International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2020): 7.803

Level of 25 (OH) Vitamin D Lower in Central Obesity Women Compared to Normal Weight Women and its Correlation with Blood Pressure

Linda Sinto[1], Wimpie Pangkahila[2], Wayan Weta[2]

[1]Postgraduate Student Biomedical Science, Anti Aging Medicine Concentration

[2, 3]Lecturer at Biomedical Science, Anti Aging Medicine Concentration

Abstract: ***Obesity is one of the biggest health challenges of this century. Central obesity cause chronic low inflammation. Vitamin D has a role in reducing inflammation. Central obesity people need higher Vitamin D, but Vitamin D utility was found lower in central obesity people. This may because the amount of Vitamin D is trapped in fat. This study was a cross sectional study compare within central obesity and normal weight woman. The sample was taken by consecutive sampling method. The subjects were 16 obese central women and 16 normal weight women. Demographic data, sun exposure and food intake containing vitamin D were obtained from interviews. Than blood pressure checked and blood sampling were taken to check fasting blood sugar levels, HDL, triglycerides, 25(OH) Vitamin D. Results showed that median level of 25 (OH) Vitamin D in the obese women group was lower than in normal weight women 7,84(7,12:10,70) VS 15,20 (10, 42: 19, 47); p=0,003. The median sun exposure in the group of central obese women was lower than women of normal weight 2, 05 (0: 5, 50) VS 17, 50 (6, 25: 25, 50); p=0,001. While food intake containing Vitamin D does not have a significant comparison. The correlation test results between 25(OH) Vitamin D levels and metabolic indicators showed no correlation in the group of normal weight women. Whereas in the group of central obese women, levels of 25 (OH) Vitamin D had a correlation with systolic, diastolic blood pressure and MAP (p=0,001, p=0.006, p=0,003, respectively) but did not have a correlation with fasting blood sugar, HDL and triglycerides. Conclusion of this study is 25 (OH) Vitamin D levels in central obese women group proved to be lower than normal weight women and had a negative correlation with blood pressure in central obese women group.***

Keywords: Vitamin D, obesity, metabolic syndrome, inflammation

1. Introduction

Aging process is a process that cannot be avoided and must be experienced by every individual, which results in gradual changes in various cells or organs of the body[1].Many factors cause people to experience the aging process faster than they should like a lifestyle. Changes in diet, namely saturated fat intake increases, while physical activity decreases. This condition will cause excessive fat accumulation in body tissues called obesity[2].

Obesity is one of the major health challenges of this century. One of the health problems experienced by obese people is vitamin D deficiency which later becomes a high risk of the occurrence of the metabolic syndrome. Bioavailability of vitamin D in obese group is low due to deposition in fat tissue. Increased fat tissue in obesity will increase leptin secretion which in turn stimulates Fibroblast Growth Factor 23 (FGF-23). FGF-23 will inhibit the synthesis of 1-alpha-hydroxylase enzyme which results in a disruption of 1, 25 (OH) D3 production[3].

In adipose cells, insulin resistence causes insulin failure in suppressing lipolysis, causing an increase in free fatty acids from adipocytes to other tissues. In the muscle, an increase in free fatty acids causes a disruption of glucose uptake in cells. This results disruption of glucose tolerance throughout the body. In the liver, increase in free fatty acids causes increase in triglyceride synthesis and liver steatosis. Pancreatic β cells will compensate for insulin resistance by increasing the production of insulin resulting in a hyperinsulinemia state. This condition will stimulate the occurrence of lipogenesis in the liver and cause more free fatty acids to accumulate in the liver for the production of triglycerides[4].

Special characteristics of metabolic syndrome are low-grade (sub-inflammatory) inflammation that occurs chronically with increased levels of TNF-α and IL-6 by adipose tissue and a decrease in adiponectin.

Molecular factors that recognized to play a role in the metabolic sub-inflammation are:

1) Endoplasmic Reticulum Stress (ERS). In conditions of excess lipids, the endoplasmic reticulum is unable to synthesize proteins properly.
2) Activation of Toll-Like Receptor (TLR4). Long chain fatty acids can activate TLR4 and cause insulin resistance and transcription of inflammatory genes.
3) Stimulation of Protein Kinase R (PKR). Excess fat stimulates PKR, induces an inflammatory signaling process (through c-Jun terminal kinase / JNK and IkB kinase/ IKK).

These three pathways have a negative effect on the insulin signaling pathway. A high-fat diet also causes changes in the intestinal microbiota, which through the process of TLR4 activation and changes from ERS result in metabolic inflammation[5].

In activated immune cells, 1α hydroxylase expression occurs so that it can activate 25 (OH) D3 into the active form of 1, 25 (OH) 2D3. Vitamin D is found to be anti-adipogenic, has an effect as an immunoregulator and can reduce inflammatory conditions in adipose tissue[3].

Positive relationship between low levels of 25 (OH) D and low HDL levels is probably caused by the role of vitamin D in maintaining adequate concentrations of apolipoprotein A-1, which is the main component of HDL, and regulation of lipase lipoprotein enzyme activity affecting blood triglyceride levels. 25(OH) D also plays a role in regulation of adipocyte intracellular calcium in the process of lipogenesis in adipose tissue. Thus the lack of vitamin D can reduce calcium absorption in adipose tissue, so that lipolysis and free fatty acids occur, the synthesis of VLDL and LDL liver also increases[6].

Mechanism that connects Vitamin D with hypertension is through a negative regulator from the Renin Angiotensin System (RAS). RAS has a role in the risk of hypertension and cardiovascular disease. Vitamin D also plays a role in vascular endothelial function or intracellular calcium muscle blood vessel concentration[7].

The mechanism of the influence of vitamin D on Diabetes Mellitus is due to the presence of vitamin D receptors in the pancreatic beta cells and serves to express enzymes 1α hydroxylase[8]. In addition, Vitamin D plays a role in the regulation of PPAR (Peroxisome Proliferative Activated Receptor), transcription factors involved in metabolism and fatty acid mobilization in adipose tissue and skeletal muscles[5,9]. Some of these mechanisms have a positive effect on insulin secretion and insulin sensitivity[10]. At the molecular level, vitamin D is useful in reducing oxidative stress levels and is anti-inflammatory[8,9].

This mechanism can explain why lower of 25(OH) Vitamin D have a correlation with incidence of metabolic syndrome.

2. Method

This study was a comparative study with an observational study design (cross sectional) with sampling using the consecutive sampling method. Subjects consisted of 32 women with 35-45 years of age and categorized into 2 groups: central obese women (BMI> 25 and abdominal circumference ≥80cm) and groups of normal weight women (BMI 18.5-25 and abdominal circumference < 80cm). The research subjects were not currently in the treatment of obesity, hypertension, diabetes mellitus, dyslipidemia. Menstrual status is still regular, not on hormones therapy or multivitamins that contains Vitamin D. After being categorized into 2 group's anamnesa is carried out regarding daily exposure to the sun and food sources of Vitamin D which are commonly consumed by daily patients. Then a blood pressure checked and blood sampling were taken to check fasting blood sugar levels, HDL, triglycerides, 25 (OH) Vitamin D.

3. Result

Subjects were 100% with levels of 25 (OH) Vitamin D <30ng/mL.

Table 1 describes the average daily source of Vitamin D in the two study groups assessed through sun exposure and food sources assessed by using the Food Frequency Questionnaire. From these results it can be seen that sun exposure in the central obesity group of women is lower than normal weight women, but there are no significant differences in food ingredients in both the central obese and normal body weight groups.

Table 2 shows the median levels of 25 (OH) Vitamin D, systolic and diastolic blood pressure, and triglycerides from each research group. Then a comparative test was performed using the Mann Whitney test. The difference was significant in the variables 25 (OH) Vitamin D (p = 0.003) and systolic blood pressure variables (p = 0.005), diastolic blood pressure (p = 0.001).

Table 3 illustrates the results of the Spearmann correlation test between levels of 25 (OH) Vitamin D with blood pressure, fasting blood sugar, HDL and triglycerides in the two study groups. The test results depicting 25 (OH) Vitamin D levels had a negative correlation with systolic blood pressure variables (p = 0.001) and diastolic (p = 0.006) in the group of central obese women.

Table 1: Comparability test of sun exposure, Vitamin D intake and hijab use

Variable	Nutrition status		p
	Central Obesity	Normal Weight	
Sun exposure score			
Mean ± SD	3,06 ± 3,55	15,37 ± 10,71	0,001*
Vitamin D from food(IU)			
Median (Q25:Q75)	545,79 (317,73:605,81)	562,26 (307,83:673,04)	0,515**
Hijab Use	56,3%	62,5%	0,719***

Note: *Mann Whitney Test; **Independent t-test; *** Chi Square Test

Table 2: Comparability test results

	Nutrition status		
Variable	Central Obesity	Normal Weight	p
25(OH) Vitamin D (ng/mL)	7,85±3,57	15,20 ± 9,05	0,003*
Blood Pressure			
Systolic (mmHg)	125,00 ± 28,75	105,00 ± 13,75	0,005*
Diastolic (mmHg)	82,50 ± 15,00	70,00 ± 8,75	0,001*
MAP (mmHg)	96,66 (86,67:106,25)	83,33 (80:90)	0,001**
Fasting Blood Sugar (mg/dl)	89.5 (82:105,25)	88.5 (84,25:95)	0,780**

HDL (mg/dl)	51 (43:67,25)	64.5 (53,50:73,50)	0,051**
Trigliserida (mg/dl)	117,00 ± 111,25	83,00 ± 51,75	0,061*

Note: *analyzed by Mann Whitney test, presented with Mean ± SD
**analyzed by Independent t-test, presented with Median (Q25:Q75)

Table 3: Correlate test of levels of 25 (OH) Vitamin D with metabolic indicators

Variable	Central Obesity		Normal Weight	
	r	p	r	p
Blood pressure				
Systolic	-0,737	0,001	0,033	0,903
Diastolic	-0,655	0,006	-0,036	0,895
MAP	-0,689	0,003	-0,028	0,917
Fasting blood sugar	0,151	0,578	0,284	0,286
HDL	-0,140	0,606	-0,007	0,978
Trigliserida	0,094	0,729	0,143	0,598

Note: MAP= Mean Arterial Pressure; HDL = High Density Lipoprotein

4. Discussion

There were significant differences in the sun exposure score between the two groups. This is consistent with the research that has been conducted, where it was found that there was a strong relationship between obesity and 25 (OH) D levels caused by the possibility of low sun exposure in obese people compared to people with normal weight because physical activity tends to be lower in obese people compared to normal weight people[11].

Results of comparative test illustrate that there were mean differences in the levels of 25 (OH) Vitamin D and blood pressure between the two groups. Although the statistical results state that the majority of the results of the examination do not have a correlation, the mean difference between the two groups states the possibility of decreasing levels of 25 (OH) Vitamin D increases fasting blood sugar and triglycerides and decreases HDL levels. This can be seen from the average fasting blood sugar and triglyceride levels in the group of central obese women, while HDL was higher in the group of normal weight women.

Correlation test results illustrate that levels of 25 (OH) Vitamin D only have a negative correlation with blood pressure. This may also be influenced by the length of the subject suffering from obesity and Vitamin D deficiency. The average age of the study subjects ranged from 40 years in the two groups where in the aging process this age is in the transition phase. This phase is the initial phase of symptoms beginning to appear.

5. Conclusion

Level of 25(OH) Vitamin D in central obesity woman lower than in normal weight woman and have a correlation with blood pressure in woman with central obesity.

References

[1] Pangkahila W. Anti aging: tetapmuda dan sehat. Buku Kompas; 2011.
[2] Sjarif DR, Lestari ED, Mexitalia M, Nasar SS. Buku ajar nutrisipediatrik dan penyakitmetabolik. Jilid I. Ikatan Dokter Anak Indonesia. Jakarta. 2011.
[3] de Souza WN, Martini LA. The role of Vitamin D in obesity and inflammation at adipose tissue. Journal of Obesity and Metabolic Research. 2015 Jul 1;2(3):161.
[4] Attie AD, Scherer PE. Adipocyte metabolism and obesity. Journal of lipid research. 2009 Apr 1;50(Supplement):S395-9.
[5] Garbossa, S.G. and Folli, F., 2017. Vitamin D, sub-inflammation and insulin resistance. A window on a potential role for the interaction between bone and glucose metabolism. *Reviews in Endocrine and Metabolic Disorders*, *18*(2), pp.243-258.
[6] Chaudhuri JR, Mridula KR, Anamika A, Boddu DB, Misra PK, Lingaiah A, Balaraju B, Bandaru VS. Deficiency of 25-hydroxyvitamin d and dyslipidemia in Indian subjects. Journal of lipids. 2013;2013.
[7] Vaidya A, Williams JS. Vitamin D in the Pathophysiology of Hypertension, Kidney Disease, and Diabetes: Examining the Relationship Between Vitamin D and the Renin-Angiotensin System in Human Diseases. Metabolism. 2012 Apr;61(4):450.
[8] Kostoglou-Athanassiou I, Athanassiou P, Gkountouvas A, Kaldrymides P. Vitamin D and glycemic control in diabetes mellitus type 2. Therapeutic advances in endocrinology and metabolism. 2013 Aug;4(4):122-8.
[9] Talaei A, Mohamadi M, Adgi Z. The effect of vitamin D on insulin resistance in patients with type 2 diabetes. Diabetology & metabolic syndrome. 2013 Dec;5(1):8.
[10] Bayani MA, Akbari R, Banasaz B, Saeedi F. Status of Vitamin-D in diabetic patients. Caspian journal of internal medicine. 2014;5(1):40.
[11] 11.Lin E, Armstrong-Moore D, Liang Z, Sweeney JF, Torres WE, Ziegler TR, Tangpricha V, Gletsu-Miller N. Contribution of adipose tissue to plasma 25-hydroxyvitamin D concentrations during weight loss following gastric bypass surgery. Obesity. 2011 Mar 1;19(3):588-94.