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Serum 25(OH)D and adipokines levels in people with abdominal obesity

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ABSTRACT

Abdominal obesity is a risk factor for cardiovascular disease and diabetes mellitus and has been associated with vitamin D deficiency. Some studies have suggested an association between obesity and adipokine levels as well as low serum 25-hydroxyvitamin D (25(OH)D) level but the underlying mechanisms of the interlink between vitamin D status and serum leptin and adiponectin concentrations are still disputed.

We included 435 residents (132 males) from St. Petersburg, Russia into this study. All subjects had physical examination and demographics noted. Blood was collected after an overnight fast and plasma glucose, insulin, serum lipids, 25(OH)D and adipokines (adiponectin and leptin) concentrations were determined at baseline in all participants.

Abdominal obesity was diagnosed in 310 (71.3%) subjects (251 females and 59 males). Vitamin D insufficiency and deficiency were found in 314 (72.2%) subjects. Mean (95% CI) age, body mass index (BMI) and serum 25(OH)D for the cohort were 47.6 ± 11.3 years; 28.7 ± 0.2 kg/m^2 and 62.5 ± 24.3 nmol/l respectively. Serum 25(OH)D level inversely correlated with body weight, waist circumference (WC) and BMI in females but not in males, was lower in diabetic than non-diabetic subjects, and was not significantly different in subjects with and without MetS. WC was positively correlated with leptin and negatively correlated with adiponectin. We found correlation between leptin and serum 25(OH)D level ($r = -0.15$, $p = 0.01$) but this finding was a characteristic seen only in women.

Our study showed a high prevalence of abdominal obesity, vitamin D deficiency and insufficiency in residents from North-West region of Russia, close association between adipokine (leptin, adiponectin) concentrations as well as vitamin D status and body composition (WC, BMI). However in our study the interlink between leptin level and 25(OH)D was found only in females. Further investigations are required to study the relationship between serum 25(OH)D level, obesity and serum adipokine levels.

Abbreviations: 25(OH)D, 25-hydroxyvitamin D; WC, waist circumference; BMI, body mass index; FPG, fasting plasma glucose; HOMA-IR, homeostasis model assessment estimates of insulin resistance; HDL, high density lipoprotein; LDL, low density lipoprotein; BP, blood pressure; DXA, dual-energy X-ray absorptiometry; MRI, magnetic resonance imaging.

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1. Introduction

It is well known that obesity, and in particular abdominal obesity, is one of the major risk factors for cardiovascular disease (CVD) and its complications [1,2]. Obesity prevalence has reached epidemic proportions and according to the World Health Organization data the number of overweight individuals is continuously increasing [3]. Recent evidence has shown that vitamin D deficiency has been associated with obesity and other components of the metabolic syndrome (impaired glucose metabolism, dyslipidemia, hypertension) [4,5] and that obese patients often have low 25-hydroxyvitamin D levels [6–10]. On the other hand vitamin D deficiency is supposed to contribute to fat accumulation [11–15]. Adipose tissue is now considered to be an active endocrine organ releasing a variety of adipokines, including serum leptin and adiponectin [16–19]. It is known that adipokine imbalance is associated with the metabolic syndrome (MetS). In a number of studies, it has been shown that elevated leptin can potentiate insulin resistance and arterial hypertension; and activated pro-inflammatory factors; while adiponectin, on the contrary, possesses cardioprotective effects [12,16,18]. In addition, leptin could regulate several stages of vitamin D synthesis to negatively affect the activity of 1-alpha-hydroxylase in the kidneys and peripheral tissues, including adipose tissue, resulting in reduced concentration of 1,25-dihydroxyvitamin D that is an active metabolite of vitamin D [20–23]. Despite the association of vitamin D deficiency and adipokine imbalance with obesity, a relationship between low 25-hydroxyvitamin D and adipokine concentrations is still to be elucidated.

The aim of this study was to investigate the association between serum 25(OH)D and adipokine concentrations in subjects with abdominal obesity compared to those of normal body weight.

2. Materials and methods

2.1. Study population

We examined 435 (132 males and 303 females) residents from North-West region of Russia in a cross-sectional study. The subjects were recruited during visits to outpatient clinics with relatives or for minor intercurrent illness. Exclusion criteria were the following: clinically significant kidney and gastrointestinal diseases, medical history of diabetes mellitus, regular insolation (every week) or intake of vitamin D supplements. All participants gave written informed consent. The study was approved by the local research ethics committee.

2.2. Data collection

All study participants underwent anthropometric measurements (waist circumference, WC; height and weight; body mass index, BMI). Measurements were performed in the morning with the participant dressed in light clothing, without shoes. The WC was measured with the patient standing and at the midpoint between the lower rib margin and the iliac crest parallel to the floor. Blood pressure was measured in the right arm in the sitting position after resting for 10 min.

Blood samples were taken from all study participants and serum aliquots were stored at −70 °C. Serum 25-hydroxyvitamin D (25(OH)D) was analyzed using a chemiluminescent immunoassay (Abbott Architect, Deerfield, IL, USA); intra-assay CV ranged from 1.6 to 5.9% whereas the inter-assay CV ranged from 2.2 to 2.6%.

Adiponectin and leptin levels were measured by ELISA with the use of ImmunoChem-2100 device (DRG Diagnostics, Marburg, Germany).

Fasting plasma glucose (FPG) was determined enzymatically using commercially available kits and auto analyzer (UniCel DxC 800, Brea, CA, USA).

Serum insulin was measured using enzyme immunoassay kits (Beckman Coulter, Brea, CA, USA). Homeostasis model assessment estimates of insulin resistance (HOMA-IR) was calculated using fasting glucose and insulin measurements [23]. Serum lipid profile (total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL) and low-density lipoprotein cholesterol (LDL)) were measured by enzymatic colorimetric assays with the analyzer COBAS INTEGRA 400/700/800 and standard kits (Roche Diagnostics, Mannheim, Germany).

2.3. Data analysis

Abdominal obesity was diagnosed according to the Guidelines of International Diabetes Federation (IDF) if WC was ≥80 cm for females and ≥94 cm for males [24]. The body mass index was considered normal if BMI was 18.5–24.9 kg/m^2, overweight – if BMI was 25.0–29.9 kg/m^2; obesity was diagnosed when BMI was ≥30.0 kg/m^2. Metabolic syndrome (MetS) was diagnosed according to the IDF criteria [24].

Vitamin D status was classified according to the Endocrine Society criteria [25] which are also used by the Russian Association of Endocrinologists and Central European countries [26]. Vitamin D deficiency was diagnosed as serum 25(OH)D level <50 nmol/l, insufficiency 50–75 nmol/l and sufficiency ≥75 nmol/L.

Biomedical research data was processed by SPSS software 17.0 for Windows (SPSS Inc, Chicago, Ill). Sample characteristics were presented as a percentage or mean ± SD. Comparison of frequency characteristics of qualitative indicators was done by nonparametric techniques using chi square (c2). Comparison of quantity indicators was performed using ANOVA. To find the correlation between the studied indicators we applied Pearson correlation analysis.

3. Results

3.1. Anthropometric and biochemical characteristics of studied group

Of the 435 participants, there were 303 (69.6%) females and 132 (30.4%) males of comparable age (49 ± 10 yrs and 47 ± 13 yrs, respectively; p > 0.05). Mean WC was 94 ± 25 cm for women and 94 ± 12 cm for men. Depending on the WC, subjects were divided into two groups: those with abdominal obesity (306 subjects, 55 males) and those without (129 subjects, 77 males).

One hundred and thirty five subjects (41 males and 94 females) had normal BMI, 160 (63 males and 97 females) were overweight and 140 (28 males and 112 females) were obese. The basic characteristics of participants are shown in Table 1.

Prevalence of abdominal obesity was higher in women than in men ($p < 0.001$), but the incidence of overweight and obesity was not different in both sexes ($p > 0.05$). The FPG ($p < 0.001$), leptin ($p < 0.001$), adiponectin ($p < 0.001$) and HDL ($p < 0.001$) levels were higher in women. There was no difference in insulin, HOMA-IR, total cholesterol, and LDL levels between sexes ($p > 0.05$). Both, systolic and diastolic BP was higher in males than in females ($p = 0.02$ and $p = 0.001$, respectively).

WC positively correlated with leptin ($r = 0.32$, $p = 0.02$), TG ($r = 0.39$, $p = 0.03$), FPG ($r = 0.28$, $p = 0.04$), insulin ($r = 0.42$, $p = 0.03$) levels and HOMA-IR value ($r = 0.40$, $p = 0.02$) and negatively correlated with adiponectin ($r = -0.25$, $p = 0.03$) and HDL ($r = 0.25$, $p = 0.03$) concentrations independently of gender.

We also found that in abdominal obese cohort there was a positive correlation between leptin and TG level ($r = 0.26$, $p = 0.04$).

Table 1
Characteristics of subjects.

	Males n = 132	Females n = 303	P-value
Age, years	47 ± 13	49 ± 10	NS
WC, cm	94 ± 12	94 ± 25	NS
Abdominal obesity (%)	60 (45)	251 (83)	0.001
Normal (%)	72 (55)	52 (17)	
BMI, kg/m^2	27.0 ± 4.1	28.2 ± 5.6	0.04
Normal weight n (%)	41 (31)	94 (31)	
Overweight n (%)	63 (48)	97 (32)	
Obese n (%)	28 (21)	112 (37)	
25(OH)D, nmol/l	68.4 ± 22.5	60.0 ± 24.6	<0.001
Deficient n (%)	30 (23)	121 (40)	
Insufficient n (%)	54 (41)	106 (35)	
Adequate n (%)	48 (36)	76 (25)	
Adiponectin, μg/ml	7.6 ± 5.7	15.8 ± 11.4	<0.001
Leptin, ng/ml	6.5 ± 7.8	30.6 ± 25.8	<0.001
FPG, mmol/l	5.0 ± 0.9	5.3 ± 1.4	<0.001
Insulin, mIU/l	13.7 ± 16.1	10.6 ± 7.8	NS
HOMA-IR	3.2 ± 4.0	2.7 ± 2.3	NS
Total cholesterol, mmol/l	5.0 ± 1.0	5.2 ± 1.1	NS
Triglyceride, mmol/l	1.3 ± 0.6	1.2 ± 0.7	NS
HDL, mmol/l	1.2 ± 0.3	1.4 ± 0.4	<0.001
LDL, mmol/l	3.2 ± 0.9	3.2 ± 0.9	NS
BP, mm Hg			
systolic	130 ± 20	126 ± 21	0.02
diastolic	81 ± 10	78 ± 13	0.001

Values are shown as n, means ± SE or percentages.
WC: waist circumference; BMI: body mass index; FPG: fasting plasma glucose; HOMA-IR: homeostasis model assessment estimates of insulin resistance; HDL: high density lipoprotein; LDL: low density lipoprotein; BP: blood pressure.

3.2. 25(OH)D levels and its relation to anthropometric and biochemical characteristics of studied group

One hundred and twenty-one (27.8%) participants had adequate vitamin D status, 32.0% had vitamin D insufficiency, and 40.2% had vitamin D deficiency. Serum 25(OH)D concentration was significantly higher in men than in women ($p < 0.001$). Women with abdominal obesity had lower serum 25(OH)D than women with normal WC ($p = 0.02$), however, the aforementioned observation was not evident in men (Fig. 1, Table 2).

Table 2
Characteristics of subjects with abdominal obesity by gender.

	Males n = 55	Females n = 251	P-value
Age, years	50 ± 11	50 ± 10	NS
WC, cm	104 ± 8	97 ± 12	<0.001
BMI, kg/m^2	30.1 ± 3.3	29.4 ± 5.1	NS
25 (OH)D, nmol/l	70.8 ± 22.1	57.7 ± 22.6	<0.001
Adiponectin, μg/ml	6.1 ± 5.3	15.4 ± 11.8	<0.001
Leptin, ng/ml	10.1 ± 9.8	33.3 ± 25.1	<0.001
FPG, mmol/l	5.3 ± 1.1	5.4 ± 1.4	NS
Insulin, mIU/l	17.4 ± 18.1	11.6 ± 7.9	0.002
HOMA-IR	4.2 ± 4.6	2.9 ± 2.4	0.02
Total cholesterol, mmol/l	5.1 ± 1.0	5.3 ± 1.1	NS
Triglyceride, mmol/l	1.6 ± 0.7	1.3 ± 0.6	0.002
HDL, mmol/l	1.2 ± 0.2	1.3 ± 0.3	<0.001
LDL, mmol/l	3.2 ± 0.9	3.3 ± 0.9	NS
BP, mm Hg			
Systolic	137 ± 20	128 ± 20	<0.001
Diastolic	85 ± 10	78 ± 10	<0.001

Values are shown as means ± SE or percentages.
WC: waist circumference; BMI: body mass index; 25(OH)D: 25-hydroxyvitamin D; FPG: fasting plasma glucose; HOMA-IR: homeostasis model assessment estimates of insulin resistance; HDL: high density lipoprotein; LDL: low density lipoprotein; BP: blood pressure.

When gender-related differences were analyzed in abdominal obese individuals, a higher adiponectin, leptin and HDL levels and a lower insulin, HOMA-IR and TG levels as well as lower systolic and diastolic BP values were noted in females compared to males (Table 3). Further, females with abdominal obesity had significantly lower 25(OH)D levels of 57.7 ± 22.6 nmol/l, compared to70.8 ± 22.1 nmol/l ($p < 0.001$) in male subjects. Moreover, vitamin D deficiency appeared significantly more prevalent in women with abdominal obesity compared to women with normal BMI and WC values ($\chi 2 = 6.8$, $p < 0.01$). Similar observation was not observed in males.

Correlation coefficients between serum 25(OH)D levels and anthropometric and biochemical parameters investigated in this study are shown in Table 3.

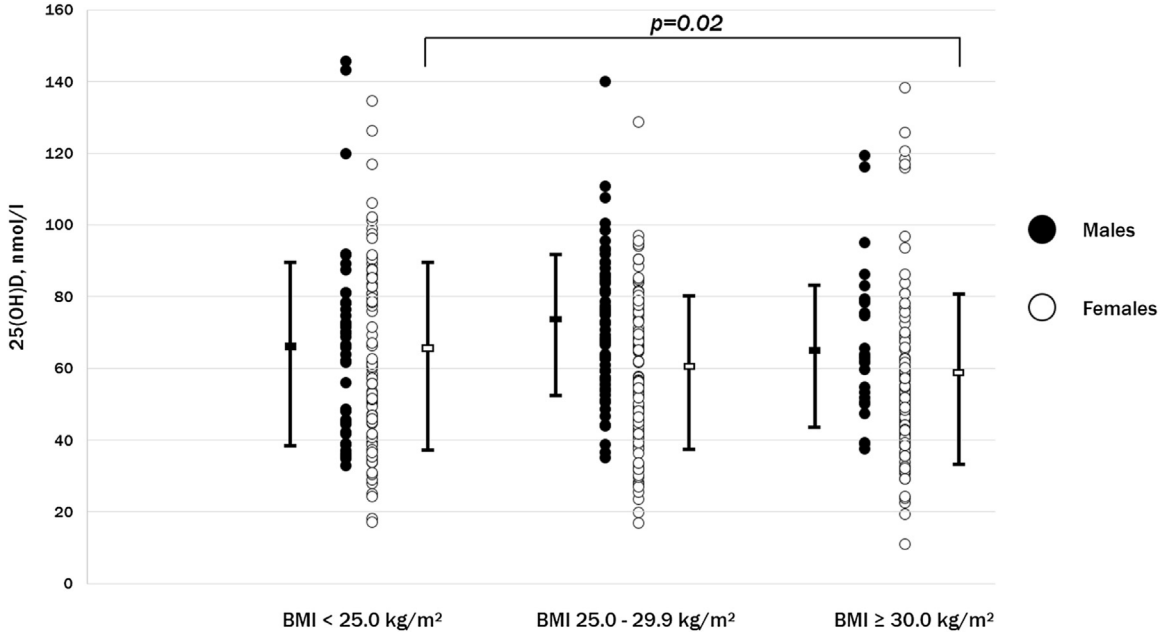

Fig. 1. Serum 25(OH)D level according to BMI in male and female cohorts. 25(OH)D: 25-hydroxyvitamin D; BMI: body mass index.

BMI and WC negatively correlated with serum 25(OH)D levels only in women ($r = -0.15$, $p = 0.02$ and $r = -0.20$, $p = 0.001$ respectively), and not in men. Serum leptin ($r = -0.15$, $p = 0.01$) as well as FPG ($r = -0.15$, $p = 0.02$), insulin ($r = -0.19$, $p = 0.002$), HOMA-IR ($r = -0.18$, $p = 0.004$) and TG ($r = -0.19$, $p = 0.002$) were correlated with serum 25(OH)D in female cohort. We did not find correlations between 25(OH)D concentration and serum adiponectin in both sexes.

In subjects with abdominal obesity we found negative correlation between 25(OH)D and leptin ($r = -0.17$, $p = 0.04$), TG ($r = -0.14$, $p = 0.002$) and BMI ($r = -0.11$, $p = 0.07$).

Regression analysis showed association between 25(OH)D concentration and WC ($R^2 = -0.15$, $p = 0.04$), insulin level ($R^2 = -0.15$, $p = 0.03$) and triglycerides ($R^2 = -0.14$, $p = 0.05$) only in women with abdominal obesity. Moreover, in subjects with vitamin D insufficiency there was a negative association between 25(OH)D and FPG ($R^2 = -0.16$, $p = 0.04$).

3.3. 25(OH)D levels in patients with type 2 diabetes mellitus

Type 2 diabetes mellitus was newly diagnosed in 35 abdominal obese subjects. The diagnosis was based on FPG level greater than 7.0 mmol/l. In the newly diagnosed subgroup of patients with type 2 diabetes mellitus serum 25(OH)D appeared significantly lower compared to values observed in subjects with normal FPG (54.3 ± 21.3 nmol/l vs 63.3 ± 24.4 nmol/l, respectively, $p = 0.04$), and did not differ from values noted in participants with abdominal obesity (60.9 ± 23.3 nmol/l, $p > 0.05$).

3.4. 25(OH)D levels in patients with metabolic syndrome

According to IDF criteria, metabolic syndrome (MetS) was diagnosed in 110 subjects with abdominal obesity (92 females; 83.6%). Anthropometric and biochemical characteristics of subgroup with newly diagnosed MetS are shown in Table 4. Prevalence of MetS was not different in female (30.4%) and male population (13.6%) ($\chi^2 = 13.6$, $p > 0.05$).

Irrespective of clinical manifestation of MetS, both adiponectin and leptin levels were higher in females than in males and in the MetS subgroup (Table 4). Serum 25(OH)D was not different in subjects with MetS compared to those without MetS. However, within the MetS group serum 25(OH)D was significantly higher in males ($p = 0.001$). A similar trend was also observed in subjects without MetS ($p = 0.07$). Correlation analysis showed a negative coefficient value for relationship between 25(OH)D and FPG ($r = -0.31$, $p = 0.03$), but none between 25(OH)D and adipokine levels irrespective of gender.

Table 3
Pearson's correlation analyzes between serum 25(OH)D and anthropometric and biochemical parameters in studied group (n = 435).

Parameters	Males (n = 132)		Females (n = 303)	
	R	P-value	R	P-value
Age	0.10	0.26	0.07	0.28
Weight	0.10	0.27	−0.17	<0.01
BMI	0.06	0.47	−0.15	0.02
WC	0.11	0.21	−0.20	0.001
Adiponectin	0.04	0.69	0.01	0.84
Leptin	0.02	0.86	−0.15	0.01
FPG	−0.02	0.83	−0.15	0.02
Insulin	−0.00	0.96	−0.19	<0.01
HOMA-IR	−0.04	0.68	−0.18	<0.01
Total cholesterol	0.11	0.20	−0.09	0.38
Triglyceride	−0.10	0.24	−0.19	<0.01
HDL	0.03	0.74	0.01	0.94
LDL	0.13	0.14	−0.08	0.18
BP				
Systolic	0.04	0.67	0.01	0.91
Diastolic	0.15	0.10	0.01	0.85

NS: non significant; WC: waist circumference; BMI: body mass index; FPG: fasting plasma glucose; HOMA-IR: homeostasis model assessment estimates of insulin resistance; HDL: high density lipoprotein; LDL: low density lipoprotein; BP: blood pressure.

4. Discussion

This study, like our other previous studies, found high prevalence of abdominal obesity in the population of the North-West region of Russia [8] and an association between serum adipokines (leptin, adiponectin) with BMI and WC [27]. It is known that adipokine imbalance in abdominal obese subjects is linked with impaired glucose metabolism, dyslipidemia and hypertension development [28]. Some population studies indicate a relationship between low serum adiponectin level and insulin resistance in patients with type 2 diabetes [29] as well as between serum leptin and BMI [30]. Interestingly, numerous studies have shown that adipokine levels could be sex-dependent [27,31]. In our study, both leptin and adiponectin levels correlated with BMI and WC independently of gender, and were higher in women than in men. In addition, we confirmed negative association between serum leptin and TG in obese subjects.

It is now an accepted fact that obese humans generally have low vitamin D status [32]. Several pathways link obesity and vitamin D deficiency [6,33,34]. Firstly, obese people with sedentary lifestyle have limited exposure to sunlight and even under ultraviolet radiation they seem to have decreased synthesis of pre-vitamin D in the skin [6]. On the other hand, many researchers agree that the accumulation and storage of 25(OH)D in adipose tissue lead to a decrease of circulating 25(OH)D level and its enhanced catabolism in the adipose tissue converting 25(OH)D to 24,25-dihydroxyvitamin D, the biologically inactive metabolite [33,34]. Furthermore, vitamin D deficiency is likely to contribute to the development of obesity, based on the expression of vitamin D receptors in adipocytes [8,11–13,21,22]. Interestingly, Drincic et al. showed that not only high fat mass but even total body size could be linked with low 25(OH)D level [35].

In comparison to other Central European studies on general population in latitudes similar to that of St. Petersburg (59^0 North latitude), our cohort presented mean 25(OH)D levels (62.5 ± 24.3 nmol/l) within the same prevalence of vitamin D deficiency and insufficiency (72.2%) [36]. We found that serum 25(OH)D level was lower in women than in men and was negatively associated with body composition (weight, BMI and WC), an association not seen in the male cohort. Some information is available from studies regarding serum 25(OH)D concentrations in men and women. A cohort study from the UK found that women had statistically higher concentrations than men in winter, while men had statistically higher concentrations in summer [37]. In the author's opinion those differences might be the results of men spending more time outdoors and women taking more oral vitamin D. Upon regression analysis, in this study serum 25(OH)D level was inversely associated with WC in obese women studied but was not associated with BMI. In our opinion the observed difference between vitamin D status in men and women and absence of the association between the body composition and 25(OH)D level among the male subjects might be explained mainly by the lower frequency of obesity cases in this group and generally low number of male subjects within the study. Our findings are consistent with previous studies reporting an inverse relationship between vitamin D levels and fat mass quantity [4,5,8,38,39].

Recent studies showed that adipokine imbalance could be associated with low serum 25(OH)D [31,40–47]. Large population studies indicated a relationship between serum 25(OH)D level and

Table 4
Characteristics of subjects with and without metabolic syndrome.

Parameters	With MetS n = 110 (M/F, 18/92)	Without MetS n = 325 (M/F, 114/211)	P-value
Age, years	51 ± 10	46 ± 12	<0.001
WC, cm	102 ± 11	87 ± 12	<0.001
Males	103 ± 11	87 ± 11	<0.001
Females	101 ± 11	88 ± 12	<0.001
BMI, kg/m^2	28.2 ± 5.2	27.4 ± 5.1	NS
25(OH)D, nmol/l	60.5 ± 25.9	64.3 ± 23.8	0.08
Males	74.2 ± 19.2	67.5 ± 23.2	NS
Females	57.9 ± 26.3	62.5 ± 24.0	0.06
Adiponectin, μg/ml	15.2 ± 14.4	12.3 ± 8.9	0.04
Males	5.7 ± 2.2	7.7 ± 5.4	NS
Females	17.2 ± 15.0	15.1 ± 9.5	NS
Leptin, ng/ml	25.8 ± 24.3	20.3 ± 22.4	<0.01
Males	6.6 ± 7.1	5.9 ± 6.0	NS
Females	29.6 ± 24.7	28.7 ± 24.1	NS
FPG, mmol/l	5.4 ± 0.7	4.8 ± 0.7	<0.001
Insulin, mIU/l	11.4 ± 9.5	10.8 ± 10.9	NS
HOMA-IR	2.8 ± 2.4	2.3 ± 2.4	0.004
Total cholesterol, mmol/l	5.3 ± 1.0	5.1 ± 1.1	NS
Triglyceride, mmol/l	1.8 ± 0.8	1.0 ± 0.5	<0.001
HDL, mmol/l	1.2 ± 0.3	1.4 ± 0.4	<0.001
LDL, mmol/l	3.3 ± 0.9	3.1 ± 0.9	NS
BP, mm Hg			
Systolic:	139 ± 19	122 ± 19	<0.001
Diastolic:	85 ± 13	76 ± 11	<0.001

Values are shown as means ± SE or percentages.
WC: waist circumference; BMI: body mass index; FPG: fasting plasma glucose; HOMA-IR: homeostasis model assessment estimates of insulin resistance; HDL: high density lipoprotein; LDL: low density lipoprotein; BP: blood pressure.

adiponectin and leptin concentrations [41–45]. On the other hand smaller studies reported a negative correlation with leptin, no relationship to adiponectin and resistin in healthy population, and no significant relationship in morbidly obese subjects as well as in children with vitamin D deficiency rickets [46,47]. While, one study from Saudi Arabia observed positive correlation between 25-hydroxyvitamin D level and leptin but only in males and did not find such an association in females [48]. Reasons for discordance in clinical data remain unclear. However some researchers are still discussing the role of vitamin D in the regulation of leptin synthesis [20–22]. Our study found a negative correlation between serum 25 (OH)D level and leptin concentration and did not suggest any association with adiponectin. Our data therefore agrees with that from other studies; for example in a study performed in the Slovak adult population' adipokine levels were significantly altered in the presence of cardiometabolic risk factors, but was not associated with vitamin D status [49].

Mechanisms of how vitamin D affects adipokines concentration are not clear. Several potential effects of vitamin D on adipokines are being discussed. Firstly, through an increase of parathyroid hormone, low 25-hydroxyvitamin D could stimulate lipogenesis, obesity and insulin resistance, the conditions that are associated with increased activity of proinflammatory cytokines (TNF-α and interleukin-1), and could be associated with adipokines disbalance [49]. Secondly, with the presence of vitamin D receptors in adipocytes, vitamin D may play a role in the regulation of adipokine gene expression in visceral adipose tissue and vitamin D deficiency may be associated with increased serum leptin and decreased adiponectin level [49]. Thirdly, vitamin D may affect adiponectin through the renin-angiotensinogen system, where an increase in angiotensin production leads to dysfunctional adipocytes and decreased adiponectin production [44]. And finally, vitamin D deficiency, can result in the up-regulation of osteocalcin, which may increase adiponectin gene expression in adipocytes [50]. We would like to add that the results of our research and the data from other studies [27,31,46] suggest the potential existence of gender-dependent factors influencing the relationship between vitamin D and serum adipokine concentrations.

This study has some limitations. First, this was a cross-sectional and not a prospective study with vitamin D supplements treatment. Secondly, we did not include instrumental methods (DXA, MRI) to assess fat and lean mass quantity. Finally, we should note that measuring only total adiponectin level, not the active high-molecular weight adiponectin, could also be a limiting factor in our study. However, this is the first study to suggest a link between vitamin D status and adipokines concentrations in subjects with and without abdominal obesity in the Russian population.

5. Conclusion

We demonstrated the presence of high prevalence of abdominal obesity and vitamin D deficiency in both female and male cohorts in the Russian population. High leptin and low adiponectin levels were associated with abdominal obesity. Serum 25(OH)D level was inversely associated with WC and BMI as well as leptin level but only in female subjects. The modulatory impact of 25(OH)D on

adipokines definitely requires further studies, as it might be disparate in apparent health and under disease-imposed burden.

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