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Prevalence of vitamin D deficiency and association with metabolic syndrome

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Abstract

Objectives: To evaluate the correlation between vitamin D (vit. D) serum concentrations and metabolic syndrome (MetS)

Methods: A cross-sectional study of 1205 participants (702 women and 503 men) from between the ages of 18 and 80 years was included in this study, multivariate linear regression analyses to examine the association between metabolic syndrome and prevalence of vitamin D deficiency (defined as $<20\ ng\ ml^{-1}$ serum vitamin D levels) Odds ratios and 95% confidence intervals were calculated for all analyses.

Results: Approximately 64% of participants were vitamin D deficient ($<20\ ng\ ml^{-1}$) with more men being deficient (68.6%) than women (61.3%). Serum vitamin D was 8% lower in individuals with metabolic syndrome. compared to individuals without metabolic syndrome. Waist circumference and HDL as well as high triglyceride levels were also significantly positively associated with vitamin D deficiency. No association was found between the other components of metabolic syndrome or diabetes and the presence of vitamin D deficiency.

Conclusions: Vitamin D deficiency is prevalent in this population. Presence of metabolic syndrome was associated with presence of vitamin D deficiency.

Introduction

Vitamin D deficiency is highly prevalent worldwide and is associated with many adverse health outcomes.[1] Vitamin D is acquired in three ways; from sun exposure, diet and supplements; however, the greatest proportion is obtained from sun exposure. For example, exposure to 0.5 minimal erythemal dose is equivalent to supplementing ~ 3000 IU of vitamin D3 [2]. One of the main physiological functions of vitamin D is to maintain calcium and phosphorous levels in the body to sustain various metabolic functions, including bone metabolism [3]. The most abundant circulating biomarker of vitamin D status is 25- hydroxyvitamin D (25(OH)D), which also has a longer half-life (25 days) compared to the active metabolite; 1,25-dihydroxyvitamin D (7 h). However, the threshold used to define vitamin D deficiency often varies according to the population and outcome of interest [4]. The most common definition for optimal 25(OH)D levels is the concentration at which it supresses the parathyroid hormone to its minimum, however, using this definition

results in a wide range of minimal optimal values from 12 ng ml − 1 to 40 ng ml − 1 . 7

The association between vitamin D levels and a range of disease outcomes could be explained by intermediate disease risk factors such as the metabolic syndrome (MetS); a constellation of risk factors, including increased obesity, hypertriglyceridemia, hypertension and diabetes. Some studies have suggested an inverse associations between serum vitamin D and MetS, while others have not confirmed this observation [5]. It is also unclear which components of the MetS might drive this association with vitamin D with some studies, suggesting obesity and others glucose haemostasis

A number of studies have shown that vitamin D levels are inversely associated with the risk of a diverse set of diseases, including several cancers, diabetes and cardiovascular diseases [6]. The association between vitamin D levels and a range of disease outcomes could be explained by intermediate disease risk factors such as the metabolic syndrome (MetS); a constellation of risk factors, including increased obesity, hypertriglyceridemia, hypertension and diabetes. Some studies have suggested an inverse associations between serum vitamin D and MetS, while others have not confirmed this observation [7]. It is also unclear which components of the MetS might drive this association with vitamin D with some studies, suggesting obesity and others glucose haemostasis

At the same time, the prevalence of MetS was measured to be ~ 26.5%, according to the International Diabetes Federation (IDF) criteria, aged 20 and over.27 Similarly, there was a high prevalence of MetS, according to both the IDF and Adult Treatment Panel (ATPIII) criteria, and its components in neighbouring regions. Given the high prevalence of these two conditions in the region and the limited epidemiological evidence in this population so far, we aimed to investigate the prevalence of vitamin D deficiency, as well as the association between MetS and its components with vitamin D.

Materials and Methods

The data for this study was consisted of 1205 participants; with 702 females and 503 males, at the time of investigation. All participants gave informed consent. Questionnaires on health, lifestyle, and diet were completed. Anthropometric measurements and body composition were also obtained. In this study, vitamin D deficiency was defined by the US Endocrine Society (USES) guidelines as o20 ng ml − 1 (50 nmol l − 1) in circulation, while individuals with 21–29 ng ml − 1 were considered insufficient, and individuals with 430 ng ml − 1 were considered sufficient [8]. To convert between ng ml − 1 and nmol l-1 of 25(OH)D, multiply the concentration of 25(OH)D in ng ml − 1 by 2.5.

Anthropometric measures

Weight, height, body mass index (BMI), and waist and hip circumference were all measured by a trained nurse. BMI was calculated as weight in kilogram divided by height in metres squared (kg m− 2). Waist and hip measurements were measured using a non-stretchable sprung measuring tape by Seca. For the hip measurement, the Seca measuring tape was placed at the widest part of the hips and measured in centimetres. Waistto-hip ratio was calculated as waist in centimetres divided by hip circumference also in centimetres.

Metabolic syndrome components

Metabolic syndrome was defined according to the new IDF definition as being centrally obese (defined as waist circumference ⩾94 cm for males and ⩾80 cm for females) as well as at least two of the following four factors: (1) raised triglycerides (⩾1.7 mmol l − 1),2 (2) reduced HDL cholesterol (o1.03 mmol l − 1 in males and o1.29 mmol l − 1 for females), (3) raised blood pressure (BP) (systolic BP ⩾130 mm Hg or diastolic BP ⩾85 mm Hg), (4) raised

fasting plasma glucose (defined as HbA1c levels⩾5.7 mmol l − 1) [9]. HbA1c was measured with turbidimetric inhibition immunoassay (TINIA). Diabetes was reported in two ways: (1) self-reported; where 15.4% reported having diabetes and (2) laboratory measurements of HbA1c levels (⩾6.5% was considered evidence for diabetes), where 17.4% of participants were considered diabetic.

Statistical analysis

To test the difference in the distributions of categorical variables, a X2 test was performed, while a t-test was performed to test the differences between a categorical and a continuous variable after stratifying by sex. A geometric mean for serum 25(OH)D was calculated by performing a oneway Analysis of Covariance (ANCOVA) adjusting for sex, age and season of blood collection. Metabolic syndrome and its components were categorised into having abnormal levels or normal levels based on the IDF definition [10]. Univariate (crude) and multivariable linear regression models were used to calculate the regression coefficients and associated 95% confidence intervals (CI). Multivariable model (Model 2) was adjusted for age (continuous), sex, ethnicity, education, physical activity and season. Participants with missing ethnicity values (n = 242) were excluded from all analyses.

Results

Descriptive Characteristics

Approximately 58% of the study population were females; mean age was similar between males and females. There were several differences between males and females with regards to anthropometric factors: men had more visceral fat (97 cm) than women (85 cm) and also reported to smoke more and exercise more frequently (39% and 19 h per week, respectively) than women (12% and 11 h per week, respectively). BMI as well as the prevalence of diabetes (~15%) and metabolic syndrome (~28%) were similar between males and females. Approximately 64% of the participants in this study were vitamin D deficient (o20 ng ml − 1) with slightly more men (69%) being vitamin D deficient compared to women (61%). Another 25% of the population had insufficient vitamin D levels. The percentage of people reported taking vitamin D supplements was high with 49% of females and 25% of males. Women were more likely to undergo vitamin D screening, and take vitamin D and calcium supplements compared to men. Females reported a higher concentration of serum 25(OH)D compared to males in both the adjusted and unadjusted mean 25(OH)D, although this difference was not statistically significant. Also, higher 25(OH)D levels were observed in older compared to younger participants. The remaining characteristics did not show differences according to 25(OH)D levels.

Associations between metabolic syndrome, vitamin D deficiency and serum 25(OH)D levels there is association between metabolic syndrome with the risk of vitamin D deficiency (o20 ng ml − 1) and serum 25(OH) D levels. There was a significant positive association between the presence of MetS and vitamin D deficiency after adjusting for Model 2). However, age was the main confounder responsible for the statistical significant association between MetS and its components with 25(OH)D concentration. The presence of MetS was associated with lower 25(OH)D. Components of the MetS that were inversely associated with serum 25(OH)D levels, including high waist circumference and elevated. While increasing levels of HDL was positively associated with 25(OH)D levels. Elevated blood pressure showed weak inverse association with vitamin D; the association was stronger with the presence of vitamin D deficiency compared to 25(OH)D levels. The association between vitamin D deficiency or 25(OH)D levels with presence of diabetes was not statistically significant.

Discussion

In this unique, we showed that the prevalence of vitamin D deficiency is very high (64%),with the majority of the examined population showing signs of deficiency. This is consistent with previous observations which estimated the prevalence of vitamin deficiency (o20 ng/ml) to range between At the same time, the percentage of participants reporting supplementation for vitamin D, especially in women was very high (49%), higher than that observed in white populations, potentially showing a greater awareness of vitamin D deficiency in this population [11]. Approximately 64% of the participants were vitamin D deficient (o20 ng ml − 1), with slightly more men being deficient compared to women. Previous evidence from studies in the Middle East region supports higher level of vitamin D in males compared to females. A Saudi Arabian study performed on 10 709 participants reported more females being severely vitamin D deficient (o10 ng ml − 1) compared to males [12]. A Bahraini and Lebanese study also reported lower mean serum vitamin D in females compared to males. One hypothesis for this difference could be explained by vitamin D supplementation, women were more likely to take vitamin D supplements compared to men. MetS was significantly associated with the risk of vitamin D deficiency. Several cross-sectional and few prospective studies have reported similar positive associations between vitamin D deficiency and the risk of metabolic syndrome [13]. In addition, prospective studies from the PROMISE cohort and the Australian Diabetes, Obesity and Lifestyle (Aus-Diab) study, reported significant inverse associations between continuous serum vitamin D and overall MetS, based on the IDF criteria [14]. Studies examining the direction of the effect between vitamin D, cholesterol and lipids suggest that lipids levels and BMI may be a cause for decreased vitamin D and not vice versa [15]. Other cross-sectional studies did not support these associations. However, this might have been due to the small sample sizes and small variation in the exposure (e.g. high levels of vitamin D in the study by Reis et al. in both men and women). Studies on Middle Eastern population where the metabolism of vitamin D could be different due to different lifestyles and darker skin pigmentation, are very limited and our results support an inverse association between the two examined phenotypes. Many mechanisms have been proposed to explain the association between vitamin D and future risk of MetS component [16]. As vitamin D is fat soluble and could be stored in adipose tissue, it can be sequestered in the subcutaneous fat in obese individuals, reducing the levels of circulating vitamin D in the blood leading to less release of vitamin D into the blood [17]. Vitamin D has also been shown to inhibit the release of cytokines from the immune cell, which is harmful to β cells. Obesity, measured as BMI, waist circumference, and waist-to-hip ratio were positively associated with vitamin D deficiency with stronger associations observed with waist circumference [18]. Conversely, several cross-sectional studies did not report a correlation between obesity and vitamin D deficiency [19]. However, these are mainly small studies with limited power to observe associations. We also showed that high triglyceride levels were associated with lower levels of vitamin D. Studies also report significant inverse associations between vitamin D with high triglyceride levels. However, when stratified by sex, only the association in men remained significant in the Ling et al. study. On the contrary, Reis et al. reported a positive trend between vitamin D deficiency and high triglyceride levels only in women. In the QSS, measurements of height, weight, and BMI were similar to this study. However over 60% of the participants had a university degree compared to 35% of the QSS, suggesting a higher representation of more educated and higher socioeconomic status individuals. Since this study is cross-sectional, the directionality of the relationship

between adiposity, vitamin D, metabolic syndrome, and diabetes could not be elucidated. The non-significant results for the association between diabetes and vitamin D deficiency, may have been due to the relatively small sample size and low response rate for some questions regarding physical activity, smoking, supplements and so on. It could also have been due to other confounding factors such as specific medication use and dosage of supplements, which were not captured. Approximately 84% of participants were missing data on medication use for hypertension and 68% of participants were missing for medication on cholesterol usage, which may have caused some misclassification. Additionally, the low levels of circulating vitamin D found in this population may have been insufficient to observe any inverse relationships with metabolic syndrome components and diabetes. To conclude, the findings from this study support a positive association between the presence of metabolic syndrome and vitamin D deficiency. We also observed that MetS components, such as obesity and high triglyceride, were inversely associated with circulating serum vitamin D levels. Future prospective studies should elucidate the potential causal association between vitamin D and MetS using Mendelian randomisation approaches or through supplementation with Vitamin D. Moreover, mechanistic studies should concentrate on identifying pathophysiological pathways and molecular mechanisms linking vitamin D deficiency and metabolic syndrome.

References

1. Gueli N, Verrusio W, et al. Vitamin D: drug of the future. A new therapeutic approach. Arch Gerontol Geriatr 2012;54(1):222-7.
2. Danik JS, Manson JE. Vitamin D and cardiovascular disease. Curr Treat Options Cardiovasc Med 2012;14(4):414- 24.
3. Andreozzi P, Verrusio W, Viscogliosi G, et al. Relationship between vitamin D and body fat distribution evaluated by DXA in postmenopausal women. Nutrition 2015;29:pii:S0899-9007(15)00526-2. DOI: 10.1016/j. nut.2015.12.029
4. Michos ED, Melamed ML. Vitamin D and cardiovascular disease risk. Current opinion in Clinical Nutrition and Metabolic Care 2008;11:7-12.
5. Wang TJ. Vitamin D and cardiovascular disease. Annu Rev Med 2016:14;67:261-72. DOI: 10.1146/annurevmed-051214-025146
6. Buffington C, Walker B, Cowan GS, et al. Vitamin D deficiency in the morbidly obese. Obes Surg 1993;3:421-4.
7. Wortsman J, Matsuoka LY, Chen TC, et al. Decreased bioavailability of vitamin in obesity. Am J Clin Nutr 2000;72:690-3.
8. Arunabh S, Pollack S, Yeh J, et al. Body fat content and 25-hydroxyvitamin D levels in healthy women. J Clin Endocrinol Metab 2003;88:157-61.
9. Hypponen E, Power C. Vitamin D status and glucose homeostasis in the 1958 British birth cohort: the role of obesity. Diabetes Care 2006;29:2244-6.
10. Hintzpeter B, Mensink GB, Thierelder W, et al. Vitamin D status and health correlates among German Adults. Eur J Clin Nutr 2008;62(9):1079-89.
11. Brenner DR, Arora P, Garcia-Bailo B, Wolever TM, Morrison H, El-Sohemy A, Karmali M, Badawi A. Plasma vitamin D levels and risk of metabolic syndrome in Canadians. Clin Invest Med 2011;34:E377.
12. Shantavasinkul PC, Phanachet P, Puchaiwattananon O et al. Vitamin D status is a determinant of skeletal muscle mass in obesity according to body fat percentage. Nutrition 2015;31(6):801-6. DOI: 10.1016/j.nut.2014.11.011
13. Ford ES, Ajani UA, McGuire LC, Liu S. Concentrations of serum vitamin D and the metabolic

syndrome among US adults. Diabetes Care 2005;28:1228-30.

14. McGill AT, Stewart JM, Lithander FE, Strik CM, Poppitt SD. Relationships of low serum vitamin D3 with anthropometry and markers of the metabolic syndrome and diabetes in overweight and obesity. Nutr J 2008;7:4.
15. Rueda S, Fernández-Fernández C, Romero F, Martínez de Osaba J, Vidal J. Vitamin D, PTH, and the metabolic syndrome in severely obese subjects. Obes Surg 2008;18:151-4.
16. Reis JP, von Mühlen D, Miller ER 3rd. Relation of 25-hydroxyvitamin D and parathyroid hormone levels with metabolic syndrome among US adults. Eur J Endocrinol 2008;159:41-8.
17. Vitezova A, Zillikens C, van Herpt T, Sijbrands E. Vitamin D status and metabolic syndrome in the elderly: the Rotterdam Study. Eur J Endocrinol 2015;172(3)327-35.
18. Zerwekh JE. Blood biomarkers of vitamin D status. Am J Clin Nutr 2008;87(Suppl.):1087-91S.
19. Boersma D, Demontiero O, Mohtasham Amiri Z, et al. Vitamin D status in relation to postural stability in the elderly. J Nutr Health Aging 2012;16(3):270-5.
20. Zheng Y1, Zhu J1, Zhou M1, Cui L1, Yao W2, Liu Y1. Meta-analysis of long-term vitamin d supplementation on overall mortality. PLoS One 2013;8(12):e82109.