Insulin Resistance in Dialysis versus Non Dialysis End Stage Renal Disease Patients without Diabetes

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Objective: *Insulin resistance is frequently recognized in uremic patients and is a predictor of cardiovascular mortality in end stage renal disease (ESRD) patients. However, sparse data are available regarding the effects of different methods of renal dialysis on insulin resistance in ESRD without diabetes. The present study was conducted to evaluate the levels of insulin resistance in dialysis versus non dialysis ESRD patients without diabetes.*

Material and Method: *A cross-sectional study was carried out in 45 non diabetic ESRD patients including continuous ambulatory peritoneal dialysis (CAPD), hemodialysis (HD), and non dialysis ESRD patients. The value of insulin resistance was obtained by homeostasis model assessment (HOMA). Estimation of the glomerular filtration rate (GFR) was obtained by the four-variable Modification of Diet in Renal Disease equation and ESRD was defined when GFR was below 15 ml/min/1.73 m^2.*

Results: *Non diabetic ESRD patients were studied: 12 patients on CAPD treatment for 67.4 months, 18 patients on HD treatment for 89.3 months, and 15 patients on conservative treatment. HOMA scores (CAPD 5.4 $\pm$ 2.3, HD 6.0 $\pm$ 1.9 vs. non dialysis 1.5 $\pm$ 0.9, $p < 0.05$) and fasting plasma insulin levels (CAPD 21.9 $\pm$ 7.7 μU/mL, HD 19.5 $\pm$ 8.4 μU/mL vs. non dialysis 4.4 $\pm$ 2.5 μU/mL, $p < 0.05$) of the CAPD and HD groups were significantly higher than the non dialysis ESRD group, with no significant differences observed between CAPD and HD groups. However, fasting plasma glucose was significantly lower in the HD group than the CAPD and non dialysis ESRD groups (CAPD 98.2 $\pm$ 10.6 mg/dL, non dialysis 93.0 $\pm$ 11.5 mg/dL vs. HD 76.2 $\pm$ 7.8 mg/dL, $p < 0.05$). All groups showed no significant differences for blood pressure, body weight, body mass index, fat free mass, body fat, and serum levels of albumin, total cholesterol, LDL-cholesterol, HDL-cholesterol, and triglycerides.*

Conclusion: *Impaired insulin sensitivity in both dialysis groups after long term dialysis was still higher than that of the non dialysis ESRD group. However, no significant differences were noted between CAPD and HD treatments.*

Keywords: *Insulin resistance, Hyperinsulinemia, End stage renal disease, Hemodialysis, Peritoneal dialysis*

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Insulin resistance is an independent predictor of cardiovascular mortality in non diabetic end stage renal disease (ESRD) patients[1]. Impaired insulin sensitivity in the absence of overt diabetes or metabolic syndrome plays a central role in the development of atherosclerotic vascular disease[2]. A line of evidence suggests that insulin resistance is involved in the pathogenesis of endothelial dysfunction, hypertension, and atherosclerosis frequently observed in kidney disease, and could induce cardiovascular complications, the most significant causes of morbidity and mortality in ESRD patients. Several clinical studies have noted impaired tissue sensitivity to insulin in diabetic kidney disease[3] and non diabetic patients exhibiting only mild to moderate reductions in renal function[4-6] and in ESRD[7,8].

Impaired insulin sensitivity has been recognized in uremic patients for many years[9,10]. Hypertension, dyslipidemia, obesity, nutritional status, and metabolic factors are widely-known to play important roles in the development of insulin resistance and altered glucose metabolism[11]. In addition, in uremic patients, previous studies reported that

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treatment with hemodialysis (HD), active vitamin D, erythropoietin, and angiotensin receptor blocker can improve the insulin insensitivity[7,12-14], but the outcomes were not confirmed by others[15,16]. Moreover, dialysis might only partly correct glucose disturbances. Indeed, sparse data exist regarding the effects of different methods of renal dialysis on insulin resistance in ESRD without diabetes. The present cross-sectional study was carried out in non diabetic patients with dialysis and non dialysis ESRD to evaluate the different degrees of insulin resistance.

Material and Method

This was a cross-sectional study in 45 non diabetic ESRD or chronic kidney disease (CKD) stage V patients according to the K/DOQI definitions[17] that used glomerular filtration rate (GFR) less than 15 ml/min/1.73 m^2. All patients were well and without any uremic symptoms before being enrolled in the present study. The patients with defined criteria and 18 years or older were collected and interviewed on medical history, use of antihypertensive medication and lipid lowering medication. The ESRD patients were assigned by modalities of dialysis or non dialysis based on individual preferences. No statistically significant differences were found between subject groups with respect to age, sex, and comorbid diseases. The continuous ambulatory peritoneal dialysis (CAPD) regimen comprised four exchanges per day of 1.36% glucose solution. HD was performed 12 hours per week with biocompatible membrane. All dialysis patients had been treated by regular HD or CAPD for more than 12 months and had adequate dialysis defined by Kt/V $\geq$1.2 in HD patients and weekly Kt/V $\geq$ 1.7 in CAPD patients. The present 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.

All patients were given a physical examination including recorded blood pressure. Body weight, height, and body mass index (BMI) were measured according to standard protocols. The nutritional parameters including total body fat and fat free mass were also evaluated by total body bioelectrical impedance (BIA) using BIA (Maltron® Bioscan 915 & 916) at a single frequency: 0.8 MA, 50 KHz.

Laboratory data

The patients were asked to fast overnight for 12 hr before clinical examination. Fasting plasma samples were drawn and processed following standardized protocols. Total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, and fasting plasma glucose levels were measured by standardized methods. Fasting plasma insulin levels were analyzed by electrochemiluminescence immunoassay (Roche Elecsys 2010, USA).

Assessment of insulin resistance using HOMA

The value of insulin resistance was obtained by homeostasis model assessment (HOMA). HOMA was calculated as the following formula: HOMA = fasting serum insulin (μU/ml) x fasting plasma glucose (mmol/L)/22.5

HOMA has a close correlation with the insulin sensitivity index by the standard euglycemic hyperinsulinemic clamp as shown by Mathew et al[18]. This index can be applied to patients with renal failure[19], assuming that normal subjects aged <35 years with normal weight have an insulin resistance of 1.

Definition of ESRD

An estimate of the GFR was obtained by the four-variable Modification of Diet in Renal Disease (MDRD) equation: GFR = 186.3 x serum creatinine$^{-1.154}$ x age$^{-0.203}$ x 0.742 if female and x 1.21 if black. ESRD was defined as GFR <15 ml/min per 1.73 m^2.

Statistical analysis

Continuous data were described as mean and standard deviations (SD). Categorical variables were described as percentages. Baseline characteristics in CAPD, HD, and non dialysis ESRD groups were examined using Chi-square test for categorical variables and ANOVA tests for continuous variables. All analyses were performed using statistical software for Windows (SPSS version 12.0, Chicago, IL). Differences were considered as significant at a p level of 0.05 or less.

Results

The clinical characteristics according to modalities of dialysis of 45 non diabetic ESRD patients are shown in Table 1, and all clinical characteristics were not different between HD, CAPD and non dialysis patients. For 61.1 to 66.7% of males with a mean age of 46.7 $\pm$ 10.4 years, 37.8% and 26.7% presented with primary renal disease of chronic glomerulonephritis and hypertension, respectively. Mean systolic and diastolic blood pressure, body weight, BMI, fat free mass, and fat mass were similar among the three groups.

As shown in Fig. 1A, the fasting plasma glucose concentration was 93.0 $\pm$ 11.5 mg/dl for non

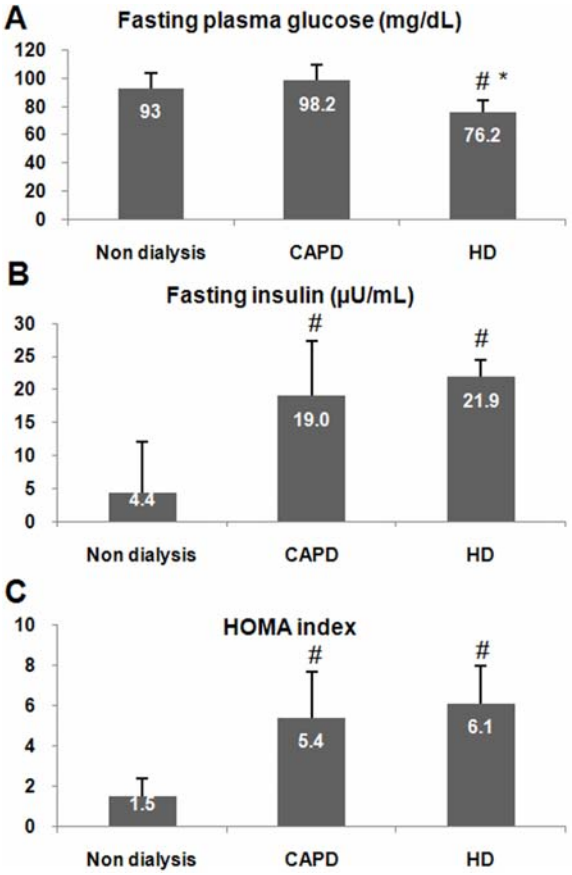

Fig. 1 Fasting plasma glucose, fasting plasma insulin, and HOMA-insulin resistance of non diabetes ESRD according to modalities of chronic renal replacement therapy. $^{\#}p < 0.05$ vs non dialysis and $^{*}p < 0.05$ vs. CAPD

dialysis ESRD patients, 98.2 ± 10.6 mg/dl for CAPD patients and 76.2 ± 7.8 mg/dl for HD patients, both of which were significantly lower in the HD group than in the CAPD and non dialysis ESRD groups ($p < 0.05$). The mean values of fasting plasma insulin were 4.4 ± 2.5 μU/ml in non dialysis ESRD subjects, 21.9 ± 7.7 μU/ml in CAPD subjects and 19.5 ± 8.4 μU/ml in HD patients. Significant differences were noted in fasting plasma insulin levels of both dialysis treatments (Fig. 1B). Insulin resistance, as measured by HOMA, was 1.5 ± 0.9 in non dialysis ESRD patients. Of interest, the values of HOMA index of CAPD and HD patients, 5.4 ± 2.3 and 6.0 ± 1.9, respectively, were significantly higher than that of non dialysis ESRD patients, suggesting high insulin resistance in dialysis patients. However, no statistically significant difference was observed in the HOMA index between HD and CAPD patients ($p > 0.05$, Fig. 1C). Moreover, no significant differences were found in the hematocrit, serum albumin, total cholesterol, HDL-cholesterol, LDL-cholesterol and triglyceride levels among groups (Table 2). However, high levels of BUN and serum creatinine were noted in the dialysis group compared with the non dialysis ESRD group. Furthermore, in CAPD patients, a trend for triglyceride to be increased, and serum albumin to be decreased was observed but with no statistically significant levels.

Discussion

The major findings of the present study were that HOMA-insulin resistance and fasting plasma insulin were significantly higher in both HD and CAPD patients compared with non dialysis ESRD patients, but not significantly different in patients with dialysis treatments. This finding provides additional evidence that impaired insulin sensitivity is relatively high after long term chronic renal replacement therapy.

Several studies have clearly shown that the sensitivity to the action of insulin with respect to glucose metabolism is markedly impaired in CKD[7]. Many mechanisms may contribute to insulin resistance in kidney disease including postreceptor defects in insulin action in muscle, adipose, and liver tissues. The defects are primarily localized to glucose uptake and metabolism by these insulin-sensitive tissues[7]. Significantly impaired insulin sensitivity presents after the course of dialysis. The result is in contrast with findings by Koyabashi et al, who showed that 4.9 to 5.4 weeks of HD or CAPD treatment resulted in a completely normalized insulin sensitivity[20]. The present study employed a different methodological approach with the previous author, who concluded that insulin resistance in uremic patients is diminished after initiating dialysis. The authors protocol studied patients had long term dialysis more than 12 months. Therefore, the authors dialysis population had still high impaired insulin sensitivity when compared with ESRD patients before initiating dialysis. Some hypotheses can explain the persistence of the apparent insulin resistance in patients with ESRD treated by dialysis. It is possible that dialysis may be unable to remove circulating factors-middle molecules or others -that could be implicated in the defect of glucose utilization observed during renal impairment[7]. A previous study demonstrated that dialysis patients displayed increased glycemic and insulinemic responses to oral glucose, suggesting an insulin-resistant state that might be due to the persistence of defective glucose transport secondary to renal insufficiency itself[21]. Thus, the present study supports this hypothesis because BUN and serum

Table 1. Patient characteristics according to the modes of renal dialysis

	Non dialysis (n = 18)	CAPD (n = 12)	HD (n = 15)
Age (yr)	48.7 ± 11.9	47.6 ± 8.9	44.4 ± 10.2
Sex (M (%))	10 (66.7)	8 (66.7)	11 (61.1)
Systolic blood pressure (mmHg)	137.2 ± 12.8	141.4 ± 19.6	134.8 ± 13.8
Diastolic blood pressure (mmHg)	83.3 ± 9.7	84.6 ± 13.9	84.1 ± 13.1
Body weight (kg)	54.4 ± 11.9	61.5 ± 3.2	56.5 ± 8.1
BMI (kg/m^2)	21.8 ± 3.6	23.0 ± 1.5	21.6 ± 2.9
Duration of dialysis (months)	-	67.4 ± 31.9	89.3 ± 39.2
Primary renal disease			
Glomerulonephritis (%)	6 (40.0)	3 (25.0)	8 (44.4)
Hypertension (%)	6 (40.0)	3 (25.0)	3 (16.6)
ADPKD (%)	1 (6.6)	1 (8.3)	-
Unknown (%)	2 (13.3)	5 (41.6)	7 (38.8)
Fat free mass (kg)	36.9 ± 7.6	41.0 ± 9.0	40.0 ± 8.3
Fat mass (kg)	15.4 ± 4.6	13.0 ± 5.1	12.9 ± 6.4

All data were presented as mean ± SD

Table 2. Biochemical variables

	Non dialysis (n = 42)	CAPD (n = 13)	HD (n = 5)
Total chlolesterol (mg/dl)	182.1 ± 41.1	176.0 ± 64.3	180.3 ± 28.7
LDL-chlolesterol (mg/dl)	111.9 ± 38.0	97.1 ± 27.2	114.4 ± 28.3
HDL-chlolesterol (mg/dl)	52.8 ± 10.8	48.1 ± 17.9	57.9 ± 12.7
Triglycerides (mg/dl)	108.7 ± 40.2	198.0 ± 35.2	106.1 ± 68.8
Hematocrit (%)	34.4 ± 7.1	32.6 ± 5.2	30.1 ± 3.8
BUN (mg/dl)	41.1 ± 12.6	57.3 ± 11.2*	58.4 ± 10.9*
Creatinine (mg/dl)	6.6 ± 2.5	8.4 ± 2.1	9.9 ± 2.5*
Albumin (g/L)	3.9 ± 0.2	3.6 ± 0.2	4.0 ± 0.7

All data were presented as mean ± SD. * vs. non dialysis $p < 0.05$.

creatinine levels were significantly high in patients with HD and CAPD.

Little data are available regarding the effect of renal replacement therapy on insulin resistance. In CAPD categories of patients, factors other than renal insufficiency per se, such as high glucose dialysate solution and inflammation process during dialysis, are likely to cause or contribute to insulin resistance. However, in the present study, the authors showed that impaired insulin sensitivity in both CAPD and HD groups was similar. Another report has shown that CAPD therapy normalized insulin resistance similar to HD therapy[20] and CAPD therapy tended to improved insulin resistance[22]. In contrast, others have observed that patients on CAPD treatment had significantly higher insulin insensitivity, assessed by HOMA index, than did patients on HD treatment[23,24].

Many factors have been implicated in the pathogenesis of insulin sensitivity. Uremic toxin, anemia with erythropoietin deficiency, metabolic acidosis, excess parathyroid hormone, 1,25 OH_2D_3 deficiency, and malnutrition have been reported to contribute to insulin resistance[25]. However, although all biochemical and nutritional parameters including hematocrit, serum albumin, and body composition, were similar in the three groups of ESRD patients, unfortunately, the authors did not measure erythopoietin and 1,25 OH_2D_3 levels, which may have been important contributing factors.

The present study had certain limitations. First, the number of ESRD patients was relatively small, as such the statistical power may not be large enough

to detect the difference between insulin resistance in ESRD patients with treatment and those of the CAPD or HD groups. Second, the cross-sectional study design cannot draw inferences regarding causality among insulin resistance in ESRD patients. Prospective clinical studies may provide a better context to define the evolution of insulin sensitivity with time in patients with treatment.

In conclusion, the present study demonstrated that dialysis patients without diabetes had high HOMA-insulin resistance and hyperinsulinemia when compared with non dialysis ESRD patients. Of particular importance in the present study is the finding that modalities of dialysis did not affect the degree of impaired insulin sensitivity.

Acknowledgment

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Potential conflicts of interest

None.

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ภาวะดื้อต่ออินสูลินในผู้ป่วยโรคไตเรื้อรังระยะสุดท้ายที่ไม่ได้เกิดจากเบาหวานระหว่างกลุ่มที่ได้รับและไม่ได้รับการบำบัดทดแทนทางไต

บัญชา สถิระพจน์, อุปถัมภ์ ศุภสินธุ์, ปณิภัทร พันธนะอังกูร, ประเจษฎ์ เรืองกาญจนเศรษฐ์, เนาวนิตย์ นาทา, อำนาจ ชัยประเสริฐ, อินทรีย์ กาญจนกูล, พรรณบุปผา ชูวิเชียร

วัตถุประสงค์: *พบภาวะดื้อต่ออินสูลินได้บ่อยในผู้ป่วยไตเรื้อรังระยะสุดท้าย และเป็นปัจจัยสำคัญในการทำนายการเกิดโรคหัวใจและหลอดเลือดของผู้ป่วยไตเรื้อรังระยะสุดท้าย อย่างไรก็ตามมีข้อมูลจำนวนจำกัดถึงวิธีการบำบัดทดแทนทางไตต่อการเกิดภาวะดื้อต่ออินสูลินในผู้ป่วยไตเรื้อรังระยะสุดท้ายที่ไม่ได้เกิดจากเบาหวาน เพื่อประเมินระดับการดื้อต่ออินสูลินในผู้ป่วยไตเรื้อรังระยะสุดท้ายที่ไม่ได้เกิดจากเบาหวานเปรียบเทียบระหว่างกลุ่มที่ได้ และไม่ได้รับการบำบัดทดแทนทางไต*

วัสดุและวิธีการ: *เป็นการศึกษาแบบตัดขวางในผู้ป่วยไตเรื้อรังระยะสุดท้ายที่ไม่ได้เป็นเบาหวาน โดยมีทั้งกลุ่มผู้ป่วยที่ได้รับการฟอกเลือด ล้างไตทางหน้าท้อง และไม่ได้รับการบำบัดทดแทนทางไต ทั้งหมดจำนวน 45 ราย ประเมินภาวะดื้อต่ออินสูลินจากวิธีโฮมาและอัตราการกรองของไตประเมินจากสูตรดัดแปลงของเอ็มดีอาร์ดี ซึ่งวินิจฉัยไตเรื้อรังระยะสุดท้ายจากอัตราการกรองของไตน้อยกว่า 15 มิลลิลิตร/นาที ต่อ 1.73 $ม^2$*

ผลการศึกษา: *มีผู้ป่วยไตเรื้อรังระยะสุดท้ายที่ได้รับการฟอกเลือดเป็นระยะเวลา 67.4 เดือน จำนวน 18 ราย ล้างไตทางหน้าท้องเป็นระยะเวลา 89.3 เดือน จำนวน 12 ราย และยังไม่ได้รับการบำบัดทดแทนทางไตจำนวน 15 ราย ผลตรวจวัดภาวะดื้อต่ออินสูลินจากโฮมา (กลุ่มล้างไตทางหน้าท้อง 5.4 $\pm$ 2.3 กลุ่มฟอกเลือด 6.0 $\pm$ 1.9 เทียบกับกลุ่มไม่ได้รับการบำบัดทดแทนทางไต 1.5 $\pm$ 0.9, $p < 0.05$) และระดับอินสูลินในเลือด (กลุ่มล้างไตทางหน้าท้อง 21.9 $\pm$ 7.7 ไมโครยูนิต/มิลลิลิตร กลุ่มฟอกเลือด 19.5 $\pm$ 8.4 ไมโครยูนิต/มิลลิลิตร เทียบกับกลุ่มไม่ได้รับการบำบัดทดแทนทางไต 4.4 $\pm$ 2.5 ไมโครยูนิต/มิลลิลิตร $p < 0.05$) ในกลุ่มที่ได้รับการบำบัดทดแทนทางไตทั้งจากการฟอกเลือดและล้างไตทางหน้าท้องสูงกว่า กลุ่มที่ไม่ได้รับการบำบัดทดแทนทางไตอย่างมีนัยสำคัญทางสถิติแต่ไม่พบความแตกต่างกันของภาวะดื้อต่ออินสูลิน และระดับอินสูลินในเลือดระหว่างกลุ่มฟอกเลือด และล้างไตทางหน้าท้อง อย่างไรก็ตามระดับน้ำตาลในเลือด หลังอดอาหารในกลุ่มฟอกเลือดต่ำกว่ากลุ่มล้างไตทางหน้าท้องและกลุ่มที่ไม่ได้รับการบำบัดทดแทนทางไต (กลุ่มล้างไตทางหน้าท้อง 98.2 $\pm$ 10.6 มิลลิกรัม/เดซิลิตร กลุ่มไม่ได้รับการบำบัดทดแทนทางไต 93.0 $\pm$ 11.5 มิลลิกรัม/เดซิลิตร เทียบกับกลุ่มฟอกเลือด 76.2 $\pm$ 7.8 มิลลิกรัม/เดซิลิตร, $p < 0.05$) นอกจากนี้ในผู้ป่วยทั้งสามกลุ่มไม่มีความแตกต่างกันของระดับความดันโลหิตน้ำหนักตัว ค่าดัชนีมวลกาย ปริมาณกล้ามเนื้อ ปริมาณไขมันในร่างกาย ระดับอัลบูมินในเลือด รวมทั้งระดับไขมัน ในเลือด*

สรุป: *ภาวะดื้อต่ออินสูลินในผู้ป่วยไตเรื้อรังระยะสุดท้ายที่ได้รับการฟอกเลือด และล้างไตทางหน้าท้องสูงกว่าผู้ป่วยไตเรื้อรังระยะสุดท้ายที่ไม่ได้รับการบำบัดทดแทนทางไต โดยภาวะดื้อต่ออินสูลินไม่มีความแตกต่างกันระหว่างกลุ่มผู้ป่วยที่ฟอกเลือด และล้างไตทางหน้าท้อง*