The Correlation of Insulin Resistance and Renal Function in Non Diabetic Chronic Kidney Disease Patients [1]

Bancha Satirapoj MD*,
Ouppatham Supasyndh MD*, Apussanee Boonyavarakul MD**,
Lersan Luesutthiviboon MD*, Panbubpa Choovichian MD*

** Division of Nephrology, **Division of Endocrinology, Department of Medicine, Phramongkutklao Hospital*

[1]*Poster presentation at Annual Dialysis Conference 2005, University of Missouri School of Medicine, Columbia, Missouri, February 28-March 2, 2005, Tampa, Florida, USA*

Introduction: *A greater degree of insulin resistance may predispose to renal injury by worsening renal hemodynamics through the elevation of glomerular filtration fraction. However, there are sparse data on the relationship between insulin resistance, glomerular filtration rate (GFR) and body composition in chronic kidney disease (CKD) without diabetes.*

Objectives: *To evaluate the relationship between insulin resistance, total body fat and GFR in CKD without diabetes.*

Material and Method: *The authors screened 84 non-diabetic CKD patients according to the K/DOQI definitions and only 78 patients were enrolled into the study (CKD stages 2-4, GFR between 15 and 90 ml/min/ 1.73 m^2). Insulin resistance was evaluated by homeostasis model assessment (HOMA-IR). Bioelectrical impedance analysis was performed to determine the percentage of total body fat. GFR was calculated by the average of creatinine and urea clearances.*

Results: *The correlation analysis showed that HOMA-IR was positively correlated with percent body fat ($r = 0.32$, $P<0.05$), BMI ($r = 0.46$, $P<0.01$), serum triglyceride (TG) ($r =0.29$, $P<0.01$), and mean arterial pressure ($r =0.25$, $P<0.05$), but not significantly correlated with GFR, age, cholesterol, HDL, uric acid and 24-hr urinary protein.*

Conclusion: *In non-diabetic CKD patients, the independent factor for insulin resistance was the amount of total body fat. The insulin level and HOMA-IR were not dependent on the GFR in the present study.*

Keywords: *Insulin resistance, Body fat, Chronic kidney disease*

J Med Assoc Thai 2005; 88(Suppl 3): S97-104
Full text. e-Journal: *http://www.medassocthai.org/journal*

Insulin resistance is closely associated with atherosclerosis and cardiovascular mortality in the general population[1,2]. Recently, an observational study strongly suggested that insulin resistance was an independent predictor of cardiovascular mortality in a cohort of non-diabetic end-stage kidney disease (ESKD) patients[3]. It is also an underlying cause of type 2 diabetes and is always accompanied with hypertension, central obesity and dyslipidemia, all of which are important risk factors for progression of chronic kidney disease (CKD)[4]. Furthermore, in the absence of overt diabetes or metabolic syndrome, it may cause endothelial dysfunction[5] and then plays a central role in the development of atherosclerotic vascular disease[6,7]. In African Americans, insulin resistance can be detected prior to the clinical expression of

Correspondence to: Satirapoj B, Division of Nephrology, Department of Medicine, Phramongkutklao Hospital, Bangkok 10400, Thailand. Phone: 02-354-7600 ext. 93310, E-mail address: Satirapoj@yahoo.com

hypertension or diabetes[8]. It has also initiated and accelerated the kidney pathology and the risk of microalbuminuria in non diabetes patients by several mechanisms. First, it altered tissue regulatory factors and vasoactive peptides such as angiotensin II, endothelin, and growth factors[9,10]. Secondly, the glomerular hemodynamics has been changed through the elevation of glomerular filtration fraction and resultant glomerular hyperfiltration[12]. In Hisayama s study, hyperinsulinemia was a significant relevant factor of kidney function in the general Japanese population[11].

Impaired tissue sensitivity to insulin is present in ESKD[13-15] and is largely responsible for the abnormal glucose metabolism complicated in this setting. Possible mechanisms accounting for the reduction in insulin-mediated glucose handling include[16]1) increased hepatic gluconeogenesis not normally suppressible following insulin release; 2) reduced hepatic and/or skeletal muscle glucose uptake; and 3) impaired intracellular glucose metabolism. A few clinical studies have noted impaired tissue sensitivity to insulin in nondiabetic patients who had only mild to moderate reductions in kidney function[17-19].

The amount and distribution of adipose tissue is normal in most populations with insulin resistance. However, some patients manifested abdominal obesity. High serum free fatty acid concentrations[20] and increased release of tumor necrosis factor (TNF)-alpha[21] from enlarged adipose cells have been implicated in the pathogenesis of obesity in patients with normal kidney function. A small clinical study showed abdominal adiposity was associated with a deteriorating insulin resistance in subjects on hemodialysis[22]. There are sparse data on the relationship between insulin resistance, glomerular filtration rate (GFR) and total body fat or phase angle in CKD patients without diabetes. The authors performed a cross-sectional study in patients with stage 2 to 4 CKD without diabetes to evaluate the relationship between insulin resistance, GFR, and total body fat or phase angle.

Material and Method

The authors conducted a cross-sectional study at the Out-Patient Clinic of Medicine Department, Phramongkutklao Hospital, Bangkok, Thailand. Eighty-four, non-diabetic CKD patients according to the K/DOQI definitions[23] was screened. Only 78 patients were enrolled into the present study (GFR between 15 and 90 ml/min/1.73 m^2). Six patients were excluded because of GFR was less than 15 ml/min/1.73 m^2. The study protocol was approved by the Ethics Committee of the Phramongkutklao Hospital and College of Medicine and written informed consent was obtained from all patients.

Patients:

The patients as defined criteria and 18 years or older were collected from interview on medical and personal history, such as history of smoking, alcohol consumption, hypertension, family history of diabetes, and use of antihypertensive or lipid lowering medications. All patients were assigned to a physical examination including blood pressure and anthropometry. Body weight, height, waist circumference and hip circumference were measured according to a standard protocol. Body mass index (BMI) and waist-hip ratio (WHR) were calculated. Bioelectrical impedance analyses was performed to determine the percentage of total body fat (%BF) by using monofrequency bioelectrical impedance analysis, BIA (Maltronfi, England) at single frequency: 0.8 MA, 50 KHz.

Biochemical Measurements:

Blood samples were collected in the morning after an overnight fast of at least 12 hours. Fasting plasma glucose level was measured by the glucose oxidase method. Fasting plasma insulin levels was analyzed by electrochemiluminescence immunoassay (Roche Elecsys 2010, USA). Serum and urinary creatinine levels were measured by Jaffe s method and urea levels were determined by kinetic test with urease method. GFR was calculated using a 24-hour urinary excretion of average creatinine and urea clearance then corrected with body surface area. Creatinine and urea clearance were calculated with the standard formula.

Assessment of Insulin Resistance Using HOMA-IR

The value of insulin resistance was obtained by homeostasis model assessment (HOMA-IR). HOMA-IR was calculated using the following formula:

HOMA-IR = [fasting plasma insulin (μU/ml) x fasting plasma glucose (mmol/L)] ÷ 22.5

HOMA-IR has a close correlation with the insulin sensitivity index by the standard euglycemic hyperinsulinemic clamp as shown by Mathew et al[24]. This index has been studied in patients with CKD[25]. Assuming that normal subjects aged <35 year with normal weight have an insulin resistance of 1.

Statistical Analysis

The SPSS software was used for statistical analysis. After testing for normality of data distribu-

tion, the data of quartile of insulin resistance group was compared using one way analysis of variance (ANOVA) with Bonferroni correction for multiple comparisons. Relationships between the variable parameters were evaluated with Pearson s correlation coefficient. All data are presented as mean ± SD. Differences were considered as statistical significant at a p level of 0.05.

Results

Baseline characteristics according to the K/DOQI definitions of 78 non-diabetic CKD patients are shown in Table 1. There were 69.2% males and the mean age of 59 years old. About 20.3 percent had a history of smoking and 15.2 percent had a family history of diabetic mellitus. The major cause of CKD in the present study was hypertension (64.1%). Mean HOMA-IR of CKD stage 2, 3 and 4 were 2.01 ± 0.85, 2.06 ± 1.48 and 2.12 ± 1.35, respectively. Mean fasting plasma insulin of CKD stage 2, 3 and 4 were 8.7 ± 5.3, 9.2 ± 6.5 and 9.2 ± 5.6 mg/dL, respectively. No statistical significant difference was noted in HOMA-IR and fasting plasma insulin levels between CKD groups (Table 2).

The correlation analysis (Table 3) demonstrated that HOMA-IR was positively correlated with % BF (r = 0.32, P<0.05), BMI (r =0.46, P<0.01), serum triglyceride (r = 0.29, P<0.01), and MAP (r = 0.25, P<0.05), but not significantly correlated with age, GFR, serum cholesterol, serum HDL cholesterol, serum uric acid and 24-hour urine protein. Mean difference of % BF, BMI, and serum triglyceride between the highest quartile and lowest quartile of HOMA-IR were 7.26 (95%CI; 1.50-13.02, p <0.05)

Table 1. Patient characteristics according to the K/DOQI definitions of 75 non- diabetic CKD patients

	Stage 4 (N=42) (mean±SD)	Stage 3 (N=31) (mean±SD)	Stage 2 (N=5) (mean±SD)	Total(N=78) (mean±SD)
Age (yr)	62.2±13.9	59.5±14.6	52.0±13.9	59.5±14.6
Gender M (%)	29 (69.0)	20 (64.5)	5 (100)	54 (69.2)
MAP (mmHg)	103.3±11.2	101.2±10.3	106.0±18.5	102.6±11.3
BMI (kg/m^2)	23.8±4.2	24.4±3.5	22.9±3.0	23.9±3.8
WHR	1.12 ± 0.10	1.10 ± 0.08	1.11 ± 0.05	1.11 ± 0.09
Primary renal disease				
-HTN (%)	28 (66.7)	19 (61.3)	3 (60.0)	50 (64.1)
-GN (%)	3 (7.5)	7 (22.6)	2 (40.1)	12 (15.4)
-Interstitial nephritis (%)	4 (9.5)	3 (9.7)	-	7 (9.0)
Family of DM (%)	5 (11.9)	6 (19.4)	1 (20.0)	12 (15.2)
Phase angle	5.30 ± 1.00	5.84 ± 1.04	6.17 ± 0.49	5.59 ± 1.03
% BF	29.6±6.7	30.1±6.6	27.8±5.6	27.8±5.6
Smoking (%)	7 (17.1)	6 (18.2)	3 (60.0)	16 (20.3)

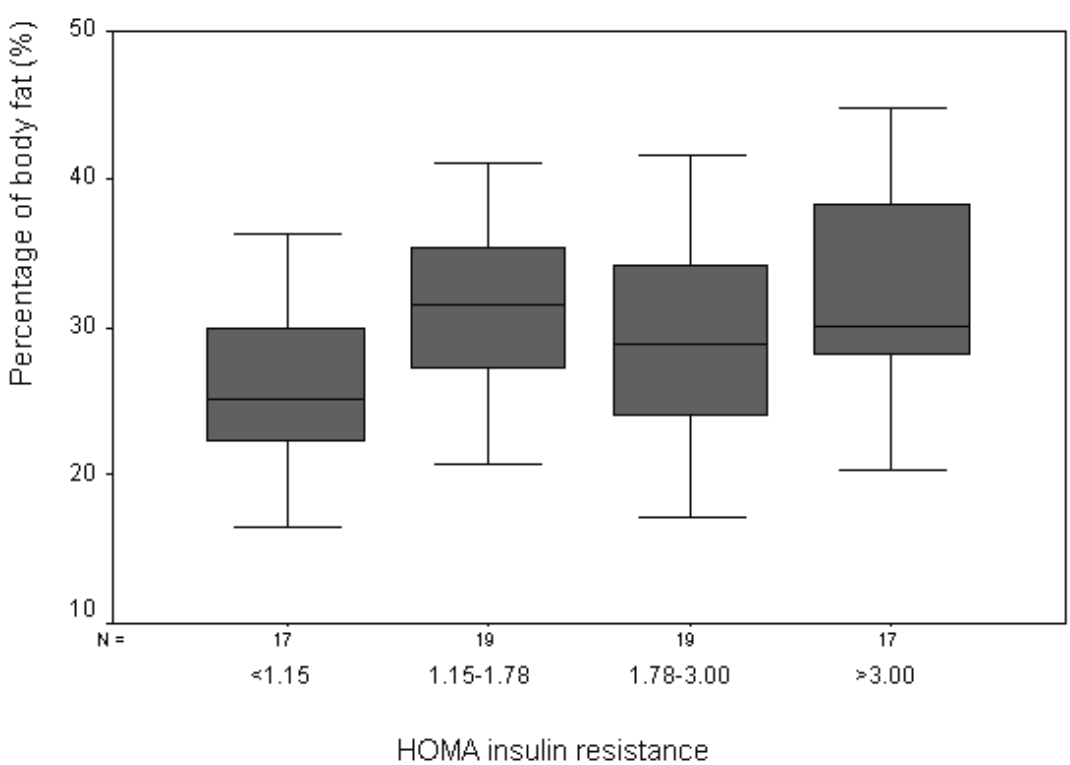
Table 2. Biochemical variables

Serum Parameters	Stage 4 (N=42) (mean±SD)	Stage 3 (N=31) (mean±SD)	Stage 2 (N=5) (mean±SD)	Total (N=78) (mean±SD)
Chlolesterol (mg/dL)	186.4±42.1	197.4±43.2	204.6±16.5	191.9±41.5
LDL (mg/dL)	116.27 ± 38.32	118.52 ± 39.18	137.80 ± 34.48	118.57 ± 38.34
HDL (mg/dL)	50.6±13.1	48.9± 16.4	48.0±10.5	49.6±14.3
Triglyceride (mg/dL)	132.1±71.7	151.3±158.0	121.2±96.4	139.0±114.3
Uric acid (mmol/L)	522.92 ± 116.78	460.40 ± 156.39	452.80 ± 124.91	492.37 ± 137.30
Insulin (U/mL)	9.2±5.6	9.2±6.5	8.7±5.3	9.2±5.8
FBS (mmol/L)	5.1±0.5	5.0±0.5	5.1±0.6	5.1±0.5
HOMA-IR	2.12±1.35	2.06±1.48	2.01±0.85	2.12±1.35

Table 3. Correlation coefficients of insulin resistance and other variable factors

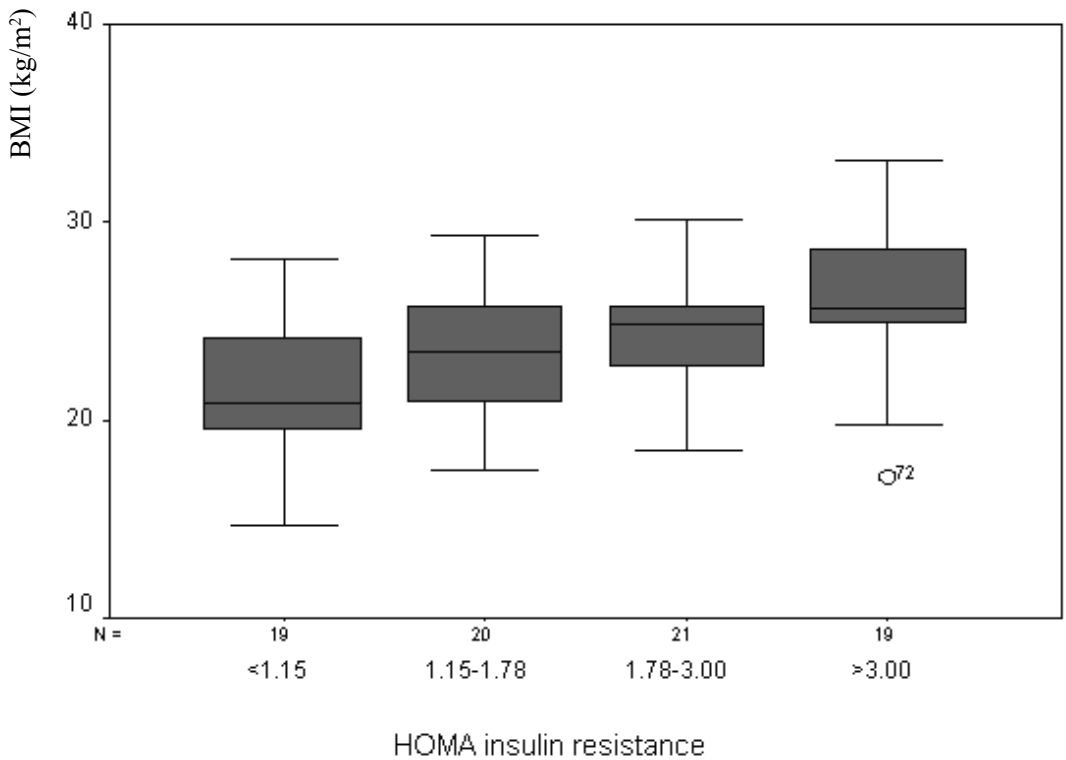
Parameters	Insulin resistance	
	HOMA-IR (N=78)	Insulin Levels
Age (yr)	0.07	0.04
MAP (mmHg)	0.25*	0.22*
BMI (kg/m^2)	0.46**	0.49**
WHR	- 0.42**	- 0.42**
Cholesterol (mg/dL)	0.01	0.01
HDL (mg/dL)	- 0.07	- 0.23*
Triglyceride (mg/dL)	0.29**	0.36**
Uric acid (mmol/L)	0.15	0.17
Phase angle (d)	0.36*	0.36**
% BF	0.32*	0.35**
Urine protein 24 hr (g)	0.01	0.03
GFR (ml/min/1.73 m^2)	- 0.01	- 0.03

* Correlation is significant at the .05 level
** Correlation is significant at the .01 level



Mean difference in highest quartile and lowest quartile 7.26 (95%CI 1.50-13.02) ($p < 0.05$)

Fig. 1 Percentage of body fat of non diabetes CKD according to quartiles of HOMA-IR



Mean difference in highest quartiles and lowest quartiles 5.46 (95%CI2.40-8.48) ($p < 0.05$)

Fig. 2 Body mass index of non diabetes CKD according to quartiles of HOMA-IR

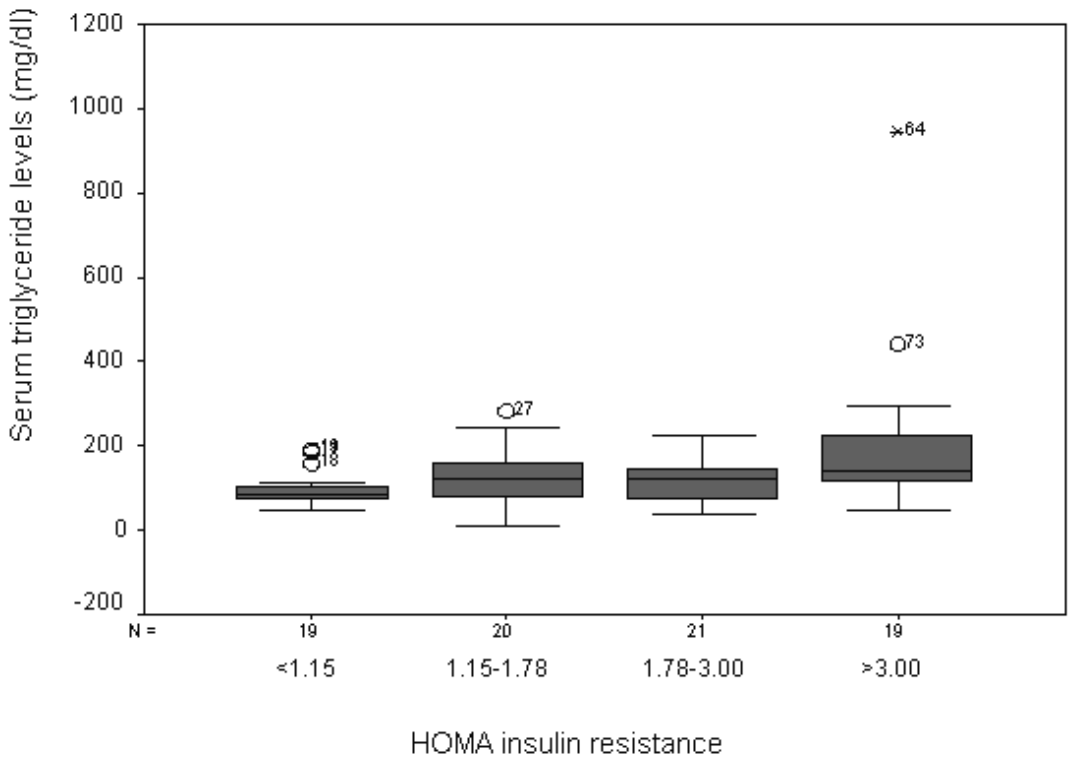
(Fig. 1), 5.46 (95%CI; 2.40-8.48, p <0.05) (Fig. 2), and 123.4 (95%CI; 28.3-218.5, p <0.05) (Fig. 3), respectively. Mean difference of GFR between the highest quartile and lowest quartile of HOMA-IR was not statistically significant (0.51; 95%CI-12.72-13.75, p>0.05).

Discussion

In the present study, insulin resistance using HOMA method was positively correlated with total body fat content, BMI, serum triglyceride, and blood pressure. These were supportedly other previous studies[22]. Conversely, declined renal function had no effect on insulin sensitivity.

Obesity is closely associated with insulin resistance and is an unfavourable factor for cardiovascular disease in the general population. The present study showed the indices of obesity such as % BF, BMI and phase angle, were associated with insulin resistance in non-diabetic CKD patients (GFR 15-90 ml/min/1.73^2). Lee P et al also found that abdominal adiposity was associated with diminished insulin sensitivity in subjects undergoing maintenance hemodialysis[22]. The possible mechanism of insulin resistance in both obese and dialysis patients may involve secreted cytokines by adipocytes (adipocytokines). Adipocytokines which can induce insulin resistance include TNF-alpha[21] and leptin[26]. In addition, high serum free fatty acid concentrations[20] may be implicated in the pathogenesis of such diseases. Hyperinsulinemia may contribute to dyslipidemia by increasing the synthesis of VLDL by the liver[27], resulting in increased concentrations of triglycerides. The authors demonstrated an existing statistical significantly difference in serum triglyceride level between the highest and lowest quartile of HOMA-IR groups. Data from the present study regarding serum triglyceride levels were in good agreement with other clinical studies [18, 28].

Several studies have clearly shown that the sensitivity to the action of insulin with respect to glucose metabolism is markedly impaired in CKD[13]. Mechanisms may contribute to insulin resistance in CKD including defects at postreceptor level of insulin



Mean difference between highest quartiles and lowest quartiles = 123.4 (95%CI 28.3-218.5) ($p < 0.05$)

Fig. 3 Serum triglycerides of non diabetes CKD according to quartiles of HOMA-IR

action in muscle, adipose, and liver tissues. The defects are primarily localized to glucose uptake and metabolism by these insulin-sensitive tissues[15,16]. Significantly impaired insulin sensitivity is present early in the course of CKD[29]. However, in early CKD patients, factors other than impaired kidney function per se, such as dyslipidemia and adverse effects of medications are likely to cause or contribute to insulin resistance.

It has been suggested that insulin resistance is associated with hypertension. As in the present study, subjects manifested with increasing mean arterial pressure and also had a high degree of HOMA-IR. At least two mechanisms favor a causal relationship between insulin resistance and the development of hypertension: the first mechanism is through an alteration of renal sodium handling, where in hyperinsulinemia may promote sodium reabsorption through an acute direct effect on renal tubules[30], and the second mechanism is that hyperinsulinemia activates sympathetic nervous system activity in some patients to raise cardiac output and stimulate peripheral vasoconstriction[31]. Additionally, the renal hemodynamic changes can facilitate renal salt and water conservation and expand blood volume which leads to a salt-sensitive hypertensive state in insulin resistant individuals[12].

There are a few limitations in the present study. First, the number of patients was relatively small and statistical power may not be adequate enough to detect the relationship between insulin resistance and the decline of GFR. Second, the cross-sectional study design cannot determine to draw inferences regarding causality among insulin resistance in CKD patients. Prospective clinical studies may provide a better context to define the evolution of the insulin sensitivity with time in patients with progressive renal disease.

In conclusion, the present study showed the independent factor for insulin resistance in non-diabetic CKD patient is the amount of total body fat, as assessed by % BF, and BMI. The insulin level and HOMA-IR are not dependent on the levels of GFR in the present study.

Acknowledgments

The authors wish to thank the Department of Medicine, Phramongkutklao Hospital and Jayanton Patumanond M.D., M.P.H., D.Sc. for his statistical advice.

References

1. Eschwege E, Richard JL, Thibult N, Ducimetiere P, Warnet JM, Claude JR, et al. Coronary heart disease mortality in relation with diabetes, blood glucose and plasma insulin levels. The Paris Prospective Study, ten years later. Horm Metab Res Suppl 1985; 15: 41-6.
2. Orchard TJ, Eichner J, Kuller LH, Becker DJ, McCallum LM, Grandits GA. Insulin as a predictor of coronary heart disease: interaction with apolipoprotein E phenotype. Ann Epidemiol 1994; 4: 40-5.
3. Shinohara K, Shoji T, Emoto M, Tahara H, Koyama H, Ishimura E, et al. Insulin resistance as an independent predictor of cardiovascular mortality in patients with end-stage renal disease. J Am Soc Nephrol 2002; 13: 1894-900.
4. RAO Goutham. Insulin resistance syndrome. American Family Physician 2001; 63: 1159-63.
5. Hsueh WA, Quinones MJ. Role of endothelial dysfunction in insulin resistance. Am J Cardiol 2003; 92(4 A): 10J-17J.
6. Ginsberg HN. Insulin resistance and cardiovascular disease. J Clin Inves 2000; 106: 453-8.
7. DeFronzo R, Ferranimi E. Insulin resistance: A multifaceted syndrome responsible for NIDDM, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetics Care 1991; 14: 173-94.
8. Falkner B. Insulin resistance in African American. Kidney Int 2003; 63(Suppl 83): S27-30.
9. Mykkanen L, Zaccaro DJ, Wagenknecht LE, Robbins DC, Gabriel M, Haffner SM. Microalbuminuria is associated with insulin resistance in nondiabetic subjects. Diabetes 1998; 47: 793-800.
10. Fujikawa R, Okubo M, Egusa G, Kohno N. Insulin resistance precedes the appearance of albiminuria in non-diabetic subjects: 6 years follow-up study. Diabetes Res Clin Pract 2001; 53: 99-106.
11. Kubo M, Kiyohara Y, Kato I, Iwamoto H, Nakayama K, Hirakata H, et al. Effect of hyperinsulinemia on renal function in a general Japanese population: The Hisayama study. Kidney Int 1999; 55: 2450-6.
12. Dengel DR, Goldberg AP, Mayuga RS, Kairis GM, Weir MR. Insulin resistance, elevated glomerular filtration, and renal injury. Hypertension 1996; 28: 127-32.
13. DeFronzo RA, Tobin JD, Rowe JW, Andres R. Glucose intolerance in uremia: Quantification of pancreatic beta cell sensitivity to glucose and tissue sensitivity to insulin. J Clin Invest 1978; 62: 425-35.
14. Hong SY, Yang DH. Insulin levels and fibrinolytic activity in patients with end stage renal disease. Nephron 1994; 68: 329-33.
15. Mak RH, DeFronzo RA. Glucose and insulin metabolism in uremia. Nephron 1992; 61: 377-82.
16. Alvestrand A. Carbohydrate and insulin metabolism in renal failure. Kidney Int 1997; 52(Suppl 62): S48-52.
17. Vareesangthip K, Tong P, Wilkinson R, Thomas TH. Insulin resistance in adult polycystic kidney disease. Kidney Int 1997; 52: 503-8.
18. Fliser D, Pacini G, Engelleiter R, Kautzky-Willer A, Prager R, Franek E, et al. Insulin resistance and hyperinsulinemia are already present in patients with incipient renal disease. Kidney Int 1998; 53: 1343-7.
19. Kato Y, Hayashi M, Ohno Y, Suzawa T, Sasaki T, Saruta T. Mild renal dysfunction is associated with insulin resistance in chronic glomerulonephritis. Clin Nephrol 2000; 54(5): 366-73.
20. Bjorntorp P. Adipose tissue as a generator of risk factors for cardiovascular disease and diabetes. Arteriosclerosis 1990; 10: 493.
21. Hotamisligil GS, Shargill NS, Spiegelman BM. Adipose expression of tumor necrosis factor-alpha: Direct role in obesity-linked insulin resistance. Science 1993; 259: 87.
22. Lee P, O Neal, Murphy B, Best J. The role of abdominal adiposity and insulin resistance in dyslipidemia of chronic renal failure. Am J Kidney Dis 1997; 29: 54-6.
23. National Kidney Foundation Kidney Disease Outcome Quality Initiative (K/DOQI) Advisory Board. K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification, and stratification. Kidney Disease Outcome Quality Initiative. Am J Kidney Dis 2002; 39(Suppl2): S32-33.
24. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: Insulin resistance and beta-cell function from fasting plasma glucose and insulin concentration in man. Diabetologia 1985; 28: 412-9.

25. Shoji T, Emoto M, Nishizawa Y. HOMA index to assess insulin resistance in renal failure patients. Nephron 2001; 89:348-9.
26. Kellerer M, Rett K, Renn W, Groop L, Haring HU. Circulating TNF-alpha and leptin levels in offspring of NIDDM patients do not correlate to individual insulin sensitivity. Horm Metab Res 1996; 28: 737-43.
27. Tobey TA, Greenfield M, Kraemer F, Reaven GM. Relationship between insulin resistance, insulin secretion, very low density lipoprotein kinetics and plasma triglyceride levels in normotriglyceridemic man. Metabolism 1981; 30: 165-171A.
28. Kobayashi S, Maesto K, Moriya H, Ohtake T, Ikeda T. Insulin resistance in patients with chronic kidney disease. Am J Kidney Dis 2005; 45: 275-80.
29. Stenvinkel P, Ottosson-Seeberger A, Alvestrand A. Renal hemodynamics and sodium handing in moderate renal insufficiency: The role of insulin resistant and dyslipidemia. J Am Soc Nephrol 1995; 5: 1751-60.
30. Defronzo RA. The effect of insulin on renal sodium metabolism. Diabetologia 1981; 21: 165-71.
31. Pratley RE, Dengel DR, Hagberg JM, Goldberg AP. Insulin resistance, hyperinsulinemia, and increased sympathetic nervous system activity associated with hypertension improve with diet and exercise. Diabetes 1994; 43 (Suppl1): 46A.
32. Lovejoy J, Windhauser M, Rood J, de la Bretonne JA. Effect of a controlled high fat versus low fat diet on insulin sensitivity and leptin levels in Africa-American and Caucasian women. Metabolism 1998; 47: 1520-4.

ความสัมพันธ์ของภาวะดื้อต่ออินซูลินกับระดับการทำงานของไตในผู้ป่วยโรคไตเรื้อรังที่ไม่ได้เป็นเบาหวาน

บัญชา สถิระพจน์, อุปถัมภ์ ศุภสินธุ์, อภัสนี บุญญาวรกุล, เลอสรรพ์ ลือสุทธิวิบูลย์, พรรณบุปผา ชูวิเชียร

บทนำ: *ภาวะดื้อต่ออินซูลินพบมีความสัมพันธ์กับ โรคเบาหวานชนิดที่ 2, โรคความดันโลหิตสูง, โรคอ้วน และโรคไขมันในเลือดสูงซึ่งทั้งหมดเป็นปัจจัยเสี่ยงต่อการเสื่อมหน้าที่ของผู้ป่วยโรคไตเรื้อรัง เมื่อภาวะดื้อต่ออินซูลินมีระดับเพิ่มขึ้นจะมีผลเพิ่มความดันหลอดเลือดในกรวยไต ส่งผลให้การทำงานของไตลดลง ขณะที่ข้อมูลแสดงความสัมพันธ์ของภาวะดื้อต่ออินซูลินกับระดับการทำงานของไต ในผู้ป่วยโรคไตวายเรื้อรังที่ไม่ได้เป็นเบาหวานค่อนข้างน้อย*

วัตถุประสงค์: *เพื่อแสดงความสัมพันธ์ของภาวะดื้อต่ออินซูลินกับระดับการทำงานของไต ในผู้ป่วยโรคไตวายเรื้อรังที่ไม่ได้เป็นเบาหวานรวมทั้งศึกษาถึงปัจจัยอื่นๆ ที่คาดว่าจะมีผลต่อระดับการทำงานของไต*

วัสดุและวิธีการ: *เป็นการศึกษาแบบเชิงพรรณนา ผู้ป่วยที่ได้รับการวินิจฉัยว่าเป็นผู้ป่วยไตเรื้อรังที่มีค่าอัตราการกรองของหน้าที่ไต (glomerular filtration rate, GFR) อยู่ระหว่าง 15 ถึง 89 มิลลิลิตรต่อนาทีต่อ 1.73 ตารางเมตร เข้ารับการตรวจรักษาพยาบาลที่โรงพยาบาลพระมงกุฎเกล้าจำนวน 79 ราย ผู้ป่วยทุกรายได้รับการตรวจภาวะดื้อต่ออินซูลินด้วยวิธี Homeostasis Model Assessment (HOMA-IR), ตรวจวัดเปอร์เซ็นต์ไขมันในร่างกายด้วยเครื่องตรวจวัดไขมัน (bioelectrical Impedance analyzer, BIA) และเก็บปัสสาวะ 24 ชั่วโมงส่งตรวจเพื่อคำนวณหาค่า GFR จากค่าเฉลี่ยของ urea clearance และ creatinine clearance*

ผลการศึกษา: *ภาวะดื้อต่ออินซูลินมีความสัมพันธ์เชิงบวกกับ ค่าดัชนีมวลกาย, ระดับไขมันไตรกรีเซอไรด์, ค่าความดันโลหิตสูงเฉลี่ย (Mean arterial blood pressure) และร้อยละของไขมันในร่างกายอย่างมีนัยสำคัญทางสถิติ ขณะที่ไม่พบความสัมพันธ์กับระดับค่า GFR, อายุ, ระดับไขมันโคเลสเตอรอล, ระดับไขมัน HDL, ค่ายูริกในเลือด และ โปรตีนในปัสสาวะ 24 ชั่วโมง*

สรุป: *จากการศึกษานี้พบว่าภาวะอ้วนจากร้อยละของไขมันในร่างกาย, ค่าดัชนีมวลกาย, และระดับไขมันไตรกรีเซอไรด์น่าจะมีปัจจัยเสี่ยงต่อภาวะดื้อต่ออินซูลินในผู้ป่วยไตวายระยะต้น ขณะที่ไม่พบความสัมพันธ์ระหว่างระดับการ ทำงานของหน้าที่ไตกับภาวะดื้อต่ออินซูลินในผู้ป่วยโรคไตเรื้อรัง*