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Association between triglyceride-glucose index and clinical outcomes among patients with chronic kidney disease: a meta-analysis

Jinli Tuo[1], Zhong Li[1] and Linshen Xie[1*]

Abstract

Purpose To identify the relationship of triglyceride-glucose (TyG) index with clinical outcomes in chronic kidney disease (CKD) patients based on current available evidence.

Methods PubMed, EMBASE, Web of Science and CNKI databases were searched up to August 31, 2024. Primary outcome was the all-cause mortality. Secondary outcomes included the coronary artery disease (CAD) mortality, CKD progression, risk of severe coronary artery stenosis (SCAS), major adverse cardiovascular event (MACE), coronary artery calcification (CAC) progression, end-stage renal disease (ESRD), and nonalcoholic fatty liver disease (NAFLD). The hazard ratio (HR) and odds ratio (OR) with 95% confidence interval (CI) were combined to assess the predictive role of TyG index for above clinical outcomes among CKD patients. All statistical analysis was performed by STATA 15.0 version.

Results Twelve studies with 26,530 cases were included. Pooled results indicated that elevated TyG index was significantly related to increased risk for all-cause mortality (HR = 1.22, 95% CI: 1.13–1.31, $P<0.001$). Besides, high TyG index was also associated with the CAD mortality (HR = 1.19, 95% CI: 1.04–1.36, $P=0.011$), occurrence of CKD progression (HR = 1.52, 95% CI: 1.36–1.70, $P<0.001$), SCAS (OR = 1.79, 95% CI: 1.13–2.83, $P=0.013$), MACE (OR = 1.68, 95% CI: 1.11–2.54, $P=0.014$), CAC progression (OR = 1.55, 95% CI: 1.06–1.76, $P=0.02$), CAD (OR = 2.865, 95% CI: 1.681–4.885, $P<0.001$), ESRD (OR = 1.49, 95% CI: 1.12–1.99, $P=0.006$) and NAFLD (OR = 4.903, 95% CI: 3.046–7.893, $P<0.001$).

Conclusion High TyG index predicts poor clinical outcomes and might serve as a novel prognostic indicator among CKD patients. However, more studies are still needed to verify above findings.

Keywords Triglyceride-glucose index, Chronic kidney disease, Clinical outcomes, Meta-analysis

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Introduction

Chronic kidney disease (CKD) is a common pathological condition characterized by the gradual damage and progressive loss of kidney function. The pathogenesis of CKD is insidious, yet its mortality rate is high [1]. Among the causes of death in CKD patients, about 50% are due to cardiovascular diseases, including acute myocardial infarction (AMI) and ischemic stroke (IS) [1]. On the other hand, diabetes and hypertension are widely recognized as the most common causes of CKD [2]. The coexistence of CKD and diabetes is very common, with about 30% of patients with type 1 diabetes and 50% of those with type 2 diabetes developing CKD [3, 4]. Notably, the prevalence of diabetes-related CKD has exceeded that of glomerulonephritis-related CKD to become the leading cause of CKD in China [5], which is also observed in some other Asian countries [6].In terms of pathophysiological mechanisms, nephrons are more susceptible to damage under hyperglycemic conditions. In this context, insulin resistance (IR) is a pathological condition in which target tissues fail to respond normally to insulin stimulation. IR occurs even when kidney function is still within the normal range, indirectly reflecting the severity of CKD. Current evidence suggests that IR is closely associated with the prognosis of CKD patients and may influence clinical outcomes through various mechanisms, including triggering inflammatory responses, increasing oxidative stress, and promoting vascular damage [7, 8].

Recently, the triglyceride-glucose (TyG) index, as a surrogate marker for insulin resistance (IR), has been shown to have significant prognostic value in various vascular diseases including the ischemic stroke, cardiovascular disease, and hypertension [9–11]. Previous meta-analyses have indicated that the TyG index is associated with a significantly increased risk of developing CKD [hazard ratio (HR) = 1.11, 95% confidence interval (CI): 1.03–1.19] [12]. However, the relationship between the TyG index and clinical outcomes in CKD patients remains unclear.

Therefore, this meta-analysis aimed to identify the association of TyG index with clinical outcomes including the mortality risk and CKD progression among CKD patients based on current research progress.

Materials and methods

The current meta-analysis was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 [13].

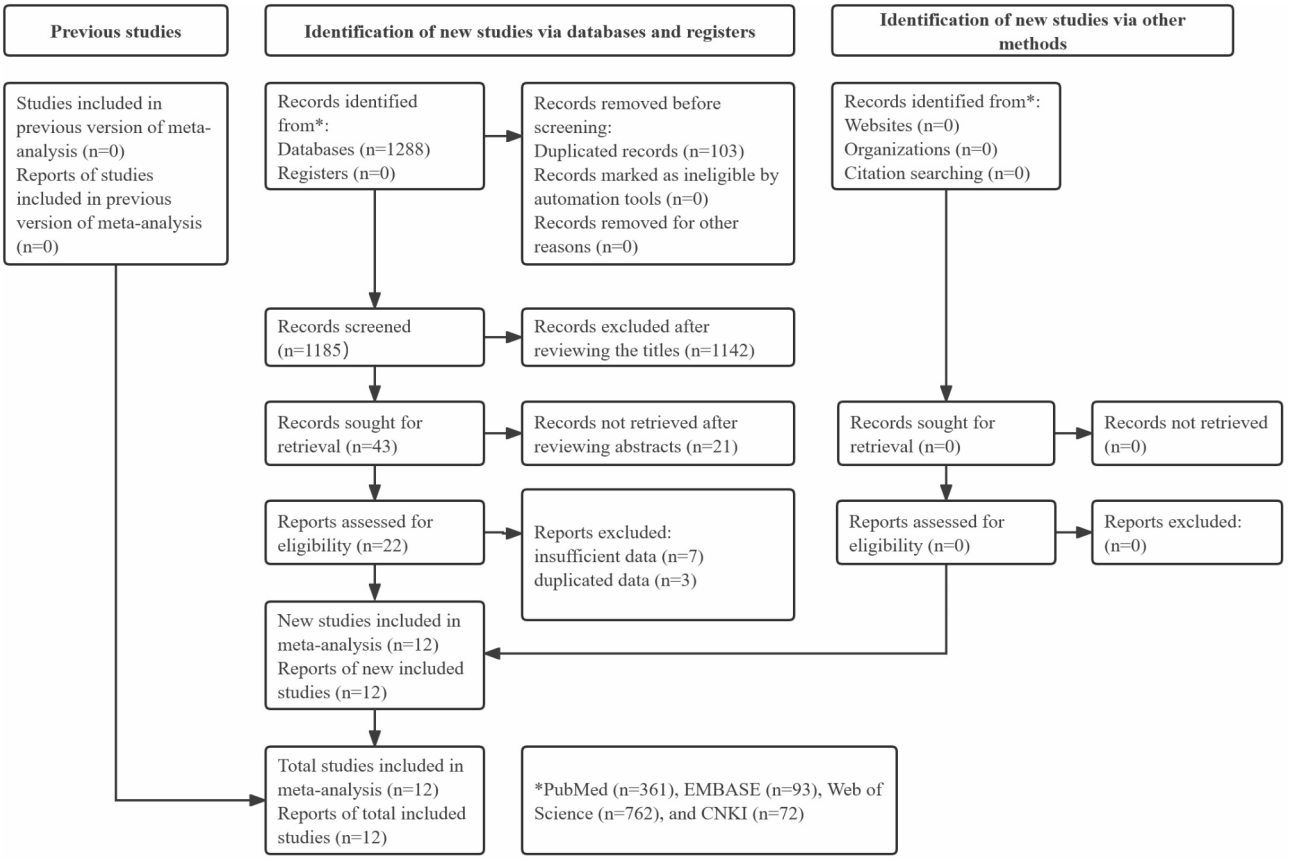

Fig. 1 Flow diagram of this meta-analysis

Literature search

We searched the PubMed, Embase, Web of Science and CNKI databases from inception up to August 31, 2024 with following terms: triglyceride glucose index, triglyceride-glucose index, TyG index, chronic kidney disease and CKD. Detailed searching strategy was as follows: (triglyceride glucose index OR triglyceride-glucose index OR TyG index) AND (chronic kidney disease OR CKD). Meanwhile, MeSH terms and free texts were applied and references of included studies were also evaluated. The search strategy and results were presented in the Supplementary file 1.

Inclusion criteria and exclusion criteria

Studies met following criteria were included: (1) prospective or retrospective cohort studies or randomized controlled trials in which patients were diagnosed with CKD; (2) the TyG index was calculated according to the formula: ln [triglyceride (mg/dL) × fasting blood glucose (mg/dL)/2]; (3) the association between TyG index and clinical outcomes after the diagnosis of CKD such as the CKD progression and mortality was explored and hazard ratios (HRs) and odds ratios (ORs) with corresponding confidence intervals (CIs) were reported or could be calculated; (4) at least one of the clinical outcomes was analyzed; (5) articles were published in English or Chinese; (6) full texts were available.

Studies met following criteria were excluded: (1) studies with insufficient or duplicated data; (2) studies with the type of letter, editorial, review, case report, animal trial or meeting abstract.

Data extraction and quality assessment

Following information was collected; the first author, year, country, sample size, studies design, patient age, CKD stage, history of diabetes mellitus, endpoints, OR and 95% CI. In this meta-analysis, primary outcome was the all-cause mortality. Secondary outcomes included the coronary artery disease (CAD) mortality, CKD progression, severe coronary artery stenosis (SCAS), major adverse cardiovascular event (MACE), coronary artery calcification (CAC) progression, end-stage renal disease (ESRD) and nonalcoholic fatty liver disease (NAFLD).

All included studies were assessed for the methodological quality according to the Newcastle-Ottawa Scale (NOS) score and studies with an NOS score ≥ 6 were defined as high-quality studies [14].

Two investigators independently performed the literature searching, selection, data collection, and quality

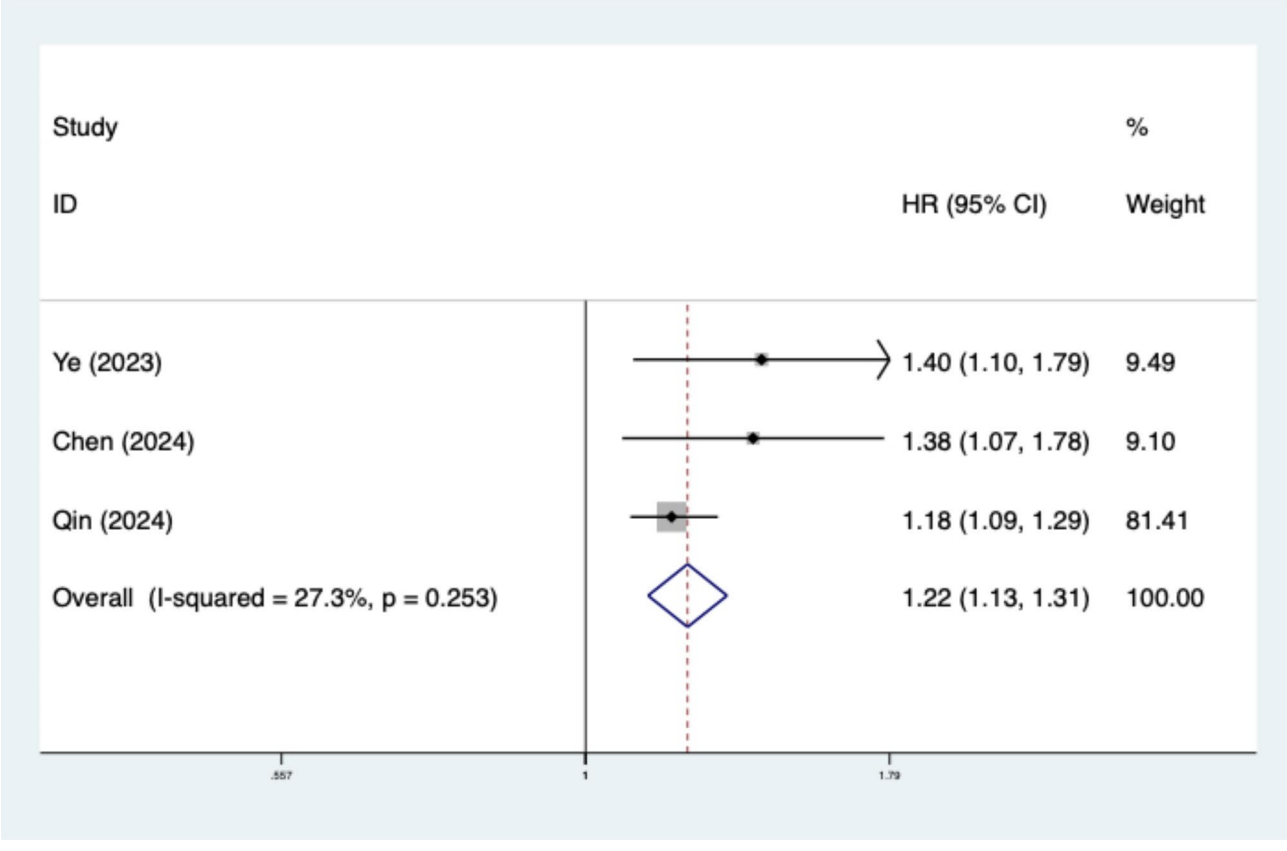

Fig. 2 The association between triglyceride-glucose index and risk of all-cause mortality among chronic kidney disease patients

Table 1 Basic characteristics of included studies

Author	Year	Country	Sample size	Study design	Age, years	Stage	History of diabetes	Endpoints	NOS
Choe [15]	2020	Republic ofKorea	819	Retrospective	64.7 ±15.3	3-5	Mixed	NAFLD	6
Chen [16]	2022	China	376	Retrospective	53.52±13.45	2-5	Mixed	MACE	7
Guan [17]	2022	China	884	Retrospective	73.66±8.57	3-5	Mixed	SCAS	6
Low [18]	2022	Singapore	1571	Prospective	57.3±11.0	1-4	Yes	CKD progression	6
Duan [19]	2023	China	179	Retrospective	52.28±11.22	1-4	Yes	CKD progression	7
Gao [20]	2023	China	1936	Retrospective	67 (59-74)	NR	Yes	ESRD	7
Ye [21]	2023	China	639	Retrospective	75.0 (66.0-83.0)	NR	Mixed	In-hospital mortality, 1-year mortality	6
Chen [22]	2024	China	1537	Retrospective	68.11±12.87	NR	Mixed	In-hospital mortality, 1-year mortality	7
Ko [23]	2024	Republic ofKorea	1154	Prospective	52.8±11.9	1-5	Mixed	CAC progression	7
Li [24]	2024	China	13517	Retrospective	57.92±13.54	NR	Mixed	MACE	7
Liu [25]	2024	China	943	Retrospective	70.96±8.78/74.42±8.42	3-5	Mixed	CAD, SCAS	6
Qin [26]	2024	China	2975	Retrospective	56.6 (55.7-57.6)	NR	NR	CAD mortality, all-cause mortality	7

NAFLD: nonalcoholic fatty liver disease; SCAS: severe coronary artery stenosis; ESRD: end-stage renal disease; CAC: coronary artery calcification; MACE: major adverse cardiovascular event; CAD: coronary artery disease; NOS: Newcastle-Ottawa Scale

Table 2 Results of meta-analysis

Items	No of studies	HR/OR	95% confidence interval	*P* value	I^2 (%)	*P* value
Primary outcome						
All-cause mortality	3	1.22 (HR)	1.13–1.31	<0.001	27.3	0.253
Secondary outcome						
CAD mortality	1	1.19 (HR)	1.04–1.36	0.011	-	-
CKD progression	2	1.52 (HR)	1.36–1.70	<0.001	0.0	0.554
SCAS	2	1.79	1.13–2.83	0.013	63.8	0.096
MACE	2	1.68	1.11–2.54	0.014	56.5	0.129
CAC progression	1	1.55	1.06–1.76	0.02	-	-
CAD	1	2.865	1.681–4.885	<0.001	-	-
ESRD	1	1.49	1.12–1.99	0.006	-	-
NAFLD	1	4.903	3.046–7.893	<0.001	-	-

NAFLD: nonalcoholic fatty liver disease; SCAS: severe coronary artery stenosis; ESRD: end-stage renal disease; CAC: coronary artery calcification; MACE: major adverse cardiovascular event; CAD: coronary artery disease; HR: hazard ratio; OR: odds ratio

assessment, and all disagreements were resolved by team discussion.

Statistical analysis

All statistical analyses were conducted by STATA (version 15.0) software. For statistical heterogeneity, the I^2 statistic and Chi-square test (Q test) were used. An I^2 value greater than 50% and a p-value less than 0.10 in the Q test were considered indicative of substantial heterogeneity. However, the choice between fixed-effect and random-effects models was not based solely on these statistics. If the included studies were judged to be clinically and methodologically homogeneous, and statistical heterogeneity was low ($I^2 \leq 50\%$), a fixed-effect model was applied, assuming a common true effect across studies. If substantial clinical or methodological heterogeneity was present, or if significant statistical heterogeneity was observed ($I^2 > 50\%$), a random-effects model was employed to account for between-study variation. Effect sizes were reported as HRs for time-to-event outcomes including the all-cause mortality, CAD mortality and CKD progression and ORs for binary outcomes including the SCAS, MACE, CAC progression, CAD, ESRD and NAFLD, both with 95% CIs. The Inverse Variance (IV) method was used under the fixed-effect model to combine the natural logarithms of HRs and ORs (log HR and log OR). The DerSimonian-Laird method was applied for the random-effects model to account for between-study heterogeneity. Additionally, the Hartung-Knapp adjustment was used to improve the accuracy of the 95% CIs, particularly when the number of studies was small. During the meta-analysis, in-hospital mortality and one-year mortality were both regarded as all-cause mortality.

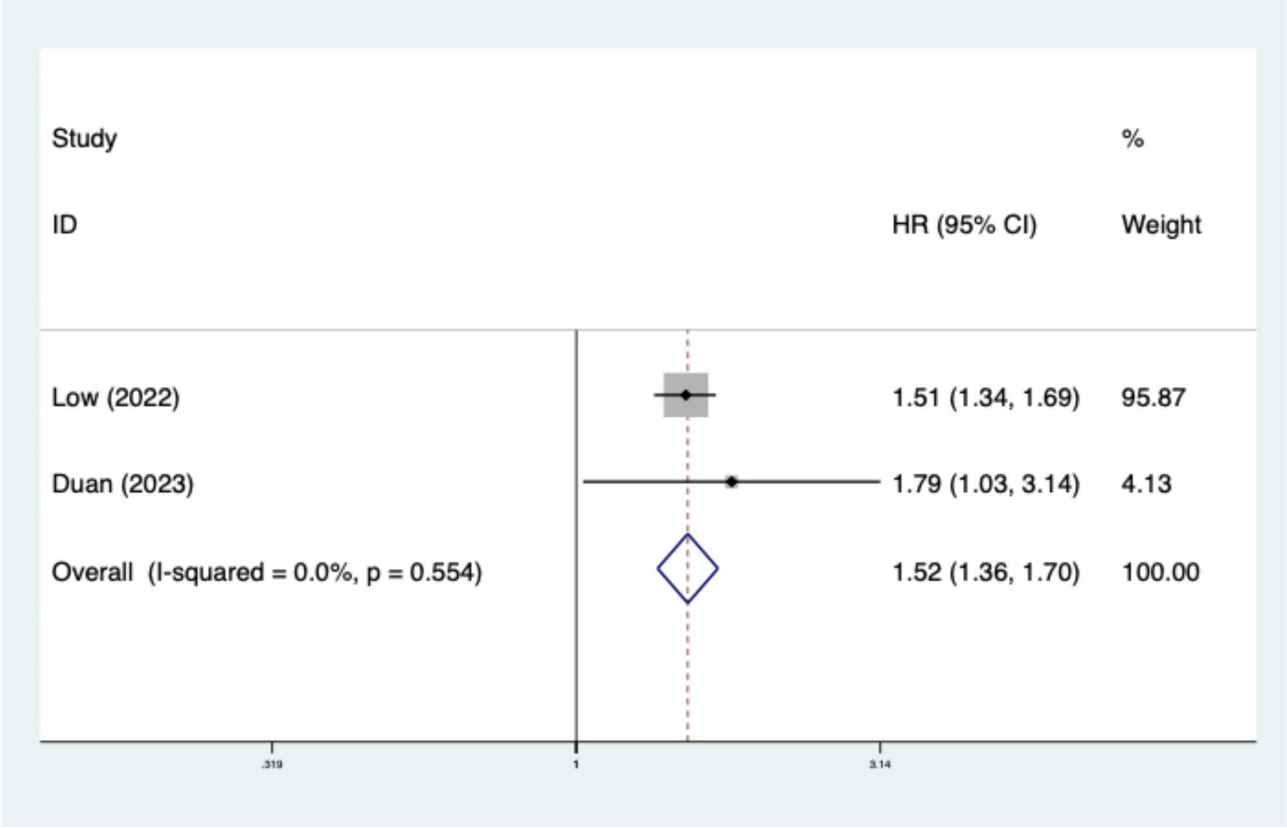

Fig. 3 The association between triglyceride-glucose index and risk of chronic kidney disease progression among chronic kidney disease patients

Results

Literature searching and selection process

As presented in Figs. 1 and 1238 records were searched from three databases and 98 duplicated records were removed. After reviewing titles and abstracts, 1118 publications were excluded. Then ten publications were excluded due to the insufficient ($n=7$) or duplication data ($n=3$). Finally, 12 studies were included in this meta-analysis [15–26]. The reasons for inclusion and exclusion of each study were presented in Supplementary Table 1.

Basic characteristics of included studies

A total of 26,530 patients were enrolled among the 11 studies with the sample size ranged from 179 to 13,517. Most studies were from China (9/12) and retrospective (10/12). Besides, three studies focused on patients with diabetes mellitus. All studies were with high-quality with the NOS score ≥ 6 (score 6: $n=5$, score 7: $n=7$). Detailed data were presented in Table 1.

Association between TyG index and primary outcomes

Three studies explored the relationship between TyG index and all-cause mortality. According to the pooled results, elevated TyG index was significantly associated with increased risk of all-cause mortality (HR = 1.22, 95% CI: 1.13–1.31, $P<0.001$; $I^2=27.3\%$, $P=0.253$) (Fig. 2) with the fixed-effect model. (Table 2)

Association between TyG index and secondary outcomes

Furthermore, high TyG index was associated with the CAD mortality (HR = 1.19, 95% CI: 1.04–1.36, $P=0.011$), occurrence of CKD progression (HR = 1.52, 95% CI: 1.36–1.70, $P<0.001$; $I^2=0.0\%$, $P=0.554$) (Fig. 3) with the fixed-effect model, SCAS (OR = 1.79, 95% CI: 1.13–2.83, $P=0.013$; $I^2=63.8\%$, $P=0.096$) (Fig. 4) with the random-effect model, MACE (OR = 1.68, 95% CI: 1.11–2.54, $P=0.014$; $I^2=56.5\%$, $P=0.129$) (Fig. 5) with the random-effect model, CAC progression (OR = 1.55, 95% CI: 1.06–1.76, $P=0.02$), CAD (OR = 2.865, 95% CI: 1.681–4.885, $P<0.001$), ESRD (OR = 1.49, 95% CI: 1.12–1.99, $P=0.006$), and NAFLD (OR = 4.903, 95% CI: 3.046–7.893, $P<0.001$). (Table 2) Therefor, the significant association of TyG index with worse prognosis was observed for all secondary outcomes.

Discussion

Although there are several relevant studies investigating the predictive role of TyG index for clinical outcomes in CKD patients, our study is the first meta-analysis to comprehensively and systematically clarify the prognostic value of TyG index in CKD, which contributes to the

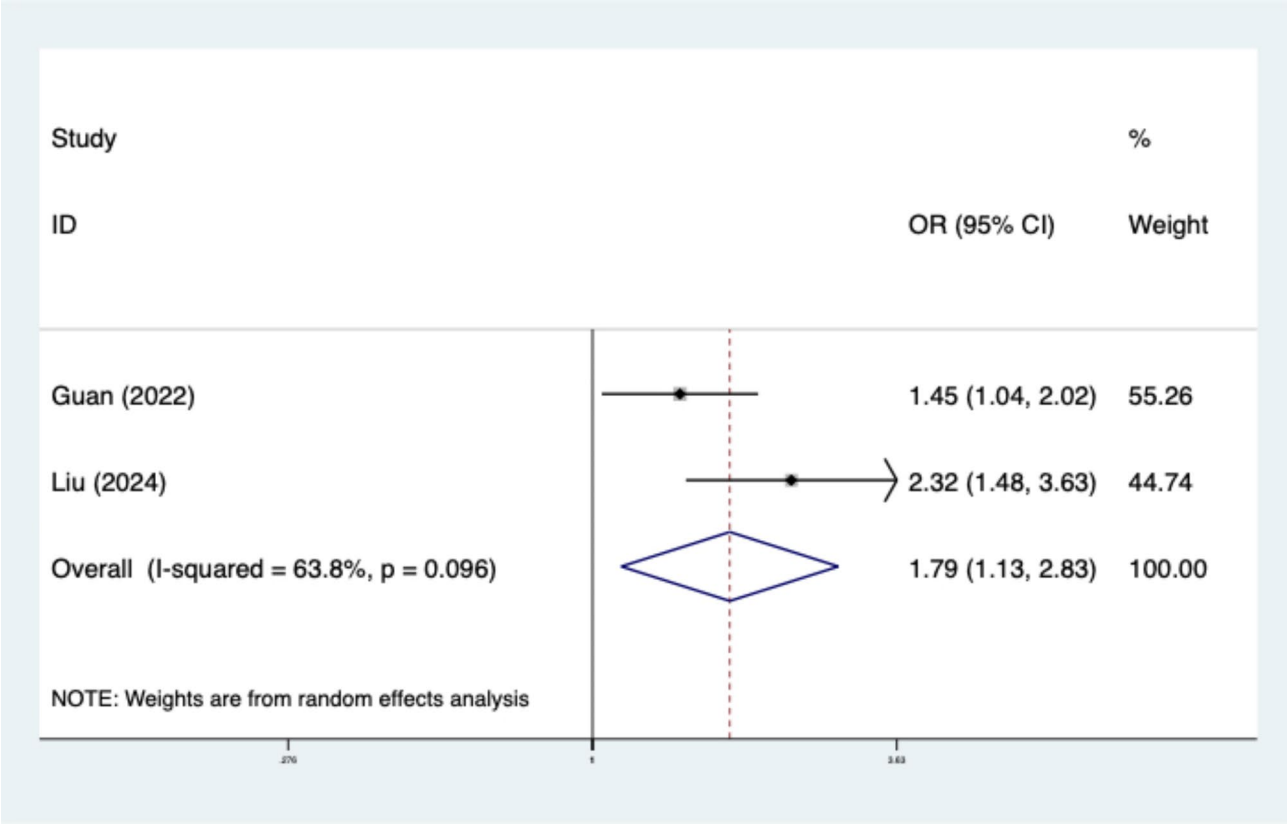

Fig. 4 The association between triglyceride-glucose index and risk of severe coronary artery stenosis among chronic kidney disease patients

application of TyG index among CKD patients and also provides the direction and thinking for future relevant studies. In this meta-analysis, it was manifested that TyG could serve as a prognostic indicator in CKD patients and patients with an elevated TyG index experienced significantly higher risk for poor clinical outcomes including the mortality risk and CKD progression. However, due to the limitations existed in included studies and this meta-analysis, more studies are needed to further verify our results.

Our results have indicated that elevated TyG index is closely related to increased risk of mortality. Based on current research advancements, the reasons why IR increases the mortality risk in CKD patients can be attributed to several complex mechanisms. First, insulin resistance leads to chronic low-grade inflammation, promoting the release of inflammatory factors such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) [27]. These inflammatory factors not only accelerate kidney damage but also increase the risk of cardiovascular diseases, which are the leading cause of death in CKD patients [27]. Second, IR causes an increase in oxidative stress, characterized by the excessive production of free radicals and a weakened antioxidant defense system [28]. Oxidative stress can lead to glomerulosclerosis and tubulointerstitial fibrosis, thus accelerating the progression of CKD. Moreover, oxidative stress damages the vascular endothelium, promoting atherosclerosis and increasing the incidence of cardiovascular events [28]. Additionally, IR promotes vascular disease through various pathways. For example, it increases the activity of vasoconstrictors such as angiotensin II and reduces the production of vasodilators such as nitric oxide, leading to elevated blood pressure and arteriosclerosis. These vascular diseases are particularly deadly in CKD patients, who are already at high risk for cardiovascular conditions [29, 30]. Furthermore, IR is associated with increased proteinuria, which is an important marker of CKD progression. As CKD progresses, kidney function continues to deteriorate, ultimately leading to ESRD, which significantly increases the risk of death [31, 32]. In summary, insulin resistance markedly raises the mortality risk in CKD patients by triggering and exacerbating these pathological processes. Our study results further demonstrate that IR primarily leads to adverse outcomes in CKD patients by increasing the risk of cardiovascular disease and CKD progression.

Improving IR can cause a positive impact on the prognosis of patients with CKD. We believe the following methods can enhance CKD outcomes by improving IR. First, improving blood glucose control and reducing IR can mitigate the damage diabetes does to the kidneys.

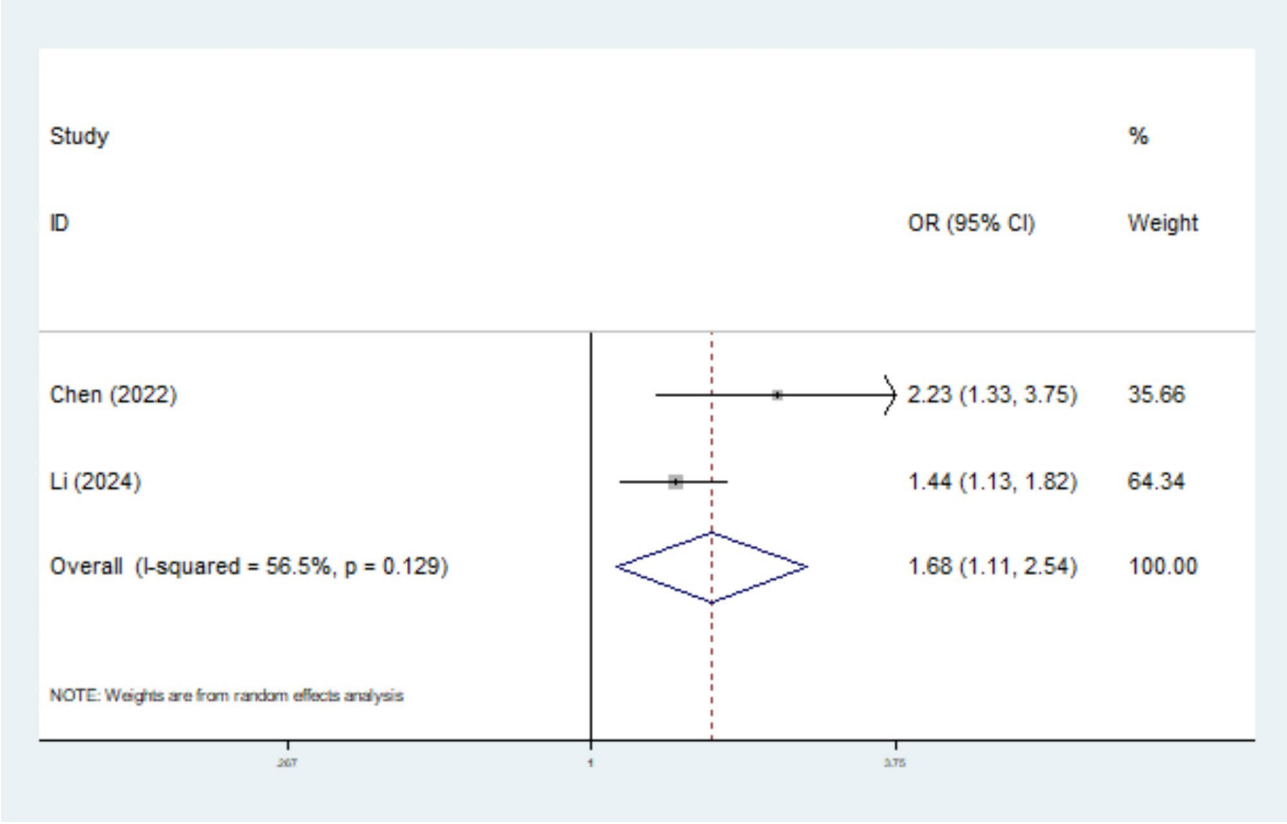

Fig. 5 The association between triglyceride-glucose index and risk of severe coronary artery stenosis among chronic kidney disease patients

Using insulin-sensitizing medications (such as metformin) and good glucose management can help control diabetes and slow CKD progression. Second, dietary improvements such as adopting a low-sugar, low-fat, high-fiber diet can help improve IR. Including antioxidant-rich foods like fresh fruits and vegetables in the diet can help reduce inflammation and oxidative stress [33]. Regular physical activity also improves insulin sensitivity, helps control weight and blood glucose levels. Aerobic exercise and strength training are recommended to enhance overall insulin sensitivity. In addition to metformin, other medications such as GLP-1 receptor agonists and SGLT2 inhibitors can also improve insulin resistance and potentially provide protection for CKD [33]. Recent studies have shown that SGLT2 inhibitors not only reduce the risk of cardiovascular events but also slow the progression of CKD, even in advanced stages [34]. Similarly, GLP-1 receptor agonists have shown promise in reducing albuminuria and improving glycemic control in CKD patients. Although their efficacy in advanced CKD stages (e.g., stages 4 and 5) is less well-studied compared to SGLT2i, emerging evidence suggests that they may offer cardiovascular and metabolic benefits without significantly compromising renal function [35, 36]. Hypertension and dyslipidemia are closely related to IR. Managing these factors through medication and lifestyle adjustments can help reduce the burden on the kidneys and improve CKD prognosis. Furthermore, weight reduction, especially in overweight or obese CKD patients, can significantly improve IR, reduce insulin requirements, and slow CKD progression. Finally, regular monitoring of blood glucose, insulin levels, and kidney function is crucial. It helps in timely adjustment of treatment plans to prevent further deterioration of the condition [8, 33, 37].

Notably, this meta-analysis highlights the potential of TyG index as a marker associated with adverse outcomes in CKD. However, the absence of subgroup analyses by CKD stage limits our ability to determine whether the TyG index is more predictive in early, moderate, or advanced CKD. Future studies should address this gap to clarify its stage-specific utility. Additionally, changes in the TyG index over time may reflect metabolic shifts, offering potential as a dynamic marker for monitoring disease progression or therapeutic response. Incorporating the TyG index into existing risk assessment tools could enhance their predictive power, providing greater clinical utility. While the TyG index is linked to IR, our study is unable to explore whether interventions targeting IR, such as metformin, SGLT2 inhibitors, or lifestyle modifications, could influence outcomes associated with a high TyG index. Investigating these interventions in

future studies would be crucial for translating these findings into clinical practice and improving patient outcomes. Therefore, more relevant studies are still needed to further address these issues.

There are some limitations in this meta-analysis. First, the overall sample size is relatively small and most studies are retrospective, which may cause some bias. Second, significant heterogeneity was observed for several endpoints. However, for most endpoints, only one or two studies were included for analysis. It is difficult to perform the sensitivity analysis and subgroup analysis to explore the source of heterogeneity. Third, we are unable to perform subgroup analysis based on some important parameters such as the history of diabetes mellitus and comorbidity without the original data. Therefore, more detailed investigations should be performed in future studies to explore the modification of these factors on the association of TyG index with clinical outcomes in CKD patients. Four, due to the limited number of included studies for each endpoint, it is unable to conduct the sensitivity analysis and publication bias detection and more studies are still needed to verify our findings. Five, the CIs for some outcomes are relatively wide such as the NAFLD and CAD, which might indicate the low statistical power or variability in the data. Five, all studies are from Asian countries, which might affect the generalizability of our conclusions. Besides, the dietary, culture, genetic and racial characteristics might cause an impact on the predictive role of TyG index for clinical outcomes among CKD patients, which should be further identified by studies from other countries.

Conclusion

High TyG index predicts poor clinical outcomes such as the all-cause mortality and CKD progression and might serve as a novel prognostic indicator among CKD patients. However, more prospective studies are still needed to verify above findings due to the limitations in this meta-analysis.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-025-03984-w .

Supplementary Material 1

Supplementary Material 2

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None.

Author contributions
Linshen Xie conceived and designed the analyses. Jinli Tuo and Zhong Li performed the literature search and selection, collected data, conducted statistical analyses and wrote the paper. All authors contributed substantially to its revision.

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None.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in studies that involved 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.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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