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ORIGINAL RESEARCH

The Association Between Estimated Glomerular Filtration Rate and All-Cause Mortality in Patients with Dysglycemia in Northeastern Thailand

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Video Abstract

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Background: Dysglycemia, including prediabetes and type 2 diabetes (T2DM), is associated with an increased risk of chronic kidney disease and mortality. However, data on the relationship between estimated glomerular filtration rate (eGFR) and all-cause mortality in dysglycemic individuals remain limited.

Methods: This 10-year retrospective cohort study included 277 individuals with dysglycemia from a health check-up clinic at Srinagarind Hospital, Thailand (2007–2017). Participants were divided into three eGFR groups: ≥90, 60–<90, and <60 mL/min/1.73 m^2. Multivariate Cox regression models adjusted for fasting plasma glucose, body mass index, total cholesterol, and hypertension were used to estimate hazard ratios (HRs) for all-cause mortality. The association between ≥40% eGFR decline and mortality was also evaluated.

Results: Over 10 years, 37 participants (13.4%) died. Lower eGFR was associated with higher mortality risk. Compared to the ≥90 group, adjusted HRs for all-cause mortality were 1.70 (95% CI 0.82–3.52) for eGFR 60–<90 and 3.74 (95% CI 1.27–11.03) for eGFR <60. A ≥40% eGFR decline significantly increased mortality risk (adjusted HR 7.14; 95% CI 3.16–16.14).

Conclusion: Dysglycemic individuals with eGFR <60 or a ≥40% eGFR decline have a significantly higher mortality risk, highlighting the need for early detection and intervention.

Keywords: dysglycemia, estimated glomerular filtration rate, mortality, type 2 diabetes, prediabetes

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Background

The global prevalence of dysglycemia, encompassing both prediabetes and type 2 diabetes mellitus (T2DM), has been steadily increasing, reflecting a growing public health concern.[1] T2DM is characterized by progressive loss of pancreatic β-cell function and an increase in insulin resistance. Notably, these pathophysiological defects are often evident even in the prediabetic state, which represents a continuum leading to full-blown diabetes.[2] This suggests that individuals with prediabetes are not only at risk for developing T2DM but also face elevated risks of other serious complications, including cardiovascular events and premature mortality. A meta-analysis published in 2021 further supported this notion, showing that prediabetes is associated with an increased risk of all-cause mortality.[3,4]

Diabetic kidney disease (DKD) is one of the most common and serious microvascular complications of T2DM, typically characterized by a persistently reduced glomerular filtration rate (GFR) below 60 mL/min/1.73 m^2 or an elevated urine albumin-to-creatinine ratio (uACR) of ≥ 30 mg/g in individuals with diabetes.[5] DKD is strongly associated with poor outcomes, including progression to end-stage kidney disease and increased mortality risk.[6,7] A study has shown that even prediabetes is linked to an increased risk of proteinuria and adverse cardiovascular outcomes, suggesting that kidney function may deteriorate even in the prediabetic stage.[8] While much of the research on the relationship

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between kidney function and mortality has focused on individuals with T2DM, fewer studies have investigated this association in individuals with prediabetes. Most of the studies conducted about prediabetes focused on uACR,[9,10] which might not be available in some hospitals. Serum creatinine, a readily available biomarker, is commonly used to assess kidney function and estimate eGFR, with the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines providing a classification system for chronic kidney disease (CKD) based on eGFR levels.[11] A US-based study employing subgroup analysis highlighted the link between reduced eGFR and all-cause mortality in individuals with dysglycemia, suggesting that even those without overt diabetes may be at risk of adverse outcomes.[8] However, there is limited data regarding the relationship between eGFR and mortality in individuals with prediabetes. Thailand has experienced a significant epidemiological transition with an increasing prevalence of diabetes mellitus and prediabetes. The northeastern region, where this study was conducted, has limited healthcare resources and higher rates of chronic kidney disease complications. Understanding the relationship between eGFR decline and mortality in this specific population is crucial for developing targeted screening and intervention strategies in resource-limited settings. This knowledge gap is particularly important given the asymptomatic nature of early kidney dysfunction in patients with dysglycemia. This study aims to examine the relationship between eGFR and mortality in individuals with dysglycemia, including both prediabetes and T2DM, in a Thai population. Our objective is to identify high-risk individuals who may benefit from early intervention, despite appearing asymptomatic.

Methods

Study Design and Participants

This retrospective cohort study was conducted at Srinagarind Hospital, a tertiary care facility located in the Northeastern region of Thailand. Participants were selected based on data extracted from health check-up clinic records, over a 10-year period from 2007 to 2017, focusing on individuals diagnosed with impaired fasting glucose (IFG) or T2DM. All participants were registered with the Bureau of Registration Administration, Thailand, which also provided mortality data, including the causes and dates of death during the study period.

Measurements

At the initial visit to the health check-up clinic, the participants' age, weight, and height were recorded. These data were used to calculate body mass index (BMI). Blood pressure was measured twice after the participant had rested for at least five minutes. Fasting blood samples were collected in the morning following an 8–12 hour fast. Serum creatinine, total cholesterol (TC), and fasting plasma glucose (FPG) were measured. FPG was assessed using the glucose oxidase method, while serum creatinine and TC were measured using enzymatic methods with an automatic autoanalyzer (Cobas Integra 800; Roche Diagnostics, Mannheim, Germany).

For participants who underwent subsequent blood tests, the most recent serum creatinine measurement was used to determine their eGFR via the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula. To calculate the percent change in eGFR, the first eGFR value was subtracted from the most recent eGFR value, divided by the first eGFR, and the result was multiplied by 100.

Operational Definitions

Prediabetes was defined according to the 2025 American Diabetes Association (ADA) guidelines as an FPG level between 100–125 mg/dL. T2DM was defined as a FPG level of ≥126 mg/dL.[12] FPG was repeated within one week in participants with abnormal results to confirm the diagnosis. Hypertension was defined as a systolic blood pressure of ≥140 mmHg or a diastolic blood pressure of ≥90 mmHg.[13]

Participants were stratified into three groups based on their eGFR values: Group 1 (eGFR ≥ 90 mL/min/1.73 m^2), Group 2 (eGFR 60- <90 mL/min/1.73 m^2), and Group 3 (eGFR < 60 mL/min/1.73 m^2). Due to the small number of participants with an eGFR below 45 mL/min/1.73 m^2, the eGFR categories were defined as described above.

Statistical Analysis

Descriptive statistics were used to present the baseline characteristics of participants, including means and standard deviations. Normality was assessed using the Shapiro–Wilk test and visual inspection of histograms. Cox regression analysis was employed to calculate the hazard ratios (HRs) for all-cause mortality across different eGFR groups. The multivariate analysis adjusted for potential confounders, including FPG, BMI, total cholesterol, and hypertension status. Age and sex were excluded from the multivariate analysis because they are components of the eGFR equation.

To assess the relationship between the percent change in eGFR and all-cause mortality, the Cox regression model was used with eGFR excluded as a variable, while the other variables remained unchanged. Kaplan-Meier survival curves were generated to estimate all-cause mortality, and group differences in survival curves were assessed using the Log rank test. A two-sided p-value of < 0.05 was considered statistically significant. All statistical analyses were performed using IBM SPSS Statistics version 28.0.0.0, provided by Khon Kaen University.

Results

A total of 1842 healthy individuals were seen at a health check-up clinic at Srinagarind Hospital between 2007 and 2017. Among them, a total of 277 individuals, 202 prediabetic individuals and 75 diabetic individuals were subsequently recruited for the study. Within this group, 136 participants had an eGFR of ≥90 mL/min/1.73 m^2, 123 participants had an eGFR between 60 and < 90 mL/min/1.73 m^2, and 18 participants had an eGFR below 60 mL/min/1.73 m^2. The mean eGFR ± standard deviation (SD) for the three groups were 104.32 ± 8.79, 76.0 ± 8.13, and 49.6 ± 11.92 mL/min/1.73 m^2, respectively. In the case of 244 participants with second serum creatinine results, the mean second eGFR ± SD were 86.83 ± 21.61, 70.21 ± 18.03, and 43.50 ± 24.11 mL/min/1.73 m^2 for each group respectively. The mean age of participants in group 3 was the highest at 61.1 years, while the mean ages of the participants in group 1 and group 2 were 49.7 and 55.9 years, respectively (Table 1).

During the 10-year study period, 37 participants died from any cause (13.4%). The mortality rate increased as eGFR declined. Using group 1 as the reference group, the unadjusted hazard ratios for all-cause mortality were 1.45 (95% CI 0.72–2.91) for group 2 and 3.14 (95% CI 1.31–8.72) for group 3. After adjusting for FPG, BMI, total cholesterol, and hypertension status, the hazard ratios for all-cause mortality were 1.70 (95% CI 0.82–3.52) for group 2 and 3.74 (95% CI 1.27–11.03) for group 3 (Table 2). Another factor that showed a statistically significant hazard ratio in the multivariate analysis was FPG, with a hazard ratio of 1.01 (95% CI 1.00–1.02).

Table 1 Baseline Characteristics of the Participants Stratified by eGFR

	Group 1 (n = 136) eGFR ≥ 90 mL/min/1.73 m^2	**Group 2 (n = 123) eGFR 60- < 90 mL/min/1.73 m^2**	**P-Value***	**Group 3 (n = 18) eGFR < 60 mL/min/1.73 m^2**	**P-Value****
eGFR (mL/min/1.73 m^2)	104.32 ± 8.79	76.04 ± 8.13	-	49.66 ± 11.92	-
2nd eGFR (mL/min/1.73 m^2)	86.83 ± 21.61 (n = 121)	70.21 ± 18.03 (n = 108)	< 0.01	43.50 ± 24.11 (n = 15)	< 0.01
Change in eGFR (mL/min/1.73 m^2)	17.16 ± 20.35 (n = 121)	5.96 ± 16.91 (n = 108)	< 0.01	5.13 ± 22.30 (n = 15)	< 0.01
eGFR drop≥40% (%)	7 (5.8)	10 (9.3)	0.33	2 (13.3)	0.24
Age (year)	49.37 ± 8.48	55.87 ± 9.45	< 0.01	61.11 ± 7.75	< 0.01
Male (%)	67 (49.3)	75 (61.0)	0.06	11 (61.1)	0.35
BMI (kg/m^2)	25.48 ± 4.00	26.49 ± 3.26	0.03	25.48 ± 2.12	1.00
Hypertension (%)	50 (36.8)	50 (40.6)	0.92	9 (50)	0.46
Fasting plasma glucose (mg/dL)	131.15 ± 44.11	122.11 ± 32.75	0.06	122.94 ± 34.91	0.50
Total cholesterol (mg/dL)	221.20 ± 47.42	222.64 ± 42.50	0.80	227.56 ± 36.00	0.60

Notes: Data presented in mean ± SD. *Compared between group 2 and group 1. **Compared between group 3 and group 1.
Abbreviations: BMI; body mass index, eGFR; estimated glomerular filtration rate.

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Table 2 Hazard Ratio and 95% CI for All-Cause Mortality According to Baseline eGFR

	Group 1 (n = 136) eGFR ≥ 90 mL/min/1.73 m^2	Group 2 (n = 123) eGFR 60- <90 mL/min/1.73 m^2	Group 3 (n = 18) eGFR < 60 mL/min/1.73 m^2
eGFR (mL/min/1.73 m^2)	104.32 ± 8.79	76.04 ± 8.13	49.66 ± 11.92
Number of deaths	14	18	5
Hazard ratio (unadjusted)	1 (Reference)	1.45 (95% CI 0.72–2.91)	3.14 (95% CI 1.13–8.72)
Hazard ratio (adjusted*)	1 (Reference)	1.70 (95% CI 0.82–3.52)	3.74 (95% CI 1.27–11.03)

Notes: eGFR presented in mean ± SD. *Variables used to perform multivariate analysis were FPG, BMI, total cholesterol, and hypertension status.
Abbreviations: FPG; fasting plasma glucose, BMI; body mass index, eGFR; estimated glomerular filtration rate.

The eGFR change was calculated for 244 participants with a subsequent serum creatinine result. Forty-five participants experienced a decline in eGFR of 40% or more. The adjusted hazard ratio for all-cause mortality, based on multivariate Cox analysis, was 7.14 (95% CI 3.16–16.14) for those with a 40% or greater eGFR decline.

In participants with eGFR < 90 mL/min/1.73m^2, the adjusted HR for all-cause mortality was 2.24 (95% CI: 1.18–4.26, p=0.014) compared to the eGFR ≥ 90 group. Furthermore, when assessed as a continuous variable, every 10 mL/min/1.73m^2 decrease in eGFR was associated with an 18% increased risk of mortality (Adjusted HR 1.18, 95% CI: 1.08–1.29, p<0.001).

The Kaplan-Meier curve demonstrated a significant divergence for group 3 compared to the other groups, with a p-value of 0.02 (Figure 1).

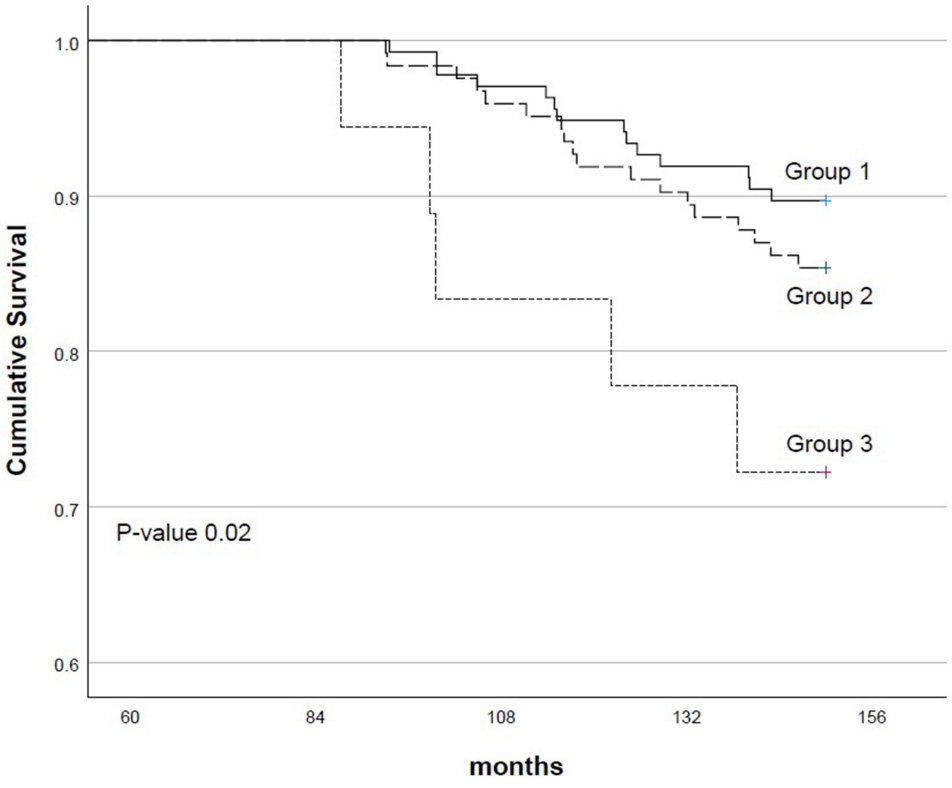

Figure 1 Kaplan-Meier survival curves for all-cause mortality stratified by baseline eGFR group. Group 1; eGFR ≥ 90 mL/min/1.73 m^2. Group 2; eGFR 60- <90 mL/min/1.73 m^2. Group 3; eGFR < 60 mL/min/1.73 m^2.
Notes: Group differences were assessed using the Log rank test (p=0.02). The follow-up period extends to 11 years to accommodate the full follow-up duration for participants enrolled early in the study period.
Abbreviation: eGFR; estimated glomerular filtration rate.

Discussion

In our study, we classified seemingly healthy individuals with dysglycemia (prediabetes or T2DM) identified at a health check-up clinic into three groups based on their eGFR. After controlling for FPG, BMI, total cholesterol, and hypertension status, we found that individuals with an eGFR of less than 60 mL/min/1.73 m^2 had significantly greater risks of all-cause mortality than those with an eGFR of 90 mL/min/1.73 m^2 or above. We also found a strong association between a decline in eGFR of 40% or more and an increased risk of all-cause mortality.

Hyperglycemia causes microvascular damage, resulting in glomerular hypertrophy and thickening of the glomerular basement membrane. Over time, this can lead to a progressive reduction in eGFR.[14] In the renal tubulointerstitial compartments, inflammation and fibrosis can also occur.[15] The presence of deteriorating kidney function may suggest a continuous inflammatory and fibrotic process that affects numerous organs and is associated with an increased risk of mortality. The higher mortality observed in the eGFR 60-<90 group compared to the reference group, though not statistically significant in our adjusted model, suggests that even a mild reduction in kidney function may carry prognostic significance in dysglycemic patients. This finding emphasizes the importance of early detection and intervention.

An eGFR can be calculated using a variety of formulas. Our study used the CKD-EPI equation, which was developed in 2009. The CKD-EPI formula was established to improve eGFR estimates, as the MDRD formula tends to underestimate kidney function in individuals with a normal eGFR of more than 90 mL/min/1.73 m^2.[16,17] The KDIGO guideline recommended using the CKD-EPI formula to calculate eGFR.[11]

Previous studies indicate that a decline of 40% or more in eGFR is a surrogate endpoint for CKD progression.[18,19] Interestingly, our study found that a decline of 40% or more in eGFR is associated with an increased risk of mortality among the dysglycemic population. The strong association between a >40% eGFR decline and mortality likely reflects rapid nephron loss. While we could not assess (uACR) in this study, rapid eGFR decline is often associated with increased proteinuria, which independently predicts cardiovascular events and mortality. In the absence of uACR data, other mechanisms may also explain our findings, including endothelial dysfunction, chronic inflammation, and mineral bone disorders associated with declining kidney function.

It is well established that DKD is associated with increased mortality in individuals with diabetes. A study conducted in Singapore by Ang, estimated the annual all-cause mortality rate of individuals with DKD to be 64.1 per 1,000 individuals, with the mortality rate rising as the severity of CKD increased.[20] Our study found that individuals with lower eGFR had a higher risk of all-cause mortality, which was consistent with previous studies. Other studies showed similar findings. Q. Jin et al found in Hong Kong that rapidly declining eGFR is associated with an increased risk of all-cause mortality.[21] In Thailand, two studies investigated the association between DKD and mortality risk, although none investigated specific eGFR levels or eGFR decline. Pratipanawatr et al found that individuals with serum creatinine levels greater than 3 mg/dL had a significantly higher risk of all-cause mortality than those with levels less than 1.5 mg/dL, with an adjusted hazard ratio of 6.85 (95% CI 4.86–9.66). Those with serum creatinine levels between 1.5 and 3.0 mg/dL had a greater risk, with an adjusted hazard ratio of 1.82 (95% CI 1.40–2.37).[22] Another study conducted in 2017 by Krairittichai et al showed an association between DKD, defined as a UACR of more than 30 mg/g, and higher cardiovascular mortality, with a hazard ratio of 1.72 (95% CI 1.03–2.88).[23]

To our knowledge, this is the first study to investigate the relationship between eGFR levels, eGFR decline, and the risk of all-cause mortality in individuals with dysglycemia, including those with prediabetes and T2DM, in Thailand. We focused on seemingly healthy adults who came to a health checkup clinic, a demographic in which physicians may be unaware of the associated mortality risk. Our findings support screening for CKD among individuals with prediabetes and T2DM, since it may help identify those who need more extensive therapy to minimize their risk of all-cause mortality while appearing healthy.

Our study's strengths included a long follow-up period and data confirmation with the Bureau of Registration Administration to establish participant status. However, several limitations must be acknowledged. First, the sample size was relatively small, particularly in the group with eGFR < 60 mL/min/1.73 m^2, which may limit the statistical power and precision of our estimates. Second, a major limitation is the absence of uACR data, which is crucial for the

comprehensive assessment of diabetic nephropathy, as it is an important independent predictor of both eGFR decline and mortality. Future prospective studies should include both eGFR and uACR for more complete risk stratification. Third, we were unable to obtain the duration between the results in participants who had subsequent blood tests. Finally, our study lacked specific data on cardiovascular events or causes of death, highlighting an important area for future research.

Conclusion

Our study demonstrated that dysglycemic individuals who have eGFR levels below 60 mL/min/1.73 m^2 or eGFR declines of 40% or more are at a higher risk of all-cause mortality. Early or more comprehensive therapy may assist individuals who meet these criteria by lowering their mortality risk.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author, Chatlert Pongchaiyakul, upon reasonable request.

Ethical Approval

This study was approved by the Khon Kaen University Human Research Ethics Committee on 31 August 2024 and reviewed in accordance with the Helsinki Declaration and the Good Clinical Practice Guidelines (HE671495). Patient consent for medical record review was waived by the committee due to the retrospective nature of the study and the use of de-identified data. All patient data were handled confidentially and in compliance with the Declaration of Helsinki.

Author Contributions

Dueanchonnee Sribenjalak: Conceptualization, Methodology, Data Curation, Formal Analysis, Writing – Original Draft, Writing – Review & Editing. Suranut Charoensri: Methodology, Data Curation, and Writing – Review & Editing. Kittrawee Kritmetapak: Methodology, Writing – Review & Editing. Chatlert Pongchaiyakul: Conceptualization, Methodology, Data Curation, Formal Analysis, and Writing – Review & Editing.

All authors have agreed on the journal to which the article will be submitted; reviewed and agreed on all versions of the article before submission, during revision, the final version accepted for publication, and any significant changes introduced at the proofing stage; agree to take responsibility and be accountable for the contents of the article.

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Disclosure

The authors declare no competing interest.

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