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Prevalence of Reduced Kidney Function by Estimated Glomerular Filtration Rate Using an Equation Based on Creatinine and Cystatin C in Metabolic Syndrome and Its Components in Korean Adults

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Background: It is known that metabolic syndrome (MetS) is associated with chronic kidney disease. We evaluated and compared the prevalence of reduced kidney function in MetS and its components by estimated glomerular filtration rate (eGFR) using an equation based on creatinine (eGFRcr), cystatin C (eGFRcys), and combined creatinine-cystatin C (eGFRcr-cys) in Korean adults.

Methods: We analyzed data from 3,649 adults who participated in a comprehensive health examination.

Results: Mean values of eGFRcys were higher compared with mean values of eGFRcr (96.1±18.2 mL/min/1.73 m^2 vs. 91.2±13.6 mL/min/1.73 m^2) in total subjects. The prevalence of reduced kidney function increased with age (9.6% for eGFRcys vs. 5.8% for eGFRcr-cys vs. 4.9% for eGFRcr, in subjects aged ≥60 years), and significantly increased with MetS, abdominal obesity, hypertension, high triglyceride, low high density lipoprotein (HDL), and high insulin resistance. The prevalence of MetS, abdominal obesity, hypertension, high insulin resistance, low HDL, and hepatic steatosis was significantly increased in subjects with reduced kidney function. This increased prevalence and the odds ratio of reduced kidney function for prevalence of MetS was highest for eGFRcys, followed by those of eGFRcr-cys, and eGFRcr.

Conclusion: The prevalence of reduced kidney function by eGFR was significantly increased in subjects with MetS and its related components. eGFRcys and eGFRcr-cys were superior to eGFRcr in detecting reduced kidney function.

Keywords: Glomerular filtration rate; Cystatin C; Creatinine; Metabolic syndrome

INTRODUCTION

Metabolic syndrome (MetS), a constellation of cardiovascular disease (CVD) risk factors [1] is associated with risk of coronary heart disease, stroke, and cardiovascular related mortality [2,3]. Moreover, MetS is associated with progression to chronic kidney disease (CKD) [4-6]. The glomerular filtration rate (GFR) is considered the most widely used index of overall kid-

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ney function and is estimated rather than measured in clinical practice. Generally, estimation of GFR from equations based on serum creatinine level is the most common method. However, creatinine is affected by age, sex, race, muscle mass, and diet [7,8].

Recently, serum cystatin C, an endogenous protein, which is freely filtered by the glomerulus, reabsorbed, and catabolized, but not secreted by the renal tubules [9], has been proposed as a potential alternative for serum creatinine as a filtration marker [10]. Cystatin C is less affected by age, race, muscle mass, and diet [7,11]. However, body mass index (BMI), diabetes, and inflammation affect cystatin C level independent of kidney function [12]. Tsai et al. [13] estimated GFR (eGFR) using equations based on creatinine (eGFRcr) and cystatin C (eGFRcys) and compared the discordance of the equations using the two filtration markers in evaluating the prevalence of reduced kidney function and incident all-cause and cardiovascular mortality among persons with diabetes in the United States. However, few studies have evaluated the prevalence of reduced kidney function in MetS or compared equations based on creatinine and cystatin C to eGFR as an indicator of kidney function especially in an Asian population.

The aim of this study was to evaluate and compare the prevalence of reduced kidney function in MetS and related metabolic components by eGFR using an equation based on eGFRcr, eGFRcys, and combined creatinine-cystatin C (eGFRcr-cys).

METHODS

Subjects

We analyzed data from 3,649 adults aged ≥20 years who participated in a comprehensive health examination at Pusan National University Yangsan Hospital in Yangsan, Korea in 2013. Of the subjects, we excluded those with liver disease (serum levels of aspartate aminotransferase or alanine aminotransferase greater than three times the upper limit of the reference range) (n=19), abnormal serum thyroid stimulating hormone (TSH) levels (TSH <0.27 μIU/mL or TSH >4.2 μIU/mL; n=473), or elevated high-sensitivity C-reactive protein (hs-CRP) level (>10.0 mg/L; n=3). Finally, 3,154 adults (1,871 men, 1,283 women) were enrolled in the study. No subject had an eGFR lower than 15 mL/min/1.73 m^2 or an eGFR higher than 200 mL/min/1.73 m^2.

Informed consent for use of the health screening data analyzed in this study was obtained from all subjects. This study was approved by the Institutional Review Board of Pusan National University Yangsan Hospital (subject no. 05-2016-025).

Anthropometric and biochemical data

Height and weight were measured with subjects barefoot and wearing light weight clothing. BMI was calculated as weight in kilograms divided by the square of height in meters. Waist circumference was measured with a soft tape measure on standing subjects midway between the lowest rib and the iliac crest. Blood pressure was measured on the right arm with subjects in a seated position after a 5-minute rest. Blood specimens were collected from the antecubital vein after an overnight fast. Fasting blood glucose (FBG) was measured by the glucose oxidase method (Synchron LX-20, Beckman Coulter Inc., Fullerton, CA, USA). Concentrations of standard liver enzymes, total cholesterol, high density lipoprotein (HDL) cholesterol, serum triglyceride (TG), γ-glutamyltransferase (GGT), and serum creatinine (Jaffe's kinetic assay) were measured using an autoanalyzer and an enzymatic colorimetric method (Hitachi 7600, Hitachi Ltd., Tokyo, Japan). The serum cystatin C level was measured by the latex agglutination test (Modular P800, Roche Diagnostics, Mannheim, Germany).

Estimating equations

GFR was estimated using equations developed by the CKD-epidemiology collaboration (CKD-EPI). eGFRcr was computed using the CKD-EPI creatinine 2009 equation [10,14]. eGFRcys and eGFRcr-cys [10,15] were used to assess the most accurate estimate of GFR and were computed using the CKD-EPI cystatin C 2012 equation and the CKD-EPI creatinine-cystatin C 2012 equation, respectively [10].

Definitions

Reduced kidney function was defined as eGFR <60 mL/min/1.73 m^2. MetS was defined according to the modified, revised National Cholesterol Education Program Adult Treatment III [16,17] as the presence of three or more of the following criteria: (1) abdominal obesity defined as waist circumference ≥90 cm in men and ≥80 cm in women; (2) impaired fasting glucose as defined by FBG ≥100 mg/dL; (3) high TG as defined by TG ≥150 mg/dL (for conversion to mmol/L, multiply by 0.01129); (4) low HDL as defined by HDL <40 mg/dL in men and <50 mg/dL in women (for conversion to mmol/L, multiply by 0.02586); and (5) blood pressure ≥ 130/85 mm Hg. The homeostasis model assessment of insulin resistance (HOMA-IR) index was calculated using the following formula:

HOMA-IR=[fasting serum insulin (mU/L)×fasting plasma glucose (mmol/L)]/22.5

High insulin resistance was defined as an HOMA-IR score higher than the 75th percentile [18]. Fatty liver index (FLI) was calculated as below [19,20], and an FLI ≥60 was considered to indicate hepatic steatosis:

FLI=[e0.953×loge (TG)+0.139×BMI+0.718×loge (GGT)+0.053×waist circumference–15.745)]/[1+e0.953×loge (TG)+0.139×BMI+0.718×loge (GGT)+0.053×waist circumference–15.745]×100

Statistical analysis

All data are presented as mean±SD for continuous variables. Median values are also indicated in the cases of TG, GGT, and hs-CRP, which had skewed distributions. Categorical variables were compared using the chi-square test. Odds ratio (OR) and corresponding 95% confidence interval (CI) as an estimate of the relative risk of reduced kidney function for the prevalence of MetS were calculated by multiple logistic regression analyses with non-adjusted data (model 1), after adjusting for age (20 to 39, 40 to 59, ≥60 years) and sex (men, women) (model

Table 1. Clinical Characteristics of the Subjects

Variable	Total	Men	Women	*P* value
Number	3,154	1,871	1,283	
Age, yr	53.2±10.2	53.2±10.2	53.2±10.3	0.961
Waist circumference, cm	85.0±9.2	88.4±7.6	79.9±8.9	<0.001
Body mass index, kg/m^2	23.9±3.0	24.5±2.8	23.1±3.2	<0.001
Systolic blood pressure, mm Hg	117.8±13.9	119.2±12.9	115.7±14.9	<0.001
Diastolic blood pressure, mm Hg	77.4±10.4	79.5±10.1	74.4±10.2	<0.001
Fasting blood glucose, mg/dL	98.3±22.5	101.2±24.6	93.9±18.3	<0.001
Insulin, μU/mL	4.97±3.23	5.11±3.47	4.77±2.83	0.003
HOMA-IR	1.24±1.00	1.31±1.09	1.14±0.84	<0.001
Total cholesterol, mg/dL	210.3±38.6	209.7±38.9	211.2±38.1	0.303
LDL, mg/dL	129.1±34.9	130.2±34.6	127.4±35.2	0.029
Triglyceride, mg/dL (median)	129.1±94.4 (106.0)	147.0±102.6 (121.0)	104.6±74.1 (86.0)	<0.001[a]
HDL, mg/dL	55.1±13.7	51.6±12.3	60.1±14.2	<0.001
AST, IU/L	28.1±10.9	30.0±11.7	25.2±9.1	<0.001
ALT, IU/L	26.7±16.2	30.7±17.1	20.9±12.7	<0.001
GGT, IU/L (median)	42.5±46.9 (28.0)	55.2±53.3 (39.0)	24.0±26.0 (18.0)	<0.001[a]
BUN, mg/dL	14.1±3.7	14.6±3.7	13.4±3.7	<0.001
Creatinine, mg/dL	0.86±0.18	0.97±0.14	0.71±0.13	<0.001
Uric acid, mg/dL	5.7±1.5	6.4±1.4	4.7±1.2	<0.001
hs-CRP, mg/dL (median)	0.15±0.35 (0.06)	0.17±0.35 (0.07)	0.12±0.35 (0.05)	<0.001[a]
Cystatin C, mg/L	0.85±0.17	0.89±0.16	0.80±0.18	<0.001
Fatty liver index	33.3±25.6	42.8±24.9	19.3±19.4	<0.001
eGFRcr, mL/min/1.73 m^2	91.2±13.6	88.7±13.3	94.9±13.2	<0.001
eGFRcys, mL/min/1.73 m^2	96.1±18.2	94.9±18.5	97.9±17.5	<0.001
eGFRcr-cys, mL/min/1.73 m^2	94.5±15.4	92.5±15.0	97.5±15.5	<0.001
Metabolic syndrome, *n* (%)	745 (23.6)	503 (26.9)	233 (18.2)	<0.001[b]

Values are expressed as mean±SD unless otherwise indicated. *P* value was calculated by independent *t* test unless otherwise indicated.
HOMA-IR, homeostasis model assessment of insulin resistance; LDL, low density lipoprotein; HDL, high density lipoprotein; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, γ-glutamyltransferase; BUN, blood urea nitrogen; hs-CRP, high-sensitivity C-reactive protein; eGFRcr, estimated glomerular filtration rate (GFR) from creatinine equation; eGFRcys, estimated GFR from cystatin C equation; eGFRcr-cys, estimated GFR from creatinine-cystatin C equation.
[a]Mann-Whitney test; [b]Chi-square test.

2), and after adjusting for age, sex, abdominal obesity (yes, no), and high fasting glucose (yes, no) (model 3), using preserved kidney function (eGFR ≥60 mL/min/1.73 m^2) as the reference category. Statistical analyses were performed with the SPSS version 18.0 (SPSS Inc., Chicago, IL, USA). A probability value less than 0.05 was considered significant.

RESULTS

Clinical characteristics

The prevalence of MetS was 26.9% in men and 18.2% in women. Men showed higher mean levels of serum creatinine (0.97±0.14 mg/dL vs. 0.7±0.1 mg/dL, P<0.001) and serum cystatin C (0.89±0.16 mg/dL vs. 0.80±0.18 mg/dL, P<0.001) than women. Mean eGFRcys was higher than mean eGFRcr (96.1±18.2 mL/min/1.73 m^2 vs. 91.2±13.6 mL/min/1.73 m^2, respectively), with mean eGFRcr-cys falling between them (94.5±15.4 mL/min/1.73 m^2) in total subjects (Table 1).

The prevalence of reduced kidney function by eGFRcr, eGFRcys, and eGFRcr-cys

With an increase in age, the prevalence of reduced kidney function increased and was highest for eGFRcys, intermediate for eGFRcr-cys, and lowest for eGFRcr (9.6% for eGFRcys vs. 5.8% for eGFRcr-cys vs. 4.9% for eGFRcr, P<0.001 in subjects aged ≥60 years) (Table 2). With regard to metabolic parameters, the prevalence of reduced kidney function was significantly increased in subjects with MetS, abdominal obesity, hypertension, high TG, low HDL, and high insulin resistance and was highest for eGFRcys, intermediate for eGFRcr-cys, and lowest for eGFRcr (Table 2).

Prevalence of MetS and its components between subjects with reduced kidney function and those with preserved kidney function by eGFRcr, eGFRcys, and eGFRcr-cys

The prevalence of MetS (34.5% vs. 23.1% in eGFRcr, 36.9% vs. 22.9% in eGFRcys, 36.7% vs. 23.1% in eGFRcr-cys), abdominal obesity (63.6% vs. 45.5% in eGFRcr, 68.9% vs. 45.0% in eGFRcys, 70.0% vs. 45.3% in eGFRcr-cys), hypertension (43.6% vs. 31.2% in eGFRcr, 42.7% vs. 31.1% in eGFRcys, 45.0% vs. 31.2% in eGFRcr-cys), high insulin resistance (41.8% vs. 24.7% in eGFRcr, 39.8% vs. 24.5% in eGFRcys, 38.3% vs. 24.7% in eGFRcr-cys), and hepatic steatosis (29.1% vs. 18.0% in eGFRcr, 27.2% vs. 17.9% in eGFRcys, 26.7% vs. 18.0% in eGFRcr-cys) was significantly increased in subjects with reduced kidney function compared with subjects with preserved kidney function. In case of low HDL, the increased prevalence was only significant in eGFRcys and eGFRcr-cys (16.4% vs. 17.9% in eGFRcr, 32.0% vs. 17.4% in eGFRcys, 30.0% vs. 17.7% in eGFRcr-cys) (Fig. 1).

Table 2. The Prevalence of Reduced Kidney Function by eGFR Based on Creatinine, Cystatin C, and Combined Creatinine-Cystatin C

Parameter	No.	eGFRcr	eGFRcys	eGFR cr-cys	*P* value
Sex					
Men	1,871	39 (2.1)	66 (3.5)	39 (2.1)	0.005
Women	1,283	16 (1.2)	37 (2.9)	21 (1.6)	0.006
Age, yr					
20–39	349	1 (0.3)	0	0	0.367
40–59	2,007	15 (0.7)	26 (1.3)	14 (0.7)	0.087
≥60	798	39 (4.9)	77 (9.6)	46 (5.8)	<0.001
Metabolic syndrome					
No	2,409	36 (1.5)	65 (2.7)	38 (1.6)	0.002
Yes	745	19 (2.6)	38 (5.2)	22 (3.0)	0.022
Abdominal obesity					
No	1,711	20 (1.2)	32 (1.9)	33 (1.5)	0.153
Yes	1,443	35 (2.4)	71 (4.9)	27 (2.7)	<0.001
Hypertension					
No	2,162	31 (1.4)	59 (2.7)	33 (1.5)	0.002
Yes	992	24 (2.4)	44 (4.4)	27 (2.7)	<0.001
High fasting glucose					
No	2,169	34 (1.6)	68 (3.1)	40 (1.8)	<0.001
Yes	985	21 (2.1)	35 (3.6)	20 (2.0)	0.057
High triglyceride					
No	2,286	36 (1.6)	67 (2.9)	40 (1.8)	0.002
Yes	868	19 (2.2)	36 (4.1)	20 (2.0)	0.023
Low HDL					
No	2,589	46 (1.8)	70 (2.7)	42 (1.6)	0.011
Yes	565	9 (1.6)	33 (5.8)	18 (3.2)	<0.001
High insulin resistance					
IR ≤75 percentile	2,366	32 (1.4)	62 (2.6)	37 (1.6)	0.002
IR >75 percentile	788	23 (2.9)	41 (5.2)	23 (2.9)	0.021
Hepatic steatosis					
FLI <30	1,693	21 (1.2)	36 (2.1)	20 (1.2)	0.012
FLI ≥60	574	16 (2.8)	28 (4.9)	16 (2.8)	0.083

Values are expressed as number (%). *P* value was calculated by chi-square test.
eGFR, estimated glomerular filtration rate; eGFRcr, estimated GFR from creatinine equation; eGFRcys, estimated GFR from cystatin C equation; eGFRcr-cys, estimated GFR from creatinine-cystatin C equation; HDL, high density lipoprotein; IR, insulin resistance; FLI, fatty liver index.

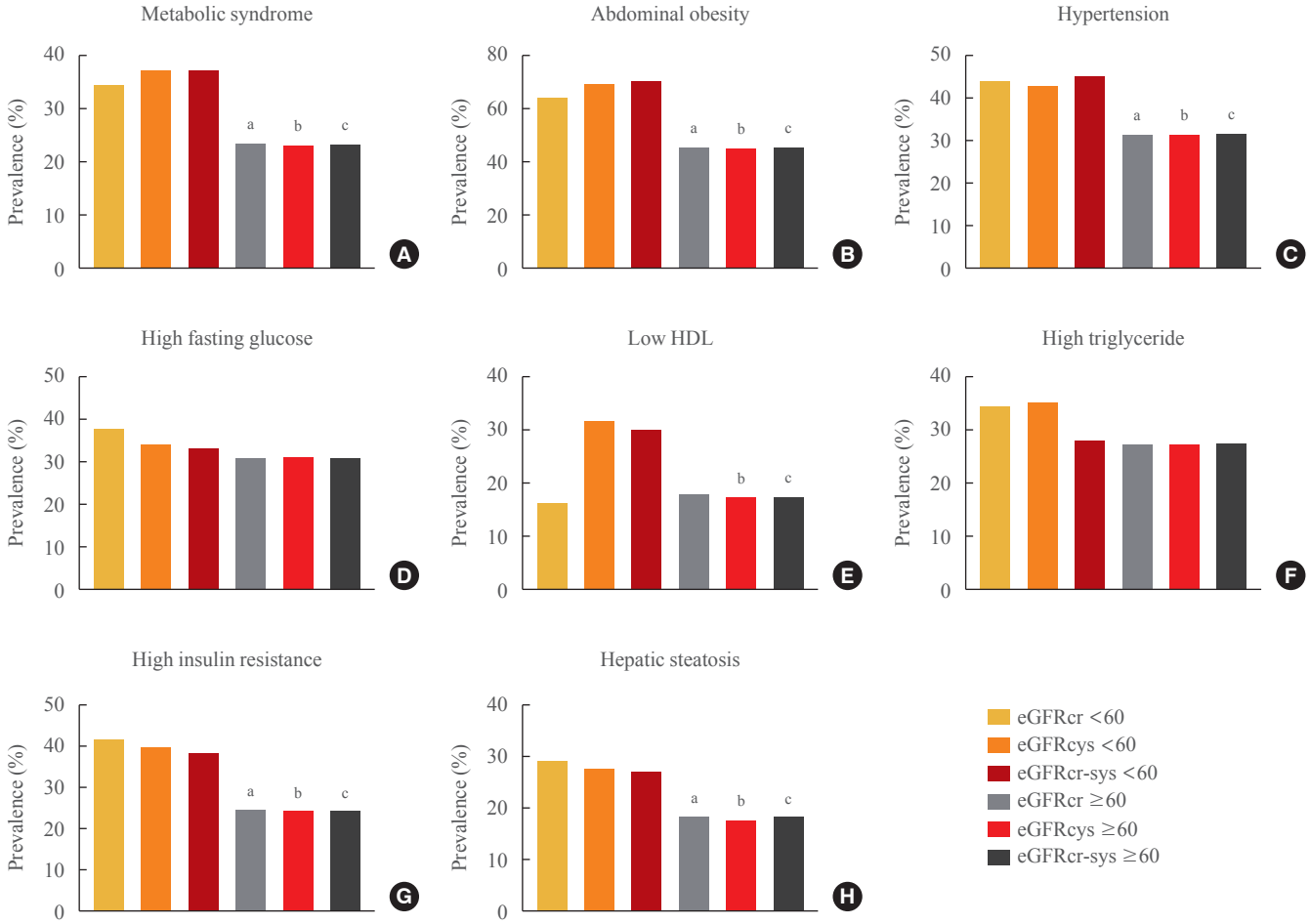

Fig. 1. Prevalence of metabolic syndrome and its components between reduced kidney function and preserved kidney function by estimated glomerular filtration rate (eGFR) based on creatinine (eGFRcr), cystatin C (eGFRcys), and combined creatinine-cystatin C (eGFRcr-cys). (A) Metabolic syndrome, (B) abdominal obesity, (C) hypertension, (D) high fasting glucose, (E) low high density lipoprotein (HDL), (F) high triglyceride, (G) high insulin resistance, and (H) hepatic steatosis. *P* value was calculated by chi-square test. [a]*P*<0.05 vs. eGFRcr <60 mL/min/1.73 m^2; [b]*P*<0.05 vs. eGFRcys <60 mL/min/1.73 m^2; [c]*P*<0.05 vs. eGFRcr-cys <60 mL/min/1.73 m^2.

Table 3. ORs (95% CI) of Reduced Kidney Function by eGFR Based on Creatinine, Cystatin C, and Combined Creatinine-Cystatin C for the Prevalence of Metabolic Syndrome

eGFR	Model 1[a]		Model 2[b]		Model 3[c]	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
eGFRcr ≥60	1 (reference)		1 (reference)		1 (reference)	
eGFRcr <60	1.75 (1.00–3.07)	0.050	1.29 (0.72–2.29)	0.380	1.03 (0.47–2.28)	0.928
eGFRcys ≥60	1 (reference)		1 (reference)		1 (reference)	
eGFRcys <60	1.97 (1.30–2.96)	0.001	1.47 (0.96–2.25)	0.070	1.57 (0.92–2.67)	0.094
eGFRcr-cys ≥60	1 (reference)		1 (reference)		1 (reference)	
eGFRcr-cys <60	1.93 (1.13–3.28)	0.015	1.42 (0.82–2.45)	0.203	1.42 (0.71–2.85)	0.315

eGFR is expressed as mL/min/1.73 m^2.
OR, odds ratio; CI, confidence interval; eGFR, estimated glomerular filtration rate; eGFRcr, estimated GFR from creatinine equation; eGFRcys, estimated GFR from cystatin C equation; eGFRcr-cys, estimated GFR from creatinine-cystatin C equation.
[a]Model 1, non-adjusted; [b]Model 2, adjusted for age and sex; [c]Model 3, adjusted for age, sex, abdominal obesity, and high fasting glucose.

OR (95% CI) of reduced kidney function for prevalence of MetS according to eGFRcr, eGFRcys, and eGFRcr-cys

In comparison of relative risk of reduced kidney function for prevalence of MetS, eGFRcys was the highest (OR, 1.97; 95% CI, 1.30 to 2.96; P=0.001), followed by eGFRcr-cys (OR, 1.93; 95% CI, 1.13 to 3.28; P=0.015), and eGFRcr (OR, 1.75; 95% CI, 1.00 to 3.07; P=0.050) in the non-adjusted model. However, it was attenuated after adjusting for age and sex (model 1) and for age, sex, abdominal obesity, and high fasting glucose (model 2) (Table 3).

DISCUSSION

In this study, we evaluated and compared the prevalence of reduced kidney function in subjects with MetS and its related parameters by eGFRcr, eGFRcys, and eGFRcr-cys in Korean adults. The mean value of eGFRcys (96.1±18.2 mL/min/1.73 m^2) was higher than the mean value of eGFRcr (91.2±13.6 mL/min/1.73 m^2). This finding was consistent with previous large scale studies [13,21] from the National Health and Nutrition Examination Survey in the United States and study [22] using data from an Italian population. This finding might be due to more disperse distributions of eGFRcys than eGFRcr and differences in non-GFR determinants, such as BMI, between serum cystatin C and creatinine levels [21,23].

Similar to previous results [21,24], there was an increase in the prevalence of reduced eGFR with age in our study. Pattaro et al. [22] reported that correlation of eGFRcys and eGFRcr was significantly different in those ≥65 years compared to in those <65 years. Tsai et al. [13] showed that the absolute difference in reduced kidney function prevalence between eGFRcys and eGFRcr was 6.9% in those aged 60 to 80 years and 10.3% in those aged 80 years or older. Similarly, the discrepancy in the prevalence of reduced kidney function between eGFRcys and eGFRcr was largest in those aged ≥60 years (38%) compared to those aged 40 to 59 years (11%) in this study. A possible reason for the increased prevalence of reduced kidney function by eGFRcys than eGFRcr in older age (9.6% vs. 5.8%, P<0.001 in aged ≥60 years respectively) could be that muscle mass and diet are significant non-GFR determinants in the case of creatinine, and eGFRcr may be confounded by cachexia and muscle wasting at older ages [8,13,21]. Furthermore, serum cystatin C is known to have a higher association with mortality and CVD than serum creatinine, especially in older adults [25-27]. Therefore, our finding suggests that cystatin C is a better filtration marker than creatinine in elderly persons [12,15].

In the present study, we demonstrated that the prevalence of reduced kidney function was significantly increased and higher when using eGFRcys than eGFRcr in subjects with MetS, abdominal obesity, hypertension, low HDL, and high insulin resistance. In addition, the prevalence of MetS, abdominal obesity, hypertension, high insulin resistance, and hepatic steatosis was significantly increased in subjects with reduced kidney function compared to those with preserved kidney function, and the percentage was higher according to eGFRcys than eGFRcr. Previous studies have demonstrated that cystatin C had stronger associations than creatinine with systolic blood pressure, weight, and BMI [12], and eGFRcys were more accurate, sensitive, and specific in overweight and obese subjects including visceral obesity compared to eGFR equations based on creatinine [28,29]. It was also shown that cystatin C may be a sensitive marker of small reductions in kidney function (preclinical kidney disease) [30], and eGFRcys may be more reliable than eGFRcr, particularly in subjects with a mild reduction in GFR, in whom changes in serum creatinine are not detected [7,31]. MetS and its related components such as obesity, hypertension, dyslipidemia, and insulin resistance were also important in increased risk of CKD [32]. The possible pathophysiologic mechanism of the strong association between MetS and advancing CKD is that obesity, insulin resistance, hypertension, dyslipidemia, and inflammation result in increased expression of adipocytokines, angiotensin, and inflammatory cytokines and have been identified as causes of renal inflammation and fibrosis [32,33]. Hepatic steatosis is now also considered to be a component of MetS [34] and has an association with advancing CKD [35]. Hence, these earlier reports may provide explanations for our findings. Diabetes is also an important factor, has a strong association with cystatin C [36,37], and is one of the non-GFR determinants for eGFRcys [12]. Tsai et al. [13] showed that the prevalence of reduced kidney function was almost three times higher in persons with diabetes compared to those without diabetes (eGFRcys 22.0% vs. 7.9%, eGFRcr 16.5% vs. 5.8%). However, in our study, there was no significant difference in the prevalence of reduced kidney function compared to preserved kidney function in subjects with high fasting glucose. Additionally, the percentage of reduced kidney function by eGFRcys (3.6%) was higher than that by eGFRcr (2.0%) in those with high fasting glucose, but showed a weak significance. The possible reason for this finding might be that enrolled subjects in this study were mostly healthy and hyperglycemia was defined by fasting glucose only. Therefore, the

number of actual diabetic patients would be very small in our data.

In comparison of relative risk of reduced kidney function for prevalence of MetS, eGFRcys was also higher than eGFRcr in a non-adjusted model, although it was attenuated after adjusting for the non-GFR determinants of both filtration markers including age, sex, abdominal obesity, and high fasting glucose.

The eGFRcr-cys has been suggested as a useful and confirmatory test for CKD, because it performed better than equations based on either of these markers alone [10,15,21]. In this study, the values of eGFRcr-cys were higher than those of eGFRcr but lower than those of eGFRcys in all performed analyses. These results support suggestion that the combination of creatinine and cystatin C is a more accurate tool for eGFR due to over-estimation using cystatin C alone or under-estimation using creatinine alone.

Our study has some limitations. First, serum cystatin C and creatinine were measured only once. Second, direct-measured GFR was not performed, and we could not compare direct-measured GFR with eGFR as determined by the three equations to assess approximation to true kidney function. Third, we could not analyze social data such as exercise, diet, smoking, and medication use for previously diagnosed hypertension and/or hyperlipidemia, which could affect the prevalence of MetS and its related components, and serum levels of creatinine and cystatin C.

In conclusion, the prevalence of reduced kidney function by eGFR was significantly increased in subjects with MetS and its related components. eGFRcys and eGFRcr-cys were superior to eGFRcr in detecting the prevalence of reduced kidney function.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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