의학석사 학위논문

Relationship of Serum 25-Hydroxyvitamin D, Parathyroid Hormone with Insulin Resistance and the Impaired Fasting Glucose in Overweight or Obese Children and Adolescents

과체중 또는 비만인 소아청소년에서 비타민 D, 부갑상선호르몬과 인슐린 저항성 및 공복혈당장애와의 관련성에 대한 연구

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ABSTRACT

Introduction: The nonclassical roles of vitamin D and parathyroid hormone (PTH) in glucose metabolism, insulin resistance, and metabolic syndrome have been proven with increasing evidence in the adult population. However, the role of PTH is controversial and pediatric studies are limited. Therefore, the aim of this study was to examine the association of 25-hydroxyvitamin D [25(OH)D] and PTH levels with insulin resistance, impaired fasting glucose, and metabolic syndrome.

Methods: Obese and overweight children and adolescents that visited Seoul National University Children's Hospital were enrolled. Correlations between 25(OH)D and PTH levels and HOMA-IR (Homeostasis Model Assessment-Insulin Resistance), fasting glucose, HbA1c, and metabolic syndrome were evaluated by linear regression and logistic regression analysis.

Results: In overweight or obese children and adolescents, adjusting for sex, puberty, and body mass index (BMI) Z-score, 25(OH)D levels were related to HOMA-IR and fasting glucose, independent of PTH levels. PTH levels were related to fasting

glucose, although this relationship was not independent of 25(OH)D levels.

Conclusions: This study revealed a significant relationship between vitamin D with fasting glucose and insulin resistance. PTH was correlated with FBG, but this relationship between PTH and fasting glucose was not independent of 25(OH)D.

Keywords: Parathyroid hormone, PTH, vitamin D, insulin resistance, fasting blood glucose, metabolic syndrome, Korean children

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LIST OF ABBREVIATIONS

BMI, body mass index

25(OH)D, 25-hydroxyvitamin D

PTH, parathyroid hormone

FBG, fasting blood glucose

IFG, impaired fasting glucose

HOMA-IR, homeostasis model assessment-insulin resistance

TG, triglyceride

HDL-C, high density lipoprotein-cholesterol

LDL-C, low density lipoprotein-cholesterol

SBP, systolic blood pressure

DBP, diastolic blood pressure

Mets, metabolic syndrome

VDR, vitamin D receptor

VDBP, vitamin D binding protein

INTRODUCTION

The role of 25-hydroxyvitamin D (25(OH)D) and parathyroid hormone (PTH) in calcium homeostasis and bone metabolism is well known. Recently, so-called 'nonclassical' novel roles of vitamin D and PTH are under investigation. Besides calcium and phosphate homeostasis, vitamin D has been found to influence muscle function, the cardiovascular system, immune response, and insulin homeostasis [1]. Vitamin D deficiency or insufficiency is associated with muscle weakness and a high incidence of various chronic diseases such as cardiovascular disease, cancer, multiple sclerosis, and diabetes [1]. Low vitamin D levels and high PTH levels are associated with nonalcoholic fatty liver disease [2]. An increasing number of population-based cross-sectional studies suggest additional nonclassical roles of 25(OH)D and PTH in insulin resistance [3, 4], insulin sensitivity [5, 6], and metabolic syndrome [3, 6-10]. Some studies have found low vitamin D levels and elevated PTH levels to be potential risk factors of metabolic syndrome [11, 12].

Previous research has shown that hyperparathyroidism patients have increased risk of hypertension [13, 14], obesity

[15-17], and diabetes mellitus [18-20] which are markers of metabolic syndrome. Emerging evidence suggests that PTH plays a role in normal insulin levels, glucose homeostasis and metabolic syndrome. An inverse relationship between PTH and insulin sensitivity has also been reported [6]. Studies that focused on elderly men in the US [21] and on the obese adult population [22] have found that elevated levels of PTH were associated with increased risk of metabolic syndrome. A study on the adolescent population reported that PTH was associated with insulin resistance, independent of vitamin D levels [4]. In contrast to these studies, others have reported that PTH has no significant relationship with insulin resistance [3] and metabolic syndrome [3,7,9,12]. While the role of vitamin D in insulin resistance and metabolic syndrome has gradually been acknowledged, studies on the association between PTH and parameters of glucose homeostasis have still shown conflicting results. It is not clear whether the observed high circulating PTH concentrations in overweight or obese individuals can further and independently contribute to the risk of developing impaired glucose tolerance and diabetes mellitus.

The aim of this study was to determine the relationships of

25(OH)D and PTH levels with the parameters of glucose homeostasis in overweight and obese Korean children. We were particularly interested in whether PTH was associated with parameters of glucose homeostasis, independent of 25(OH)D levels. We also examined the relationships of 25(OH)D and PTH levels with metabolic syndrome in subjects over 10 years of age.

MATERIALS AND METHODS

1. Study population

This study protocol was approved by the Institutional Review Board of Seoul National University Hospital (IRB No. H-1306-091-497) and the requirement for informed consent was waived. Between August, 2009 and June, 2013, overweight (Body mass index [BMI]: 85~95 percentile) or obese (BMI>95 percentile) children and adolescents under 18 years of age who visited Seoul National University Children's Hospital for obesity, growth or puberty examinations were included. Patients with underlying diseases or on medication were excluded. Serum calcium levels were within the normal range

for all included participants. Medical records were retrospectively reviewed for 215 Korean children and adolescents.

2. Physical examination

All participants underwent puberty staging by a pediatric endocrinologist. Height (cm) was measured twice with a Harpenden stadiometer (Holtain Ltd, Crymmynch, Wales, UK) to the first decimal place. Weight was measured to the first decimal place with a digital scale. Waist circumference (WC, cm) was measured to the first decimal place. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). BMI percentile and BMI Z-score were assigned according to the 2007 Korean National Growth Charts [23]. Blood pressure was measured twice using an automatic sphygmomanometer in the right arm with the subject seated after 5 minutes of resting.

3. Laboratory measurements

Fasting glucose and insulin levels, and HbA1c, HDL-cholesterol, LDL-cholesterol, TG, 25(OH)D, and PTH levels were measured with blood samples obtained by venipuncture after 12

hours of fasting. Plasma glucose levels (mg/dL) were measured enzymatically with the Hitachi Automatic Analyzer 7600 (Hitachi, Tokyo, Japan). Insulin levels (μU/mL) were measured with a 1470 Wizard gamma counter and an immunoradiometric assay kit (Biosource, Fleurus, Belgium). Serum 25(OH)D levels were measured using an ^{125}I-labeled radioimmunoassay (DiaSorin, Stillwater, MN, USA). The inter-assay coefficient of variation (CV) was 10.8% and the intra-assay CV was 9.4%. Serum iPTH levels were measured using a standard ELSA-PTH immunoradiometric assay (CIS Bio International, Sorgues, France). The inter-assay CV was 4.6%, and the intra-assay CV was 4.3%.

4. HOMA-IR, IGF and vitamin D deficiency

Homeostatic Model Assessment-Insulin Resistance (HOMA-IR) was calculated as fasting glucose [mg/dL] x insulin [mU/L]/405 and was used to evaluate insulin resistance. Impaired fasting glucose (IFG) was defined as fasting glucose level ≥ 100mg/dL. Vitamin D deficiency was defined as serum 25(OH)D level < 20ng/mL.

5. Metabolic syndrome

Subjects 10 years of age or older (n=73) were evaluated for metabolic syndrome. We adopted the International Diabetes Federation definition of metabolic syndrome in children and adolescents [24]. Participants were classified as having metabolic syndrome if they had central obesity (WC≥90 percentile) plus any two of the following four factors: 1) triglycerides ≥150mg/dL in children 10~15.9 years of age, or triyglycerides ≥150mg/dL or on treatment for high TG in adolescents 16 years of age or older, 2) HDL-cholesterol <40mg/dL in children 10~15.9 years of age, or HDL-cholesterol <40mg/dL (male) or < 50mg/dL (female) or on treatment for low HDL-cholesterol in adolescents 16 years of age or older, 3) systolic blood pressure (SBP) ≥130mmHg or diastolic blood pressure (DBP) ≥85mmHg in children 10~15.9 years of age, or SBP≥ 130mmHg or DBP≥ 85mmHg or on treatment for hypertension in adolescents 16 years of age or older, 4) fasting glucose ≥ 100mg/dL or known type 2 diabetes mellitus.

6. Statistical analysis

SPSS version 21.0 for Windows (SPSS institute, Chicago, IL, USA) was used for all statistical analyses. Variables were tested for normal distribution. PTH level, insulin level, and HOMA-IR showed skewed distributions and were accordingly log-transformed for analysis. All continuous variables are described as mean ± SD. The student's *t* test was used to compare the mean value of continuous variables, and the chi-squared test was used to compare categorical variables between two groups with normally distributed data. The relationships between 25(OH)D and HOMA-IR, FBG, HbA1c and the relationships between PTH and HOMA-IR, FBG, HbA1c were evaluated by univariate analysis. The relationship between 25(OH)D and PTH in patients with IFG and metabolic syndrome was evaluated by binary logistic regression analysis. Multivariate linear regression analysis was subsequently performed and included all independent variables that were significant in univariate analysis and previously known covariates. A $P < 0.05$ was considered significant.

RESULTS

Clinical characteristics of subjects (Table 1)

The mean age of the study subjects (84 males) was 9.2 ± 2.4 years old, with 54.9% of the subjects being prepubertal (n = 118). The mean BMI was 24.5 ± 3.7 (17.8–39.0) with 70.2% of the subjects being obese (n = 151). The percentages of subjects with serum 25(OH)D level < 20ng/mL (the vitamin D–deficient group) was 44% (n = 92), 20 to < 30ng/mL (the vitamin D–insufficient group) was 40% (n = 88), and ≥ 30ng/mL (the vitamin D–sufficient group) was 16% (n = 35). Two subjects had 25(OH)D levels < 10ng/mL. No clinical symptoms of vitamin D deficiency such as rickets, hypocalcemia, or hypophosphatemia were observed in the vitamin D–deficient group. Twenty–six (12.1%) of the total 215 subjects were diagnosed with IFG. Twenty–one (29.6%) of 71 subjects older than 10 years of age were diagnosed with metabolic syndrome.

Comparison between the vitamin D-deficient group and the vitamin D-nondeficient group (Table 1)

Subjects were classified into the vitamin D-deficient group (n = 92) and the vitamin D-nondeficient group (serum 25(OH)D level ≥ 20ng/mL, n = 123). The proportion of males, puberty, obesity, and IFG did not differ between the two groups. The vitamin D-deficient group had higher levels of PTH (P = 0.020), HOMA-IR (P = 0.038), TG (P = 0.028) than the vitamin D-nondeficient group. The proportion of metabolic syndrome among subjects older than 10 years of age was higher in the vitamin D-deficient group than in the vitamin D-nondeficient group (63.9 vs. 46.7%), although without statistical significance. No differences in the BMI z-score, FBG, total cholesterol, HDL-cholesterol, and LDL-cholesterol levels, and systolic and diastolic BP were found between the two groups.

Table 1. Clinical characteristics of the study participants

	Total	25(OH)D (ng/dL)		*P* value
		< 20 (deficiency)	≥ 20	
No (%)	215 (100%)	92 (42.8%)	123 (57.2%)	
Season (spring/summer/fall/winter)	42/59/55/59	27/21/12/32	21/32/43/27	0.894
Male (%)	84(39)	47(38.2)	37(40.2)	0.779
Age (yrs)	9.2 ± 2.4	9.7 ± 2.6	8.8 ± 2.1	0.006
Pre/pubertal (n)	118/97	47/45	71/52	0.406
BMI (kg/m^2)	24.3 ± 3.5	23.8 ± 3.0	25.0 ± 4.0	0.752
Overweight /obese	84/151	19/73	16/107	0.14
25(OH)D (ng/mL)	22.5 ± 6.6	16.3 ± 2.7	27.1 ± 4.6	<0.001
PTH (pg/mL) *	25.4 ± 11.4	27.4 ± 12.0	23.8 ± 10.7	0.02
FBG (mg/dL)	90.3 ± 8.7	91.5 ± 8.3	89.4 ± 8.7	0.085
IFG (%) *	26 (12.1)	15 (16.3)	11 (8.9)	0.138
Insulin (uIU/mL) *	15.2 ± 10.0	16.9 ± 10.9	14.0 ± 9.2	0.057
HOMA-IR *	3.4 ± 2.4	3.8 ± 2.6	3.1 ± 2.1	0.038
HbA1c (%)	5.3 ± 0.3	5.4 ± 0.3	5.3 ± 0.3	0.024
Total cholesterol (mg/dL)*	175.6 ± 26.5	174.8 ± 27.5	176.1 ± 25.8	0.718
TG (mg/dL)*	102.8 ± 44.0	110.3 ± 47.0	97.0 ± 40.8	0.028
HDL-C (mg/dL)*	51.4 ± 10.1	49.9 ± 10.0	52.6 ± 10.1	0.051
LDL-C (mg/dL)*	122.8 ± 25.3	112.7 ± 25.7	112.9 ± 25.4	0.946
SBP (mmHg)	117.1 ± 10.2	118.6 ± 10.2	115.8 ± 10.1	0.142
DBP (mmHg)	60.45 ± 7.8	60.7 ± 7.2	60.2 ± 8.3	0.751

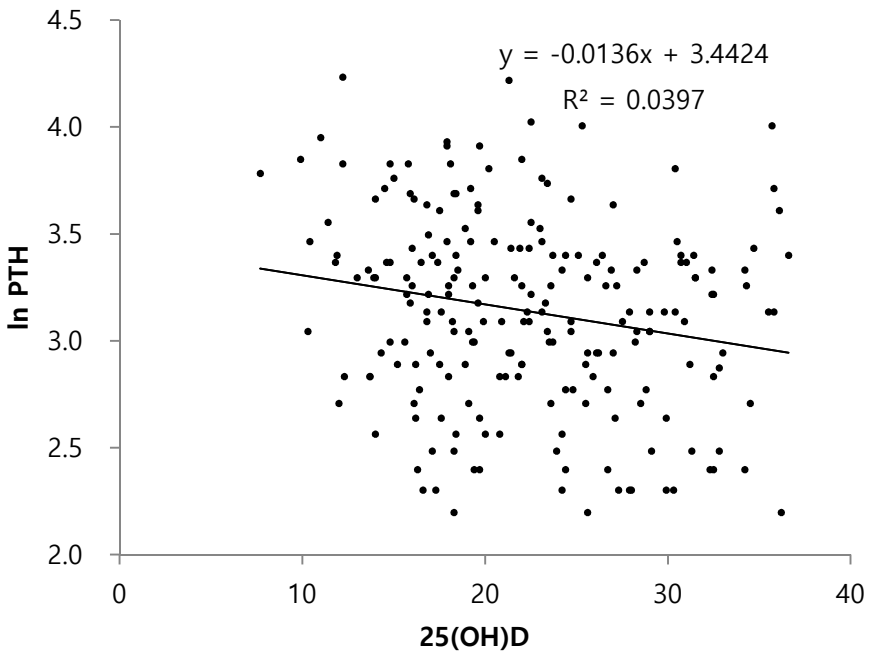
Abbreviations: BMI, body mass index; 25(OH)D, 25-hydroxyvitamin D; PTH, parathyroid hormone; FBG, fasting blood glucose; IFG, impaired fasting glucose; HOMA-IR, homeostasis model assessment – insulin resistance; TG, triglyceride; HDL-C, high density lipoprotein-cholesterol; LDL-C, low density lipoprotein-cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure. Data are expressed as median ±SD.

* The analysis was performed using natural log-transformed values.

Relationship between 25(OH)D and ln_PTH

25(OH)D and PTH level had significant negative correlation for the total population (β = − 0.01, r^2 =0.041, P = 0.003). (Figure 1) There was a significant negative correlation between 25(OH)D and PTH level for male (β = − 0.244, r^2 =0.065, P = 0.026), female (β = − 0.193, r^2 = 0.031, P = 0.027), and prepubertal (β = − 0.221, r^2 = 0.062, P = 0.016) group. For pubertal adolescents 25(OH)D and PTH level had negative correlation, however was not significant (β = − 0.174, r^2 =0.016, P = 0.089).

Figure 1. Relationship between 25(OH)D and ln _PTH



Relationship between vitamin D and markers of glucose homeostasis (Table 2)

25(OH)D levels were inversely related with log-transformed PTH levels (r^2 = 0.041, P = 0.003, Fig 1). 25(OH)D levels also had a significant negative correlation with FBG levels(P = 0.007). This negative correlation was still significant even after adjustment for puberty, gender and BMI-Z score (P = 0.016) and PTH (P = 0.043). 25(OH)D was negatively related to HOMA-IR (P = 0.03), with significance after adjustment for puberty, gender, BMI Z-score (P = 0.028), and PTH (P = 0.029). The presence of metabolic syndrome did not have significant relationship with serum 25(OH)D levels in children over 10years of age. HbA1c and the presence of IFG did not have significant correlations with serum 25(OH)D levels.

Table 2. Relationship of 25(OH)D with markers of glucose homeostasis and metabolic syndrome

	Unadjusted			Adjusted for puberty, gender, BMI-Z			Adjusted for puberty, gender, BMI-Z, PTH		
	B	*SE*	*P*	*B*	*SE*	*P*	*B*	*SE*	*P*
FBG	-1.85	0.091	0.007	-0.163	0.09	0.016	-0.138	0.091	0.043
Insulin [a]	-0.123	0.006	0.073	-0.124	0.006	0.063	-0.132	0.006	0.054
HOMA-IR[a]	-0.148	0.007	0.03	-0.146	0.006	0.028	-0.150	0.088	0.029
HbA1c	-0.127	0.004	0.088	-0.126	0.004	0.095	-0.107	0.004	0.160
IFG[a]	-0.059	0.034	0.083	-0.053	0.035	0.131	-0.039	0.035	0.268
MetS[b]	-0.049	0.041	0.233	-0.051	0.042	0.232	-0.042	0.043	0.331

Abbreviations: BMI, body mass index; 25(OH)D, 25-hydroxyvitamin D; PTH, parathyroid hormone; FBG, fasting blood glucose; IFG, impaired fasting glucose; HOMA-IR, homeostasis model assessment – insulin resistance; Mets, Metabolic syndrome

a. The analysis was performed using natural log-transformed values.

b. Metabolic syndrome was evaluated for subjects 10 years of age or older with data on weight circumference, BP, FBG, TG, and HDL (n=65)

Relationship between PTH and markers of glucose homeostasis (Table 3)

Serum PTH levels had positive correlation with FBG levels (P = 0.046). This positive correlation was significant after adjustment for puberty, gender, and BMI Z-score (P = 0.033). However after adjusted for 25(OH)D, the correlation between PTH and FBG levels was not significant (P = 0.094). Insulin, HOMA-IR, HbA1c, the presence of IFG and metabolic syndrome did not have significant relationships with PTH levels.

Table 3. Relationship between PTH and markers of glucose homeostasis and metabolic syndrome

	Unadjusted			Adjusted for puberty, gender, BMI-Z			Adjusted for puberty, gender, BMI-Z, 25(OH)D		
	B	*SE*	*P*	*B*	*SE*	*P*	*B*	*SE*	*P*
FBG	0.136	1.204	0.046	0.143	1.311	0.033	0.115	1.346	0.094
Insulin [a]	0.025	1.541	0.719	-0.006	1.525	0.935	-0.056	1.508	0.408
HOMA-IR [a]	0.068	0.098	0.322	0.016	0.096	0.818	-0.016	0.097	0.813
HbA1c	0.121	0.049	0.105	0.129	0.49	0.086	0.112	0.050	0.144
IFG [a]	0.901	0.490	0.066	0.954	0.507	0.060	0.832	0.571	0.108
MetS[b]	0.794	0.607	0.191	0.644	0.635	0.310	0.480	0.658	0.466

Abbreviations: BMI, body mass index; 25(OH)D, 25-hydroxyvitamin D; PTH, parathyroid hormone; FBG, fasting blood glucose; IFG, impaired fasting glucose; HOMA-IR, homeostasis model assessment – insulin resistance; Mets, Metabolic syndrome

a. The analysis was performed using natural log-transformed values.

b. Metabolic syndrome was evaluated for subjects 10 years of age or older with data on weight circumference, BP, FBG, TG, and HDL (n=65).

DISCUSSION

This study demonstrated that 25(OH)D was inversely related to fasting glucose and HOMA-IR in overweight and obese Korean children and adolescents. PTH was positively correlated with fasting glucose, but this correlation was not significant after adjusting for serum 25(OH)D levels.

Vitamin D is known to activate the protein synthesis of the pancreatic β cells and also increase calcium influx into the β cells which in turn, stimulates the conversion of proinsulin to insulin [25]. The active form of vitamin D, 1,25(OH)2D binds to intracellular Vitamin D receptors (VDRs). The VDR is a nuclear hormone receptor which forms a heterodimer with the retinoid X receptor (RXR) and binds to the vitamin D response element (VDRE) that exists on the promoter of the target gene. VDRs are found not only in the bone, kidney, and intestine but also in the islet β cells of the pancreas which control insulin release [26]. Also, the presence of VDRE in the human insulin gene [27] and transcriptional activation of the human insulin gene by 1,25(OH)2D [28] has been reported. An increase in serum vitamin D levels could increase the insulin secretion of

pancreatic β cells, which would result in decreased serum glucose levels. This is consistent with the inverse relationship between fasting glucose and 25(OH)D levels in this study, despite serum insulin levels not being significantly correlated with 25(OH)D levels.

VDRs exist in almost all body cells [29]. Besides the effects of vitamin D on glucose homeostasis mediated by autocrine and paracrine functions in pancreatic β cells, the effects of vitamin D on insulin sensitivity of the peripheral tissues such as cardiac and skeletal myocytes, and hepatocytes have been reported [5, 30]. The presence of VDR in these cells could explain the relationship between HOMA-IR and 25(OH)D level observed in this study.

Cross-sectional studies [3-5,7,11,12] and a prospective study in the adult population [31] revealed that vitamin D levels are related to insulin resistance and insulin sensitivity. An inverse relationship between vitamin D status and insulin resistance and the risk of impaired fasting glucose in Korean children and adolescents has been previously reported in children 10 years of age or older [32]. The result of the present study which

includes children less than 10 years of age is consistent with these previous studies. This result is of clinical significance considering that a high percentage of Korean children have vitamin D deficiency (89.1% in spring, 53.7% in summer, 63.9% in fall and 90.5% in winter) [33].

The obese population is known to have lower circulating 25(OH)D levels. This is explained by several reasons, including decreased sun exposure and vitamin D sequestration in adipose tissue [34]. Vitamin D supplementation in Iran children aged 10 to 16 years was found to reduce insulin resistance [35] In a recent study, the relationship between obesity and 25(OH)D levels was explained by the vitamin D binding protein (VDBP) [36]. Obese children had low total serum vitamin D levels, despite having similar amounts of bioavailable free-form 25(OH)D compared to normal weight children. This was explained by lower serum VDBP levels in the obese children [36]. VDBP concentrations are inversely associated with hyperinsulinemia and insulin resistance [37]. A possible mechanism explaining the relationship between vitamin D levels and insulin resistance is that obesity increases insulin resistance, which decreases VDBP, which consequently

decreases the serum total 25(OH)D level. However, VDBP was not measured in this study and further studies are needed to determine the role of VDBP.

PTH is primary regulated by serum calcium levels. When serum calcium levels decrease, a signal is transmitted by the calcium-sensing receptor (CaSR), which increases PTH secretion. When there is a risk of hypocalcemia due to vitamin D deficiency, PTH stimulates 1α-hydroxylase in the kidneys and increases the production of 1,25(OD)2D, resulting in increased serum calcium levels. The inverse relationship between PTH and vitamin D was once again observed in this study. Therefore, PTH could be related to insulin resistance and glucose metabolism indirectly by increasing active 1,25(OH)2D.

However, the possibility of PTH being associated to insulin sensitivity independent of vitamin D has been proposed. Primary hyperparathyroidism is associated with impaired glucose tolerance, insulin sensitivity, and diabetes [2]. Primary hyperparathyroidism patients have been found with decreased insulin sensitivity with insulin sensitivity improving after these patients undergo parathyroidectomy [2]. Also, insulin-stimulated glucose uptake decreased secondary to a PTH-

induced elevation in intracellular calcium both in vivo and in vitro [13, 21]. PTH treatment of differentiated 3T3-L1 adipocytes suppresses insulin-stimulated glucose uptake and insulin signaling via the cAMP pathway, potentially through the phosphorylation of IRS-1 at serine 307 [38].

Although several studies show that elevated PTH levels are related to insulin sensitivity [5, 6] and metabolic syndrome [4, 8, 10], independent of vitamin D, there have also been studies that report that PTH levels do not have a significant relationship with parameters of glucose homeostasis or metabolic syndrome after adjustment for confounders including the 25(OH)D level [5, 7, 9, 12].

PTH was negatively correlated only to fasting glucose in this study, however there was no significant association between PTH levels and serum glucose levels when the parameters were adjusted for serum 25(OH)D levels. The positive correlation between serum PTH levels and FBG levels shown in this study could be explained by the negative correlation between 25(OH)D and PTH.

To our knowledge, this is the first study on the relationship between PTH and glucose homeostasis in Asian children, and

the first study to investigate the relationship between vitamin D and parameters of glucose homeostasis in the Asian population including children less than 10 years of age.

The present study includes some limitations. Since this study was cross-sectional, temporal relationships could not be determined. We did not consider physical activity of the subjects which could be a confounding factor in this study. We included the BMI data of the participants; however, fat mass was not examined i. Since fat mass is directly associated with vitamin D levels and insulin resistance [39], not examining fat mass might be considered a weakness of this study. Patients with abnormalities in calcium levels were excluded from this study. Thus, the effect calcium levels had on vitamin D and PTH levels was not considered in this study.

In conclusion, this study revealed a significant relationship between vitamin D with FBG and insulin resistance, which was consistent with previous studies of the adult and adolescent population. PTH was correlated with FBG levels, but this correlation was not independent when adjusted for 25(OH)D levels. Further studies, including prospective and case-

controlled studies in a larger population are needed to verify these findings.

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초 록

서론: 성인을 대상으로 한 연구에서 비타민 D는 포도당 대사 및 인슐린 저항성, 그리고 당뇨 및 대사증후군과 관련이 있다는 보고가 많다. 그러나 PTH의 역할에 대해서는 논란이 있으며 소아에서 연구는 거의 없다. 그러므로 본 연구에서는 혈중 비타민D 및 PTH 농도와 인슐린 저항성, 공복혈당장애, 그리고 대사증후군이 상관관계가 있는지 알아보고자 한다.

방법: 서울대학교 어린이병원을 방문한 215명의 과체중 또는 비만인 소아청소년을 대상하였다. 후향적 의무기록 조사를 통해 키, 체중, 25(OH)D, PTH, HOMA-IR, 공복혈당, HbA1c 대사증후군의 항목 등의 자료를 수집하였다. 선형회귀분석 및 로지스틱 회귀분석을 통한 통계분석을 시행하였다.

결과: 과체중 또는 비만인 소아청소년에서 비타민D 농도는 인슐린 저항성과 공복혈당과 관련이 있었으며 이는 성별, 사춘기 여부, 체질량지수 Z-값, PTH를 보정하여도 통계적으로 유의하였다. PTH 농도는 공복혈당과 관련이 있었으나 비타민D 농도를 보정하였을 때 유의하지 않았다.

결론: 과체중 또는 비만인 소아청소년에서 비타민 D의 농도는 공복혈당 및 인슐린 저항성과 관련이 있으며 PTH의 공복혈당

과의 관련성은 비타민D 농도의 영향으로 볼 수 있다. 비타민D 결핍이 많은 우리나라 소아청소년에서 뼈와 칼슘대사뿐만 아니라 혈당과 관련해서도 비타민D의 중요성이 강조될 필요가 있다.

주요어: 비타민D, 부갑상선호르몬, 인슐린저항성, 공복혈당, 대사증후군, 한국소아청소년

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