ORIGINAL ARTICLE

Chronic kidney disease in type 2 diabetes: Identifying key risk factors from Johor national diabetes registry study

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ABSTRACT

Introduction: Chronic kidney disease (CKD) is a common and important complication among patients with, contributing to substantial morbidity and adverse clinical outcomes. This study aimed to determine the factors associated with CKD among patients with T2DM in Johor, Malaysia.

Materials and Methods: This was a registry-based cross-sectional study using secondary data from the Malaysian National Diabetes Registry (NDR). The study included patients with T2DM captured in the Johor NDR during the 2020-2021 audit cycle. CKD status was determined by calculating the estimated glomerular filtration rate (eGFR) using the CKD Epidemiology Collaboration (CKD-EPI) formula based on serum creatinine and relevant patient variables extracted from the registry. Patients were operationally classified as having CKD if the available registry-derived eGFR was <60 mL/min/1.73 m². Sociodemographic, clinical, biochemical, diabetes-related complication, and medication-related variables were analysed. Univariate analysis was performed using Chi-square test or Fisher's exact test, and variables with p<0.05 were entered into multivariable logistic regression to identify factors independently associated with CKD among patients with T2DM.

Results: A total of 10,117 patients with T2DM were included, of whom 2,387 (23.6%) had CKD. Factors independently associated with CKD were age ≥60 years (aOR: 2.97, 95% CI: 2.60, 3.38), hypertension (aOR: 1.37, 95% CI: 1.10, 1.70), diabetes duration >10 years (aOR: 1.84, 95% CI: 1.58, 2.14), abnormal triglyceride (aOR: 1.59, 95% CI: 1.42, 1.79), abnormal high-density lipoprotein (HDL) (aOR: 1.26, 95% CI: 1.13, 1.40), diabetic retinopathy (aOR: 1.22, 95% CI: 1.06, 1.40), cerebrovascular disease (aOR: 1.40, 95% CI: 1.06, 1.85), and diabetic foot ulcer (aOR: 2.40, 95% CI: 1.68, 3.41). Medication-related associations were observed but should be interpreted cautiously because of potential confounding by indication and reverse causation.

Conclusion: CKD affected nearly one-quarter of patients with T2DM in this Johor registry-based study. Older age, hypertension, longer diabetes duration, lipid abnormalities, diabetic retinopathy, cerebrovascular disease, and diabetic foot ulcer were independently associated with CKD. These findings provide useful real-world evidence to support earlier risk identification and future development of a locally derived CKD risk prediction model among Malaysian patients with T2DM.

KEYWORDS:
Type 2 Diabetes Mellitus, Chronic Kidney Disease, Diabetic Kidney Disease, National Diabetes Registry

INTRODUCTION

Type 2 diabetes mellitus (T2DM) remains a major global public health problem and is a well-recognised cause of chronic vascular and renal complications. In Malaysia, diabetes continues to impose a substantial burden with 15.6% of Malaysian adults having diabetes.[1] Among patients with T2DM, chronic kidney disease is clinically important because it is associated with increased morbidity, mortality, cardiovascular complications and healthcare utilisation.[2]

Chronic kidney disease (CKD) is defined as abnormalities of kidney structure or function, which present for at least three months.[3] In Malaysian clinical practice, patients are classified as having CKD when the estimated glomerular filtration rate (eGFR) is below 60 mL/min/1.73 m² for a minimum of three months.[4] Among individuals with T2DM, CKD is of particular importance because chronic hyperglycaemia promotes renal injury through interrelated metabolic and hemodynamic disturbances, together with inflammation and oxidative stress, which collectively accelerate structural and functional decline.[5]

The National Diabetes Registry (NDR) of Malaysia provides a valuable source of real-world clinical data, particularly from public primary care clinics, allowing evaluation of chronic disease patterns, complications and treatment practices in routine care settings.[6] Since its establishment in 2009, NDR reports have captured heterogeneity across states in patient characteristics and diabetes care performance, indicating that regional differences in demographic composition and clinical outcomes. Thus, underscoring the relevance of state-specific analyses.

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Therefore, this study aimed to determine factors associated with CKD among patients with T2DM in Johor using data from the NDR. Identifying these factors will provide the foundation for future development of a locally derived predictive model for CKD risk among Malaysian patients with T2DM. This analysis represents an early step toward identifying reliable, evidence-based predictors for inclusion in a future risk prediction tool tailored to the clinical and demographic context of Malaysia. This study aligns with the United Nations Sustainable Development Goal 3 (SDG 3) by addressing the burden of non-communicable diseases (NCDs), particularly type 2 diabetes and CKD. By identifying risk factors with CKD, it supports prevention and early intervention strategies. The findings also complement the 13th Malaysia Plan, which emphasizes strengthening NCD management, promoting data-driven healthcare, and improving early detection to enhance health outcomes in Malaysia.

MATERIALS AND METHODS

Study design and data source

This was a registry-based cross-sectional study using secondary data from the NDR. The NDR of Johor captures standardised information such as patient demographics, clinical characteristics, biochemical profile, medication use and diabetes-related complications from public healthcare facilities across Johor state of Malaysia.

Study population and eligibility criteria

The study population comprised patients with T2DM recorded in the Malaysian NDR under Johor state during the 2020–2021 audit period. Patients were eligible for inclusion if they had a documented diagnosis of T2DM and were registered in the NDR. Cases were excluded if they had missing serum creatinine data, as serum creatinine was required to calculate eGFR using the CKD-EPI formula (Chronic Kidney Disease Epidemiology Collaboration) for determination of CKD status. Repeated or overlapping records referring to the same individual in the annual audited registry list were also excluded.

Outcome variable

The dependent variable in this study was CKD. For this registry-based cross-sectional analysis, CKD status was operationally determined using the eGFR calculated from serum creatinine values and other relevant variables available in the NDR audit dataset. The eGFR was calculated using the CKD-EPI equation. Patients with an eGFR <60 mL/min/1.73 m² were classified as having CKD, whereas those with an eGFR ≥60 mL/min/1.73 m² were classified as not having CKD.[4] Although the clinical diagnosis of CKD conventionally requires evidence of persistent kidney abnormality for at least three months, longitudinal data were unavailable to confirm the persistence of reduced eGFR in the analysed registry.

Independent variables

The independent variables included sociodemographic, anthropometric measurements, clinical characteristic, biochemical, diabetes-related complications, and medication-related factors available in the registry.

Sociodemographic variables comprised age, sex, ethnicity, and smoking status. Clinical characteristics included duration of diabetes, body mass index (BMI), mean arterial pressure (MAP), hypertension, and dyslipidaemia. Biochemical parameters included glycated hemoglobin (HbA1c), total cholesterol, triglycerides, low-density lipoprotein (LDL) cholesterol and high-density lipoprotein (HDL) cholesterol. Diabetes-related complications recorded in the registry included retinopathy, cerebrovascular disease, diabetic foot ulcer and ischaemic heart disease.

Medication-related variables included use of oral hypoglycaemic agents such as metformin, sulfonylureas, alpha-glucosidase inhibitors, meglitinides, and glitazones; insulin; antihypertensive medications such as angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB), beta blockers, calcium channel blockers, diuretics, alpha blockers, and centrally acting agents; lipid-lowering agents such as statins and fibrates; and antiplatelet agents such as acetylsalicylic acid and ticlopidine.

In this study, all variables were categorised into categorical formats to facilitate descriptive and inferential analyses. For binary variables, the reference or normal category was generally coded as 0, while the abnormal or risk category was coded as 1. Continuous variables, including anthropometric and biochemical parameters, were categorised based on established guideline thresholds or clinically meaningful cut-off points before analysis.

Data management

Data cleaning was performed prior to analysis. Duplicate and overlapping records were removed, and records with missing serum creatinine values were excluded because CKD status could not be determined without serum creatinine-derived eGFR. Serum creatinine values were extracted from the registry and used to calculate eGFR for each patient. CKD status was then derived from the calculated eGFR values. As all independent variables were analysed in categorical format, missing categorical covariate values were handled using the imputation approach adopted from a previous registry-based study.[7] Binary variables were coded numerically, with reference or normal categories generally coded as 0 and abnormal or risk categories coded as 1. Missing values were estimated on this numeric scale. Imputed values greater than 0.5 were subsequently recorded as 1, while imputed values less than 0.5 were recoded as 0. All data were anonymized prior to analysis.

Statistical analysis

Data were analysed using IBM SPSS software version 30. A two-tailed approach was applied, and a p-value <0.05 was considered statistically significant. As all study variables were categorical, descriptive results were presented as frequencies and percentages. Univariate analysis was performed to examine the association between each independent variable and CKD status using Chi-square test or Fisher's exact test, as appropriate, based on expected cell counts.

Variables with p-value less than 0.05 in the univariate analysis were subsequently entered into the multivariable logistic regression model to determine factors independently associated with CKD among patients with T2DM. Adjusted

odds ratios (aORs) with 95% confidence intervals (95% CI) were reported. Collinearity among variables was assessed using variance inflation factors. Model fit and performance were assessed using the Omnibus test of model coefficients, Hosmer–Lemeshow goodness-of-fit test, Cox and Snell R^2, Nagelkerke R^2, classification accuracy, and the area under the receiver operating characteristic (ROC) curve. Sensitivity analyses were not performed in this study.

Ethical approval

Ethical approval for this study was obtained from the Medical Research Ethics Committee, Ministry of Health Malaysia (NMRR ID-25-03586-8SK (IIR)) and the Research Ethics Committee, UiTM (REC/01/2026 (PG/EX/8)).

RESULTS

A total of 14,747 Johor NDR records from the 2020–2021 audit dataset was initially screened. After removal of duplicate or overlapping records (n=3,584) and records with missing serum creatinine values (n=1,046), 10,117 patients with T2DM were included in the final analysis. The extent of missingness for other study variables ranged from 5.7% to 20.0%. Of the 10,117 patients with T2DM included in the final analysis, 2,387 patients had CKD, giving an overall prevalence of 23.6% (95% CI: 22.8, 24.4), while 7,730 patients did not have CKD (76.4%; 95% CI: 75.6, 77.2).

The baseline characteristics of the study population are presented in Table I. Most patients were aged 60 years and above (61.7%), female (60.9%), Malay (69.8%), and non-smokers (94.4%). In terms of anthropometric profile, 86.5% had increased waist circumference, while 46.0% were obese and 31.4% were overweight. A high proportion had hypertension (86.5%) and dyslipidaemia (84.8%) as comorbidities. With regard to diabetes duration, approximately one-third had lived with diabetes for more than 10 years. Poor glycaemic control was observed in 40.6% of patients. Abnormal triglyceride, HDL, and LDL levels were present in 34.6%, 40.6%, and 51.9% of patients, respectively. In addition, diabetic complications such as retinopathy, cerebrovascular disease, diabetic foot ulcer, and ischaemic heart disease were also recorded in a proportion of the study population. Metformin, statins, ACE inhibitors, and calcium channel blockers were among the most commonly used medications.

Univariate analysis demonstrated that multiple variables across sociodemographic, anthropometric measurement, clinical characteristic, biochemical, diabetes-related complication, and medication-related domains were significantly associated with CKD among patients with T2DM, as presented in Table II. Significant associations were observed for age group (p<0.001), ethnicity (p<0.001), body mass index (p<0.001), hypertension (p<0.001), dyslipidaemia (p<0.001), duration of diabetes (p<0.001), mean arterial pressure (p<0.001), HbA1c classification (p=0.011), total cholesterol (p=0.049), triglyceride (p<0.001), HDL (p<0.001) and LDL (p<0.001). In addition, diabetes-related complications, including diabetic retinopathy (p<0.001), cerebrovascular disease (p<0.001), diabetic foot ulcer (p<0.001), and ischaemic heart disease (p<0.001), were significantly associated with CKD. Several medication-related variables also showed significant associations with CKD status, namely metformin (p<0.001), sulfonylurea (p<0.001), insulin (p<0.001), aspirin (p<0.001), ticlopidine (p=0.041), ACE inhibitors (p<0.001), angiotensin receptor blockers (p<0.001), beta blockers (p<0.001), calcium channel blockers (p<0.001), diuretics (p<0.001), alpha blockers (p<0.01) statins (p=0.016), and fibrates (p=0.027). In contrast, gender, smoking status, waist circumference, alpha-glucosidase inhibitor use, meglitinides, glitazones, and centrally acting antihypertensives were not significantly associated with the presence of CKD in the univariate analysis.

The multivariable logistic regression analysis results are presented in Table III, and the adjusted effects of statistically significant factors are illustrated in Figure 1. After adjustment for confounders, several factors remained independently associated with CKD among patients with T2DM. Older age was a strong predictor, with patients aged 60 years and above having nearly three times the odds of CKD compared with those aged below 60 years (aOR 2.97, 95% CI: 2.60–3.38, p<0.001). Hypertension was also independently associated with CKD (aOR 1.37, 95% CI: 1.10–1.70, p=0.006). Patients with diabetes duration of more than 10 years had higher odds of CKD compared with those with diabetes duration of less than 5 years (aOR 1.84, 95% CI: 1.58–2.14, p<0.001), whereas duration of 5–10 years was not statistically significant.

Among biochemical variables, abnormal triglyceride level (aOR 1.59, 95% CI: 1.42–1.79, p<0.001) and abnormal HDL level (aOR 1.26, 95% CI: 1.13–1.40, p<0.001) remained independently associated with CKD. Poor HbA1c control showed an inverse association with CKD (aOR 0.70, 95% CI: 0.61–0.81, p<0.001), while intermediate HbA1c control was not significant. Complication-related factors independently associated with CKD were diabetic retinopathy (aOR 1.22, 95% CI: 1.06–1.40, p=0.005), cerebrovascular disease (aOR 1.40, 95% CI: 1.06–1.85, p=0.019), and diabetic foot ulcer (aOR 2.40, 95% CI: 1.68–3.41, p<0.001). Ischaemic heart disease did not remain statistically significant in the final model.

Several medication-related variables also remained associated with CKD in the adjusted model. Nonuse of insulin was associated with higher odds of CKD among T2DM patients (aOR 1.55, 95% CI: 1.36–1.76, p<0.001), as were nonuse of angiotensin receptor blockers (aOR 1.23, 95% CI: 1.01–1.50, p=0.042), beta blockers (aOR 1.61, 95% CI: 1.44–1.80, p<0.001), calcium channel blockers (aOR 1.23, 95% CI: 1.09–1.39, p<0.01), diuretics (aOR 1.49, 95% CI: 1.32–1.69, p<0.001), and alpha blockers (aOR 2.25, 95% CI: 1.86–2.72, p<0.001). In contrast, nonuse of metformin (aOR 0.20, 95% CI: 0.17–0.22, p<0.001) and nonuse of ACE inhibitors (aOR 0.88, 95% CI: 0.79–0.99, p=0.029) showed inverse associations with CKD.

A forest plot of statistically significant factors associated with chronic kidney disease among patients with T2DM in Johor, Malaysia is presented in Figure 1. The figure shows adjusted odds ratios and 95% confidence intervals derived from the final multivariable logistic regression model.

Table I: Characteristics of patients with type 2 diabetes mellitus registered in the National Diabetes Registry of Johor, Malaysia (N=10,117)

Variables	Frequency, n (%)
Sociodemographic characteristics	
Age, years:	
<60	3871 (38.3%)
≥60	6246 (61.7%)
Gender:	
Female	6162 (60.9%)
Male	3955 (39.1%)
Ethnicity:	
Malay	7059 (69.8%)
Chinese	2112 (20.9%)
Indian	893 (8.8%)
Others	53 (0.5%)
Smoking status:	
Yes	564 (5.6%)
No	9553 (94.4%)
Anthropometric measurements	
Waist circumference, cm	
> 90cm in men, > 80cm in women	8756 (86.5%)
< 90cm in men, < 80cm in women	1361 (13.5%)
BMI, kg/m2	
Normal	2136 (21.1%)
Overweight	3175 (31.4%)
Obese	4657 (46.0%)
Underweight	149 (1.5%)
Clinical characteristics	
Hypertension status:	
Present	8750 (86.5%)
Absent	1367 (13.5%)
Dyslipidaemia status:	
Present	8580 (84.8%)
Absent	1537 (15.2%)
Duration of diabetes, years	
< 5	2997 (29.6%)
5 – 10	3922 (38.8%)
> 10	3198 (31.6%)
Mean Arterial Pressure, mm Hg	
Between 65 – 100	5889 (58.2%)
Less than 65, more than 100	4228 (41.8%)
Biochemical profile	
HbA1c classifications	
Good control	3965 (39.2%)
Intermediate control	2040 (20.2%)
Poor control	4112 (40.6%)
Total Cholesterol, mmol/l	
< 4.5mmol/l	4371 (43.2%)
> 4.5mmol/l	5746 (56.8%)
Triglyceride, mmol/l	
< 1.7mmol/l	6620 (65.4%)
> 1.7mmol/l	3497 (34.6%)
HDL, mmol/l	
<1.0 mmol/l (Male), <1.2 mmol/l (Female)	4112 (40.6%)
>1.0 mmol/l (Male), >1.2 mmol/l (Female)	6005 (59.4%)
LDL, mmol/l	
<2.6 mmol/l	4871 (48.1%)
>2.6 mmol/l	5246 (51.9%)
Complications	
Diabetic Retinopathy	
Present	1535 (15.2%)
Absent	8582 (84.8%)
Cerebrovascular	
Present	297 (2.9%)
Absent	9820 (97.1%)
Diabetic Foot Ulcer (DFU)	
Present	179 (1.8%)
Absent	9938 (98.2%)

Table I: Characteristics of patients with type 2 diabetes mellitus registered in the National Diabetes Registry of Johor, Malaysia (N=10,117)

Variables	Frequency, n (%)
Ischaemic Heart Disease (IHD)	
Present	1054 (10.4%)
Absent	9063 (89.6%)
Medications	
Metformin	
Use	8467 (83.7%)
Nonuse	1650 (16.3%)
Sulfonylurea	
Use	4265 (42.2%)
Nonuse	5852 (57.8%)
Alpha-glucosidase Inhibitor	
Use	75 (0.7%)
Nonuse	10042 (99.3%)
Meglitinides	
Use	21 (0.2%)
Nonuse	10096 (99.8%)
Glitazones	
Use	86 (0.9%)
Nonuse	10031 (99.1%)
Insulin	
Use	3278 (32.4%)
Nonuse	6839 (67.6%)
Acetylsalicylic Acid (Aspirin)	
Use	2195 (21.7%)
Nonuse	7922 (78.3%)
Ticlopidine	
Use	37 (0.4%)
Nonuse	10080 (99.6%)
ACE Inhibitors	
Use	5234 (51.7%)
Nonuse	4883 (48.3%)
Angiotensin Receptor Blocker (ARB)	
Use	716 (7.1%)
Nonuse	9401 (92.9%)
Beta Blockers	
Use	2819 (27.9%)
Nonuse	7298 (72.1%)
Calcium Channel Blockers (CCB)	
Use	6431 (63.6%)
Nonuse	3686 (36.4%)
Diuretics	
Use	1927 (19.0%)
Nonuse	8190 (81.0%)
Alpha Blockers	
Use	648 (6.4%)
Nonuse	9469 (93.6%)
Centrally Acting Antihypertensives	
Use	13 (0.1%)
Nonuse	10104 (99.9%)
Statins	
Use	8810 (87.1%)
Nonuse	1307 (12.9%)
Fibrates	
Use	158 (1.6%)
Nonuse	9959 (98.4%)

Notes: BMI, body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table II: Univariate analysis of factors associated with chronic kidney disease among patients with type 2 diabetes mellitus in Johor, Malaysia (N=10117)

Variables	CKD		χ2 (df)	p-value
	Absent (n=7730) n (%)	Present (n=2387) n (%)		
Sociodemographic characteristics				
Age, year:				
<60	3424 (88.5%)	447 (11.5%)	504.742 (1)	<0.001*
≥60	4306 (68.9%)	1940 (31.1%)		
Gender:				
Female	4745 (77.0%)	1417 (23.0%)	3.129 (1)	0.077
Male	2985 (75.5%)	970 (24.5%)		
Ethnicity:				
Malay	5294 (75.0%)	1765 (25.0%)	32.670 (3)	<0.001*
Chinese	1654 (78.3%)	458 (21.7%)		
Indian	739 (82.8%)	154 (17.2%)		
Others	43 (81.1%)	10 (18.9%)		
Smoking status:				
Yes	435 (77.1%)	129 (22.9%)	0.173 (1)	0.678
No	7295 (76.4%)	2258 (23.6%)		
Anthropometric measurements				
Waist circumference, cm				
> 90cm in men, > 80cm in women	6695 (76.5%)	2061 (23.5%)	0.112 (1)	0.737
< 90cm in men, < 80cm in women	1035 (76.0%)	326 (24.0%)		
BMI, kg/m2				
Normal	1547 (72.4%)	589 (27.6%)	29.926 (3)	<0.001*
Overweight	2429 (76.5%)	746 (23.5%)		
Obese	3647 (78.3%)	1010 (21.7%)		
Underweight	107 (71.8%)	42 (28.2%)		
Clinical characteristics				
Hypertension status:				
Present	6501 (74.3%)	2249 (25.7%)	159.763 (1)	<0.001*
Absent	1229 (89.9%)	138 (10.1%)		
Dyslipidaemia status:				
Present	6482 (75.5%)	2098 (24.5%)	23.077 (1)	<0.001*
Absent				
1248 (81.2%)	289 (18.8%)			
Duration of diabetes, years				
< 5	2553 (85.2%)	444 (14.8%)	365.398 (2)	<0.001*
5 – 10	3093 (78.9%)	829 (21.1%)		
> 10	2084 (65.2%)	1114 (34.8%)		
Mean Arterial Pressure, mm Hg				
Between 65 – 100	4412 (74.9%)	1477 (25.1%)	17.278 (1)	<0.001*
Less than 65, more than 100	3318 (78.5%)	910 (21.5%)		
Biochemical profile				
HbA1c classifications				
Good control	2973 (75.0%)	992 (25.0%)	9.041 (2)	0.011*
Intermediate control	1557 (76.3%)	483 (23.7%)		
Poor control	3200 (77.8%)	912 (22.2%)		
Total Cholesterol, mmol/l				
< 4.5mmol/l	3298 (75.5%)	1073 (24.5%)	3.887 (1)	0.049*
> 4.5mmol/l	4432 (77.1%)	1314 (22.9%)		
Triglyceride, mmol/l				
< 1.7mmol/l	5213 (78.7%)	1407 (21.3%)	58.181 (1)	<0.001*
> 1.7mmol/l	2517 (72.0%)	980 (28.0%)		
HDL, mmol/l				
< 1.0 mmol/l (Male), <1.2 mmol/l (Female)	3018 (73.4%)	1094 (26.6%)	34.843 (1)	<0.001*
> 1.0 mmol/l (Male), >1.2 mmol/l (Female)	4712 (87.6%)	1293 (21.5%)		
LDL, mmol/l				
< 2.6 mmol/l	3649 (74.9%)	1222 (25.1%)	11.620 (1)	<0.001*
> 2.6 mmol/l	4081(77.8%)	1165 (22.2%)		
Complications				
Diabetic Retinopathy				
Present	1014 (66.1%)	521 (33.9%)	107.475 (1)	<0.001*
Absent	6716 (78.3%)	1866 (21.7%)		
Cerebrovascular				
Present	189 (63.6%)	108 (36.4%)	27.678 (1)	<0.001*
Absent	7541 (76.8%)	2279 (23.2%)		

Table II: Univariate analysis of factors associated with chronic kidney disease among patients with type 2 diabetes mellitus in Johor, Malaysia (N=10117)

Variables	CKD		χ2 (df)	p-value
	Absent (n=7730) n (%)	Present (n=2387) n (%)		
Diabetic Foot Ulcer (DFU)				
Present	93 (52.0%)	86 (48.0%)	60.431 (1)	<0.001*
Absent	7637 (76.8%)	2301 (23.2%)		
Ischaemic Heart Disease (IHD)				
Present	701 (66.5%)	353 (33.5%)	63.936 (1)	<0.001*
Absent	7029 (77.6%)	2034 (22.4%)		
Medications				
Metformin				
Use	7028 (83.0%)	1439 (17.0%)	1253.911 (1)	<0.001*
Nonuse	702 (42.5%)	948 (57.5%)		
Sulfonylurea				
Use	3465 (81.2%)	800 (18.8%)	95.680 (1)	<0.001*
Nonuse	4265 (72.9%)	1587 (27.1%)		
Alpha-glucosidase Inhibitor				
Use	62 (82.7%)	13 (17.3%)	1.643 (1)	0.200
Nonuse	7668 (76.4%)	2374 (23.6%)		
Meglitinides				
Use	17 (81.0%)	4 (19.0%)	0.241 (1)	0.623
Nonuse	7713 (76.4%)	2383 (23.6%)		
Glitazone				
Use	70 (81.4%)	16 (18.6%)	1.198 (1)	0.274
Nonuse	7660 (76.4%)	2371 (23.6%)		
Insulin				
Use	2184 (66.6%)	1094 (33.4%)	257.290 (1)	<0.001*
Nonuse	5546 (81.1%)	1293 (18.9%)		
Acetylsalicylic Acid (Aspirin)				
Use	1507 (68.7%)	688 (31.3%)	93.369 (1)	<0.001*
Nonuse	6223 (78.6%)	1699 (21.4%)		
Ticlopidine				
Use	23 (62.2%)	14 (37.8%)	4.179 (1)	0.041*
Nonuse	7707 (76.5%)	2373 (23.5%)		
ACE Inhibitors				
Use	4089 (78.1%)	1145 (21.9%)	17.750 (1)	<0.001*
Nonuse	3641 (74.6%)	1242 (25.4%)		
Angiotensin Receptor Blocker (ARB)				
Use	483 (67.5%)	233 (32.5%)	34.222 (1)	<0.001*
Nonuse	7247 (77.1%)	2154 (22.9%)		
Beta Blockers				
Use	1834 (65.1%)	985 (34.9%)	279.136 (1)	<0.001*
Nonuse	5896 (80.8%)	1402 (19.2%)		
Calcium Channel Blockers (CCB)				
Use	4708 (73.2%)	1723 (26.8%)	100.148 (1)	<0.001*
Nonuse	6200 (82.0%)	664 (18.0%)		
Diuretics				
Use	1238 (64.2%)	689 (35.8%)	195.284 (1)	<0.001*
Nonuse	6492 (79.3%)	1698 (20.7%)		
Alpha Blockers				
Use	308 (47.5%)	340 (52.5%)	320.217 (1)	<0.001*
Nonuse	7422 (78.4%)	2047 (21.6%)		
Centrally Acting Antihypertensives				
Use	9 (69.2%)	4 (30.8%)	0.372 (1)	0.542
Nonuse	7721 (76.4%)	2383 (23.6%)		
Statins				
Use	6697 (76.0%)	2113 (24.0%)	5.758 (1)	0.016*
Nonuse	1033 (79.0%)	274 (21.0%)		
Fibrates				
Use	109 (69.0%)	49 (31.0%)	4.900 (1)	0.027*
Nonuse	7621 (76.5%)	2338 (23.5%)		

*Statistically significant at α=0.05 , Statistical analysis: Chi-Square test, Fisher's Exact Test
Notes: BMI, body mass index; CKD, Chronic kidney disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein

Table III: Multivariable analysis of factors associated with chronic kidney disease among patients with type 2 diabetes mellitus in Johor, Malaysia (N = 10,117)

Variables	B (SE)[b]	Wald (df)[b]	p-value[b]	aOR[b]	95% CI (Lower, Upper)
Age:					
Age ≥60 years	1.087 (0.067)	260.043 (1)	<0.001*	2.97	2.60, 3.38
Age <60 years	REF				
Ethnicity					
Chinese	-0.420 (0.402)	1.095 (1)	0.295	0.66	0.30, 1.44
Indian	-0.625 (0.409)	2.330 (1)	0.127	0.54	0.24, 1.19
Malay	-0.125 (0.397)	0.099 (1)	0.753	0.88	0.41, 1.92
Others	REF				
BMI					
Overweight	-0.017 (0.074)	0.055 (1)	0.815	0.98	0.85, 1.14
Obese	-0.116 (0.072)	2.640 (1)	0.104	0.89	0.77, 1.02
Underweight	0.232 (0.207)	1.252 (1)	0.263	1.26	0.84, 1.89
Normal	REF				
Hypertension					
Present	0.311 (0.112)	7.702 (1)	0.006*	1.37	1.10, 1.70
Absent	REF				
Dyslipidaemia					
Present	0.075 (0.081)	0.851 (1)	0.356	1.08	0.92, 1.26
Absent	REF				
Duration of diabetes, years					
5 –10	0.122 (0.073)	2.773 (1)	0.096	1.13	0.98, 1.30
>10	0.607 (0.077)	61.962 (1)	<0.001*	1.84	1.58, 2.14
<5	REF				
Mean Arterial Pressure, mmHg					
65 - 100	-0.105 (0.056)	3.510 (1)	0.061	0.90	0.81, 1.01
<65, >100	REF				
HbA1c classification					
Good control	REF				
Intermediate control	-0.095 (0.074)	1.621 (1)	0.203	0.91	0.79, 1.05
Poor control	-0.353 (0.071)	24.958 (1)	<0.001*	0.70	0.61, 0.81
Total Cholesterol, mmol/L					
> 4.5 mmol/L	0.070 (0.083)	0.703 (1)	0.402	1.07	0.91, 1.26
≤ 4.5 mmol/L	REF				
HDL					
<1.0 mmol/l (Male), <1.2 mmol/l (Female)	0.228 (0.056)	16.653 (1)	<0.001*	1.26	1.13, 1.40
>1.0 mmol/l (Male), >1.2 mmol/l (Female)	REF				
LDL					
> 2.6 mmol/L	-0.054 (0.055)	0.967 (1)	0.325	0.95	0.85, 1.06
≤ 2.6 mmol/L	REF				
Triglyceride, mmol/l					
> 1.7mmol/l	0.466 (0.058)	63.863 (1)	<0.001*	1.59	1.42, 1.79
< 1.7mmol/l	REF				
Diabetic Retinopathy					
Present	0.199 (0.071)	7.856 (1)	0.005*	1.22	1.06, 1.40
Absent	REF				
Cerebrovascular disease					
Present	0.335 (0.143)	5.464 (1)	0.019*	1.40	1.06, 1.85
Absent	REF				
Diabetic Foot Ulcer (DFU)					
Present	0.874 (0.180)	23.533 (1)	<0.001*	2.40	1.68, 3.41
Absent	REF				
Ischaemic Heart Disease (IHD)					
Present	0.154 (0.081)	3.647 (1)	0.056	1.17	1.00, 1.37
Absent	REF				
Metformin					
Nonuse	-1.633 (0.065)	623.577 (1)	<0.001*	0.20	0.17, 0.22
Use	REF				
Sulfonylurea					
Nonuse	-0.024 (0.063)	0.149 (1)	0.699	0.98	0.86, 1.10
Use	REF				
Insulin					
Nonuse	–0.436 (0.066)	43.324 (1)	<0.001*	1.55	1.36, 1.76
Use	REF				
Acetylsalicylic acid					
Nonuse	0.085 (0.066)	1.672 (1)	0.196	1.09	0.96, 1.24
Use	REF				

Table III: Multivariable analysis of factors associated with chronic kidney disease among patients with type 2 diabetes mellitus in Johor, Malaysia (N = 10,117)

Variables	B (SE)[b]	Wald (df)[b]	p-value[b]	aOR[b]	95% CI (Lower, Upper)
Ticlopedine					
Nonuse	-0.193 (0.398)	0.236 (1)	0.627	0.82	0.38, 1.80
Use REF					
ACE Inhibitors					
Nonuse	–0.127 (0.058)	4.742 (1)	0.029*	0.88	0.78, 0.99
Use	REF				
Beta Blocker					
Nonuse	0.475 (0.058)	67.132 (1)	<0.001*	1.61	1.44, 1.80
Use REF					
ARB					
Nonuse	0.205 (0.101)	4.143 (1)	0.042*	1.23	1.01, 1.50
Use	REF				
Diuretics					
Nonuse	0.401 (0.064)	39.068 (1)	0.001*	1.49	1.32, 1.69
Use	REF				
Calcium Channel Blocker					
Nonuse	0.205 (0.062)	10.833 (1)	<0.001*	1.23	1.09, 1.39
Use	REF				
Alpha-Blockers					
Nonuse	0.809 (0.097)	70.092 (1)	<0.001*	2.25	1.86, 2.72
Use	REF				
Statins					
Nonuse	-0.004 (0.094)	0.002 (1)	0.965	1.00	0.83, 1.20
Use	REF				
Fibrates					
Nonuse	0.210 (0.103)	0.190 (1)	0.590	1.063	0.853, 1.327
Use	REF				

Notes: aOR = Adjusted Odds Ratio, REF = reference, *Statistically significant at α=0.05, bStatistical analysis: Multiple Binary Logistic Regression Test, Method: Backward LR, Omnibus test of the model coefficient was significant, P < 0.001 and the model was valid, P = 0.157 for the Hosmer and Lemeshow test. The overall correct percentage based on the classification table was 81.2%, and the coefficient of determination was 20.2%. No multicollinearity among the variables was detected in the final model. No influential outlier (Cook's influential statistic). The area under the Receiver Operating Characteristic (ROC) curve was 79.1%.
BMI: Body mass index; DM: Diabetes mellitus; HDL: High-density lipoprotein; LDL: Low-density lipoprotein

The final model of multivariable logistic regression was statistically significant overall (Omnibus test: χ^2=2281.028, df=23, p<0.001). The model demonstrated acceptable fit, with a non-significant Hosmer–Lemeshow goodness-of-fit test (p=0.157). The explained variance was 20.2% according to Cox and Snell R^2 and 30.4% according to Nagelkerke R^2. The model correctly classified 81.2% of cases, with acceptable discriminative ability of 0.791 (95% CI: 0.780–0.801, p<0.001).

DISCUSSION

In this study, the prevalence of CKD among patients with T2DM registered in the National Diabetes Registry of Johor was 23.6% (95%CI: 22.8%, 24.4%). The prevalence in this study is close to the Thailand Primary care study by Jitraknatee et al., which reported 24.4%.[8] Conversely, the Kedah-based study in Malaysia by Mohd Zuki et al., reported higher CKD prevalence among T2DM (38.6%).[9] This underscores how regional variations in population risk profiles and data collection can yield markedly different estimates even within the same country.[10] The observed differences in international and regional prevalence estimates are explained by substantial heterogeneity in CKD definition underlying study populations, and sources of data. In addition, registry-based prevalence estimates may vary depending on audit completeness, biochemical availability, and the proportion of patients with long-standing diabetes.

Several factors were found to be significantly associated with CKD in the multivariable analysis. Older age was one of the strongest factors independently associated with CKD in this study, with patients aged 60 years and above having almost three times the odds of CKD compared with those aged below 60 years. This finding is consistent with a 12-year retrospective cohort study reported that advancing age independently increased the risk of developing CKD among patients with T2DM.[11] In addition, a systematic review by Fenta et al., also reported older age as consistent predictor for CKD across included studies.[12] Biologically, advancing age is associated with nephron loss, vascular stiffening, leading to susceptibility to microvascular and macrovascular injury, all of which may accelerate renal decline in patient with diabetes.[13-14] The association between advancing age and CKD among people with T2DM is robust across multiple study designs and populations, reinforcing the view that older age functions as both a biological risk factor and a marker of cumulative disease exposure.[15-16]

Hypertension remained independently associated with CKD among patients with T2DM in the final model. Recent studies have similarly shown that hypertension is an independent predictor of CKD in people with T2DM. For example, Abdullah et al., found that poor blood pressure control had significantly higher odds of CKD (AOR = 2.63, 95% CI 1.48–4.68) among patients with T2DM.[17] Another study in tertiary hospital identified concomitant hypertension as important

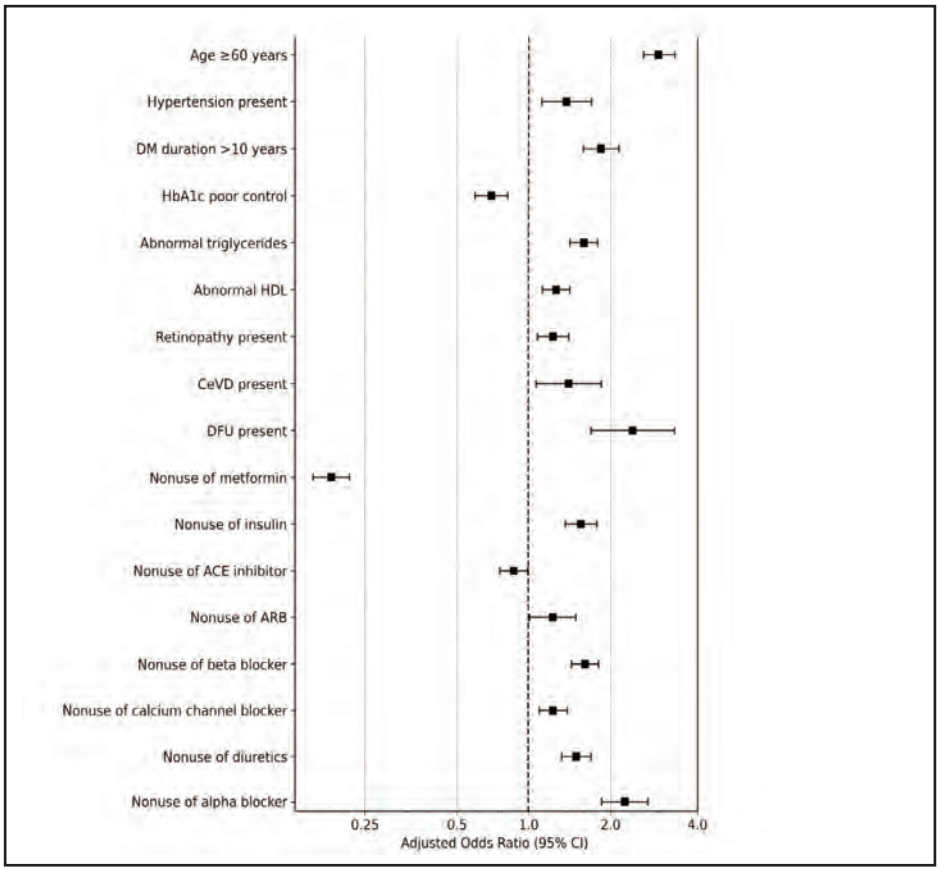

Fig. 1: Forest plot of statistically significant factors associated with chronic kidney disease among patients with type 2 diabetes mellitus in Johor

risk factor of CKD among diabetes patients.[18] It is also concordant with current KDIGO guidance, which emphasizes diabetes and hypertension as the dominant drivers of CKD progression worldwide.[19] The association is clinically plausible because elevated systemic and intraglomerular pressure contributes to glomerular injury, endothelial dysfunction, increased glomerular permeability, albumin leakage, and ultimately progressive nephron loss.[20] In patients with T2DM, the coexistence of hyperglycaemia and hypertension likely produces synergistic renal damage, making blood pressure control central to CKD prevention and progression delay.

Longer duration of diabetes was independently associated with CKD in this study, particularly among patients with diabetes duration exceeding 10 years. This finding is consistent with recent literature conducted in a large nationwide study in China.[21] Similarly, a local study found that longer diabetes duration more than 10 years had higher odd (AOR = 1.27, 95% CI 1.21–1.33) of CKD among patients with T2DM.[10] This association is biologically plausible because longer disease duration reflects prolonged cumulative exposure to hyperglycaemia, oxidative stress, and progressive microvascular injury over time.[22] Additionally, a longer duration of diabetes may indicate prolonged exposure to coexisting risk factors including hypertension which may collectively contribute to the progression of kidney injury.

This study also found that abnormal triglyceride and abnormal high-density lipoprotein (HDL) levels were significantly associated with CKD among patients with T2DM. This finding is supported by recent literature showing that triglyceride-related metabolic abnormalities are positively associated with albuminuria and CKD in patients with T2DM.[23] In addition, higher HDL has been reported to be associated with a lower incidence of CKD progression in patients with T2DM, reflecting that reduced HDL may contribute to worsening renal outcome.[24] Lipid abnormalities may contribute to renal injury through lipotoxicity, oxidative stress, endothelial dysfunction and inflammation, thus promote progressive kidney injury and fibrosis.[25] Collectively, these findings highlight the important role of dyslipidaemia within the broader metabolic environment associated with chronic kidney disease among diabetes patients.

In this study, the inverse association was found between poor HbA1c control and CKD among patients with T2DM. This contrasts with the broader literature that usually places poor glycaemic control among the major drivers of diabetic kidney disease. Thus, the inverse relationship warrants cautious interpretation. The paradoxical finding has been reported and recent consensus literature stated that HbA1c tends to underestimate true plasma glucose in advanced CKD when compared with continuous glucose monitoring, further supporting the possibility of reverse association in cross-sectional studies.[26] The cross-sectional design prevents

assessment of temporality in which glycaemic control measured at the time of registry assessment may not reflect long-term glycaemic exposure preceding CKD development. In addition, Tang et al., demonstrated that carbamylation and anaemia modify the association between HbA1c and renal outcome in patients with diabetes, supporting the view that HbA1c should be interpreted carefully in this population.[27] These factors affect HbA1c levels independently of true plasma glucose concentrations and may lead to underestimation of glycaemic exposure in patients with more advanced renal impairment.

Diabetic retinopathy was significantly associated with CKD among patients with T2DM in the present study. The finding is consistent with previous literature which reported that diabetic retinopathy associated with an increase prevalence of CKD among patients T2DM.[14] A study in China found that increasing retinopathy severity corresponding to more severe glomerular lesions.[28] This association is clinically plausible because diabetic retinopathy and diabetic kidney disease are both microvascular complications that share common pathogenic pathways, including chronic hyperglycaemia, endothelial dysfunction, inflammation and progressive microvascular damage affecting both retinal and renal circulation.

The association of cerebrovascular disease and increased risk of CKD in this study is consistent with existing cohort study of adult T2DM.[29] The American Heart Association highlighted the close bidirectional relationship between CKD and cerebrovascular disease, including shared risk factors and worse outcomes when both conditions coexist.[30] In this study, Diabetic foot ulcer (DFU) showed as one of the strongest associations with CKD. This finding is parallel with recent literature, which highlighted that individual with diabetes-related foot disease have a substantially greater burden of CKD and poorer cardio-renal outcome than those without foot complications.[31-32] This relationship is clinically relevant as both share common mechanisms, including microvascular injury, chronic inflammation and impaired healing, thus, leading to both renal and foot complications.[33]

Medication-related findings in this study should be interpreted cautiously because medication use in routine clinical care is not randomly assigned. Prescribing decisions for glucose-lowering and antihypertensive medications are commonly influenced by kidney function, albuminuria, blood pressure control, cardiovascular comorbidities, disease severity, contraindications and clinician judgement. Therefore, the observed medication-related associations may reflect underlying clinical status and treatment patterns rather than independent causal effects on CKD.

With regard to glucose-lowering medication, nonuse of metformin was associated with lower odds of CKD in patients with T2DM, should be interpreted cautiously. Metformin prescribing is strongly influenced by renal function, and its use is commonly reduced or withheld as kidney function declines. Thus, the lower odds observed among patients with nonuse of metformin may reflect reverse causation, confounding by contraindication, or treatment channeling rather than true beneficial effect of avoiding metformin.[22] A recent study in Hong Kong, showed the opposite pattern, with metformin usage associated with lower risk of major adverse kidney events and overt diabetic nephropathy.[34] The present study found that nonuse of insulin was associated with higher odds of CKD among patients with T2DM. The evidence indicates that insulin is often necessary in T2DM because of progressive beta-cell failure, thus, appropriate insulin therapy can slow CKD progression by achieving better glycaemic control.[35]

The nonuse of antihypertensive medication classes, namely angiotensin receptor blocker (ARB), beta blockers, calcium channel blocker (CCB), alpha blockers and diuretics, were associated with higher odds of CKD among patients with T2DM. The clinical rationale for this association is supported for ARBs, as current diabetes and CKD guideline consistently recommend ARBs or ACE inhibitors as first-line antihypertensive therapy in patients with diabetes due to its renal protective effects.[3] The other antihypertensive agents are commonly used as add-on therapy when blood pressure is not adequately controlled.[36] Therefore, the observed higher odds of CKD among nonusers of these medications reflects the importance of adequate antihypertensive treatment.[37] A paradoxical inverse association was observed for nonuse of ACE inhibitors, which may reflect confounding by indication and reverse causation. Thus, it is unlikely to reflect true biological protective effects. Plausible explanation, whereby patients with more severe renal disease, albuminuria and hypertension are preferentially prescribed ACE inhibitors, causing ACE inhibitors use to appear associated with CKD in observational analyses.[22,38] This confounding by indication commonly seen in pharmacoepidemiology, where treatment allocation is influenced by patient's baseline prognosis and disease severity.[39-40]

This study has several strengths. Firstly, it used a relatively large registry-based sample of patients with T2DM from public primary care clinics in Johor, which enhances the relevance of the findings to routine clinical practice. Secondly, the study utilised real-world data from the National Diabetes Registry, allowing assessment of multiple sociodemographic, clinical, biochemical, complication-related, and medication-related factors within a single population. Thirdly, the use of routinely collected variables increases the practical value of the findings, particularly as an early step toward identifying candidate predictors for future CKD risk prediction model development in Malaysian patients with T2DM.

LIMITATIONS

However, several limitations should be acknowledged. As this was a cross-sectional study based on registry data, causal relationships cannot be established. The use of secondary data may also be subject to missing information, recording errors, and possible misclassification of some variables. In addition, CKD status was determined using the eGFR calculated from the single serum creatinine value available for each patient in the registry dataset. As repeated serum creatinine measurements were unavailable, persistence of reduced eGFR for at least three months could not be verified. This may have resulted in misclassification, particularly

among patients with transient reductions in kidney function, and may have overestimated CKD prevalence or influenced the observed associations. Medication-related findings should also be interpreted with caution. Because medication use was assessed cross-sectionally, the temporal relationship between prescribing and CKD status could not be established. Therefore, the observed medication-related associations may be affected by confounding by indication, confounding by contraindication, treatment channelling and reverse causation. Finally, as the study was based on patients receiving care in public primary care facilities in Johor, the findings may not be fully generalisable to private healthcare settings.

CONCLUSION

CKD affected nearly one-quarter of patients with T2DM in this Johor registry-based study. Older age, hypertension, longer diabetes duration, lipid abnormalities, diabetic retinopathy, cerebrovascular disease, and diabetic foot ulcer were independently associated with CKD. These findings highlight the multifactorial nature of CKD in T2DM and provide real-world evidence that may support earlier risk identification, improved clinical management, and future development of a locally derived CKD risk prediction model among Malaysian patients with T2DM.

CONFLICT OF INTEREST

The authors declared no conflict of interest in this study.

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REFERENCES

1. National Health Morbidity Survey. Fact sheet on National Health and Morbidity Survey (NHMS).2023 [cited Jan 2026]. Accessed from: https://iku.gov.my/images/nhms2023/fact-sheet-nhms2023.pdf.
2. Tangri N, Svensson MK, Bodegård J, Eryd SA, Thuresson M, Gustafsson S, et al. Mortality, health care burden, and treatment of CKD: a multinational, observational study (OPTIMISE-CKD). Kidney360 2024; 5(3): 352-62.
3. Kidney Disease: Improving Global Outcomes CKDWG. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int 2024; 105(4S): S117-314.
4. Ministry of Health Malaysia. Clinical practice guidelines on management of chronic kidney disease 2nd edition. Second Edition ed: Malaysian Health Technology Assessment Section (MaHTAS) 2018.
5. Aghadavod E, Khodadadi S, Baradaran A, Nasri P, Bahmani M, Rafieian-Kopaei M. Role of oxidative stress and inflammatory factors in diabetic kidney disease. Iran J Kidney Dis 2016; 10(6): 337.
6. Chandran A, Abdullah M, Abdul F. National diabetes registry report 2013–2019. Putrajaya, Malaysia: Ministry of Health Malaysia. 2020.
7. Salleh MMM, Isa MR, Yasin MM, Azahar NM, Lutpi MRM. Association between body mass index and diabetes mellitus control classification among patients with type 2 diabetes mellitus: evidence from the National Diabetes Registry of Muar District Health Office, Malaysia, from January 2021 to July 2023. Malays Fam Physician 2025; 20: 23.
8. Jitraknatee J, Ruengorn C, Nochaiwong S. Prevalence and risk factors of chronic kidney disease among type 2 diabetes patients: a cross-sectional study in primary care practice. Sci Rep 2020; 10(1): 6205.
9. Mohd Zuki A, Isa M. Chronic kidney disease staging among type II diabetes mellitus patients in Kedah, Malaysia: prevalence and its factor associated. J Public Health Issues Pract 2022; 6(1): 198.
10. Wan KS, Hairi NN, Mustapha F, Mohd Yusoff MF, Mat Rifin H, Ismail M, et al. Prevalence of diabetic kidney disease and the associated factors among patients with type 2 diabetes in a multi-ethnic Asian country. Sci Rep 2024; 14(1): 7074.
11. Jairoun AA, Ping CC, Ibrahim B. Predictors of chronic kidney disease survival in type 2 diabetes: a 12-year retrospective cohort study utilizing estimated glomerular filtration rate. Sci Rep 2024; 14(1): 9014.
12. Fenta ET, Eshetu HB, Kebede N, Bogale EK, Zewdie A, Kassie TD, et al. Prevalence and predictors of chronic kidney disease among type 2 diabetic patients worldwide, systematic review and meta-analysis. Diabetol Metab Syndr 2023; 15(1): 245.
13. Fountoulakis N, Thakrar C, Patel K, Viberti G, Gnudi L, Karalliedde J. Increased arterial stiffness is an independent predictor of renal function decline in patients with type 2 diabetes mellitus younger than 60 years. J Am Heart Assoc 2017; 6(4): e004934.
14. Lee GW, Lee CH, Kim SG. Association of advanced chronic kidney disease with diabetic retinopathy severity in older patients with diabetes: a retrospective cross-sectional study. J Yeungnam Med Sci 2023; 40(2): 146-55.
15. Liu W, Du J, Ge X, Jiang X, Peng W, Zhao N, et al. The analysis of risk factors for diabetic kidney disease progression: a single-centre and cross-sectional experiment in Shanghai. BMJ Open 2022; 12(6): e060238.
16. Yang Y, Yang B, Wang B, Zhou H, Yang M, Liu B. Prediction factors and models for chronic kidney disease in type 2 diabetes mellitus: a review of the literature. Clin Transl Discov 2024; 4(5): e355.
17. Abdullah Fi, Rahman NAA, Kamaluzaman QA, Aris MAM, Basri MAMM. Prevalence of chronic kidney disease and its associated factors among type-2 diabetes mellitus patients at Kuantan Primary Health Clinics. IIUM Med J Malaysia 2025; 24(1): 142-50.
18. Joshi R, Subedi P, Yadav GK, Khadka S, Rijal T, Amgain K, et al. Prevalence and risk factors of chronic kidney disease among patients with type 2 diabetes mellitus at a tertiary care hospital in Nepal: a cross-sectional study. BMJ Open 2023; 13(2): e067238.
19. Kidney Disease: Improving Global Outcomes. KDIGO 2020 clinical practice guideline for diabetes management in chronic kidney disease [cited Jan 2026]. Accessed from: https://kdigo.org/wp-content/uploads/2021/07/KDIGO-2020-Diabetes-in-CKD-GL.pdf.
20. Shan J, Cao Z, Yu S. Advances in understanding diabetic kidney disease progression and the mechanisms of acupuncture intervention. Int J Gen Med 2024; 17: 5593-609.
21. Shi L, Xue Y, Yu X, Wang Y, Hong T, Li X, et al. Prevalence and risk factors of chronic kidney disease in patients with type 2 diabetes in China: cross-sectional study. JMIR Public Health Surveill 2024; 10: e54429.

22. de Boer IH, Khunti K, Sadusky T, Tuttle KR, Neumiller JJ, Rhee CM, et al. Diabetes management in chronic kidney disease: a consensus report by the American Diabetes Association (ADA) and Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int 2022; 102(5): 974-89.
23. Li X, Wang Y. Associations of the TyG index with albuminuria and chronic kidney disease in patients with type 2 diabetes. PLoS One 2024; 19(10): e0312374.
24. Nakada K, Tanaka K, Kimura H, Saito H, Oda A, Watanabe S, et al. High-density lipoprotein cholesterol and kidney disease progression in patients with type 2 diabetes mellitus: the Fukushima Cohort Study. BMJ Open Diabetes Res Care 2025; 13(6): e005581.
25. Han Y-Z, Du B-X, Zhu X-Y, Wang Y-Z-Y, Zheng H-J, Liu W-J. Lipid metabolism disorder in diabetic kidney disease. Front Endocrinol (Lausanne) 2024; 15: 1336402.
26. Rhee CM, Gianchandani RY, Kerr D, Philis-Tsimikas A, Kovesdy CP, Stanton RC, et al. Consensus report on the use of continuous glucose monitoring in chronic kidney disease and diabetes. J Diabetes Sci Technol 2025; 19(1): 217-45.
27. Tang M, Berg A, Rhee EP, Allegretti AS, Nigwekar S, Karumanchi SA, et al. The impact of carbamylation and anemia on HbA1c's association with renal outcomes in patients with diabetes and chronic kidney disease. Diabetes Care 2023; 46(1): 130-7.
28. Wang Q, Cheng H, Jiang S, Zhang L, Liu X, Chen P, et al. The relationship between diabetic retinopathy and diabetic nephropathy in type 2 diabetes. Front Endocrinol (Lausanne) 2024; 15: 1292412.
29. Kaze AD, Jaar BG, Fonarow GC, Echouffo-Tcheugui JB. Diabetic kidney disease and risk of incident stroke among adults with type 2 diabetes. BMC Med 2022; 20(1): 127.
30. Kelly DM, Ademi Z, Doehner W, Lip GY, Mark P, Toyoda K, et al. Chronic kidney disease and cerebrovascular disease: consensus and guidance from a KDIGO controversies conference. Stroke 2021; 52(7): e328-46.
31. Alkhami F, Rubin S, Borderie G, Foussard N, Larroumet A, Blanco L, et al. Increased risk of renal events in people with diabetic foot disease: a longitudinal observational study. Diabetes Metab 2024; 50(4): 101536.
32. Lan NS, Dwivedi G, Fegan PG, Game F, Hamilton EJ. Unravelling the cardio-renal-metabolic-foot connection in people with diabetes-related foot ulceration: a narrative review. Cardiovasc Diabetol 2024; 23(1): 437.
33. Bonnet J-B, Sultan A. Narrative review of the relationship between CKD and diabetic foot ulcer. Kidney Int Rep 2022; 7(3): 381-8.
34. Yang A, Shi M, Wu H, Lau ES, Cheung JT, Zhang X, et al. Clinical outcomes following discontinuation of metformin in patients with type 2 diabetes and advanced chronic kidney disease in Hong Kong: a territory-wide, retrospective cohort and target trial emulation study. EClinicalMedicine 2024; 71: 102568.
35. Sloan L, Cheng AY, Escalada J, Haluzík M, Mauricio D. The role of basal insulins in the treatment of people with type 2 diabetes and chronic kidney disease: a narrative review. Diabetes Obes Metab 2024; 26(4): 1157-70.
36. Sindhu D, Sharma GS, Kumbala D. Management of diabetic kidney disease: where do we stand?: a narrative review. Medicine (Baltimore) 2023; 102(13): e33366.
37. Georgianos PI, Agarwal R. Hypertension in chronic kidney disease—treatment standard 2023. Nephrol Dial Transplant 2023; 38(12): 2694-703.
38. American Diabetes Association Professional Practice Committee. Chronic kidney disease and risk management: standards of care in diabetes—2025. Diabetes Care 2025; 48: S239-51.
39. Fu EL, van Diepen M, Xu Y, Trevisan M, Dekker FW, Zoccali C, et al. Pharmacoepidemiology for nephrologists (part 2): potential biases and how to overcome them. Clin Kidney J 2021; 14(5): 1317-26.
40. Assimon MM. Confounding in observational studies evaluating the safety and effectiveness of medical treatments. Kidney360 2021; 2(7): 1156-9.