers almost all body parts. Jeddah is one of the largest cities in Saudi Arabia and is undergoing rapid urbanization. As such, air pollution in Jeddah can also contribute to the high prevalence of VitD deficiency; since polluted air reduces ultra-violet B ray penetration, resulting in diminished vitamin D synthesis *(Yang et al., 2021)*.

In the current study, in addition to the high prevalence of central obesity among the studied women (46%), the prevalence of obesity (BMI ≥ 30 kg/m2) was 30.5%, while that of overweight (BMI between 25-29.99 kg/m2) was 28% (data not shown in the results section). Waist circumference was negatively correlated with 25(OH)D, and the prevalence of central obesity was greater in women with

severe VitD deficiency, though statistically insignificant.

A recent report *(Hajhashemy et al., 2022)* confirmed that increased waist circumference was linked to a higher risk of VitD deficiency and insufficiency in adults. Therefore, the excessive prevalence of obesity may explain the pervasiveness of VitD deficiency among the studied women.

Many theories have explained the cause of the inverse relationship between increased adiposity, especially abdominal obesity, and low VitD plasma levels. It was reported that 25(OH)D was diluted in greater tissue volume in obese subjects, in line with the volumetric dilution hypothesis *(Drincic et al. 2012)*. Other factors include the increased VitD metabolic clearance, with enhancement of its uptake by adipose tissues *(Savastano et al., 2017)*, or decreased expression of the principal hepatic VitD 25-hydroxylase enzyme in obesity *(Roizen et al., 2019)*. Another theory suggested that the inadequate sunlight exposure among obese subjects, due to the limited outdoor activity and associated osteoarthritis, contributes to VitD deficiency *(Stein et al., 2009)*.

In the present study, a negative correlation was observed between 25(OH)D and both systolic and diastolic BP, though statistically insignificant.

Many studies have addressed the association of lower 25(OH)D levels with higher BP readings and higher prevalence of hypertension *(Kunutsor et al., 2013* and *Ke et al., 2015)*. It was reported that, in addition to the significant inverse association of baseline 25(OH)D levels with the risk of incident hypertension in apparently healthy populations, the risk of future hypertension is decreased by 12 % for every 10 ng/mL increase in circulating 25(OH)D levels *(Kunutsor et al., 2013)*. An 8-year follow-up study reported that low VitD concentrations were associated with the development of hypertension in healthy middle-aged participants without any chronic illnesses *(Karadeniz et al., 2021)*. Therefore, VitD deficiency demonstrated in the current study could be regarded as a potential risk factor for the future development of hypertension in this relatively young group of women.

He and Hao (2019) reported the possible mechanisms by which hypertension could be induced with VitD deficiency. Firstly, due to the activation of the renin-angiotensin-aldosterone system, and secondly, due to hyperparathyroidism which is a consequence of VitD deficiency, causing hypertension. The last is due to endothelial dysfunction induced by VitD deficiency, with the resultant reduced nitric oxide synthesis in the blood vessels.

Our study showed a higher prevalence of impaired fasting glucose in the severe VitD-deficient women, as compared to that in the mild to moderate deficiency individuals. In addition, FBG was negatively correlated with 25(OH)D. However, neither finding reached the level of statistical significance.

Vitamin D was reported to be inversely correlated with FBG, and individuals with VitD deficiency (age ranged from 30 to 60 years) showed higher risk for development of type 2 diabetes mellitus when compared to individuals with normal VitD status *(Anwar et al., 2018)*. A significant negative correlation was also

demonstrated between 25(OH)D levels and FBG in individuals with type 2 diabetes (age ranged from 36 to 94 years) *(Huu et al., 2021)*.

On the other hand, in a study that involved relatively young participants (35-50 years old), no correlation was found between serum 25(OH)D and FBG in both type 2 diabetic patients and healthy individuals *(Islam et al., 2020)*.

Moreover, a prospective study showed an inverse association between serum 25(OH)D levels and 10-year risk of developing hyperglycemia, insulin resistance and metabolic syndrome, *(Forouhi et al., 2008)*. Such findings might explain the absence of significance in our results, and could point to the possible future development of disturbed glucose homeostasis upon following up these participants.

The proposed mechanisms by which VitD contributes to glucose homeostasis involve modulation of insulin secretion and sensitivity. VitD receptors are expressed by pancreatic β cells, and VitD deficiency can reduce the conversion of pro-insulin into insulin by β cells *(Schmitt et al., 2018)*. In an experimental model of non-obese type 2 diabetes, it was reported that VitD deficiency impairs both glucose-stimulated insulin secretion as well as insulin sensitivity. In addition, VitD deficiency decreases β-cell mass by reducing β-cell proliferation *(Park et al., 2016)*.

Another noteworthy finding demonstrated in the present study was the significantly higher prevalence of dyslipidemia, in the form of hypertriglyceridemia and reduced HDL-C, in the severe VitD-deficient women as compared to those with mild to moderate deficiency. Also, it was found that 25(OH)D levels were negatively correlated with TG and positively correlated with HDL-C, though statistically insignificant. The incidence of dyslipidemia in VitD-deficient subjects was reported to be higher than those without VitD deficiency *(Chaudhuri et al., 2013)*. Other studies have found similar association between VitD levels and each of TG and HDL-C in the elderly *(Vitezova et al., 2015* and *Liu et al., 2020)*. The lack of significance of such associations in our study could be attributed to the recruitment of young, premenopausal participants rather than elderly individuals.

Numerous mechanisms that could explain VitD-mediated reduction of TG levels are suggested. VitD, through its stimulating effect on intestinal calcium absorption, increases plasma calcium levels. Elevated plasma calcium has a suppressive effect on TG microsomal transfer protein, thus decreasing hepatic TG synthesis and secretion *(Cho et al., 2005)*. Another mechanism is related to VitD-suppressive action on serum parathyroid hormone (PTH) concentration. Low serum PTH levels can reduce TG levels through enhancing its peripheral removal. It is to be noted that the association between 25(OH)D and TG could also be explained by insulin resistance *(Pittas and Dawson-Hughes, 2010)* which could affect lipoprotein metabolism.

VitD deficiency could be linked to increased risk of coronary artery disease through its contribution in the development of unfavorable lipid profile

(Wang et al., 2012). Moreover, it was reported that plasma HDL-C levels increased after parenteral administration of VitD *(Liyanage et al., 2017)*.

Several studies found a significant inverse association between VitD levels and the incidence of metabolic syndrome *(Oosterwerff et al., 2011*; *Vitezova et al., 2015* and *Evrim et al., 2016)*. Moreover, a meta-analysis reported an association between VitD deficiency and risk of metabolic syndrome in the adult population *(Hajhashemy et al., 2021)*.

CONCLUSION

The high prevalence of vitamin D deficiency in apparently healthy premenopausal Saudi women is quite alarming. An inverse association of vitamin D levels with almost all the components of the metabolic syndrome was determined, although statistically insignificant. Significance of such relationship was possibly obscured by the relatively young age of the studied population. Therefore, prospective studies are needed to confirm these findings and to assess vitamin D deficiency as a predictor for the development of metabolic syndrome.

ACKNOWLEDGMENTS

We would like to thank the Ministry of Higher Education in Jeddah, Saudi Arabia for funding this project.

Conflict of interest: All authors declare that they have no conflict of interest.

REFERENCES

1. **AlFaris NA, Alkehayez NM, Almushawah FI, Alnaeem AN, Alamri ND and Almudawah ES (2019):** Vitamin D deficiency and associated risk factors in women from Riyadh, Saudi Arabia. Scientific Reports, 9:20371.
2. **Altowijri A, Alloubani A, Abdulhafiz I and Saleh A (2018):** Impact of nutritional and environmental factors on vitamin D deficiency. Asian Pac J Cancer Prev. 19(9):2569-2574.
3. **Alzaheb RA and Al-Amer O (2017):** Prevalence and predictors of hypovitaminosis D among Female University students in Tabuk, Saudi Arabia. Clinical Medicine Insights Women's Health, 10:1-7.
4. **Anwar T, Rahman MM, Mollah FH and Biswas SK (2018):** Association of serum vitamin D3 with newly diagnosed type 2 diabetes mellitus. Bangabandhu Sheikh Mujib Med Univ J., 11(1): 99-101
5. **Ardawi MM, Qari MH, Rouzi ARA, Maimani AA and Radaddi RM (2011):** Vitamin-D status in relation to obesity, bone mineral density, bone turnover markers and vitD receptor genotypes in healthy Saudi pre- and postmenopausal women. Osteoporosis Int., 22:463-475.
6. **Balkhyoor H and Al Kadi H (2014):** The prevalence of the metabolic syndrome and its components in a group of apparently healthy Saudi women. International Journal of Current Research, 6(4): 6266-6269.
7. **Chaudhuri JR, Mridula KR and Anamika A. (2013):** Deficiency of 25-hydroxyvitamin D and dyslipidemia in Indian subjects. J Lipids, 2013:1-7.
8. **Cho HJ, Kang HC, Choi SA, Ju YC, Lee HS and Park HJ (2005):** The possible role of Ca2+ on the activation of microsomal triglyceride transfer protein in rat hepatocytes. Biological & pharmaceutical bulletin. 28(8):1418-23.
9. **de la Guía-Galipienso F, Martínez-Ferran M, Vallecillo N, Lavie CJ, Sanchis-Gomar, F and Pareja-Galeano H (2021):** Vitamin D and cardiovascular health. Clin. Nutr., 40, 2946–2957.
10. **Drincic AT, Armas LA, Van Diest EE and Heaney RP (2012):** Volumetric dilution, rather than sequestration best explains the low

vitamin d status of obesity. Obesity, 20, 1444–1448.

11. **Evrim N, Atalay E, Gürsoy G, Bayram M and Salman RB (2016):** Relationship between levels of vitamin d and metabolic syndrome parameters in patients with metabolic syndrome and healthy individuals. Turkiye Klinikleri J Endocrin., 11(2):36-45.
12. **Forouhi NG, Luan JA, Cooper A, Boucher BJ and Wareham NJ (2008):** Baseline serum 25-hydroxy vitamin D is predictive of future glycemic status and insulin resistance. Diabetes, 57: 2619-25.
13. **Ganji V, Sukik A, Alaayesh H, Rasoulinejad H and Shraim M (2020):** Serum vitamin d concentrations are inversely related to prevalence of metabolic syndrome in Qatari women. Biofactors, 46, 180–186.
14. **Grundy SM, Brewer HB, Cleeman JI, Smith SC and Lenfant C (2004):** Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. Circulation, 109:433-438.
15. **Hajhashemy Z, Foshati S and Saneei P (2022):** Relationship between abdominal obesity (based on waist circumference) and serum vitamin D levels: a systematic review and meta-analysis of epidemiologic studies. Nutrition Reviews, 80(5): 1105-1117.
16. **Hajhashemy Z, Shahdadian F, Moslemi E, Mirenayat FS and Saneei P (2021):** Serum vitamin D levels in relation to metabolic syndrome: A systematic review and dose–response meta-analysis of epidemiologic studies. Obesity Reviews, 22:e13223.
17. **He S and Hao X (2019):** The effect of vitamin D3 on blood pressure in people with vitamin D deficiency: A system review and meta-analysis. Medicine (Baltimore), 98(19):e15284.
18. **Huu TT, Tran HD, TN Tran and Hoang BB (2021):** Relationship between vitamin D status and the relevant parameters of glucose in patients with type 2 diabetes. Diabetes Metab Syndr Obes., 14: 2489-2494.
19. **Islam MT, Mozaffor M, Islam ME and Roy HL (2020):** Correlation between vitamin D3 and fasting blood sugar in type 2 diabetes mellitus patients and normal individuals in a Bangladeshi population. JAIM, 18 (9): 65-68.
20. **Karadeniz Y, Özpamuk-Karadeniz F, Ahbab S, Ataoˇglu E and Can G (2021):** Vitamin D deficiency is a potential risk for blood pressure elevation and the development of hypertension.Medicina, 57: 1297.
21. **Ke L, Mason RS, Kariuki M, Mpofu E and Brock KE (2015):** Vitamin D status and hypertension: a review. Integr Blood Press Control, 8: 13–35.
22. **Kunutsor SK, Apekey TA and Steur M (2013):** Vitamin D and risk of future hypertension: meta-analysis of 283,537 participants. Eur J Epidemiol., 28:205-221.
23. **Liu L, Cao Z, Lu F, Liu Y, Lv Y, Qu Y, Gu H, Li C, Cai J, Ji S, Li Y, Zhao F and Shi X (2020):** Vitamin D deficiency and metabolic syndrome in elderly Chinese individuals: evidence from CLHLS. Nutrition & Metabolism, 17:58.
24. **Liyanage GC, Lekamwasam S, Weerarathna TP and Liyanage CE (2017):** Effects of high-dose parenteral vitamin D therapy on lipid profile and blood pressure in patients with diabetic nephropathy: A randomized double-blind clinical trial. Diabetes Metab Syndr., 11 (2): S767-S770.
25. **Myers MG (2006):** Automated blood pressure measurement in routine clinical practice. Blood Press Monit., 11:59-62.
26. **Oosterwerff MM, Eekhoff EM, Heymans MW, Lips P and van Schoor NM (2011):** Serum 25-hydroxyvitamin D levels and the metabolic syndrome in older persons: a population-based study. Clin Endocrinol (Oxf), 75:608-613.
27. **Park S, Kim DS and Kang S (2016):** Vitamin D deficiency impairs glucose-stimulated insulin secretion and increases insulin resistance by reducing PPAR-expression in nonobese Type 2 diabetic rats. J. Nutr. Biochem., 27: 257–265.

28. **Pittas AG and Dawson-Hughes B (2010):** Vitamin D and diabetes. J Steroid Biochem Mol Biol., 121(1-2):425-429.

29. **Roizen JD, Long C, Casella A, O'Lear L, Caplan I, Lai M, Sasson I, Singh R, Andrew J Makowski , Simmons R and Levine MA (2019):** Obesity decreases hepatic 25-hydroxylase activity causing low serum 25-hydroxyvitamin D. J Bone Miner Res., 34(6):1068-1073.

30. **Saklayen MG (2018):** The Global Epidemic of the Metabolic Syndrome. Current Hypertension Reports, 20(2): 12.

31. **Sarma D (2019):** Vitamin D-Public health significance. Endocrinol Metab Int J., 7(5):132-138.

32. **Savastano S, Barrea L,Savanelli MC, Nappi F, Di Somma C, Orio F and Colao A (2017):** Low vitamin D status and obesity: Role of nutritionist. Reviews in Endocrine & Metabolic Disorders, 18(2):215-225.

33. **Schmitt EB, Nahas-Neto J, Bueloni-Dias F, Poloni PF, Orsatti CL and Petri-Nahas EA (2018):** Vitamin D deficiency is associated with metabolic syndrome in postmenopausal women. Maturitas, 107:97–10.

34. **Solanki P and Gohil P (2014):** Role of vitamin D in human diseases and disorders - An overview. IJPR, 4(2): 34-42.

35. **Stein EM, Strain G, Sinha N, Ortiz D, Pomp A, Dakin G, McMahon DJ, Bockman R and Silverberg SJ (2009):** Vitamin D insufficiency prior to bariatric surgery: risk factors and a pilot treatment study. Clinical Endocrinol.,71(2): 176–83.

36. **Vitezova A, Zillikens MC, Van HT, Sijbrands EJ, Hofman A, Uitterlinden AG, Franco OH and Kiefte-de Jong JC (2015):** Vitamin D status and metabolic syndrome in the elderly: the Rotterdam study. Eur J Endocrinol., 172(3):327-35.

37. **Wang H, Xia N, Yang Y and Peng D (2012):** Influence of vitamin D supplementation on plasma lipid profiles: A meta-analysis of randomized controlled trials. Lipids Health Dis., 11, 42.

38. **Yang C, Li D, Tian Y and Wang P (2021):** Ambient air pollutions are associated with vitamin D status. Int. J. Environ. Res. Public Health, 18: 6887.

الارتباط بين مستوى فيتامين "د" وعناصر متلازمة الأيض في النساء قبل انقطاع الطمث

حنان بالخيور[1]، حنان أحمد القاضي[2]، جيهان ابراهيم السلاموني[2,3]،

محمد صالح عرضاوي[4]

[1]قسم الدبلوم الصحي المساعد، مدينة الملك عبدالله الطبية، مكة المكرمة، المملكة العربية السعودية

[2]قسم الفسيولوجي، كلية الطب، جامعة الملك عبد العزيز، جدة، المملكة العربية السعودية

[3]قسم الفسيولوجي، كلية الطب، جامعة عين شمس، القاهرة، جمهورية مصرالعربية

[4]قسم العلوم المرضية، كلية فقيه للعلوم الطبية، جدة، المملكة العربية السعودية

خلفيـــة البحـــث: يعتبـــر نقـــص فيتـــامين (د) مشـــكلة واســـعة الانتشـــار ومتزايـــدة، وقـــد تســـاهم فـــي حـــدوث العديـــد مـــن الأمـــراض بمـــا فـــي ذلـــك متلازمـــة الأيـــض والتـــي تشـــتمل علـــى مجموعـــة مـــن عوامـــل الخطـــر, ممـــا يزيـــد مـــن احتماليـــة الإصـــابة بالعديـــد مـــن الامراض المزمنة، مثل مرض السكري وأمراض القلب والأوعية الدموية.

هـــدف البحـــث: هـــدفت هـــذه الدراســـة إلـــى تحديـــد مســـتوى فيتـــامين (د) لـــدى النســـاء الســـعوديات فـــي فتـــرة مـــا قبـــل انقطـــاع الطمـــث وتقيـــيم ارتباطـــه بمكونـــات متلازمـــة الأيض.

مـــنهج البحـــث: أجريـــت هـــذه الدراســـة المقطعيـــة فـــي مركـــز التميـــز لأبحـــاث هشاشـــة العظام، بجامعة الملك عبد العزيز، جدة، المملكة العربية السعودية.

تـــم اختيـــار 205 امـــرأة- تتـــراوح اعمـــارهن بـــين20 الـــى 45 ســـنة- بشـــكل عشـــوائي. تـــم قيـــاس ضـــغط الـــدم وقيـــاس الـــوزن والطـــول ومحـــيط الخصـــر وحســـاب مؤشـــر كتلـــة الجســـم. كمـــا تـــم جمـــع عينـــات الـــدم الصـــائم لقيـــاس كـــل مـــن 25-هيدروكســـي فيتـــامين د، جلوكـــوز الـــدم الصـــائم، الـــدهون الثلاثيـــة، والكوليســـترول الحميـــد. وقـــد اســـتُخدِم البرنـــامج القـــومي للتثقيـــف بالكوليســـترول – الاجتمـــاع الثالـــث لعـــلاج البـــالغين المعدل- لتشخيص متلازمة الأيض.

النتـــائج: كـــان نقـــص فيتـــامين (د) واســـع النطـــاق، حيـــث كـــان لـــدى 92.2٪ مـــن النســـاء مســـتويات 25-هيدروكســـي فيتـــامين (د) أقـــل مـــن 50 نـــانومول / لتـــر. وكانـــت متلازمـــة الأيـــض منتشـــرة فـــي 7.8٪ مـــن الحـــالات. كـــذلك اظهـــر هـــذا البحـــث وجـــود علاقـــة عكســـية بـــين مســـتوى فيتـــامين (د) وجميـــع عناصـــر متلازمـــة الأيـــض- باســـتثناء الكوليســـترول الحميـــد والـــذي ارتـــبط ايجابيـــا مـــع مســـتوى فيتـــامين (د)- مـــع ملاحظـــة انـــه لم يكن لأي من هذه العلاقات أي دلالة احصائية.

والجـــدير بالـــذكر أن معـــدل انتشـــار متلازمـــة الأيـــض وعناصـــرها المختلفـــة (باســـتثناء ضـــغط الـــدم المرتفـــع) كـــان أكبـــر فـــي الســـيدات اللاتـــي يعـــانين مـــن وجـــود نقـــص شـــديد فـــي مســـتوى فيتـــامين (د) (أقـــل مـــن 12,5 نـــانومول/لتر) بالمقارنـــة بالســـيدات اللاتـــي يعـــانين مـــن نقـــص بســـيط الـــى متوســـط بمســـتوى هـــذا الفيتـــامين (12,5-49,99 نـــانومول/لتر) وقـــد كانـــت هـــذه العلاقـــة ذات دلالـــة إحصـــائية فقـــط مـــع عنصـــري ارتفـــاع الدهون الثلاثية وانخفاض الكوليسترول الحميد.

الاســـتنتاج: يعتبـــر الانتشـــار الواســـع لـــنقص فيتـــامين (د) لـــدى النســـاء الســـعوديات فـــي فتـــرة مـــا قبـــل انقطـــاع الطمـــث أمـــر مقلـــق للغايـــة ممـــا يشـــير الـــى وجـــود حاجـــة ماســـة إلـــى اتخاذ التدابير اللازمة لتصحيحه.

وقـــد أكـــدت هـــذه الدراســـة وجـــود ارتبـــاط ســـلبي بـــين مســـتويات فيتـــامين (د) وتقريبًـــا جميـــع مكونـــات متلازمـــة الأيـــض مـــع عـــدم وجـــود دلالـــة احصـــائية لهـــذا الارتبـــاط، ربمـــا بســـبب العمـــر الصـــغير نســـبيًا للســـيدات اللاتـــي تـــم اجـــراء الدراســـة عليهن.

لـــذلك فإنـــه هنـــاك حاجـــة لإجـــراء دراســـات مســـتقبلية لتأكيـــد هـــذه النتـــائج وتقيـــيم نقص فيتامين كعامل خطورة قد ينبئ بحدوث متلازمة الايض.

الكلمات الدالة: فيتامين (د)، متلازمة الايض، فترة ما قبل انقطاع الطمث.