Prevalence and Factors Associated with Microalbuminuria and Abnormal Renal Function in Thai Obese Adults

Supawadee Eiamthanasinchai MD*, Nopparat Laowahutanont MD**,
Preyanuj Yamwong MD*, Tanyarat Teerapornlertratt MD**

** Department of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand*
*** Division of Nephrology, Department of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand*

Objective: *To determine the prevalence and factors associated with microalbuminuria and abnormal renal function in Thai obese adults.*

Material and Method: *A cross-sectional study data was collected from 86 patients during October 2010 to February 2011 at the obesity clinic and outpatient department of Siriraj Hospital. Obese patients with body mass index ≥ 25 kg/m^2 participated in the present study. Exclusion criteria were the patients who refused participation or patients with end stage renal disease who were under treatment with hemodialysis or continuous ambulatory peritoneal dialysis. A questionnaire was used for collecting information on demographic data, e.g., age, sex, residence, educational level, underlying diseases and drugs use, family history of obesity, family history of renal disease, smoking, or alcohol drinking; height, weight, body mass index, waist circumference, blood pressure, blood chemistry test and urine analysis were collected. The abnormal function of the kidney was assessed by presence of microalbuminuria or estimated glomerular filtration rate below 90 mL/min/1.73 m^2.*

Results: *The prevalence of microalbuminuria in obese patients was 28% and prevalence of chronic kidney disease stage 2 or more was 22%. Factors associated with microalbuminuria were FBS ≥ 126 mg/dL (OR = 6.2, 95% CI: 1.7-22.1), hyperuricemia (serum uric acid ≥ 7 mg/dL)(OR = 3.2, 95% CI: 1.0-9.8). Factors associated with chronic kidney disease stage 2 or more were age ≥ 55 years (OR = 7.8, 95% CI: 2.5-24.1), Angiotensin II receptor blocker (ARB) use (OR = 4.1, 95% CI: 1.3-12.3) and hyperuricemia (serum uric acid ≥ 7 mg/dL)(OR = 4.5, 95% CI: 1.5-13.5).*

Conclusion: *Early identification of obesity and metabolic syndrome and modifying pattern of life style behavior in obese adults carrying risk factors might be beneficial in preventing and delaying the progression of chronic kidney disease in Thailand.*

Keywords: *Obesity, Microalbuminuria, Chronic kidney disease*

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Obesity is an exploding health problem that affects adults worldwide, including Thailand. The increasing prevalence of obesity is primarily because of an increase in the quantity and energy content of foods ingested and a reduction in regular physical activity that seems to be occurring in most areas of the world. Obesity has proven to be an independent risk factor for chronic kidney disease (CKD) in many studies even after accounting for accompanying confounding variables such as diabetes and hypertension[1]. Obese individuals are at increased risk for metabolic syndrome. Obesity and, often, overweightness are associated with hemodynamic, physiological, structural, and pathological changes in the kidney[2,3]. These changes include an increase in hemodynamics such as an increase in effective plasma flow, glomerular filtration rate, filtration fraction and albuminuria. Anatomical changes associated with obesity include elevation in glomerular size, increased mesangial matrix and mesangial cell proliferation, reduced number and hypertrophy of podocytes, increased width of podocyte foot processes, and a higher prevalence of global sclerosis and segmental sclerosis. There is an increased incidence of focal and segmental glomerulosclerosis type lesions in obese persons and this pathology is often associated with glomerulomegaly[3]. Moreover, obese individuals are also more likely to develop various

Correspondence to:
Yamwong P, Department of Medicine, Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand.
Phone: 08-9775-0773
E-mail: preyanuj.yam@mahidol.ac.th

cancers, including renal cell carcinoma. Individuals with obesity, diabetes mellitus, and hypertension have an increased prevalence of calcium oxalate and uric acid stones as well as elevated serum urate and altered urate metabolism[3]. Obesity is also associated with a higher incidence of proteinuria, increased prevalence of CKD, more rapid progression of renal failure in individuals with a preexisting renal disease, and more frequent development of end stage renal disease (ESRD).

The defining components of metabolic syndrome are high waist circumference, elevated triglycerides, elevated blood pressure (BP), elevated fasting glucose, and reduction in high density lipoprotein cholesterol (HDL-c). An increase in the number of patients with metabolic syndrome worldwide has occurred during the past decade. Using the recommendations for Asians populations detailed in the modified National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III), a previous study reported that 23% of those aged more than 35 years living in Thailand had metabolic syndrome[4]. Epidemiologic studies have also linked metabolic syndrome with an increased risk of CKD[5,6]. To date, there are few studies about the association between obesity and chronic kidney disease in developing countries where genetic and environmental backgrounds are different from those in Western countries[7,8]. The purpose of this study was to determine the prevalence and factors associated with abnormal renal function in obese patients in Siriraj Hospital.

Material and Method

A cross sectional study was conducted during October 2010 to February 2011 in obesity clinic and outpatient department of Siriraj hospital. All obesity patients who were aged more than 18 years and had body mass index of more than 25 kg/m^2 were enrolled in this study. The patients who rejected participation, patients with end stage renal disease (ESRD) who was under treatment with hemodialysis or continuous ambulatory peritoneal dialysis, those with other causes of renal impairment or at risk of having false positive albuminuria were excluded from the present study.

Definitions

Abnormal renal function in the present study is defined as one of the followings:

1. Chronic kidney disease (CKD) stage $\geq$ 2: Estimated glomerular filtration rate (eGFR) < 90 mL/min/1.73 m^2.

An estimation of the GFR was obtained by the Modification of diet in renal disease formula (MDRD) as follows:

$$eGFR\ (mL/min/1.73\ m^2) = 186.3 \times (sCr)^{-1.154} \times (Age)^{-0.203} \times (0.742 \text{ if female})$$

2. Microalbuminuria is defined as urine albumin excretion $\geq$30-300 mg/day.

Body mass index (BMI) is calculated by the formula as follows:

$$BMI = \frac{\text{Body weight (kg)}}{(\text{Height})^2\ (m^2)}$$

Metabolic syndrome is defined using modified NCEP ATP III criteria which includes individuals with three or more of the following five components: (1) abdominal obesity (waist circumference > 90 cm for men, or > 80 cm for women); (2) high TG ($\geq$ 150 mg/dL); (3) low HDL-c (men < 40 mg/dL or women < 50 mg/dL); and (4) High BP (systolic BP $\geq$ 130 or diastolic BP $\geq$ 85 mmHg or treatment of hypertension); and (5) High fasting glucose (FBS $\geq$ 110 mg/dL).

Waist circumference is measured with a flexible tape placed on a horizontal plane at the level of the iliac crest as seen from the anterior view.

Overweight is defined as a BMI of 23 to 24.9 kg/m^2

Obesity is defined as a BMI of $\geq$ 25 kg/m^2

Severe obesity is defined as a BMI $\geq$ 40 kg/m^2

The following items were recorded: age, sex, residence, educational status, comorbid disease (e.g. diabetes mellitus, hypertension, dyslipidemia, coronary artery disease), medication, family history of obesity, family history of kidney disease, history of smoking or alcohol drinking, body weight, height, BMI, waist circumference, blood pressure, complete blood count (CBC), blood chemistry including serum creatinine, electrolytes, albumin, cholesterol, triglyceride, urinalysis, urine albumin excretion (UAE).

Statistical analysis

A prevalence of microalbuminuria and decreased renal function were estimated. Categorical variables were summarized using frequency and percentages while continuous variables were summarized using mean $\pm$ standard deviations (SD) unless otherwise indicated. Comparison between groups with and without microalbuminuria was done using t-test for continuous variables and Chi-square test for categorical variables. The relationship between an elevation of UAE and covariates were assessed

using a simple logistic regression model and reported as crude odds ratios (OR) with 95% confidence intervals (CI). Multivariate binary logistic regression was performed to correct for confounders. Results are presented as adjusted ORs with 95% CIs. P-values were two sided, and $p < 0.05$ was considered to indicate statistical significance. All analyses were performed using SPSS statistical package version 13.0.

Results

From October 2010 to February 2011, 86 patients were included in the present study. The mean age was 46.1 ± 13 years and 58 (67.4%) were female. Of the entire study group, 64 (74.4%) had hypertension, 35 (40.7%) had diabetes mellitus and 43 (50%) had dyslipidemia as co-morbid diseases. Moreover, 48.8% had family history of obesity and 15% had family history of kidney disease. Demographic data are shown in Table 1. The prevalence of microalbuminuria was 28% and prevalence of chronic kidney disease stage 2 or more was 22%. The biochemical characteristics of subjects classified by microalbuminuria status are shown in Table 2. Percentage with diabetes mellitus, BMI ≥ 40 kg/m^2, waist circumference ≥ 120 cm, fasting plasma glucose, HbA_{1C} and serum uric acid were higher ($p < 0.05$) in subjects with microalbuminuria (Table 1 and Table 2).

Comparisons of the clinical features and biochemical data of subjects classified by decreased renal function status are shown in Table 3. There was a significant relationship between the percentage of comorbid disease (diabetes mellitus, dyslipidemia), medication usage (ARB, diuretic, lipid lowering drugs) and serum uric acid level and the prevalence of chronic kidney disease more than or equal to stage 2 (Table 3).

Using multivariate analysis, the predictors for microalbuminuria were higher fasting blood sugar (FBS ≥ 126 mg/dL) (Odd ratios 6.2; 95% CI 1.7-22.1; p = 0.005), and higher serum uric acid level (uric acid ≥ 7 mg/dL) (Odd ratios 3.2; 95% CI 1.0-9.8; p = 0.04) as shown in Table 4. Furthermore, the predictors for presence of CKD more than or equal stage 2 were older age (age ≥ 55 years) (Odd ratios 8.9; 95% CI 2.3-33.9; p = 0.001), higher serum uric acid level (uric acid ≥7 mg/dL)(Odd ratios 8.3; 95% CI 1.8-37; p = 0.006), and the usage of ARB)(Odd ratios 9.4; 95% CI 2-44; p = 0.004) by using

Table 1. Baseline characteristics of the study patients with and without microalbuminuria

Characteristics	Normo-albuminuria (n = 62)	Microalbuminuria (n = 24)	p-value
Age (years)	46.1 ± 13.8	45.8 ± 11.3	0.93
Gender			
Male (%)	20 (32.3%)	8 (33.3%)	
Female (%)	42 (77.7%)	16 (66.7%)	
Waist circumference (cm)	116.6 ± 16.6	124.2 ± 16.9	0.06
≥ 120 cm	10 (30.6%)	13 (54.2%)	0.04
BMI (kg/m^2)	38.6 ± 7.8	42.0 ± 8.6	0.08
≥ 40 (kg/m^2)	10 (30.6%)	13 (54.2%)	0.04
Diabetes Mellitus	20 (32.3%)	15 (62.5%)	0.02
Hypertension	45 (72.6%)	19 (79.2%)	0.53
Dyslipidemia	31 (50%)	12 (50%)	1.00
Drugs used ACEI	12 (19.4%)	10 (41.7%)	0.33
ARB	16 (25.8%)	5 (20.8%)	0.63
Beta-blocker	13 (21%)	8 (33.3%)	0.23
Calcium channel blocker	25 (40.3%)	7 (29.2%)	0.33
Diuretic	11 (17.7%)	9 (37.5%)	0.52
Lipid lowering drugs	24 (38.7%)	12 (50%)	0.34
Family history of obesity	32 (51.6%)	10 (41.7%)	0.40
Family history of kidney disease	9 (14.5%)	4 (16.7%)	0.80
Tobacco use (%)	13 (21%)	4 (16.7%)	0.85

Abbreviations: ACEI, angiotensin converting enzyme inhibitors; ARB, angiotensin II receptor blocker; DM, diabetes mellitus; BMI, body mass index
Continuous data are shown as mean ± SD

Table 2. The biochemical characteristics of the patients

Characteristics	Normoalbuminuria (n = 62)	Microalbuminuria (n = 24)	p-value
FBS (mg/dL)	104.0 ± 26.0	122.3 ± 32.0	0.02
≥ 126 mg/dL, n (%)	6 (9.7%)	9 (40.9%)	0.04
HbA_{1C} (%)	6.1 ± 0.9	6.6 ± 0.9	0.03
Uric acid (mg/dL)	6.2 ± 1.6	7.1 ± 1.54	0.02
≥ 7 mg/dL, n (%)	17 (27.9%)	11 (52.4%)	0.02
Cholesterol (mg/dL)	184.1 ± 38.7	186.0 ± 54.8	0.82
Triglyceride (mg/dL)	155.6 ± 52.1	139.9 ± 64.6	0.07
HDL-c (mg/dL)	51.5 ± 13.7	49.5 ± 15.3	0.57
LDL-c (mg/dL)	108.5 ± 36.1	109.1 ± 46.6	0.95
eGFR ≥ 90 mL/min/1.73 m^2, n (%)	50 (80.6%)	14 (63.6%)	0.11

FBS, fasting blood sugar; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate
Continuous data are shown as mean ± SD

Table 3. Comparisons of the clinical features and biochemical data between the patients with and without CKD more than or equal to stage 2

Characteristics	Normal renal function (n = 67)	CKD stage ≥ 2 (n = 19)	p-value
Age, years	43.5 ± 12.9	56.3 ± 6.7	< 0.001
Gender			
Male	24 (35.8%)	4 (21.1%)	0.22
Female	43 (64.2%)	15 (78.9%)	
Waist circumference ≥ 120 cm	25 (37.3%)	7 (36.8%)	0.97
BMI ≥ 40 kg/m^2	25 (37.3%)	7 (36.8%)	0.97
Diabetes Mellitus	22 (32.8%)	13 (68.4%)	0.01
FBS (mg/dL)	106.8 ± 28.5	117.0 ± 29.3	0.15
HbA_{1c} (%)	6.2 ± 0.9	6.5 ± 1.1	0.13
Hypertension	47 (70.1%)	17 (89.5%)	0.09
Dyslipidemia	28 (41.8%)	15 (78.9%)	0.01
Drugs used ACEI	16 (23.9%)	6 (31.6%)	0.49
ARB	12 (17.9%)	9 (47.4%)	0.01
Diuretic	11 (16.4%)	9 (47.4%)	0.01
Lipid lowering drugs	23 (33.3%)	13 (68.4%)	0.01
Family history of obesity	32 (47.8%)	10 (52.6%)	0.71
Family history of kidney disease	12 (17.9%)	1 (5.3%)	0.17
Tobacco use	13 (19.4%)	4 (21.1%)	0.39
Microalbuminuria, n (%)	14 (21.5%)	8 (42.1%)	0.13
Cholesteral (mg/dL)	186.8 ± 42.6	177.8 ± 46.9	0.43
Triglyceride(mg/dL)	118.8 ± 52.4	134.8 ± 69.3	0.28
HDL-c (mg/dL)	49.4 ± 12.8	56.4 ± 17.2	0.06
LDL-c (mg/dL)	112.8 ± 37.4	94.1 ± 41.9	0.07
Uric acid (mg/dL)	6.1 ± 1.5	7.4 ± 2.0	0.01

multivariate analysis (Table 5).

Discussion

Microalbuminuria is common in Thai obese patients with a prevalence of 28% that comparable with the previous studies[9,10]. Gojaseni et al[9] previously reported a cross-sectional study of 559 nondiabetic hypertensive patients in Thailand in which 17.4% were found to be obesity (BMI ≥ 30 kg/m^2) and found that prevalence of microalbuminuria in obesity was 26.9%

Table 4. Predictors for the presence of microalbuminuria (univariate and multivariate analysis)

Variables	NAU (n = 62)	MAU (n = 24)	Odds ratio^{+} (95% CI)	p-value	Odds ratio^{++} (95% CI)	p-value
FBS ≥ 126 mg/dL	6 (9.7%)	9 (40.9%)	6.5 (1.95-21.4)	0.04	6.2 (1.7-22.1)	0.005
Uric acid ≥ 7 mg/dL	17 (27.9%)	11 (52.4%)	2.9 (1.02-7.92)	0.02	3.2 (1.0-9.8)	0.040
DM, n (%)	20 (32.3%)	15 (62.5%)	3.5 (1.3-9.3)	0.02	-	-
BMI ≥ 40 kg/m^2	19 (30.6%)	13 (54.2%)	2.7 (1.0-7.0)	0.04	-	-
Waist circumference ≥ 120 cm	19 (30.6%)	13 (54.2%)	2.7 (1.0-7.0)	0.04	-	-

NAU, normoalbuminuria; MAU, microalbuminuria
$^{+}$ Odd ratios of univariate analysis of presence of microalbuminuria
$^{++}$ Odd ratios of multivariate analysis of presence of microalbuminuria

Table 5. Predictors for the presence of CKD ≥ stage 2 (univariate and multivariate analysis)

Variables	CKD stage 1 (n = 67)	CKD stage ≥ 2 (n = 19)	Odds ratio^{+} (95% CI)	p-value	Odds ratio^{++} (95% CI)	p-value
Age ≥ 55 years	12 (17.9%)	12 (63.2%)	7.8 (2.5-24.1)	< 0.001	8.9 (2.3-33.9)	0.001
Uric acid ≥ 7 mg/dL	17 (25.8%)	11 (61.1%)	4.5 (1.5-13.5)	0.01	8.3 (1.8-37)	0.006
ARB	12 (17.9%)	9 (47.4%)	4.1 (1.3-12.3)	0.01	9.4 (2.0-44)	0.004
DM, n (%)	22 (32.8%)	13 (68.4%)	4.4 (1.4-13.2)	0.01	-	-
Dyslipidemia, n (%)	28 (41.8%)	15 (78.9%)	5.2 (1.5-17.4)	0.01	-	-
Diuretic	11 (16.4%)	9 (47.4%)	4.5 (1.5-13.8)	0.01	-	-
Lipid lowering drugs	23 (33.3%)	13 (68.4%)	4.1 (1.3-12.3)	0.01	-	-

CKD, chronic kidney disease; ARB, angiotensin II receptor blocker
$^{+}$ Odd ratios of univariate analysis of presence of CKD ≥ stage 2
$^{++}$ Odd ratios of multivariate analysis of presence of CKD ≥ stage 2

and that microalbuminuria is independently associated with obesity (odds ratio = 2.24, 95% CI 1.33-3.76). Menno et al[10] also reported that the prevalence of microalbuminuria in overweight patients was 35% while in obese patients it was 24.7%. It was shown that obesity, by several mechanisms, can lead to glomerular hyperfiltration, and subsequently, developed early histological changes together with the development of albuminuria[11]. Furthermore, microalbuminuria in nondiabetic patients might be part of insulin resistance syndrome. Many factors associated with microalbuminuria (e.g. hypertension, obesity, hyperglycemia, hyperlipidemia) are well known components of insulin resistance syndrome (metabolic syndrome). Therefore, one could argue that insulin resistance is the key pathophysiologic mechanism to link between all of the above-mentioned risk factors and microalbuminuria. In the present study, the predictors for microalbuminuria were hyperglycemia (FBS ≥ 126 mg/dL), HbA_{1C} > 6.5%, severe obesity (BMI ≥ 40 kg/m^2), waist circumference ≥120 cm, and hyperuricemia. When using multivariate analysis, hyperglycemia and hyperuricemia were statistical significance associated with the presence of microalbuminuria.

The prevalence of obese patients with estimated GFR < 90 mL/min/1.73 m^2 in the present study was 22%. There are no data on the prevalence of decreased renal function below CKD stage 2 for comparison. Obesity has proven to be an independent risk factor for CKD in many studies[12-15] even after accounting for accompanying confounding variables such as diabetes and hypertension. For example, in a five year follow-up of a large cohort of 5,897 hypertensive adults participating in the Hypertension Detection and Follow-up Program with no CKD at baseline, the incidence of CKD at year 5 was 28% in the normal BMI group, 31% in the overweight group (BMI 25-30 kg/m^2), and 34% in the obese group (BMI > 30 kg/m^2). The risk in overweight and obese subjects persisted despite excluding participants with diabetes at baseline[12]. Similar observations were made by Foster et al who analyzed the Framingham Health study cohort

and found that obese individuals had a 68% increased risk of developing CKD stage 3[16]. Obesity is not only an important risk factor for CKD, but also accelerates the progression of the disease. Othman et al found the frequency of progression in CKD was significantly higher in obese subjects though the rate of progression was not significantly different from non obese subjects[17]. In the present study, the predictors for CKD stage $\geq$ 2 (eGFR < 90 mL/min/1.73 m^2) were older age (age $\geq$ 55 years) (Odds ratio 8.9; 95% CI 2.3-33.9; p = 0.001), hyperuricemia (serum uric acid $\geq$ 7 mg/dL) (Odd ratios 8.3; 95% CI 1.8-37; p = 0.006), and the usage of ARB) (Odd ratios 9.4; 95% CI 2-44; p = 0.004) by using multivariate analysis that consistent with previous study[18]. Domrongkitchaiporn et al previously reported a cohort study over a period of 12 years (1985 to 1997) in 3,499 employees (aged 35 to 55 yr) of the Electric Generation Authority of Thailand and found that the adjusted odds ratio for future development of decreased kidney function (eGFR < 60 mL/min/1.73 m^2) were 2.57 (1 to 6.8) for systolic hypertension (> 159 mmHg), 1.82 (1.12 to 2.98) for hyperuricemia (> 6.29 mg/dL), 1.68 (1.02 to 2.77) for elevated BMI (> 24.9 kg/m^2) compared with subjects with systolic BP < 140 mmHg, serum uric acid < 4.5 mg/dL and BMI 20.8 to 22.8 kg/m^2[18].

It has been accepted that screening for microalbuminuria is cost-effective for the prevention of progressive kidney disease in diabetic patients. The present study reported the prevalence of microalbuminuria in obese patients to be high enough to make screening worthwhile. Moreover, the screening method is simple and with an acceptable cost. The cardinal intervention for obesity related disease is weight loss[19]. Weight loss ameliorates the hyperfiltration and proteinuria associated with obesity associated disease. Weight loss, through low calorie diet and increased physical activity in severely obese subjects, reduced low grade inflammation (MCP-1, IL-6, TNF-α) and macrophage infiltration in adipose tissue[1]. A meta-analysis of weight loss intervention trials in CKD demonstrated that non-surgical weight loss interventions did not change glomerular filtration rate or creatinine clearance, but did normalize proteinuria. Surgical interventions resulted in normalization of glomerular filtration rate in patients with hyperfiltration and caused significant decreases in microalbuminuria[1,20,21]. Significant weight loss does dramatically decrease proteinuria and improve comorbid cardiovascular risk factors such as improved blood pressure, insulin sensitivity and lipid profile.

The authors conclude that early identification of obesity and metabolic syndrome and modifying pattern of life style behavior in obese adults carrying risk factors might be beneficial in preventing CKD in Thailand.

The present study has some limitations. Firstly, urinary albumin was measured on only a single occasion. Thus, the authors cannot exclude the possibility of false positive/negative test. Secondly, a cross-sectional study design is limited in its ability to estimate causal relationships between risk factors and microalbuminuria as well as to decreased renal function. Further longitudinal studies of the natural course of microalbuminuria in obese patients will answer these questions. Prognostic value of the occurrence of microalbuminuria in this population remains to be determined in prospective cohort studies. Thirdly, the low number of participants in the present study included only individuals that received treatment in our hospital. Fourthly, the authors used eGFR rather than directly measured GFR to define CKD. This process requires that the serum creatinine (sCr) be calibrated by an indirect method to US population data. The relationship between sCr and GFR varies with race, and the modification of diet in the renal disease formula has not been validated in a Thai population. Despite these limitations, our cross-sectional data are consistent with other published series.

Conclusion

Early identification of obesity and metabolic syndrome and modifying pattern of life style behavior in obese adults carrying risk factors might be beneficial in preventing and slowing the progression of chronic kidney disease in Thailand.

Potential conflicts of interest

None.

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การศึกษาความชุกและปัจจัยที่เกี่ยวข้องกับการทำงานที่ผิดปกติของไตในผู้ใหญ่ไทยที่มีภาวะอ้วนในโรงพยาบาลศิริราช

สุภาวดี เอี่ยมธนะสินชัย, นพรัตน์ เลาหุตานนท์, ปรียานุช แย้มวงษ์, ธัญญารัตน์ ธีรพรเลิศรัฐ

วัตถุประสงค์: *เพื่อศึกษาหาความชุกและปัจจัยที่เกี่ยวข้องกับการทำงานที่ผิดปกติของไตในผู้ใหญ่ไทยที่มีภาวะอ้วน*

วัสดุและวิธีการ: *ได้ทำการรวบรวมบันทึกข้อมูลทางคลินิกแบบ cross-sectional study โดยเก็บข้อมูลระหว่างเดือนตุลาคม พ.ศ. 2553 ถึง กุมภาพันธ์ พ.ศ. 2554 ที่คลินิกโรคอ้วนและห้องตรวจผู้ป่วยนอกภาควิชาอายุรศาสตร์ในโรงพยาบาลศิริราช โดยผู้ป่วยต้องมีอายุมากกว่า 18 ปีและมีดัชนีมวลกายมากกว่าหรือเท่ากับ 25 kg/m^2 เกณฑ์การคัดออกจากการศึกษาคือผู้ป่วยที่ปฏิเสธการเข้าร่วมวิจัยหรือผู้ป่วยโรคไตวายเรื้อรังที่ได้รับการฟอกเลือดด้วยเครื่องไตเทียมหรือการฟอกทางหน้าท้องแล้ว โดยใช้แบบสอบถามในการเก็บข้อมูลอาทิเช่น อายุ, เพศ, ภูมิลำเนา, การศึกษา, โรคประจำตัว, ยาที่ใช้ประจำ, ประวัติโรคอ้วนในครอบครัว, ประวัติโรคไตในครอบครัว, การสูบบุหรี่, การดื่มสุรา, ส่วนสูง, น้ำหนัก, ดัชนีมวลกาย, เส้นรอบเอว, ความดันโลหิต, ผลตรวจเลือดทางห้องปฏิบัติการและผลตรวจปัสสาวะ การทำงานที่ผิดปกติของไตในการศึกษานี้ได้รับการประเมินจากการมี microalbuminuria หรือ estimated glomerular filtration rate ที่น้อยกว่า 90 mL/min/1.73m^2*

ผลการศึกษา: *ความชุกของภาวะ microalbuminuria ในผู้ป่วยโรคอ้วนเท่ากับ 28% และความชุกของภาวะ chronic kidney disease stage 2 ขึ้นไปเท่ากับ 22% จากการศึกษานี้พบว่าปัจจัยที่มีผลต่อการเกิด microalbuminuria คือ FBS ≤ 126 mg/dL (OR = 6.2, 95% CI: 1.7-22.1), hyperuricemia (serum uric acid ≤ 7 mg/dL) (OR = 3.2, 95% CI: 1.0-9 .8) ส่วนปัจจัยที่มีผลต่อการเกิด chronic kidney disease ระยะที่ 2 ขึ้นไปคือ อายุที่มากกว่าหรือเท่ากับ 55 ปี (OR = 7.8, 95% CI: 2.5-24.1), การได้รับยา Angiotensin II receptor blocker (ARB) (OR = 4.1, 95% CI: 1.3-12.3) หรือ hyperuricemia (serum uric acid ≤ 7 mg/dL) (OR = 4.5, 95% CI: 1.5-13.5)*

สรุป: *ภาวะอ้วนจัดว่าเป็นปัญหาที่สำคัญซึ่งทำให้เพิ่มความเสี่ยงต่อการเกิดโรคไต การป้องกันภาวะอ้วนส่วนใหญ่สามารถควบคุมได้โดยการปรับปรุงพฤติกรรมสุขภาพ การให้คำแนะนำและการรักษาภาวะอ้วนที่เหมาะสมอาจมีประโยชน์ในการชะลอการดำเนินโรคไปสู่โรคไตวายเรื้อรังในประเทศไทยได้*