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ORIGINAL ARTICLE

Triglyceride-Glucose Index as an Indicator for Early Prediction and Progression of Chronic kidney Disease in Hypertension Patients

Ezzat Mostafa Mohamed[1], Sara Mohammed Hassan[*1], Fatma M. Attia Elsayed [1], Fatma Atef Ibrahim[2], Reda Abd Elmoniem Kamel [1]

[1] Internal Medicine Department, Faculty of Medicine, Zagazig University, Egypt.
[2] Clinical Pathology Department, Faculty of Medicine, Zagazig University, Egypt.

***Corresponding author:**
Sara Mohammed Hassan
Email:
Saraspot2010@gmail.com

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ABSTRACT

Background: In order to apply evidence-based therapies that can prevent the progression of chronic kidney disease (CKD) to advanced stages, routine screening in hypertensive individuals is essential at the time of diagnosis. This study aim to early prediction and monitoring of progression of CKD in hypertensive patients using Triglyceride-Glucose Index (TyG index). **Methods:** This retrospective case control study included 85 hypertensive patients with CKD and a similar number of hypertensive patients without CKD. The patients data were followed up for 6 months, eGFR and TyG index were evaluated. **Results:** Patients without CKD had considerably lower diastolic blood pressure than those with CKD. Patients without CKD experienced a considerably shorter mean duration of hypertensive than individuals without CKD. CKD patients had lower HDL and greater cholesterol. Compared to patients with CKD, those without CKD had a much higher eGFR. Patients with CKD had a considerably higher TyG index level than individuals without CKD. TyG index demonstrated 100% sensitivity and accuracy in predicting CKD in hypertension patients, and its diagnostic performance in predicting CKD in hypertensive patients at cutoff was greater than 8.91. **Conclusion:** TyG index is a useful biomarker for the prediction of CKD in hypertensive patients and identifying individuals who are at risk of CKD.

Keywords: Chronic kidney Disease; Hypertension ; Triglyceride-Glucose Index

INTRODUCTION

According to contemporary epidemiological statistics, around one in four American adults, or 65 million adults, suffer with hypertension. Increased cardiovascular morbidity and mortality, such as heart failure, stroke, myocardial infarction, and cardiovascular death, have been associated with elevated systemic arterial pressures. Approximately one-fourth of adults worldwide suffer from hypertension, one of the main public health challenges. For many illness conditions, it is still one of the most reversible causes [1].

However, many studies have been conducted on the relationship between hypertension and chronic kidney disease (CKD). In recent decades, molecular genetic studies have clarified the link between hypertension and the development of chronic kidney disease [2].

According to the US Renal Data System, the second most frequent cause of end-stage kidney disease (ESKD) requiring renal replacement therapy is hypertension. after diabetes mellitus. As a result, clinicians have been increasingly interested in "hypertension-attributed" nephropathy, and it is now understood that lowering systemic blood pressure can either halt or stop the evolution of nephropathy in non-diabetics [3].

In recent years, it has been demonstrated that the Triglyceride-Glucose (TyG) index is helpful in determining the risk of adult insulin resistance [3]. Numerous studies have examined the connection between TyG index and CKD risk; a recent meta-analysis revealed that, regardless of known risk variables, a higher TyG index was linked to an increased risk of CKD[3,4].

Regardless of the BP subtype, high TyG levels are linked to the new onset of CKD. It's interesting to note that both normotensive and isolated diastolic hypertension (IDH) patients exhibit this connection. IDH, which is commonly ignored and not considered hypertension, is the most common kind of hypertension in young to middle-aged males. High TyG levels are linked to the likelihood of developing CKD in the future, even in individuals with normal blood pressure and IDH, which is a highly important discovery [1].

Thus, the Triglyceride-Glucose Index was used in this study. (TyG index) to predict and track the development of CKD in hypertensive individuals early.

METHODS

This retrospective case-control study was conducted between December 2024 and July 2025 through the collaborative efforts of the Clinical Pathology and Internal Medicine Departments (Nephrology Unit) at Zagazig University Hospitals, in addition to Ahmed Maher Teaching Hospital. A total of 170 hypertensive participants were enrolled and categorized into two equal groups: Group 1 (n = 85), comprising patients diagnosed with hypertensive chronic kidney disease (CKD), and Group 2 (n=85), consisting of hypertensive patients without CKD.

Hypertensive people aged 30 years or older who had either maintained renal function (eGFR > 60 mL/min/1.73 m²) or CKD, as indicated by an eGFR < 60 mL/min/1.73 m², were eligible to participate in this study. Written informed consent was given by each participant, and all clinical data were accessible. People with severe renal impairment (eGFR < 30 mL/min/1.73 m^2), those on hemodialysis, and patients with comorbid conditions that could confound the study outcomes, including thyroid disorders, infectious diseases, pregnancy, coronary atherosclerosis, heart disease, heart failure, secondary hypertension, familial hyperlipidemia, or those receiving hypoglycemic medications we're excluded from the study.

Ethical consideration:

The study was approved by the Academic and Ethical Committee of Zagazig University. (IRB#475/1- July-2024). This study was conducted in accordance with the Declaration of Helsinki, which is the World Medical Association's Code of Ethics for research involving human subjects.

Operational design:

All participants underwent a standardized assessment that included a detailed medical history, clinical examination, and calculation of BMI using the standard formula (kg/m²). Blood pressure was measured according to established clinical protocols. Renal ultrasonography was conducted to assess kidney morphology and rule out obstruction. Additional imaging studies, including CT, MRI, echocardiography, ECG, and chest X-ray, were performed when clinically indicated.

Laboratory investigations comprised a comprehensive panel of biochemical and hematological tests. Renal function was assessed via serum creatinine and urea levels, while liver function was evaluated using AST, ALT, albumin, and total bilirubin. Blood samples (5 mL) were collected in anticoagulant-free tubes, allowed to clot, centrifuged, and analyzed using a semi-automated analyzer (Cobass 8000). Fasting blood glucose was measured using the glucose oxidase method. Complete blood count (CBC) including hemoglobin, platelet count, and total white blood cells was performed on 2.5 mL of blood collected in EDTA tubes and analyzed using an automated hematology analyzer. Random blood glucose was also recorded. Following a 12-hour fast, lipid profiles (total cholesterol, triglycerides, HDL, and LDL) were measured using EDTA-treated samples, centrifuged at 2,500–3,000 rpm, and analyzed.

The recommended lipid thresholds were LDL < 100 mg/dL, HDL ≥ 50 mg/dL for females and ≥ 40 mg/dL for males, triglycerides < 150 mg/dL, and total cholesterol < 200 mg/dL. In addition, the triglyceride-glucose (TyG) index was calculated using the formula: TyG = log [fasting triglycerides (mg/dL) × fasting blood glucose (mg/dL)/2], with cut-off values of 8.8 for males and 8.7 for females [6].

TyG is calculated using the formula: TyG = log (fasting triglycerides) (TG) (mg/dL) × fasting blood glucose (FBG) (mg/dL)/2).

CKD-EPI Equation for Estimating GFR on the Natural Scale Expressed for Specified Sex, Standardized Serum Creatinine and Standardized Serum Cystatin C (From New Eng J Med 2021)

Sex	Serum Creatinine (mg/dL)	Equation
Female	≤0.7	$GFR = 142 \times (Scr/0.7)^{-0.241} \times 0.9938^{Age} \times 1.012$
Female	>0.7	$GFR = 142 \times (Scr/0.7)^{-1.200} \times 0.9938^{Age} \times 1.012$
Male	≤0.9	$GFR = 142 \times (Scr/0.9)^{-0.302} \times 0.9938^{Age}$
Male	>0.9	$GFR = 142 \times (Scr/0.9)^{-1.200} \times 0.9938^{Age}$

Estimated Glomerular Filtration Rate (eGFR) calculation:

The CKD-EPI equation is a formula used to estimate glomerular filtration rate (GFR) using serum creatinine and other variables, with the 2021 version, GFR = 142 × min(Scr/κ, 1)^α × max(Scr/κ, 1)^-1.200 × 0.9938^Age × 1.012 [if female], being a widely used form that omits the race variable. Other forms exist, including those that incorporate serum cystatin C or a different sex factor, to provide more accurate GFR estimates.

Study outcome:

The primary outcome of the study was the change in the triglyceride-glucose (TyG) index, defined as the difference between baseline and six-month follow-up values. Changes were categorized using a cut-off value of 0: a value ≥ 0 indicated an increase, while a value < 0 indicated a decrease in the TyG index. Renal function was evaluated using the estimated glomerular filtration rate (eGFR). For patients with a baseline eGFR ≥ 60 mL/min/1.73 m², the renal endpoint was defined as a ≥30% reduction resulting in a final eGFR < 60 mL/min/1.73 m². For those with a baseline eGFR < 60 mL/min/1.73 m², the outcome was a ≥50% decline in eGFR or progression to end-stage renal disease requiring dialysis. Both baseline and six-month values of eGFR and TyG index were analyzed to assess changes and their association with renal function decline.

Statistical analysis:

IBM SPSS statistical software for social research, version 23, was used. The median and IQR were shown for non-parametric data, whereas the mean, standard deviations were shown for parametric data. Qualitative characteristics were also shown as figures and percentages. We used the independent t-test, chi-square test, one-way ANOVA test, Mann-Whitney test, and/or Fisher exact test. An acceptable margin of error of 5% and a 95% confidence interval were determined. The following p-value was deemed significant since P-values >0.05, P≤0.05, and P≤0.01 denote non-significant, significant, and extremely significant, respectively.

RESULTS

According to the current study, patients without CKD had a substantially higher mean BMI than patients with CKD (P-value=0.05). Patients without CKD smoked substantially less than those with CKD (P-value≤ 0.05). However, there is no discernible variation in age or sex distribution between hypertension individuals with chronic renal disease and those without (Table 1). Individuals with and without chronic kidney disease (CKD) had significantly lower mean diastolic blood pressure, but marginally different systolic blood pressure. Compared to patients with CKD, those without CKD experienced a considerably shorter mean duration of hypertension. Antihypertensive medication (excluding all drugs that can affect lipid profile e.g. beta blockers) use was significantly higher in 83.5% of patients with CKD than in 62.9.4% of individuals without CKD, indicating a substantial difference between the two groups of patients. (Table 1).

The mean hemoglobin level was significantly higher in patients without CKD than in those with CKD (p≤ 0.05). Patients with and without chronic renal impairment do not differ substantially in their white blood cell and platelet counts (p>0.05) (Table 2).

Patients with and without chronic renal failure had significantly different serum albumin levels (p≤0.05). Serum ALT and AST levels did not significantly differ between those with and without CKD, as seen in Table (2).

There was a significant difference in the triglyceride and LDL levels between the research groups; those without CKD had lower levels than those with CKD (p = 0.001). The groups under investigation differed significantly in terms of HDL and cholesterol: CKD patients had higher cholesterol and lower HDL (p<0.05) (Table 3). Compared to patients with CKD, individuals without CKD showed significantly lower mean urea and creatinine levels (p <0.001). However, those

without CKD had a considerably higher mean GFR (p <0.001) than those with CKD (Table 4& Fig 1). Patients with CKD had a substantially higher mean TyG index level than those without CKD (p <0.001) (Table 4). When used as a diagnostic tool to predict chronic kidney disease in hypertensive individuals, the TyG index demonstrated 100% sensitivity, specificity, positive predictive value, negative predictive value, and accuracy at a threshold of greater than 8.91 (Table 5) & (Fig 2).

Table 1: Comparison between hypertensives with CKD and without CKD as regards demographic and basic characteristics data

Basic characteristics	With CKD (N=85)		Without CKD (N=85)		Test	p-value	Sig.
	No.	%	No.	%			
Sex **Male** **Female**	**40** **45**	**47%** **53%**	**42** **43**	**49.4%** **50.6%**	**1.399§**	**0.237**	**NS**
Age (days) **Mean ± SD** **Median (Range)**	**61.08 ± 9.55** **60 (41 – 75)**		**61.05 ± 13.17** **61 (35 – 85)**		**-0.248•**	**0.804**	**NS**
BMI (kg/M^2) **Mean ± SD** **Median (Range)**	**23.13 ± 2.42** **22 (20.5 – 25.60)**		**25.39 ± 3.48** **23 (22.5 – 28.60)**		**3.218***	**0.05**	**S**
Current smoker	**31**	**36.4%**	**25**	**29.4%**	**1.435§**	**0.030**	**S**
SBP (mmHg) **Mean ± SD** **Median (Range)**	**133.61 ± 15.70** **130 (110 – 160)**		**130 ± 12.87** **130 (110 – 160)**		**-0.961•**	**0.337 (NS)**	**NS**
DBP (mmHg) **Mean ± SD** **Median (Range)**	**86.11 ± 9.34** **90 (70 – 100)**		**80.27 ± 8.44** **80 (70 – 100)**		**-2.628•**	**0.009 (S)**	**S**
Duration (years) **Mean ± SD** **Median (Range)**	**14.05 ± 7.01** **11 (5 – 30)**		**4.33 ± 2.01** **4 (1 – 9)**		**-6.786•**	**<0.001**	**HS**
Antihypertensive drugs (excluding all drugs that can affect lipid profile)							
One drug **two drugs** **three drugs**	**6** **62** **17**	**7%** **73%** **20%**	**31** **48** **6**	**36.5%** **56.5%** **7%**	**1.213§**	**<0.001**	**HS**
Antiplatelet drugs	**3**	**4.7%**	**4**	**5.5%**	**3.145§-**	**0.61**	**NS**

* Independent samples Student's t-test. • Mann Whitney U test. § Chi-square test.
Significant(S): p≤ 0.05; Non-Significance (NS): p>0.05, High significance (HS) : p ≤ 0.001

Table 2: Comparison between hypertensives with CKD and without CKD as regards complete blood count & liver function tests

Complete blood count	With CKD (N=85)	Without CKD (N=85)	Test•	p-value	Sig.
Hb (g/dl)					
Mean ± SD	**10.59 ± 1.90**	**11.47 ± 2.85**	**-2.361**	**0.018**	**S**
Median (Range)	**10.55 (7.40 – 14.40)**	**12.10 (0 – 15.40)**			
WBC (x103/cc)					
Mean ± SD	**7.37 ± 2.25**	**8.13 ± 2.18**	**-0.377**	**0.706**	**NS**
Median (Range)	**6.95 (4 – 14.80)**	**7.70 (4.40 – 14.40)**			
Platelet (x103/cc)					
Mean ± SD	**218.38 ± 70.98**	**233.86 ± 95.40**	**-0.377**	**0.706**	**NS**
Median (Range)	**212.50 (100 – 400)**	**226 (100 – 473)**			
Liver function tests					
ALT (U/L)					
Mean ± SD	**18.47 ± 10.72**	**20.48 ± 9.64**	**-1.115**	**0.265**	**NS**
Median (Range)	**15.90 (5 – 45)**	**18.30 (6.80 – 45)**			
AST (U/L)					
Mean ± SD	**22.36 ± 9.78**	**25.18 ± 9.31**	**-1.431**	**0.153**	**NS**
Median (Range)	**20.70 (8.10 – 51.40)**	**26.20 (8.10 – 49.20)**			
albumin (g/dl)					
Mean ± SD	**3.1 ± 0.1**	**3.9 ± 0.2**	**1.812**	**0.03**	**S**
Median (Range)	**2.7 (2.3 – 3.2)**	**3.4 (2.9 – 4.1)**			

• Mann Whitney U test. Hb: hemoglobin; WBC: white blood cells
Significant(S): p≤ 0.05; Non-Significance (NS): p>0.05; High significance (HS): p ≤ 0.001

Table 3: Comparison between hypertensives with CKD and without CKD as regards lipid profile

Lipid profile	With CKD (N=85)	Without CKD (N=85)	Test	p-value	Sig.
Cholesterol (mg/dl)					
Mean ± SD	181.32 ± 35.44	152.98 ± 39	-0.949*	0.04	S
Median (Range)	181.85 (120 – 259.40)	175.65 (109 – 256)			
Triglycerides (mg/dl)					
Mean ± SD	155.28 ± 60.93	118.51 ± 47.43	2.394•	0.001	HS
Median (Range)	110.75 (58.50 – 352)	106.60 (58.50 – 252.80)			
HDL (mg/dl)					
Mean ± SD	44.37 ± 12.81	54.40 ± 15.17	1.265•	0.03	S
Median (Range)	42.75 (29.70 – 87.80)	43.65 (24.70 – 87.80)			
LDL (mg/dl)					
Mean ± SD	159.49 ± 28.51	114.11 ± 29.64	2.744•	0.001	HS
Median (Range)	115.15 (72.20 – 214.80)	112.80 (66.20 – 214.80)			

* Independent samples Student's t-test. • Mann Whitney U test.
HDL: high density lipoprotein; LDL: low density lipoprotein
Significant(S): p≤ 0.05; Non-Significance (NS): p>0.05; High significance (HS): p ≤ 0.001

Table 4: Comparison between hypertensives with CKD and without CKD as regards renal function tests & TyG index

Renal function tests	With CKD (N=85)		Without CKD (N=85)		Test	p-value	Sig.
	No.	%	No.	%			
Urea (mg/dl)							
Mean ± SD	55.62 ± 33.45		18.86 ± 7.32		-6.427•	<0.001	HS
Median (Range)	52.50(18.90 – 193)		20 (8.10 – 32.50)				
Creatinine (mg/dl)							
Mean ± SD	2.69 ± 1.77		0.90 ± 0.17		-7.124•	<0.001	HS
Median (Range)	2 (1.02 – 8.50)		0.86 (0.60 – 1.23)				
GFR (mL/min/1.73m^2)							
Mean ± SD	30.57 ± 14.34		84.41 ± 15.72		15.175*	<0.001	HS
Median (Range)	31.15(6.40– 59.40)		81.50(63 – 122.30)				
TyG index							
Mean ± SD	13.667 ± 3.303		8.1 ± 0.49		-7.298	<0.001	HS
Median (Range)	12(10.060–16.283)		7(6.356 – 9.210)				

* Independent samples Student's t-test. • Mann Whitney U test. § Chi-square test.
Significant(S): p≤ 0.05; non-Significance (NS): p>0.05; High significance (HS) : p ≤ 0.001
GFR: glomerular filtration rate

Table (5): Diagnostic performance of TyG index in predication of chronic kidney disease among hypertensives patients: ROC curve analysis.

Cut-off Values	SN % (95% CI)	SP % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	Accuracy (95% CI)	AUROC (95% CI)	p-value	Sig
TG index >8.91	**100% (90.3-100)**	**100% (90.3-100)**	**100% (90.3-100)**	**100% (90.3-100)**	**100% (90.3-100)**	**1.000 (0.950-1.000)**	**<0.001**	**HS**

ROC curve: Receiver Operating Characteristic curve; SN: Sensitivity; SP: Specificity; PPV: Positive Predictive Value; NPV: Negative Predictive Value; AUROC: Area Under Receiver Operating Characteristic curve; 95%CI: 95% Confidence Interval; p< 0.05 is significant

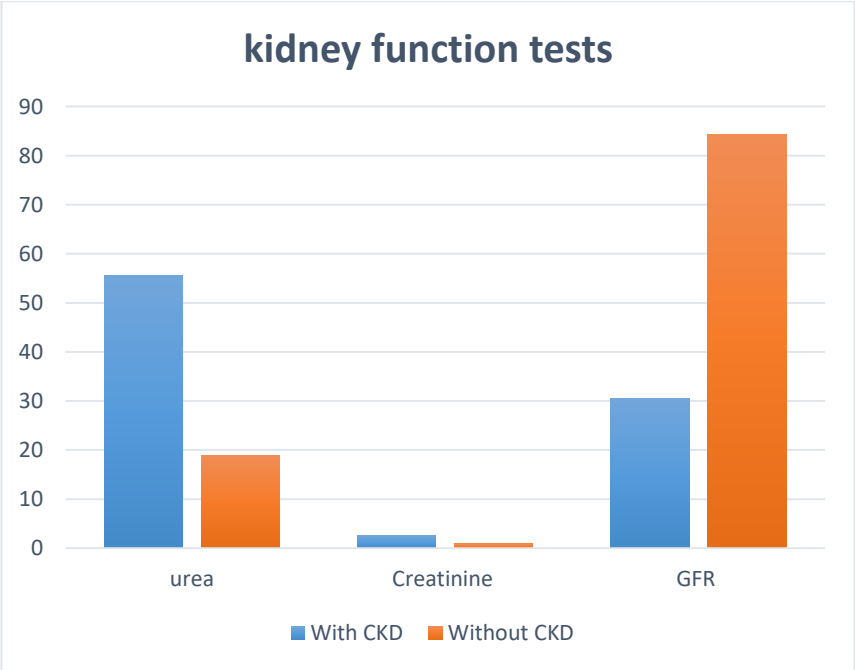

Figure 1: Renal function tests in studies groups.

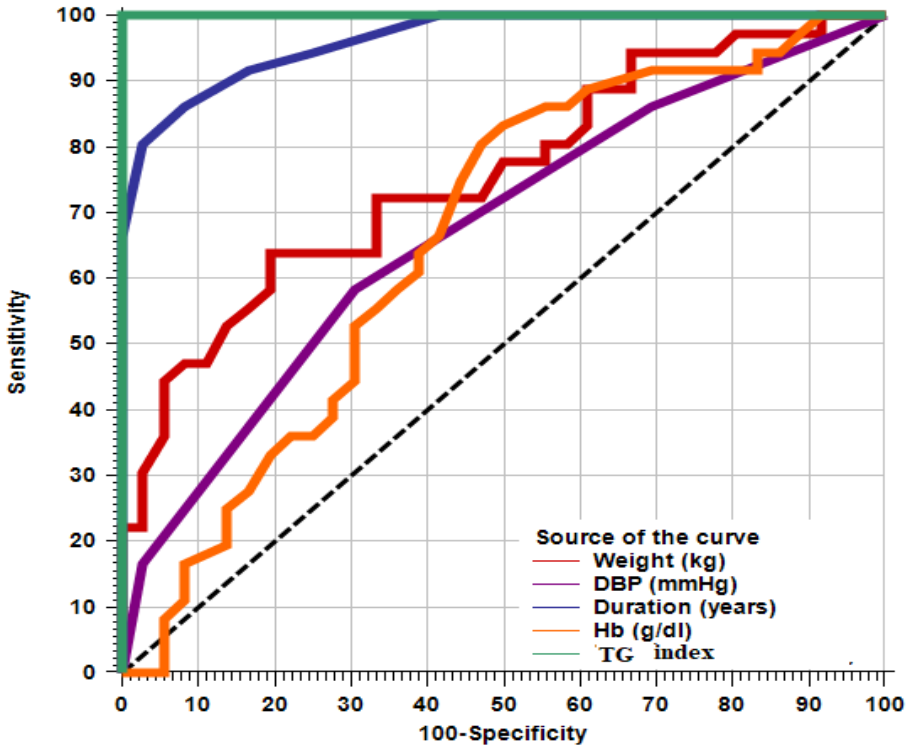

Figure 2: Receiver operating characteristic (ROC) curve of Weight (kg), DBP (mmHg), Duration (years), Hb (g/dl) and TyG index in predication of chronic kidney disease among hypertensive patients.

DISCUSSION

Many hypertensive people with chronic kidney disease (CKD) have incorrect diagnoses, and do not profit from early therapies as a result. Furthermore, CKD is usually diagnosed using blood urea and serum creatinine (sCr) levels; however, sCr has been shown to have little predictive value [7].

In patients with hypertension, CKD progression can be effectively prevented by regulating changes in baseline TyG, which is a consistent predictor of CKD progression [8].

This retrospective case control study, which aimed to predict and monitor the early course of CKD in hypertensive patients, included 85 hypertension patients with CKD and an equivalent number of CKD-free hypertensive patients. The mean age of those with and without chronic kidney disease (CKD) was 61.08 (± 9.55) and 61.05 (± 13.17) years, according to our study.

Malnutrition and muscle wasting are prevalent in CKD patients and may have significant effects on survival, physical function, and other outcomes. In terms of sex distribution, compared to 49.4% of patients without CKD, 47% of CKD patients were male. Individuals with a low body mass index (BMI) and chronic kidney disease (CKD) are more likely to die than those who have a normal BMI Heerspink et al. [9]. According to our research, patients without CKD had a substantially higher mean BMI than those with CKD (Mean±SD: 25.39 ± 3.48 vs. 23.13 ± 2.42, respectively, p-value<0.001).

Similar to our findings, Rafiee et al. [10] They conducted a survey study to evaluate blood pressure control trends and predictors in 8,829 adult participants with hypertension without (n = 7,178) and with (n = 1,651) who took part in the National Health and Nutrition Examination Survey and had chronic kidney disease. The results showed that hypertensive people without CKD were less likely to have a BMI of less than 30 kg/m2 than CKD patients.

As previously stated, renal function is severely impacted by uncontrolled hypertension. The risk of developing ESKD was approximately 15 times higher for those with a baseline blood pressure measurement close to 180/100 mm Hg than for those with a baseline blood A blood pressure reading of roughly 110/70 mm Hg was reported by a major health screening registry [11]. According to the current study, diastolic blood pressure was noticeably greater in CKD patients than in non-CKD patient.

Similar to our findings, Yacoub et al. [12] found that Smoking significantly increases the risk of CKD (OR = 1.6, p = 0.009, 95% CI = 1.12-2.29). When compared to nonsmokers, current smokers have an increased risk of having CKD (OR = 1.63 p = 0.02, 95% CI = 1.08-2.45), while former smokers did not have a statistically significant difference. The risk increased with high cumulative quantity (OR among smokers with > 30 pack-years was 2.6, p 0.00, 95% CI = 1.53-4.41). Smoking increased the risk of CKD the most for those classified as hypertensive nephropathy (OR = 2.85, p = 0.01, 95% CI = 1.27-6.39).

Of the 10,271 hypertension patients enrolled at the time of the cross-sectional analysis, 3227 had CKD, defined as having an estimated GFR of less than 60 mL/min/1.73 m2 and/or a urine albumin-to-creatinine ratio of 30 mg/g or higher. Patients without chronic renal disease had lower ambulatory systolic and diastolic blood pressure than the mean sleeping systolic blood pressure at night (125.0 ± 17.9 vs. 117.5 ± 13.1 mm Hg, P <.001) [13].

Anemia is prevalent in CKD and is linked to negative consequences. As CKD worsens, hemoglobin levels decrease for a variety of reasons, but one of the main causes is the kidneys' inability to produce enough erythropoietin. By preventing intestinal iron absorption and the release of stored iron for erythropoiesis, hepcidin-mediated iron limitation further exacerbates anemia[14]. Hemoglobin levels were considerably lower in CKD patients than in non-CKD individuals in the current study.

Our study analysis showed that serum albumin was significantly higher in patients without CKD than patients with CKD (Mean ±SD: 3.9 ± 0.2 Vs 3.1 ± 0.1 respectively, p-value<0.001), in agreement with that Cheng et al. [15] found a negative and non-linear association between Albumin and renal function decline as well as renal prognosis in Japanese CKD patients through a secondary analysis of a prospective cohort study in which a total of 954 participants were non-selectively and consecutively collected from the research of CKD-ROUTE in Japan between November 2010 and December 2011.

Similar to our findings, Guo et al. [16] In order to evaluate the relationship between anemia and CKD, they carried out a cross-sectional study in a hospital. There were 163 predialysis patients with CKD at all stages

and 163 healthy controls without CKD. The results showed that compared to non-CKD controls, hemoglobin levels were considerably lower in CKD patients. (P-value <0.001). Additionally, 13.5% of non-CKD controls and 47.8% of CKD cases had anemia (Odds ratio 5.88, P-value <0.001).

Our findings showed a significantly significant difference in the levels of LDL and triglycerides across the research groups; CKD patients had higher amounts than those without CKD. In relation to HDL and cholesterol, there was a substantial difference between the groups under study: patients with CKD had lower HDL and greater cholesterol. Chruściel et al. [17] revealed findings about the lipid profile in CKD patients that were exactly the same as our findings. There was no significant change in the mean serum TC between the CKD and control groups, while the mean HDL was considerably lower in CKD patients according to a recent study that sought to identify the prevalence and pattern of dyslipidemia in CKD patients.

Also, Miazgowski. et al. [18] who treated patients with CKD who were 60 years of age or older. There were 22.0% of cases and 5.0% of controls with hypercholesterolemia, 73.0% with low HDL and 8.0% with low HDL, 33.0% with high LDL and 7.0% with high LDL, and 15.0% with hypertriglyceridemia and 1.0% with low HDL. The only characteristic in which the two groups differed statistically significantly was HDL. Consistent with our results, a study carried out by Keerthana et al [19] to assess CKD patients' blood lipid profiles and compare them to healthy controls. The results demonstrated that CKD patients' serum HDL levels were considerably lower than those of the control group.

Also, Powell-Wiley et al. [20] assessed renal function and lipid metabolism parameters in an older patient with hypertension. Elderly hypertension patients showed lower significant levels of HDL and higher significant levels of urea and creatinine than controls. According to the findings of this study, treating arterial hypertension lowers cardiovascular morbidity and mortality as well as the incidence of renal failure. Therefore, management of arterial hypertension should concentrate on both lowering blood pressure and treating related lipid disorders.

Bunout et al. [21] demonstrated that, in comparison to controls, CKD patients had considerably higher levels of all lipid profile indicators, with the exception of HDL cholesterol.

Also agree with our results Medford et al. [22] discovered a considerable rise in TG and LDL mean values in CKD patients.

On the same way Pearson et al. [23] discovered that, in comparison to control, TC, TG, and LDL were considerably elevated in CKD patients. Additionally, a study by Sakoda et al. [1] demonstrated that blood TC and TG levels in CRF patients were lower following hemodialysis and considerably higher prior to dialysis than in control volunteers. Furthermore, the level of LDL was significantly higher before hemodialysis than in controls but did not significantly decrease after hemodialysis.

Patients with CKD had a substantially higher mean TyG index level than those without CKD in the current study (Mean ±SD: 13.667± 3.303 and 8.1 ± 0.491, respectively, p-value<0.001).

Another study that contradicts ours is a cross-sectional study conducted by Ou et al. [24]. It looked into the connection between renal failure (eGFR <30 mL/min/1.73 m2) and the TyG index. This study looked at the impact of the TyG index on CKD in a number of earlier investigations. Likewise, earlier cross-sectional studies in individuals with hypertension showed a strong correlation between elevated TyG levels and CKD [25].

López-Jaramillo et al. [26] confirmed the relationship between CKD and the TyG index, which may be explained by IR, a special diabetic indication that more properly represents the body's metabolic disorders. However, individuals with hypertension also have metabolic illnesses in addition to diabetes because one of the elements of the metabolic syndrome is elevated blood pressure. This has important implications for how individuals with chronic renal disease are treated overall. This finding could significantly alter clinical practice by enabling real-time TyG index monitoring and an

accurate evaluation of the risk of CKD in hypertensive patients.

By altering the body's metabolic processes, IR modifies the pathophysiological mechanisms behind the correlation between the TyG index and CKD. The selective suppression of insulin receptors in podocytes or proximal tubules, which are expressed by many insulin-sensitive cell types in the kidney, results in proteinuria, renal disease, and hyperglycemia. Insulin signaling is necessary to preserve podocyte function and glomerular integrity [27].

The mechanism behind the association between the TyG index and CKD risk is still unknown. Nonetheless, a number of plausible theories have been put forth. One suggestion is that IR's detrimental effects on the kidney are involved. Research points to the involvement of anomalies in the adipocytokine-induced insulin signaling pathway. Failure of this system may result in increased vasoreactivity, angiogenesis, and decreased NO-mediated vasodilation, all of which could contribute to the deterioration of renal function [28].

One of the few research that emphasizes The TyG index is currently significant in predicting the beginning of CKD in hypertensive people. Our findings suggest that additional study is required to assess the significance of TyG and other critical biomarkers in at-risk patients. The current study does have many drawbacks, though. Its retrospective design precluded the inference of causality. The found relationships need to be confirmed by prospective research. Because the study group included only those with hypertension, the generalizability of our findings may be constrained. To confirm our findings, more research in a variety of populations is required. Errors in diagnosis could happen throughout the chart review process for electronic health records. The study's findings may not be as broadly applicable to the general community because the sample number of patients included was small and came from only one facility.

CONCLUSION

In hypertensive patients, the TyG index is a helpful biomarker for CKD prediction. According to our findings, TG index variability could be a valuable tool for determining who is at risk for developing chronic kidney disease (CKD), especially in patients with hypertension. In order to prevent chronic kidney disease (CKD) and its associated issues, the TyG index is a simple and reasonably priced biomarker with important implications for clinical practice and public health policy.

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Data availability: Upon reasonable request, the corresponding author will make the datasets created and/or be examined during the current investigation available.

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