Visceral adiposity is associated with altered myocardial glucose uptake measured by ^{18}FDG-PET in 346 subjects with normal glucose tolerance, prediabetes, and type 2 diabetes

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Directed by Professor Mijin Yun

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ABSTRACT

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(Directed by Professor Mijin Yun)

Purpose: To evaluate the glucose uptake in hearts among subjects with normoglycemia, impaired fasting glucose (IFG) and type 2 diabetes mellitus (T2D) using [^{18}F] fluorodeoxyglucose- positron emission tomography (^{18}FDG-PET) and investigate its association with various clinical and laboratory profiles.

Methods and results: In a total of 387 participants (control, N=75; IFG, N=242; T2D, N=70), enrolled from the health promotion center at university-affiliated tertiary-care hospital, we assessed the myocardial glucose uptake by ^{18}FDG-PET. Visceral and subcutaneous fat areas were evaluated by abdominal computed tomography (CT) and laboratory parameters including glucose, insulin, uric acid, free fatty acids (FFA), and triglycerides were measured. Myocardial glucose uptake was significantly decreased in subjects with T2D compared to control or IFG groups (P for trend=0.001). Individuals in the lowest tertile of myocardial glucose uptake tended to be having T2D (P=0.002). Correlation analysis showed that myocardial glucose uptake was related to body mass index, visceral and subcutaneous fat areas, HOMA-IR, uric acid and fasting/postprandial serum levels of glucose, insulin, triglycerides, and FFA. In a multiple linear regression model, visceral fat area (Standardized β [STD β]=-0.31, P<0.001), fasting FFA (STD β=-0.34, P<0.001), and uric acid levels (STD β=-0.21, P=0.007) were independent determinants of

myocardial glucose uptake. Multiple logistic analyses demonstrated that decreased myocardial glucose uptake was associated with the risk of diabetes (odds ratio= 2.56; 95% confidence interval: 1.15-5.70, P=0.021).

Conclusion: Myocardial glucose uptake evaluated by ^{18}FDG-PET may predict T2D and was significantly associated with visceral adiposity and fatty acid or uric acid metabolism.

Key words : Myocardial metabolism, Type 2 diabetes mellitus, Positron emission tomography, Fluorodeoxyglucose

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I. INTRODUCTION

Type 2 diabetes (T2D) is a chronic burgeoning disorder worldwide characterized by hyperglycemia, insulin resistance, and increased lipid and lipoprotein abnormalities[1,2]. Previous studies have shown that dysfunctional adiposity such as excess visceral fat is independently associated with risks of prediabetes and T2D[3]. T2D also has effects on long-term dysfunction and damage in various organs including eye, kidney, and heart[4]. The heart requires constant sources of energy including mainly free fatty acids (FFA) and glucose for continuous pumping and has known to be highly flexible in energy substrate metabolism[5,6]. In T2D heart, alterations in myocardial substrate metabolism characterized by increased myocardial fatty acid metabolism and concurrently decreased glucose metabolism have been demonstrated[6-9].

Positron emission tomographic (PET) represents increased uptakes of [^{18}F] fluorodeoxyglucose (^{18}FDG), a glucose analog, which is taken up by tissues via glucose transporters[10,11]. Recent studies have reported that the disturbances of carbohydrate, fat, and protein metabolism altered biodistribution of FDG in patients with T2D[12,13]. In addition, myocardial glucose uptake may differ according to the

status of whole-body insulin resistance such as impaired fasting glucose (IFG) and T2D, compared with normoglycemic control, as myocardial insulin sensitivity. Yet little research has been performed in relationship with myocardial glucose uptake using PET, adiposity, and other metabolic profiles in subjects with normoglycemia, IFG, and T2D.

Therefore, in the current study, we aimed to investigate myocardial glucose uptake by ^{18}FDG-PET and its association with various clinical and laboratory parameters according to glycemic status.

II. MATERIALS AND METHODS

1. Patient Population

Between Jan 2008 and July 2014, a total of 387 individuals aged over 25 years enrolled from the health promotion center at university-affiliated tertiary-care hospital. We studied 75 healthy control subjects, 242 subjects with IFG, and 70 subjects with T2D defined by the 2011 revision of the American Diabetes Association (ADA) guidelines[14]. Patients with cardiovascular disease, previous or present abnormal renal or hepatic function, and other endocrine disorders were excluded.

2. Clinical and laboratory study and abdominal computed tomography

Body mass index (BMI) was defined as weight divided by the square of the height (kg/m^2). Blood pressure was obtained by averaging three times of blood pressure records in the sitting position after at least 5 minutes of rest. Blood samples were collected from each participant after overnight fasts. The fasting and 2-hour postprandial glucose, insulin, FFA, and triglycerides after a conventional meal were measured. Plasma glucose was measured using the glucose oxidase method. Plasma total cholesterol, triglyceride, high density lipoprotein cholesterol, free fatty acid, lipoprotein(a), and uric acid were assayed using a Hitachi 7600 auto analyzer (Hit-

achi Instruments Service, Tokyo, Japan). Low density lipoprotein cholesterol was calculated using the Friedewald equation [LDL-C (mg/dL) = TC (mg/dL) − HDL-C (mg/dL) − TG (mg/dL)/5]. Serum glycated albumin was determined by an enzymatic method as previously described[15]. HbA_{1c} was measured by high-performance liquid chromatography using Variant™ II Turbo (Bio-Rad Laboratories, Hercules, CA, USA). The reference ranges for HbA_{1c} was 4.0% to 6.0%, and for glycated albumin was 11.0% to 16.0%. Homeostatsis Model Assessment of Insulin Resistance (HOMA-IR) was calculated using the following formula: [(Fasting plasma insulin (μU/mL) * Fasting plasma glucose (mg/dL))/405][16]. An index of adipose tissue insulin resistance (Adipo-IR) was calculated as [Fasting plasma FFA (mmol/L) * Fasting plasma insulin (pmol/L)][17,18].

The abdominal adipose tissue areas were quantified by computed tomography (CT) (Tomoscan 350; Philips, Mahwah, NJ, USA). Total abdominal and visceral adipose tissue (VAT) area were measured at the L3-L4s level by a 10 mm CT slice scan in a supine position. VAT area was measured as intra-abdominal cavity at the internal aspect of the abdominal and oblique muscle walls and the posterior aspect of the vertebral body. The subcutaneous adipose tissue (SAT) area was acquired from the total adipose tissue area by subtracting the VAT area.

3. ^{18}FDG-Positron Emission Tomography and image analysis

All patients fasted for more than six hours prior to the ^{18}FDG-PET/CT scan. 5.5 MBq/kg of body weight of ^{18}F-FDG were intravenously injected over 2 min. Serum glucose levels at the time of FDG injection did not exceed 150 mg/dL. After a 1-hour equilibration period, a low-dose CT scan for attenuation correction was performed, followed by acquisition of ^{18}FDG-PET images from the skull base to the mid-thigh in the 3D mode at 3 min per bed position using a standard PET-CT scanner (Gemini, Philips Medical Systems or Discovery STe, GE Healthcare). PET images were reconstructed with attenuation correction. Two-dimensional regions of interest (ROI) were drawn the transaxial images to measure the standardized uptake

value (SUV) of the entire left ventricular myocardium within an inner edge; SUV = (peak kBq/mL in ROI) / (injected activity/g body weight). Myocardial ^{18}FDG uptake was expressed as the maximal SUV (SUVmax). We also obtained the SUV of liver from the circular ROI in the central area of the right lobe. Liver ^{18}FDG uptake was determined by averaging these values. The values of SUV of the heart to liver ^{18}FDG uptake ratio (Heart SUV/ Liver SUV [$SUV_{Heart/Liver}$]) values were used to estimate myocardial glucose uptake.

4. Statistical Analysis

All continuous variables were presented as mean ± standard deviation (SD) and categorical variables were presented as proportions. Differences were analysed using analysis of variance (ANOVA) for continuous variables and Chi-square test for categorical variables. Comparisons of myocardial glucose uptake across the status of diabetes were calculated with the Jonckheere- Terpstra trend test. Pearson's correlation coefficients were calculated to examine the relationships between myocardial glucose uptake and metabolic variables. Multiple linear regression analysis was performed to determine the independent relationship of the studied variables and Standardized β was represented as the coefficient β. The odds ratios (ORs) and 95% confidence intervals (CIs) for the factors associated with the risk of T2D were calculated using multivariate logistic regression analysis. In the Pearson's correlation, multiple linear regression, and logistic regression analysis, myocardial glucose uptake ($SUV_{Heart/Liver}$) was log-transformed. The receiver operating characteristic (ROC) curve of myocardial glucose uptake was used to assess the optimal cut-off value for the prediction of T2D with the areas under the curve (AUC). A $P < 0.05$ was considered statistically significant. Statistical analyses were performed using PASW Statistics version 20.0 for Windows (SPSS Inc., Chicago, IL, USA).

III. RESULTS

1. Baseline Characteristics of Study Population

Table 1. Characteristics of the study subjects

	NGT (N=75)	IFG (N=242)	T2D (N=70)	P
Age *yrs*	50.4 ± 10.6	58.8 ± 9.9	63.2 ± 10.7	< 0.001
Female *n* (%)	37 (49.3)	109 (45.0)	21 (30.0)	0.031
SBP (mmHg)	116.7 ± 11.9	120.8 ± 14.0	127.7 ± 14.6	< 0.001
DBP (mmHg)	74.2 ± 9.7	76.0 ± 10.2	78.0 ± 11.3	0.112
BMI ($kg/m^{2)}$	23.2 ± 3.1	24.2 ± 3.0	25.8 ± 3.9	< 0.001
Visceral fat area ($cm^{2)}$	104.3 ± 46.1	129.9 ± 59.6	195.7 ± 91.8	< 0.001
Subcutaneous fat area (cm^2)	140.9 ± 37.8	142.4 ± 56.7	157.8 ± 76.5	0.361
HbA_{1c} (%)	5.5 ± 0.2	5.8 ± 0.2	7.1 ± 1.2	< 0.001
Glycated albumin (%)	11.0 ± 1.2	11.8 ± 1.8	16.4 ± 6.3	< 0.001
Fasting glucose (*mg/dL*)	90.2 ± 8.1	97.0 ± 9.9	121.3 ± 35.5	< 0.001
Postprandial glucose (*mg/dL*)	98.2 ± 11.5	123.6 ± 36.9	214.0 ± 63.5	< 0.001
Fasting insulin ($\mu U/mL$)	5.7 ± 3.3	7.0 ± 4.2	9.4 ± 7.4	0.001
Postprandial insulin ($\mu U/mL$)	20.9 ± 17.4	38.5 ± 37.5	55.9 ± 51.7	< 0.001
HOMA-IR	1.3 ± 0.8	1.7 ± 1.0	2.9 ± 2.4	< 0.001
Adipo-IR	26.9 ± 20.4	30.3 ± 23.1	40.2 ± 36.3	0.083

Total cholesterol (*mg/dL*)	190.8 ± 36.4	186.9 ± 39.3	162.5 ± 42.0	0.029
HDL cholesterol (*mg/dL*)	49.8 ± 11.8	49.3 ± 12.4	43.5 ± 10.60	0.001
LDL cholesterol (*mg/dL*)	113.6 ± 33.7	111.3 ± 34.4	94.1 ± 32.1	< 0.001
Fasting TG (*mg/dL*)	105.0 ± 71.2	114.4 ± 58.2	132.4 ± 70.3	0.029
Postprandial TG (*mg/dL*)	96.4 ± 73.9	104.9 ± 50.0	131.3 ± 80.0	0.051
Fasting FFA (*μEq/L*)	683.7 ± 381.1	626.1 ± 242.0	652.2 ± 286.3	0.569
Postprandial FFA (*μEq/L*)	173.2 ± 183.0	135.9 ± 106.0	196.3 ± 162.3	0.055
Lipoprotein(a) (*mg/dL*)	21.6 ± 24.2	23.9 ± 25.6	29.1 ± 33.8	0.246
Uric acid (*mg/dL*)	5.0 ± 1.4	5.5 ± 1.3	5.5 ± 1.0	0.020
Statin users	5 (6.8)	48 (19.8)	32 (45.7)	< 0.001

Data were shown as mean ± standard deviation (SD) or n(%).
NGT, Normal glucose tolerance; IFG, Impaired fasting glucose; T2D, Type 2 diabetes; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; BMI, Body mass index; HOMA-IR, Homeostatsis model assessment of insulin resistance; Adipo-IR, An index of adipose tissue insulin resistance; HDL, High density lipoprotein; LDL, Low density lipoprotein; TG, Triglycerides; FFA, Free fatty acid

For all subjects, the mean age of patients was 58.9 ± 10.9 years, 43.2 % were women, and the average BMI was 24.2 ± 3.0 kg/m^2. Among T2D patients, oral hypoglycemic agent users and insulin users were 56.9 % and 10.0 %, respectively. The proportion of subjects with dipeptidyl peptidase-4 inhibitors was 32.8 %, subjects with thiazolidinedione was 13.8 %, subjects with metformin was 46.6 %, and subjects with sulfonylurea was 22.4 % in subjects using oral hypoglycemic agents. Subjects with IFG (N=242) or T2D (N=70) tended to be older, to have higher systolic blood pressure, more obese, and to have metabolically unhealthy factors compared to subjects with normal glucose tolerance (NGT; N=75). Subjects

with IFG or T2D were more likely to have higher levels of uric acid and larger visceral fat area compared to subjects with NGT.

2. Myocardial glucose uptake in NGT, IFG, and T2D

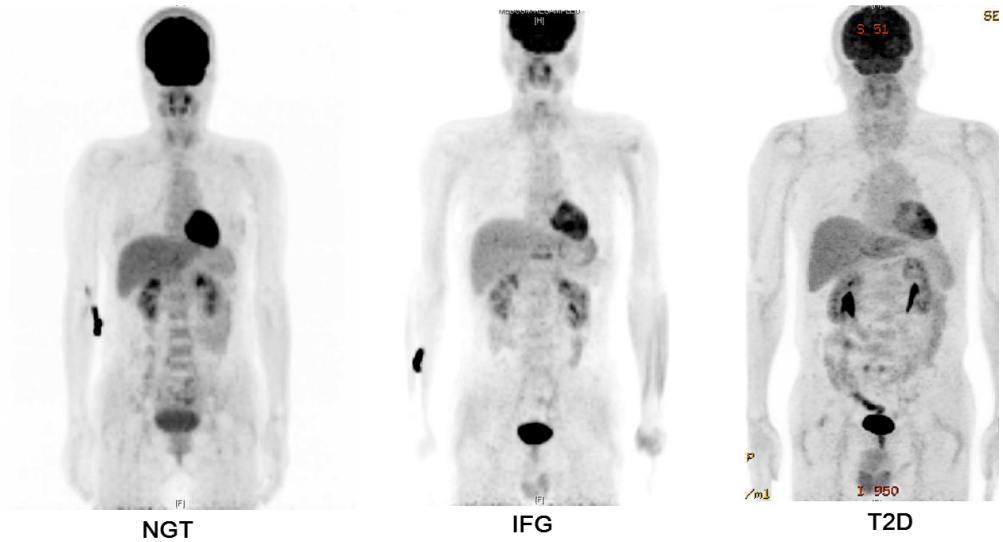

Figure 1. Altered myocardial glucose uptake of FDG in subjects by diabetic status. Each subject has median value within the highest tertile of myocardial glucose uptake in NGT, IFG, and T2D.

Figure 1 presents the status of myocardial glucose uptake in subjects by glycemic status. Compared to NGT or IFG groups, myocardial glucose uptake significantly tended to be decreased in subjects with T2D (NGT, IFG, T2D; mean $SUV_{Heart/Liver}$ =1.89, 1.60, 1.12, respectively; P for trend=0.001, Figure 1, 2). The proportion of lowest tertile of myocardial glucose uptake was significantly higher in patients with T2D (P=0.002).

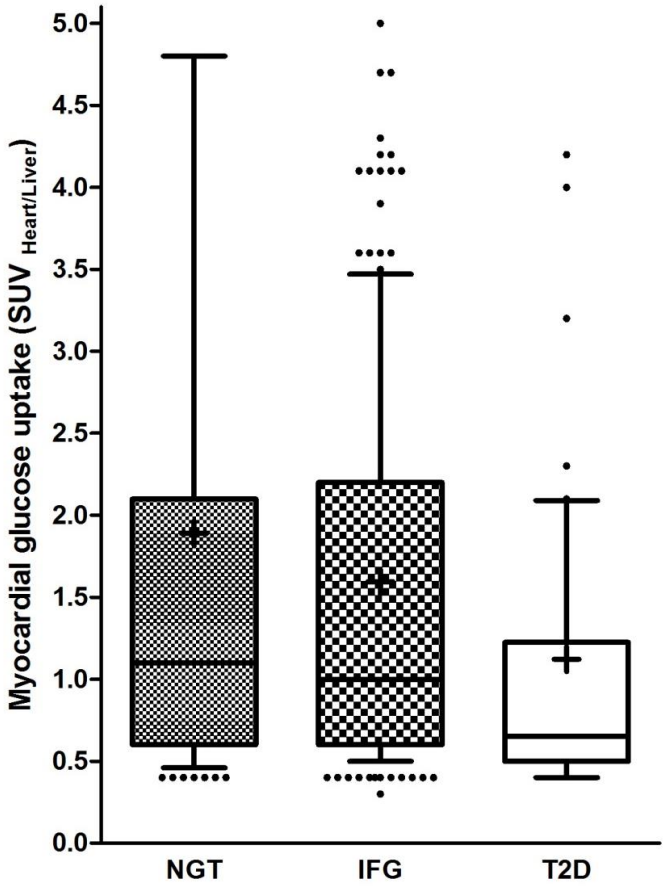

Figure 2. Myocardial glucose uptake by glycemic status Each + and horizontal line in the box indicate mean and median value of myocardial glucose uptake.
Bar indicates 10 to 90 percentiles.

3. Relationship between myocardial glucose uptake and metabolic measures

Table 2. Correlation between myocardial glucose uptake and metabolic factors

	r	P
Age *yrs*	-0.02	0.757
SBP *mmHg*	-0.03	0.540
DBP *mmHg*	-0.03	0.571

BMI kg/m^2	-0.18	< 0.001
Visceral fat area cm^2	-0.24	< 0.001
Subcutaneous fat area cm^2	-0.16	< 0.001
HbA_{1c} %	-0.14	0.005
Glycated albumin %	-0.03	0.601
Fasting glucose *mg/dL*	-0.18	< 0.001
Postprandial glucose *mg/dL*	-0.13	0.013
Fasting insulin *μU/mL*	-0.12	0.035
Postprandial insulin *μU/mL*	-0.12	0.042
HOMA-IR	-0.15	0.006
Adipo-IR	-0.32	< 0.001
Total cholesterol *mg/dL*	0.00	0.995
HDL cholesterol *mg/dL*	0.02	0.776
LDL cholesterol *mg/dL*	0.04	0.499
Fasting triglycerides *mg/dL*	-0.13	0.010
Postprandial triglycerides *mg/dL*	-0.22	0.003
Fasting FFA *μEq/L*	-0.30	< 0.001
Postprandial FFA *μEq/L*	-0.16	0.031
Lipoprotein(a) *mg/dL*	0.04	0.309

SBP, Systolic blood pressure; DBP, Diastolic blood pressure; BMI, Body mass index; HOMA-IR, Homeostatsis model assessment of insulin resistance; Adipo-IR, An index of adipose tissue insulin resistance; HDL, High density lipoprotein; LDL, Low density lipoprotein; FFA, Free fatty acids

To assess the relationship between myocardial glucose uptake and metabolic measures, univariate analysis was performed in Table 2. We observed myocardial glucose uptake correlated negatively with BMI, HbA_{1c}, fasting/postprandial levels of glucose, insulin, TG, HOMA-IR, Adipo-IR, and uric acid. Moreover, statistically significant inverse relations were found between myocardial glucose uptake and both visceral fat areas ($r=-0.24$, $P<0.001$), subcutaneous fat areas ($r=-0.16$, $P<0.001$), and fasting FFA ($r=-0.30$, $P<0.001$).

Table 3. Analysis of myocardial glucose uptake in association with metabolic parameters

	Model 1		Model 2		Model 3	
R^2	0.066		0.250		0.277	
	β	P	β	P	β	P
Visceral fat area cm^2	-0.30	< 0.001	-	-	-0.31	< 0.001
Fasting FFA *μEq/L*	-	-	-0.34	< 0.001	-0.34	< 0.001
Uric acid *mg/dL*	-	-	-0.26	< 0.001	-0.21	0.007
Postprandial glucose *mg/dL*	-	-	-0.15	0.031	-	-
Postprandial triglycerides *mg/dL*	-	-	-0.21	0.002	-	-

Model 1: adjusted for age, sex, BMI, and subcutaneous fat area; Model 2: adjusted for age, sex, fasting/postprandial glucose, insulin, C-peptide, FFA, triglycerids, HbA_{1c}, glycoalbumin, total cholesterol, HDL cholesterol, LDL cholesterol, lipoprotein(a), HOMA-IR, Adipo-IR, uric acid; Model 3: adjusted for age, sex, and variables derived from model 1 and 2.

Multiple linear regression analysis was performed to determine whether myocardial glucose uptake was independently associated with various factors of body composition in addition with age, sex (Model 1), laboratory parameters in addition with age, sex (Model 2), and variables derived from model 1 and model 2 (Model 3; Table 3). In model 3, we found that visceral fat area (β=-0.31, P<0.001), fasting FFA (β=-0.34, P<0.001), and uric acid levels (β=-0.21, P=0.007) were remained to be as independent determinants of myocardial glucose uptake after adjustment for effective body composition and laboratory parameters from model 1and model 2.

4. Assessment of diabetes risk in an association with myocardial glucose uptake

We further investigated significant determinants for the risk of diabetes by multiple logistic regression analyses. After sequential adjustment for confounding covariates, subjects with diabetes had significantly elevated odds ratios (ORs) for having decreased myocardial glucose uptake (Model 5, OR=2.56, P=0.021) and increased visceral fat area (1.02, P=0.008). The optimal cut-off value of myocardial glucose uptake for predicting diabetes was 0.726 (AUC 0.644, [95% CI]=[0.575-0.713]) with sensitivity of 62.8 % and specificity of 67.1 % (P=0.035, Figure 2).

IV. DISCUSSION

In the present study, we used ^{18}FDG-PET and abdominal CT to examine myocardial glucose uptake and visceral/ subcutaneous adipose tissue area, respectively in subjects with normoglycemia, IFG, and T2D. Our results demonstrated that myocardial glucose uptake was markedly decreased by 40.7 %, while area of visceral fat was increased by 87.6 % in patients in T2D compared with normoglycemic control. Myocardial glucose uptake was negatively associated with levels of plasma FFA, uric acid, and visceral fat area. Furthermore, alteration of myocardial glucose uptake evaluated by ^{18}FDG-PET can be a risk factor of T2D.

Previously, hyperglycemia, hyperinsulinemia, and disturbances of carbohydrates, fatty acids, and protein metabolism all have been demonstrated an association that has been found in prediabetes and T2D[2,4]. Therefore, impaired glucose uptake and metabolism in not only skeletal muscle but also in heart, which consistently requires sources of energy from mainly FFA and glucose, could have a correlation with insulin resistance in the status of prediabetes and T2D, contributing to the development of hyperglycemia[19]. Moreover, visceral fat accumulation has been shown to correlate with insulin resistance in subjects with obesity and T2D[20,21]. These findings are in line with the results of the current study that myocardial glucose uptake was significantly decreased, on the other hand, visceral adiposity

was increased with elevated levels of plasma FFA in patients with T2D.

Consistent with previous studies, we revealed that fasting FFA presented to be an independent predictor for myocardial glucose uptake[22,23]. There have been conflicting findings of the uncertain relation between insulin resistance and myocardial glucose uptake in diabetic patients because relationship between a direct effect of myocardial insulin resistance on myocardial glucose uptake and independence of increased plasma FFA is not clear[12,23-26]. For instance, there were reports that suppressed serum FFA concentrations by acipimox, a potent nicotinic acid deriavative, effected the glucose uptake in myocardium[23], and decrease in the FFA levels by rosiglitazone therapy was associated with the improvement in myocardial glucose uptake[27]. However, Yokoyama et al. showed whole-body glucose disposal rate (GDR) was independently related to myocardial FDG uptake, whereas FFA was not[25]. Similar to this, Hicks et al. reported that relation between myocardial FDG uptake and GDR was significant than between myocardial FDG uptake and FFA in subjects with diabetes[28]. Our study revealed myocardial glucose uptake independently related with not subcutaneous, but visceral fat tissue area, uric acid metabolism as well as FFA in multiple linear regression analysis. Various studies reported that increased visceral adipose tissue, not abdominal subcutaneous adipose tissue expected increased in insulin resistance[29-31]. Moreover, visceral fat accumulation turned out to be closely related to the increased production of uric acid which may be linked to increased TG synthesis by liver in obese subjects[32]. Also, an increased serum uric acid was found to be independently associated with insulin resistance and increased risk factors of metabolic syndrome even in the normal subjects[33]. Relation between serum uric acid concentration and insulin resistance seemed to be appeared by decreased urinary uric acid clearance but further study of the exact mechanism would be warranted[34]. Of note, we found that myocardial glucose uptake showed a gradual decrease in proportion in subjects with insulin resistant IFG and T2D. Collectively, myocardial glucose uptake appears to be affected by insulin resistance in an association with visceral adiposity, uric acid

metabolism, and increased FFA itself.

Previous studies showed that in the normal heart, under fasting conditions, FDG uptake image showed variable myocardial glucose uptake because FFA is primary fuel whereas glucose utilization is relatively low for myocardial oxidative metabolism compared to glucose-loading conditions[35-37]. However, in T2D heart, regulation of glucose metabolism is differ from normal heart, so that prior studies showed that myocardial FDG uptake was significantly decreased in diabetic patients compared to normal subjects consistent with our results[13,35]. Cardiac myocytes utilize glucose via mostly insulin-sensitive glucose transporters (GLUT4), responsible for more than 50 % of all body glucose uptake[38]. Reduced expression and mutations of GLUT4 have been associated with diabetes[39,40]. Therefore, the probable mechanisms of decreased myocardial glucose uptake seem to be related with the presence of insulin resistance in patients with T2D[13]. Also, recent studies reported that influences of glucagon-like peptide-1 (GLP-1) caused improvement on myocardial glucose metabolism in diabetic patients[41,42]. Several studies have reviewed that chronic shift of myocardial substrate preference has been noted in the diabetic heart, resulting in prominent decreased glucose and lactic acid oxidation and increased fatty acid oxidation[6-9]. Effects of diabetes on myocardial metabolism are very complex with systemic metabolic disturbances of hyperglycemia, increased FFA and down-regulation of glucose transporters, increased insufficient energy utilization of fatty acid oxidation, lipid accumulation, and lipotoxicity in cardiomyocyte[6,43,44]. Accordingly, these may lead to diabetic cardiomyopathy[43,45]. Further investigation in relationship between myocardial glucose uptake and cardiac function will be needed.

The present study has several distinguishable strengths. It is the first study to investigate myocardial glucose intake evaluated using ^{18}FDG-PET accompanied with subcutaneous/ visceral adiposity by abdominal CT and other metabolic parameters in the point of view of insulin sensitivity according to glycemic status. It can be speculated that myocardial glucose uptake was decreased with insulin

resistance, demonstrating visceral adiposity, hyperuricemia, and elevated FFA in patients with T2D. ^{18}FDG-PET is recently widely performed to detect of malignant tumors or ischemic heart disease for medical examination. Based on these results, furthermore, myocardial glucose uptake assessed by ^{18}FDG-PET may be a useful tool to consider the risk for T2D.

The current study has several limitations. First, this cross-sectional study design is insufficient to lead the causal relationship in the development of impaired myocardial glucose uptake. Another limitation is that in the present study, we did not assess cardiac function in our patients. Correlation between myocardial glucose uptake and cardiac function would be an interesting topic of investigation.

V. CONCLUSION

In conclusion, we confirmed that myocardial glucose uptake was decreased in patients with T2D and there was an association with increased levels of free fatty acids, uric acid, and visceral adiposity. Importantly, alteration of myocardial glucose uptake evaluated by ^{18}FDG-PET can be a risk factor of T2D. Additional studies would be needed to confirm the relationship of myocardial glucose metabolism on the development of diabetes and cardiac function.

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ABSTRACT (IN KOREAN)

양전자방출단층촬영술 시행 시 보이는 공복 상태 심근의 FDG 섭취와 정상, 당뇨 전 단계, 제 2형 당뇨병 환자의 내장 지방과의 연관성에 대한 연구

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조관형

연구목적: 정상환자와 당뇨 전 단계, 그리고 제 2형 당뇨환자에서 양전자방출단층촬영 시행 시에 관찰되는 심근의 FDG 섭취 정도를 다양한 임상적 특징과 혈액학적 검사와 비교하여 그 임상적 의미를 알아보고자 하였다.

대상 및 방법: 건강검진을 목적으로 양전자방출단층촬영술을 시행한 총 387명을 정상군, 당뇨 전 단계, 제 2형 당뇨병의 세 군으로 구분하여 공복시의 심근 포도당 섭취, 내장 및 피하 지방 영역 분석, 혈액 검사를 시행하였으며 이에 대한 유의미한 차이와 관련성을 조사하였다.

결과: 심근의 FDG 섭취는 제 2형 당뇨병 환자 그룹에서 유의하게 감소하였고(P for trend=0.001), 다변량 선형 회귀 분석에서는 내장 지방 면적(β=−0.31, P<0.001), 공복 FFA(β=−0.34, P<0.001) 및 요산 수치(β=−0.21, P=0.007)와 심근의 FDG 섭취와의 관련성이 보고되었다.

결론: 심근의 FDG 섭취는 제 2형 당뇨병을 예측할 수 있고, 내장 지방 및 지방산 그리고 요산의 대사와 유의미한 관련이 있음이 보였다.

핵심되는 말 : 심근 대사, 양전자방출단층촬영술, 제 2형 당뇨병