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# Increased risk of new-onset type 2 diabetes in people with chronic kidney disease

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## Abstract

**Purpose** This study investigated whether people with chronic kidney disease (CKD) are at the risk of new-onset type 2 diabetes.

**Methods** A cohort comprising 16,624 people with CKD, and an age- and sex-matched control cohort of 66,496 persons without any clinical kidney disease were identified from the Taiwan National Health Insurance Database during the period of 2000–2010. Both cohorts were followed up to 2011 to evaluate the incidence and hazard ratio (HR) of developing new-onset type 2 diabetes. Diseases were identified based on diagnosis coding.

**Results** The incidence of type 2 diabetes was 1.51-fold higher in the CKD cohort than in the control cohort (16.9 versus 11.2 per 1,000 person-years) with an adjusted hazard ratio of 1.17 (95% confidence interval, (CI)1.10–1.24). In the multivariate Cox regression model considering the competing-risk death, the adjusted subhazard ratio of type 2 diabetes was 1.30 (95% CI1.22–1.38) for the CKD cohort compared to the control cohort.

**Conclusions** People with CKD patients are at an increased risk of developing new-onset type 2 diabetes. Close surveillance for diabetes should be considered for these people.

**Keywords** Diabetes · Incidence · Insulin resistance · Chronic kidney disease

## Introduction

Diabetes mellitus, responsible for over 40% of patients starting renal replacement therapy, is a continuously growing serious health problem worldwide with a high risk of subsequent morbidity and mortality [1, 2]. People with chronic kidney disease (CKD) and diabetes have higher mortality risks than those without diabetes [3].

The relationship between insulin resistance and CKD is bidirectional. Insulin resistance and concomitant hyperinsulinemia may present not only in people with different stages of CKD, but also in people with normal glomerular filtration rate (GFR), irrespective of the type of renal disease [4, 5]. There is no difference in the mean insulin sensitivity indexes among people with different types of renal disease,

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suggesting that the impairment of renal function rather than the specific type of renal disease causes insulin resistance [5]. The metabolic abnormalities and uremic toxins might contribute to insulin resistance in people with CKD [6, 7]. On the other hand, insulin resistance appears to be associated with worsening renal function [8, 9].

Older age, obesity, insulin resistance, hypertension, physical inactivity, hepatitis C infection, and chronic inflammation are shared risk factors for diabetes, associated with the development of CKD [8, 10–13]. The unanswered question is whether people with CKD but without diabetes are at an increased risk of developing diabetes. The aim of this study was to evaluate the risk of new-onset type 2 diabetes in people with CKD, using the claims data of Taiwan National Health Insurance (TNHI).

## Methods

### Data source

Launched in March 1995, the TNHI Program has covered 99% of all 23.74 million people in Taiwan since 1996 [14]. We used the insurance data to conduct the population-based retrospective cohort study to evaluate the diabetic risk among people with CKD but without diabetes. Diseases were coded using the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM). This study was performed in compliance with guidelines of the Declaration of Helsinki and was approved by the Research Ethics Committee of China Medical University Hospital in Taiwan (CMUH-104-REC2-115). Since this study involved retrospective analysis of existing data, consent is not required. All data were de-identified and analyzed anonymously.

### Sampled cohorts

From the 2000–2010 claims data, we identified patients aged 18 years and older with newly diagnosed CKD (ICD-9-CM codes 581, 582, 585, 587, 403, 404) including nephrotic syndrome (ICD-9-CM code 581), without diabetes, as the CKD cohort. The first dates of CKD diagnosis for people free of diabetes were defined as the index dates. We excluded people with the medical history of type 1 diabetes (ICD-9 codes 250.x1, 250.x3), type 2 diabetes (ICD-9-CM codes 250.x0 and 250.x2), end-stage renal disease (ESRD) (ICD-9-CM code 585.6), renal transplantation (ICD-9-CM codes V42.0, 996.81), acute pancreatitis (ICD-9 code 577.0), chronic pancreatitis (ICD-9 code 577.1), pancreatic cancer (ICD-9 code 157), impaired fasting glucose or impaired glucose tolerance (ICD-9 code 790.2), or gestational diabetes (ICD-9 codes 648.0, 648.8) at baseline. The index date for comparison controls was randomly appointed a month and day with the same index year of the matched CKD cases. For each person with CKD, four comparison controls were randomly selected from individuals without clinical kidney disease and diabetes and were matched by age (every 5 years), gender, and the year of index date using the same exclusion criteria. Baseline comorbidities, including hyperlipidemia (ICD-9 code 272), hypertension (ICD-9 codes 401–405), and obesity (ICD-9 code 278.0), and medication uses of steroid, diuretics, and statin were identified. We defined those who had these medications for at least 90 days as medication users. Glucocorticoids, diuretics, and statins are medicines which might induce hyperglycemia [15, 16]. Thus, uses of these three classes of medication were included as covariates. We followed both cohorts until the development of type 2 diabetes, or censored for death, withdrawal from the TNHI program, or the end of December 31, 2011.

### Statistical analysis

Demographic characteristics and the prevalence of comorbidity were compared between two cohorts. We used Chi-square test or Fisher exact test to examine categorical variables and Student's $t$ test to examine continuous variables. The Kaplan–Meier analysis method was used to calculate the cumulative incidence of type 2 diabetes for the 2 cohorts, and log-rank test was applied to assess the difference. The incidence density rates of type 2 diabetes were presented in events per 1000 person-years for both cohorts. Univariate and multivariate Cox proportion hazards regression analyses were used to estimate crude hazard ratios (HRs) and adjusted hazard ratios (HRs) of type 2 diabetes and related 95% confidence intervals (CIs) for the CKD cohort compared with controls. In addition, because of higher mortality rates in people with CKD, we considered deaths as the competing risk events and calculated subhazard ratio (SHR) of developing type 2 diabetes for the CKD cohort.

All analyses were performed using SAS 9.4 (SAS Institute Inc, Cary, North Carolina), and the Kaplan–Meier survival curve was plotted using the R software (R Foundation for Statistical Computing, Vienna, Austria). A two-tailed $p$ value of $< 0.05$ was considered statistically significant.

## Results

We identified 16,624 people with newly diagnosed CKD and 66,496 controls as the age- and gender-matched cohorts. Both cohorts were composed of 40.9% patients older than 65 years of age, and 57.1% were men (Table 1). Comorbidities and medications were more common in the CKD cohort than in controls ($p < 0.01$).

**Table 1** Characteristics compared between chronic kidney disease cohort and control cohort

| | Chronic kidney disease | | | | $p$ value |
|---|---|---|---|---|---|
| | No | | Yes | | |
| | $N$=66,496 | | $N$=16,624 | | |
| | $n$ | % | $N$ | % | |
| Age, years | | | | | |
| 20–49 | 20,740 | 31.2 | 5185 | 31.2 | 0.95 |
| 50–64 | 18,564 | 27.9 | 4641 | 27.9 | |
| ≥65 | 27,192 | 40.9 | 6798 | 40.9 | |
| Mean (SD)[a] | 58.3 | 16.8 | 58.9 | 16.7 | |
| Gender | | | | | |
| Female | 28,600 | 43.0 | 7150 | 43.0 | 0.97 |
| Male | 37,856 | 57.0 | 9474 | 57.0 | |
| Comorbidity | | | | | |
| Hyperlipidemia | 15,557 | 23.4 | 7048 | 42.4 | <0.001 |
| Hypertension | 29,484 | 44.3 | 12,098 | 72.8 | <0.001 |
| Obesity | 1033 | 1.55 | 456 | 2.74 | <0.001 |
| Medication | | | | | |
| Steroid use | 10,085 | 15.2 | 3166 | 19.0 | <0.001 |
| Diuretics | 9173 | 13.8 | 4193 | 25.2 | <0.001 |
| Statin | 11,734 | 17.7 | 5556 | 33.4 | <0.001 |

*SD* standard deviation

[a]Student's *t* test,

The mean follow-up periods for estimating incident type 2 diabetes in CKD and control cohorts were 5.60 ± 3.51 and 6.32 ± 3.32 years, respectively. The cumulative proportion of new-onset type 2 diabetes was 3.1% greater in the CKD cohort than in the control cohort ($p < 0.001$ by log-rank test) (Fig. 1).

The overall incidence density of type 2 diabetes was 1.5-fold higher in the CKD patients than that in controls (16.9 versus 11.2 per 1000 person-years) (Table 2). Compared with controls, the adjusted HR of type 2 diabetes was 1.17 (95% CI1.10–1.24) for the CKD cohort. People with nephrotic syndrome also had a 1.16-fold increased risk of type 2 diabetes compared with the controls (95% CI 1.04–1.30) (data not shown).

The mortality was higher in the CKD cohort than in controls (12.2% ($n$ = 2029) vs. 6.81% (4526); data not shown). Table 2 also shows the adjusted subhazard ratio (SHR) of developing type 2 diabetes for people with CKD was 1.30 (95% CI1.22–1.38) after considering the competing risk of death.

Table 3 shows variables with significant association with the development of type 2 diabetes. The risk increased with age (adjusted HR1.01, 95% CI1.01–1.01) and was greater in men (adjusted HR1.11, 95% CI1.05–1.16). Hyperlipidemia (adjusted HR2.14, 95% CI2.01–2.27), hypertension (adjusted HR1.90, 95% CI1.79–2.03), obesity (adjusted HR1.28, 95% CI1.10–1.49), steroid use (adjusted HR1.13, 95% CI1.05–1.20), and diuretic use (adjusted HR1.88, 95% CI1.77–1.99) were significant factors for the development of type 2 diabetes. On the other hand, the use of statins was associated with a decreased risk of developing type 2 diabetes [adjusted HR0.62 (95% CI0.58–0.67)].

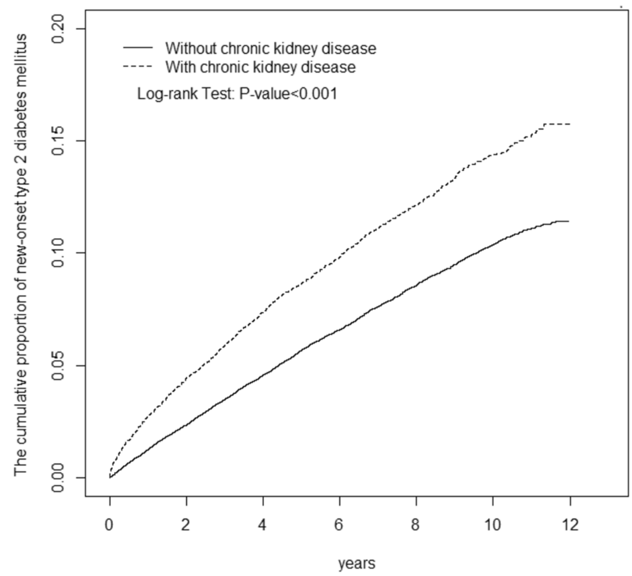


**Fig. 1** The cumulative proportion of new-onset type 2 diabetes mellitus for the chronic kidney disease cohort and the control cohort without clinical kidney disease

**Table 2** Incidence (per 1000 person-years) and hazard ratios of type 2 diabetes mellitus

| | Chronic kidney disease | |
|---|---|---|
| | No | Yes |
| | ($N$=66,496) | ($N$=16,624) |
| All | | |
| No. of event | 4692 | 1578 |
| Incidence rate | 11.2 | 16.9 |
| cHR (95% CI) | 1 (reference) | 1.51 (1.42, 1.60)* |
| aHR (95% CI)[a] | 1 (reference) | 1.17 (1.10, 1.24)* |
| cSHR (95% CI) | 1 (reference) | 1.69 (1.59, 1.79)* |
| aSHR[a] (95% CI) | 1 (reference) | 1.30 (1.22, 1.38)* |

*cHR* crude hazard ratio, *aHR* adjusted hazard ratio, *cSHR* crude subhazard ratio, *aSHR* adjusted subhazard ratio

*$p < 0.001$

[a]Adjusted for age, gender, and comorbidity of hyperlipidemia, hypertension, obesity, and medication use of steroid, diuretics and statin

**Table 3** Hazard ratios for type 2 diabetes mellitus in association with age, gender, comorbidities, and medication use in univariate and multivariable Cox regression models

| Variable | Crude | | Adjusted[a] | |
|---|---|---|---|---|
| | HR | (95% CI) | HR | (95% CI) |
| Age, years | 1.02 | (1.02, 1.02)** | 1.01 | (1.01, 1.01)** |
| Gender | | | | |
| Female | 1.00 | (Reference) | 1.00 | (Reference) |
| Male | 1.07 | (1.02, 1.13)* | 1.11 | (1.05, 1.16)** |
| Chronic kidney disease | 1.51 | (1.42, 1.60)** | 1.17 | (1.10, 1.24)** |
| Comorbidity | | | | |
| Hyperlipidemia | 2.17 | (2.06, 2.28)** | 2.14 | (2.01, 2.27)** |
| Hypertension | 2.51 | (2.38, 2.65)** | 1.90 | (1.79, 2.03)** |
| Obesity | 1.48 | (1.27, 1.72)** | 1.28 | (1.10, 1.49)** |
| Medication | | | | |
| Steroid use | 1.54 | (1.44, 1.64)** | 1.13 | (1.05, 1.20)** |
| Diuretics | 1.97 | (1.86, 2.09)** | 1.88 | (1.77, 1.99)** |
| Statin | 1.33 | (1.26, 1.41)** | 0.62 | (0.58, 0.67)** |

*HR* hazard ratio

*$p<0.01$, **$p<0.001$

[a]Adjusted for age, gender, and comorbidities of hyperlipidemia, hypertension, obesity, and medication use of steroid, diuretics, and statin

## Discussion

The present study demonstrated that people with CKD were at an elevated risk of new-onset diabetes, independently of comorbidities, and medication use. The risk was still present among people with nephrotic syndrome.

Insulin resistance in people with CKD has been recognized since decades ago [17]. Using the euglycemic clamp technique, DeFronzo et al. found that advanced CKD was associated with impairment of insulin-mediated glucose metabolism [17]. They suggested that the primary site of insulin resistance in people with CKD is located in the peripheral tissues. Friedman et al. also discovered that the increase in insulin-stimulated glucose transport is impaired in isolated skeletal muscle fibers of people with advanced CKD presumably by affecting post-receptor signaling pathway [18]. Possible mechanisms of insulin resistance in people with CKD include uremic carbamylation of signaling proteins, acidosis, altered apolipoprotein profile, oxidative stress, accumulation of free fatty acids, and inflammation [6, 7, 19, 20].

Previous studies have shown that insulin resistance exists in people with different stages of CKD. Using minimal-model technique, Fliser at al. found that insulin resistance and hyperinsulinemia may present early in the course of renal disease, even in people with normal GFR [5]. Their study also showed that mean insulin sensitivity index is not significantly different between patients with IgA nephropathy and autosomal dominant polycystic kidney disease, and it is not correlated with GFR [5]. These findings suggest that the impaired renal function rather than the specific type of renal disease causes insulin resistance. However, other studies reported that the severity of insulin resistance is correlated with GFR [6, 21]. Conflicting findings on the correlation between the severity of insulin resistance and GFR could be explained by different methods for quantitating insulin resistance.

Lorenzo et al. showed that individuals in the upper and lower range of GFR are at a higher risk of type 2 diabetes [22]. Sahakyan et al. found a positive independent relationship between serum cystatin C and the incidence of type 2 diabetes [23]. These results are consistent with our finding that CKD might be associated with the development of diabetes. On the contrary, Pham et al. showed that although lower GFR was associated with insulin resistance, the risk of impaired glucose tolerance and new-onset diabetes were not increased in older adults [24]. The difference in the study populations may explain the different results.

Our data showed that CKD is not the only risk factor associated with the development of new-onset type 2 diabetes. Some comorbidities are also contributors of developing type 2 diabetes, including hyperlipidemia, hypertension, and obesity, which are components of metabolic syndrome. These three comorbidities are well-known risk factors relating to the development of type 2 diabetes [25]. In addition, the use of statins was associated with a decreased risk of developing new-onset type 2 diabetes. This finding is inconsistent with previous studies [16]. The reason of discrepancy is unknown.

This study was strengthened by using a large population data to investigate the subsequent risk of new-onset type 2 diabetes in people with CKD. However, the present study has several limitations. First, information on personal characteristics, such as body height, body weight, and lifestyle, was unavailable in this database. Nevertheless, information on obesity was available to be used as a covariate in the data analysis based on the ICD diagnosis coding. In addition, the association between body mass index and diabetes is weaker in Asians than in Europeans [26]. Second, information on laboratory data such as levels of plasma glucose, glycated hemoglobin, C-reactive protein, and GFR was also unavailable. Although laboratory information was unavailable, a previous study has reported that the diabetes diagnosis, not specific to people with CKD, is valid in this claims data [27]. In our study, 75% people with new-onset diabetes received hypoglycemic medications when this disorder was diagnosed. Thus, the diagnosis of diabetes was reliable. Third, both type 2 diabetes and renal impairment can be asymptomatic for years. It is possible that type 2 diabetes was thus undiagnosed for a period of time and caused renal damage before a clinical diagnosis is made [28]. In addition, it is likely that people with CKD could visit clinics more often than controls, receiving diabetes screening more often.

In conclusion, this study showed that people with CKD patients were at an elevated risk of developing new-onset type 2 diabetes. Close surveillance for diabetes should be performed for these people. Life style or pharmacological interventions also should be considered for people with CKD. Further prospective studies are necessary to confirm our findings.

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## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

**Ethical approval** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

**Informed consent** For this type of study, formal consent is not required.

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