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Associations of 25-hydroxyvitamin D Deficiency with metabolic components and Adiponectin among obese young adults, Saudi Arabia

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Vitamin D insufficiency/deficiency prevalent in all age groups across the world, is common in obesity. This cross-sectional study is to evaluate the relationship between levels of adiponectin, circulating 25(OH)D, and its effect on metabolic biomarker among overweight/obese female students. Three hundred female students; with mean age20.9±3.2years were randomly selected from Jouf University during 2016–2017 year. Anthropometric and biochemical indices were determined. The study showed 19%of the female's student were either overweight or obese (15%and4%, respectively). The frequency of MS diagnosis among the students was17%, with13% and 4% had either three or four risk factors, respectively. Overweight/obese subjects had significantly worse anthropometric and biochemical characteristics, including waist/hip ratio, blood pressure (BP), fasting blood glucose (BG), insulin, insulin resistance (HOMA-IR), triglyceride levels (TG), low-density lipoprotein cholesterol levels (LDL-C), leptin, adiponectin, leptin/adiponectin ratio and high-density lipoprotein cholesterol levels (HDL-C) compared with normal weight. Of the subjects diagnosed with MS, 59%had mild and 8.6% had severe25(OH)D deficiency. There was negative association between 25(OH)D and both FBG and HOMA-IR among young women obese/overweight. The study suggested that low level of adiponectin was strongly correlated with low25(OH)D levels. Also, the prevalence of MS tends to increase with high occurrence rate of low circulating 25(OH)D levels that is, known cause poor glycemic control and prediction of cardiovascular outcomes.

Keywords: Vitamin D deficiency, adiponectin, leptin, blood glucose, insulin resistance and lipid profile

INTRODUCTION

The deficiency of Vitamin D is prevalent among the age groups across the world. Hypovitaminosis D, which is deemed to occur at a lower level of 25OHD in blood < 50 mmol/l, is common in obese (Bassil et al., 2013).Several studies have found serum 25OHD levels greater than 50 nmol/l to be prevalent (in up to 90% of the

population) with lower proportion of the population have 25(OH)D levels < 25nmol/l in South Asia, Middle East and North Africa (Bassil et al., 2013). Low vitamin D status which indicated by circulating 25(OH)D is linked to metabolic risk factors such as inflammation, adipokines, insulin resistance, abnormalities of lipid profile, and high (BP) in adults 9Cheng et al., 2010). However, many studies have evaluated the relationship between 25(OH)D concentrations and metabolic components in children and adolescents. Some workers suggested that vitamin D is isolated in excess fatty tissue, resulting in lack of biological availability [Pramyothin et al., 2011]. In a study of Pittas et al., found that individuals who suffered from CVD were more vitamin D deficient than those without CVD [Pittas et al., 2010]. In a different study that followed young adults for more than 20 years, vitamin D level, in combination with vitamin D supplementation, was inversely related to the incidence of MS in the study population [Fung et al., 2012]. It also, suggested that lifestyle factors and having high levels of body fat mass might contribute to this development.

Adipokines secretes by adipose tissue, has a vital role on body weight, blood glucose, and lipid metabolism. Adiponectin, a collagen-like protein has anti-inflammatory, antiatherogenic and antidiabetic properties. Increased circulating of adiponectin is associated with lower risk of impaired glucose tolerance and myocardial infarction, indicating that the atherosclerosis index was early (Wolfson et al., 2009). In obesity, adiponectin was declined and may be involved in type-2 diabetes and cardiovascular disease pathology [Wolfson et al., 2009]. Adipose tissues secrete leptin, a cytokine-like molecule that regulates the fatty mass and body weight by inhibiting eating and stimulating energy consumption (Jéquier et al., 2002). Leptin was increases in obesity, type-2 diabetes, MS and hypertension. Several studies have found that leptin is the biomarker of obesity, IR, MS and cardiovascular disease in adult (Jéquier et al., 2002).

Hyperlipidaemia, which considered to be an independent CVD risk factor, forms a potential link between low vitamin D levels and CVD. Dyslipidaemia, known to be a disorder of lipoprotein metabolism, is the result of excess TG, TC, LDL-C, and is due to the suppression of HDL-C. A favourable serum lipid profile is correlated with high levels of 25(OH)D which regulates the appropriate use of apolipoprotein A-1 (ApoA1), since it is a component of HDL. Salehpou et al., (2012) conducted a study on obese individuals who received vitamin D supplement (25µg/d); the results of this study suggested that significantly elevated levels of HDL-C and Apo A-1 lowered body fat in obese individuals. The objective of the current study was to evaluate the relation between adiponectin and circulating 25(OH)D levels, and its effect on metabolic biomarker among overweight/obese.

MATERIALS AND METHODS

Subjects

This cross-sectional study, which took place between November 2016 and June 2017, was performed on group of 300 overweight/obese female students; with mean age 20.9±3.2 years. Subjects were selected randomly from female students attended in Jouf University, Sakaka, Saudi Arabia. The exclude criteria: liver disease, kidney disease, diabetes mellitus, use of any medication that could affect bone health or vitamin D status within the previous three years, history of pregnancy, lactation convulsions, thyroid-parathyroid diseases, adrenal disease. All participates gave their informed consent before inclusion in this study. The study was approved by the Medical Research Ethics Committee of the Faculty of Medicine, King Saud University.

Data collection.

Each subject completed a self-reporting questionnaire that revealed their socio-economic status, age, smoking habits, meal and snack frequencies, and level of physical activity, as well as the educational and occupational status of each of their parents.

Dietary data.

For each subject, the following data were recorded: the frequency of meals and snacks, and daily intake of vegetables, fruits, fatty foods, sugars, milk, mushrooms, fish, and crustaceans. Lifestyle practices, such as physical activity (exercise) and history of vitamin D supplements, were also recorded.

Nutrient analysis.

Daily dietary intake was recorded over three consecutive days to insure energy and nutrient intake. A software program, purchased from ESHA (Trumbo et al., 2002), was used to analyse the nutritional content of each subject's diet. The dietary reference intakes (DRI), which are the recommended dietary intakes for a healthy diet,

were calculated for each subject, adjusting for local practices, gender, age, weight, height, and physical activity levels (Hambidge, 2010).

Anthropometric measurements.

Anthropometric data, including weight and height, were recorded (to the nearest0.1kg and0.5cm, respectively) using a beam balance scale (Adam Equipment Inc., USA) after removal of outer garments and shoes; body mass index (BMI) was calculated (body weight/kg/(body height/ m)2) [Woo, 2009]. Body weight was categorised according to BMI values using the National Institutes of Health guidelines: (i) normal with BMI range 18.5-24.9, (ii) overweight with BMI range 25.0-29.9, and (iii) obese with BMI > 30 [Pi-Sunyer, 1998]. Waist and hip circumferences(cm) were measured and the weight-to-hip ratio (WHR) was calculated [Nagy et al., 2008]. Systolic and diastolic blood pressures were recorded, using the average of two measurements that were taken within 15-minuts interval using standardised mercury sphygmomanometer. Subjects whose BP measurements were ≥130/85mmHg were classified as being hypertensive [NHBPEP Working Group, 2004].

Diagnostic criteria.

MS was diagnosed when three or more of the following criteria: abdominal obesity (WC≥88 cm), elevated FBG (≥ 100 mg/dl or diagnosed with diabetes, or taking an oral hypoglycaemic or insulin medication), reduced HDL-C level (≤40 mg/dl), elevated TG level (≥150mg/dl) and elevated BP (SBP≥130mmHg, DBP≥85mmHg or taking antihypertensive medication) [Zimmet et al., 2007].

Biochemical analyses.

Blood samples were drawn after fasting for more than 12-hrs and the serum was separated and frozen at-20°C. Glucose concentration was estimated using a commercially available glucose kit (Randox Laboratories Ltd., UK.) based on the glucose oxidase method. Serum TC, LDL-C, HDL-C and TG, creatinine, uric acid, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities were estimated calorimetrically using the available kits (cited in Randox Laboratories Ltd., UK). Serum 25(OH)D and insulin levels were measured by enzyme-linked immunosorbent assay (ELISA) (IDS, Tyne and Wear, UK). The inter- and intra-assay variabilities for 25(OH)D were 5.2% and 4.3%, respectively, and the inter- and intra-assay variabilities for insulin were 5.7%, and 3.1%, respectively. Insulin resistance was calculated using HOMA-IR (insulin [mU/L) ×glucose[mg/dl]/405) [Matthews et al., 1985]. Serum adiponectin and leptin were measured by ELISA. the intra-assay and inter-assay coefficients of variation for adiponectin was 5.4% and 8.5% respectively and intra-assay and inter-assay coefficients of variation for leptin was 7.4% and 9.3% respectively (Ani Biotech Oy, Orgenium Laboratories Division, Vantaa, Finland). The reading was taken using an ELISA microplate reader (VERSA Max, Molecular Devices Corporation, MN, USA). Vitamin D status was classified as follows: (i) deficient (vitamin D levels < 20 ng/ml), (ii) insufficient (vitamin D levels in the range 20 – 29 ng/ml), and (iii) sufficient (vitamin D levels >30 ng/ml) [Holick et al., 2005].

Statistical Analysis.

Data were expressed as mean ± standard deviation (SD) and data were analysed using SPSS version 12.0 software (SPSS, Chicago, III), using the appropriate statistical tests (chi-square test, Student's t-test, correlation coefficient, odds ratio). The P values were considered significant for $P < 0.05$.

RESULTS

Socio-demographic characteristics of the study group are described in Table 1. The mean age of the subjects was 20.9±3.2 years. The parents of the subjects had been educated to at least University standard in 66%(fathers) and36%(mothers) of the cases. Most fathers (65%) were employed whereas few mothers (29%) were employed. Family history of MS was 64%. The study showed 19% of the subjects were either overweight/obese (15% and 4%, respectively). The frequency of MS diagnosis of the subjects was17%, with 13% and 4% of the subjects having three or four risk factors, respectively (Table 1).

Overweight\obese subjects had statistically significant of anthropometric and biochemical parameters compared to normal weight subjects (Table 2). A higher levels of fasting blood glucose, insulin, HOMA-IR, leptin, leptin/adiponectin ratio, TG, LDL-C, W/H ratio, SBP and DBP in overweight/obese female students; but lower adiponectin and HDL-C levels. Also, overweight/obese subjects had significantly lower levels of 25(OH)D ($p<0.01$) relative to the normal weight.

Fifty-nine percent of the students that were diagnosed as having MS had mild 25(OH)D deficiency and 8.56% had severe 25(OH)D deficiency (Table 3).

Table 1. Socio-demographic characteristics and Metabolic risk factor Syndrome of the studied group.

	No (300)	%
Mean age (years)	20.9 ±2.1	
Range	18-25	
Father`s education		
Head formal education	198	66 %
No formal education	34%	34%
Mother`s education		
Formal education	108	36%
No formal education	192	64%
Father`s occupation		
Employed	195	65%
Un employed	105	35%
Mother's occupation		
Employed	87	29%
Un employed	213	71%
Family income		
Sufficient and saving	147	49%
Sufficient	132	44%
Insufficient	21	7%
Family history of metabolic syndrome	192	64%
Risk factor for MS	No (36)	
o/Risk factor	126	42%
+1/Risk factor	66	22%
+2/Risk factor	57	19%
+3/Risk factor	39	13%
+4/Risk factor	12%	4%
Total	300	100

Values are presented as age-adjusted mean + percentage (%) for Socio-demographic of all participants (N = 300) & metabolic (MS) risk factors in the studied group of total No of students =36

Table 2. Comparison of Anthropometric measurements, Blood pressure, Biochemical characteristics of the study groups

Parameters	Normal weight No:243 (mean ± SD)	Overweight/obese No:57 (mean ± SD)	
	Normal (18.5-24.9)	(overweight;25-29.9) (Obese; ≥30)	P value
Anthropometric measurements			
Waist circumference (cm)	67.2 ±0.6	91.81 ±7.1	< 0.05
Hip circumference (cm)	76.1±3.1	98.9±7.8	< 0.05
BMI (kg/m^2)	23.1 ±0.2	31.6 ±0.1	< 0.01
Blood pressure			
SBP (mm Hg)	118 ±5.3	133.7 ±7.2	< 0.01
DBP (mm Hg	69.8 ±9.3	83.8 ±3.4	< 0.01
Biochemical characteristics			
FBG (mg/dl)	86±0.4	130±1.1	< 0.01
TG (mg/dl)	99 ±0.2	159±3.1	< 0.01
HDL-C (mg/dl)	50 ±0.4	31±2.1	< 0.01
LDL-C (mg/dl)	76±0.1	138±2.4	< 0.01
25OHD (ng/ml)	36.1±2.7	12.7±0.3	< 0.01
HOMA-IR	1.4±1.5	15.4±3.61	< 0.01
Leptin (ng/ml)	5.3± 3.6	22.6 ± 6.8	< 0.01
Adiponectin (µg/ml)	16.8 ± 5.3	6.8 ± 3.3	< 0.01
Ratio Leptin/Adiponectin	0.42 ± 1.9	3.82± 2.1	< 0.01

Table 3 Comparison of Anthropometric Measurements, Blood pressure, Biochemical Characteristics of the Study Groups as Regard to Deficiency of 25-hydroxyvitamin D and with or without Metabolic Syndrome

Parameters	Normal weight No:243 (mean ± SD)	Overweight/obese No:57 (mean ± S.D)		without MS (mean ± S.D)	With MS (mean ± S.D)	
	Normal (18.5-24.9)	(overweight;25-29.9) (Obese; ≥30)	P value	No=218	No=82	P value
Anthropometric measurements						
Waist circumference (cm)	67.2 ±0.6	91.81 ±7.1	< 0.05	79 ± 3.1	99.7±4.7	< 0.05
Hip circumference (cm)	76.1±3.1	98.9±7.8	< 0.05	86.2 ± 5.1	101.5±3.8	< 0.05
BMI (kg/m^2)	23.1 ±0.2	31.6 ±0.1	< 0.01	20 ± 4.3	30±2.6	< 0.05
Blood pressure						
SBP (mm Hg)	118 ±5.3	133.7 ±7.2	< 0.01	119 ± 8.7	138.9 ±8.6	< 0.05
DBP (mm Hg)	69.8 ±9.3	83.8 ±3.4	< 0.01	70.9 ± 9.5	83.9 ±8.5	< 0.05
Biochemical characteristics						
FBG (mg/dl)	86±0.4	130±1.1	< 0.01	87 ± 0.6	129 ±9.4	< 0.05
TG (mg/dl)	99 ±0.2	159±3.1	< 0.01	88 ± 9.6	155±1.8	< 0.05
HDL-C (mg/dl)	50 ±0.4	31±2.1	< 0.01	50 ± 8.3	28±1.8	< 0.05
LDL-C (mg/dl)	76±0.1	138±2.4	< 0.01	79 ± 6.1	136±9.2	< 0.05
25OHD (ng/ml)	36.1±2.7	12.7±0.3	< 0.01	39.6 ± 4.4	11.3±5.4	< 0.01
HOMA-IR	1.4±1.5	15.4±3.61	< 0.01	1.2 ± 0.4	12.0±0.24	< 0.01
Leptin (ng/ml)	5.3± 3.6	22.6 ± 6.8	< 0.01	4.9 ± 2.6	28.3 ± 6.1	< 0.01
Adiponectin (µg/ml)	16.8 ± 5.3	6.8 ± 3.3	< 0.01	17.9 ± 4.3	5.9 ± 2.4	< 0.01
Ratio Leptin/Adiponectin	0.42 ± 1.9	3.82± 2.1	< 0.01	0.28 ± 1.3	5.1± 2.8	< 0.01

Values are presented as age-adjusted means ± standard deviation (SD), BMI-body mass index, SBP-systolic blood pressure, DBP-diastolic blood pressure FBG-fasting blood glucose, TG-triglycerides, HDL-C - high density lipoprotein cholesterol, LDL-C - low density lipoprotein cholesterol, 25(OH)D - 25-hydroxy vitamin D, HOMA-IR - insulin resistance index. significance at p<0.001

Table 4. Correlations of Serum 25OHD and Odd's Ratio (95%CI) with Metabolic Variables

Parameters	Serum 25(OH)D		Metabolic syndrome	
	Correlation coefficients (r)	P value	Odd's Ratio (95%CI)	P value
BMI (kg/m^2)	-0.694	< 0.01	5.92	< 0.01
Abdominal obesity	-0.653	< 0.01	5.26	< 0.01
FBG	-0.537	< 0.01	3.11	< 0.01
HOR	-0.312	< 0.01	3.22	< 0.01
TG	-0.322	< 0.05	3.01	< 0.05
HDL-C	0.395	< 0.01	3.42	< 0.01
LDL-C	-0.434	< 0.01	4.33	< 0.01
SBP	-0.301	< 0.05	4.11	< 0.01
DBP	-0.261	< 0.05	3.39	< 0.01
Leptin (ng/ml)	-0.431	< 0.01	4.26	< 0.01
Adiponectin (µg/ml)	-0.461	< 0.01	4.84	< 0.01
Leptin/Adiponectin	-0.411	< 0.01	4.73	< 0.01
Low 25(OH)D	___	___	2.87	< 0.01
Family History	___	___	2.87	< 0.05
Life style	___	___	2.01	< 0.05

Correlations of Serum 25OHD with Metabolic variable using ANOVA test. BMI-body mass index, FBG- fasting blood glucose, TG-triglycerides, HDL-C - high density lipoprotein cholesterol, LDL-C - low density lipoprotein cholesterol, 25(OH)D - 25-hydroxy vitamin D, HOMA-IR - insulin resistance index. **significance at p<0.001. *significance at p<0.05

Table 5. Comparison of Macronutrient Consumption Between Subjects with and Without Metabolic Syndrome

Macronutrients	Without MS (Mean ± SD)	With MS (Mean ± SD)	P value
Energy (Kcal/d)	1798 ± 361	1998 ± 689	< 0.05
Carbohydrate (g/d)	210 ± 44	298 ± 99	<0.01
Protein (g/d	69 ± 11.3	70 ± 12.1	
Fat (g/d)	70 ± 22.1	85 ± 11.5	<0.01
Saturated Fat (g/d)	19.2 ± 22.1	39.9 ± 8.9	< 0.01
Polyunsaturated Fat (omega 3/d)	15.8 ± 10.8	13.8 ± 6.5	
Monounsaturated Fat (g/d)	23.7 ± 12.2	20.8 ± 6.6	
Sugar (g/d)	68 ± 21.3	80± 97.8	< 0.05
Cholesterol (mg/d)	151 ± 314	187 ± 84	< 0.05
Dietary Fiber (g/d)	7.8 ±8.8	2.6 ±4.4	< 0.05

Comparison of Macronutrient Consumption Between Subjects with and Without Metabolic Syndrome. Values are presented as age-adjusted means ± SD participants (N = 300). **significance at p<0.001. *significance at p<0.05

Serum 25(OH)D levels were negatively associated with BMI, FBG, leptin, leptin/adiponectin ratio, LDL-C, TG and BP, and positively associated with adiponectin and HDL-C (P<0.01) (Table 4). In addition, MS had pronounced impact on elevating leptin and inhibition adiponectin levels that was additive to overweight/obesity. Otherwise, leptin/adiponectin ratio, that had postulated as a biomarker, was increased in subjects with MS than without.

Table (4) details the association between MS and different risk factors. It can be observed that the primary predisposing risk factor for MS was abdominal obesity (OR:5.92). The second

predisposing risk factor for MS was abdominal obesity, as indicated by WC (OR:5.26), followed by leptin (OR:4.84), Leptin/adiponectin ratio(OR:4.73), adiponectin (OP:4.26), hypertension (OR:4.11), low HDL-C (OR:3.42), FBG (OR:3.11) and vitamin 25(OH)D (OR:2.87).

The levels of carbohydrates and sugar consumption were significantly higher in subjects with MS compared to those without (Table 5). Consumption of dietary saturated fats were significantly more by subjects with MS than those without, but dietary fibre was consumed significantly less among subjects with MS than those without.

In overweight/obese, the daily dietary intake of vitamin D was 198±136.6 IU/day and in normal weight was 190±129.6 IU/day. The intake of vitamin D as a percental of DRI was 101.0±69.2% in overweight/obese subjects and 89.9±49.8% in normal subjects. Among the groups study, no statistically significant in vitamin D intake was showed either based on daily intake or on the percent of DRI.

DISCUSSION

Vitamin D has a vital role of public health and human well-beings and its deficiency has been linked to several metabolic disorders including Cancer, autoimmune diseases, obesity, type-2 diabetes, hypertension and cardiovascular disease and there is a relationship between low serum 25(OH)D, HOMA-IR, type-2 diabetes and low adiponectin in adults (Ashra ET AL., 2011). In the finding Leptin was increased and adiponectin decreased in overweight/obese female students relative to normal weight**.** There was a strong correlation between leptin with abdominal obesity and inverse to adiponectin. Furthermore, leptin, leptin-to-adiponectin ratio and adiponectin were strongly correlated with MS. These results had additive effects to overweight/obesity among female students. The most important outcomes are the combination of risk factors with increased leptin or lack of adiponectin that was found in overweight/obesity among female students. A cross-sectional study by (Jackson et al., 2016), conducted on adolescents aged 12–19years showed that abdominal obesity was the predominate risk factor for IR and there is an inverse correlation between adiponectin and obesity and IR which is linked to risk factors of cardiac disease.

The results of our study showed that low serum 25(OH)D associated with abdominal obesity, adiponectin, HOMA-IR as well as leptin/adiponectin ratio. Leptin and adiponectin have adverse effects on inflammation and insulin resistance, the high level of leptin increases the expression of pro-inflammatory and vasoconstrictive factors (Aleffi et al., 2005); while adiponectin stimulates anti-inflammatory cytokines production and enhanced the sensitivity of peripheral insulin (Ouchi and Walsh, 2007). Al-Daghri et al., [2013] observed that a positive correlation between BMI and; 25(OH)D and adiponectin in type-2 diabetes. The fact is that these adipokines has a potential role as a link between 25(OH)D and IR.

The current study showed 25(OH)D levels to be significantly lower in subjects who had been diagnosed with MS compared to those without MS. The finding also, observed that subjects who had been diagnosed with MS were mildly 25(OH)D deficient more frequently than those without. Additionally, vitamin D deficiency was more prevalent in obese subjects who had been diagnosed with MS (60.9%) compared to obese subjects without (33.3%). Previous studies have elucidated an inverse association between serum 25(OH)D levels and MS (Prasad et al., 2012). A cross-sectional study of 101 healthy subjects living in urban areas found a risk of MS increases three-fold in subjects with low levels of vitamin D (23.37% vs. 8.3% P<0.001) (Prasad et al., 2012).

Interestingly, after ameliorating lifestyle intervention in children obese /adults, adiponectin is the most predictive indicator for improving metabolic disorder, while changes in leptin were not related to positive metabolic outcomes (García-Hermoso et al., 2016). However, the clustering of many unfavorable biomarkers strongly requires early intervention not only in childhood obesity, but also in normal weight of subjects whose feature appear the risk.

The underlying mechanisms of the association between 25(OH)D status and dyslipidaemia are poorly understood. Previous studies have demonstrated that being either overweight/ obese was strongly associated with dyslipidaemia (elevated TG and lower HDL-C) in Asian-Indian adolescents living in urban area [Stewart et al., 2008]. In the third National Health and Nutrition Examination Survey [Stewart et al., 2008], adults whose 25(OH)D level was in the lowest quartile had the highest risk of elevated serum TG levels (≥150 mg/dl); this demonstrates that the health dangers of low vitamin D levels. Similarly, in obese subjects, 25(OH)D levels below 50 nmol/l were associated with lower HDL-C and high TG levels (Rammos et al., 2008). In

the present study, it was observed that the level of 25(OH)D was positively correlated with HDL-C (*P*< 0.01) and negatively correlated with TG levels and LDL-C(*P*<0.05). The female students in our study who had been diagnosed with MS had significantly elevated levels of TC, LDL-C, TG and BP, and significantly reduced levels of HDL-C compared to those without. Several epidemiological studies found that serum 25(OH)D levels to be negatively associated with BMI (Youssef et al., 2012; Mauss et al., 2015); excessive weight is a major component of MS, which may be attributed to lower 25(OH)D levels. However, in a cross-sectional study conducted on children, it was found that there is a negative correlation between 25(OH)D and BMI, FBG, LDL-C and TG, and positive correlation with HDL-C (*P*<0.01) (Mauss et al., 2015).

Insulin resistance is strongly associated with obesity and type-2 diabetes, hypertension, hyperlipidaemia and CVD [Peterson et al., 2014]. There is evidence from both clinical and non-clinical studies that glucose homeostasis might affect the level of vitamin D (Peterson et al., 2014). In pervious study that focused on pancreatic β-cells and the effect of vitamin D receptors, it was found that glucose intolerance, impaired synthesis and secretion of insulin, as well as an increased risk of type-2 diabetes, may be affected by insufficient 25(OH)D levels [Co et al., 2015]. Peterson et al., found that vitamin D deficiency is associated with different metabolic disorders, and the authors concluded that low levels of vitamin D is a risk factor for MS (Co et al., 2015). Our findings illustrated that significant increased odds ratio between high levels of TC, TG and LDL-C in subjects that had serum 25(OH)D levels below 20 ng/ml when compared to subjects that had normal serum 25(OH)D levels. The lower levels of 25(OH)D may be attributed to poor dietary intake of vitamin D as well as insufficient exposure to sunlight, despite the strong natural light of Saudi Arabia.

Considering the associations between MS and different predisposing factors, the finding found that obesity was the strongest predisposing risk factor for MS (OR:5.92). The second strongest predisposing risk factor for MS was abdominal obesity (OR:4.26). Unsurprisingly, WC, which is typically used as a surrogate measure of abdominal obesity, is the main criteria when determining MS risk. We also, found that the hypertension to be a strong risk factor for MS (OR:4.11). Previous studies of children living in urban areas have demonstrated that obesity is a major risk factor in the development of childhood hypertension (Sahoo et al., 2015). In addition, we found that lifestyle is a risk factor for MS, with OR:2.87. previous scientists indicated that regular physical activity and eating healthy diet have positive influence on health status, reducing the risk of obesity and thus reducing the risk of cardiac disease (Mauss et al., 2015). While elevated blood lipid levels increase the risk of hypertension, increasing physical activity lowers that risk; this is because exercise increases energy requirements and decreases fat deposition (Bombak, 2014). Skilton et al., 2008) found the prevalence of obesity to be increased by eating calorie dense food and failing to take adequate exercise While MS can be treated, in part, pharmacologically, the most important strategy for reducing the risk of MS and heart disease is to make lifestyle changes that promote physical activity and reduce excess weight.

An analyses of macronutrients showed that the consumption of carbohydrates and sugar were significantly higher in subjects with MS compared to those without, Whereas subjects with MS consumed less dietary fibre than without. Rapid changes in contemporary lifestyle habits can be seen in Saudi Arabia that are contributing to: (i) increased consumption of foods that are high in carbohydrates and sugars, and (ii) lack of adequate physical activity; the combined result of these lifestyle changes is an increase the prevalence of MS in the Saudi Arabian population (Al Junaibi et al., 2013).These results are consistent with studies that found sedentary lifestyle and poor dietary habits to be important predictors for development of MS (Al Junaibi et al., 2013).

CONCLUSION

In our study, it suggested that low level of adiponectin was strongly correlated with low 25(OH)D levels. Also, the prevalence of MS tends to increase with the high occurrence rate of low circulating 25(OH)D levels that is, known cause poor glycemic control. This relationship is unclear and may be a sign of future prediction of cardiovascular outcomes. Further studies are needed in the young population who are overweight/obese to assess the relationship between circulating adiponectin levels, low 25(OH)D and IR for adverse cardiovascular and metabolic outcomes. We suggest requiring strategies efforts to prevent the tendency of deficiency and should be improved in early and middle age by increasing outdoor physical activity

and by strengthening dietary vitamin D in the diet part of the Saudi diet.

CONFLICT OF INTEREST

The authors declared that present study was performed in absence of any conflict of interest.

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AUTHOR CONTRIBUTIONS

This work was carried out in collaboration between all authors. Author HMA suggested the idea, designed the study, wrote the protocol and wrote the first draft and final of the manuscript. Authors AFY performed statistical analysis and chair wrote the first draft of the manuscript. Authors AHA, OKR and MME supervised the study and managed the literature searches. Authors MAE, SBA, OKR and MME follow up the group study and collected the samples and performed the data analyses. All authors read and approved the final revision of manuscript.

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