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Chronic kidney disease among patients with hypertension in sub-Saharan Africa: a systematic review and meta-analysis

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Abstract

Introduction Chronic kidney disease is defined by the presence of kidney damage or decreased kidney function for at least three months, irrespective of the cause. Hypertensive kidney disease is one of the long-term complications of poorly controlled hypertension. It is the second leading cause of developing chronic kidney disease, next to diabetic mellitus.

Methods The literature was searched using an international electronic database (PubMed, Google Scholar, Hinari, and Open Google) to get significant studies on chronic kidney disease among hypertensive patients. This study is conducted to determine the pooled prevalence and associated factors of chronic kidney disease among hypertensive patients up to May 20, 2024. Heterogeneity between studies was checked using I^2 test statistics, and small study effects were checked using graphical and Egger's statistical tests at a 5% significance level. Subgroup analysis and sensitivity analysis were checked. A random-effects model was used to guess the pooled effect size across studies.

Result In this meta-analysis, 16 studies in sub-Saharan Africa were included with a total of 6648 participants who fulfilled the inclusion criteria. The estimated prevalence of CKD among hypertension patients was found to be 29.01% (95% CI: 23.03–34.99, $I^2 = 97.10\%$) in sub-Saharan Africa. Age greater than 60 years old (OR = 2.36; 95% CI: 1.02–3.71, I2 = 99.11%), uncontrolled blood pressure (OR = 6.57; 95% CI: 2.44–10.71, $I^2 = 97.38\%$), hypertensive patients with diabetes comorbidity (OR = 3.27; 95% CI: 1.65–4.89, $I^2 = 95.79\%$), Bing overweight (OR = 2.75; 95% CI: 1.04–4.46, $I^2 = 98.22\%$), and proteinuria (OR = 4.64, 95% CI: 4.09–5.18, $I^2 = 0.00\%$).

Conclusion Hypertension is one of the major causes of chronic kidney disease. Most patients living with hypertension develop CKD over time in sub-Saharan Africa. The highest prevalence of CKD among hypertension was observed in West Africa and Middle Africa.

Keywords Chronic kidney disease, Hypertension

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Introduction.

Background

Chronic kidney disease is defined as damage or a glomerular filtration rate (GFR) < 60 mL/min/1.73 m [2] for 3 months or more, irrespective of cause [1, 2]. It is a progressive kidney disorder that affects > 10% of the general population worldwide [3, 4]. Globally, the pattern of disease burden in the twenty-first century has dramatically shifted in favor of chronic illnesses [5, 6]. Noncommunicable diseases are future threats, even though infectious diseases constitute the main cause of death in low-income nations [7]. Chronic kidney disease significantly increases the burden of cardiovascular disease and the risk of death [8, 9].

The pathophysiology of hypertension in chronic kidney disease involves many factors, including a reduced number of functioning nephrons, sodium retention and volume expansion, activation of the sympathetic nervous system, and hormonal factors [10]. Endothelial dysfunction may contribute to the increased peripheral resistance by several mechanisms that lead to the enhancement of constriction and vascular remodeling [11]. Glomerular capillary hypertension is translated into increased mechanical stress affecting glomerular cells [12]. The relationship between hypertension and chronic kidney disease is bidirectional in nature, as they are cause and effect, vice versa [11].

Chronic kidney disease is a global burden that challenges both industrialized and developing nations [7, 13, 14]. It became one of the leading causes of death and suffering [3]. The global expected prevalence of CKD is 13.4% (11.7–15.1%), and patients with end-stage kidney disease demanding kidney replacement therapy are estimated between 4.902 and 7.083 million [15]. Globally, CKD increases due to the increasing prevalence of diabetes mellitus, hypertension, and obesity [16, 17]. The age-standardized prevalence of CKD stages 3 to 5 ranged from 7.6 to 13.7% in Central and Eastern Europe, respectively [18]. In Africa, the overall prevalence of CKD was 15.8% in the general population and 32.3% in high-risk populations, and it was significantly higher in sub-Saharan Africa compared to North Africa. The prevalence of CKD is higher in developing countries than in the developed world [17].

Hypertensive kidney disease is one of the long-term complications of poorly controlled hypertension [19–21]. It is the second leading cause of developing chronic kidney disease, next to diabetic mellitus [22–24]. It is a key pathogenic factor contributing to kidney function deterioration [25, 26]. The complicated relationship between hypertension and chronic kidney disease (CKD) presents a global challenge for the prevention of hypertension-related CKD [27]. The overall global pooled prevalence of chronic kidney disease among hypertensive patients was 34.97% [28]. Hypertension is the cause of approximately 30% of end-stage kidney disease findings in the United States; still, there has been an argument as to whether benign hypertension is a cause of chronic kidney disease [29]. The prevalence of CKD is higher among hypertensive patients in Africa, particularly for those in urban areas than in rural areas [30].

The global implications of hypertension-related kidney disease extend beyond clinical concerns, impacting healthcare systems, economies, and, most importantly, the quality of life of affected individuals [27]. Nowadays in developing countries, including Africa, the community suffers from morbidity, mortality, and the economic burden of dialysis. So this review was conducted to estimate the pooled magnitude of CKD among hypertension and its determinants up to May 20, 2024.

Methods

Reporting and registration protocol

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist [31] was used.

Search strategy

The literature was searched using an international electronic database (PubMed, Google Scholar, Hinari, Midline, Science Direct, Web Science, Open Google, and different repositories) to identify published reports on chronic kidney disease among hypertensive patients in sub-Saharan Africa up to May 20, 2024. During the search, Medical Subject Headings (MeSH), as well as the plain text, were used for the following keywords: chronic kidney disease" OR "hypertensive nephropathy' 'OR "chronic kidney failure" OR "kidney impairment' 'OR" end-stage kidney/renal disease' 'OR "kidney/ renal insufficiency" " OR kidney injury", OR" kidney damage," renal failure" OR AND "Hypertension" OR "hypertensive"(tiab) OR "high blood pressure," AND "sub-Saharan Africa, Africa, " and all sub-Saharan African countries by name. We have followed the search protocol described in the previous publication and used Boolean operators such as "AND" and "OR, "which were used to combine search terms.

Eligibility criteria

We used the CoCoPop (Condition, Context, and Population) approach for prevalence studies to declare inclusion and exclusion criteria.

Inclusion criteria

- Only cross-sectional studies.
- CKD among hypertensive patients in adults.

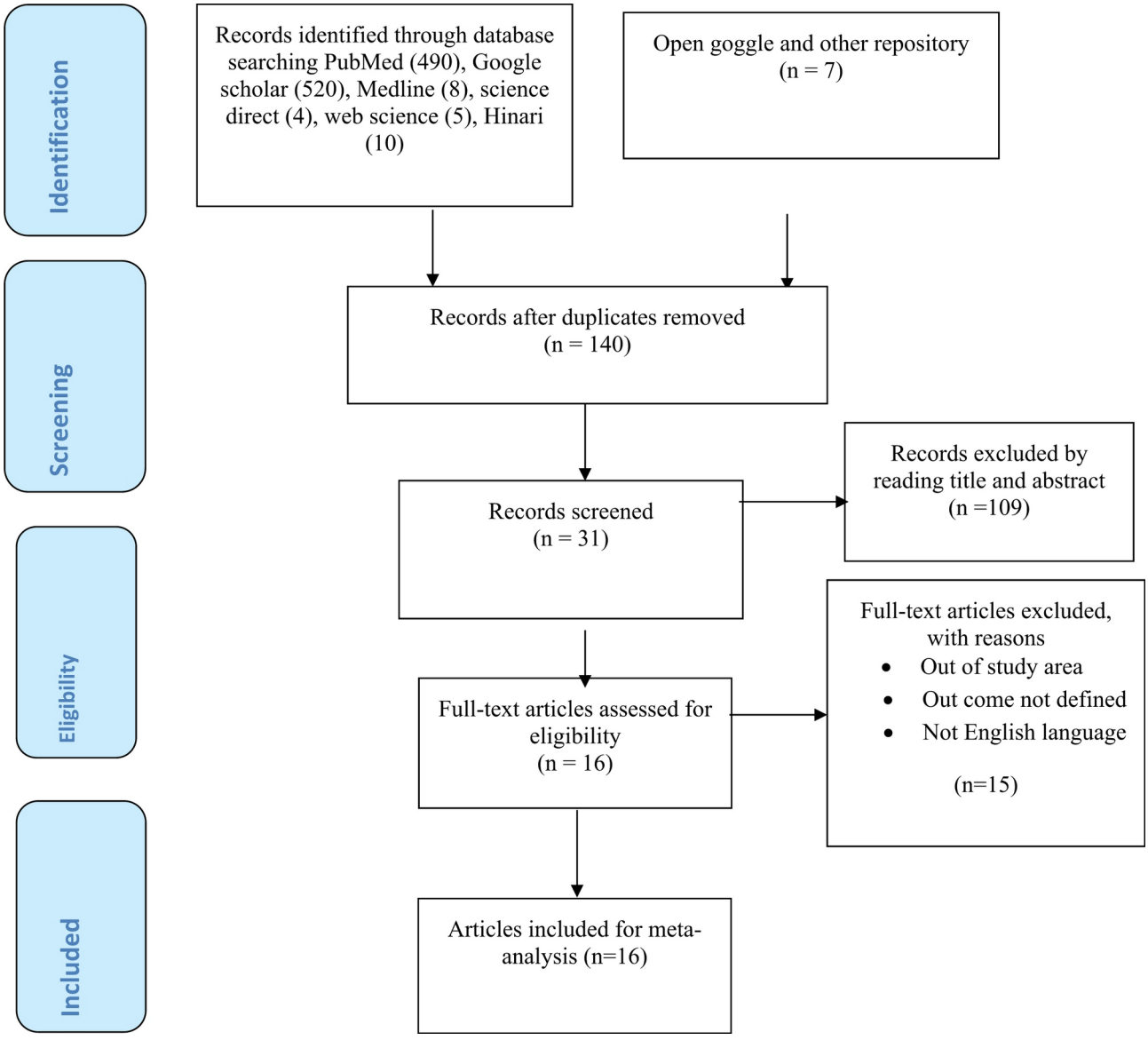

Fig. 1 PRISMA flow diagram of article selection for systematic review and meta-analysis of the prevalence of CKD among hypertensive patients in sub-Saharan Africa

- Articles published in peer-reviewed journals or grey literature.
- Articles published in English.

Exclusion criteria

- Studies not fully accessible.
- Poor quality as per the stated criteria.
- Studies with no report of prevalence.

Outcome measurement

This review has two main outcomes. The first outcome of interest is the pooled prevalence of chronic kidney disease among hypertensive patients, and the second is its determinants.

Study selection

Different electronic databases were screened by two independent authors to review studies.

Studies were exported to Endnote20 software, and then duplicate articles were removed. The full text of the selected citation was downloaded and assessed in detail against the inclusion criteria by two reviewers. Any disagreements or issues among the reviewers during the study selection process were solved through discussion.

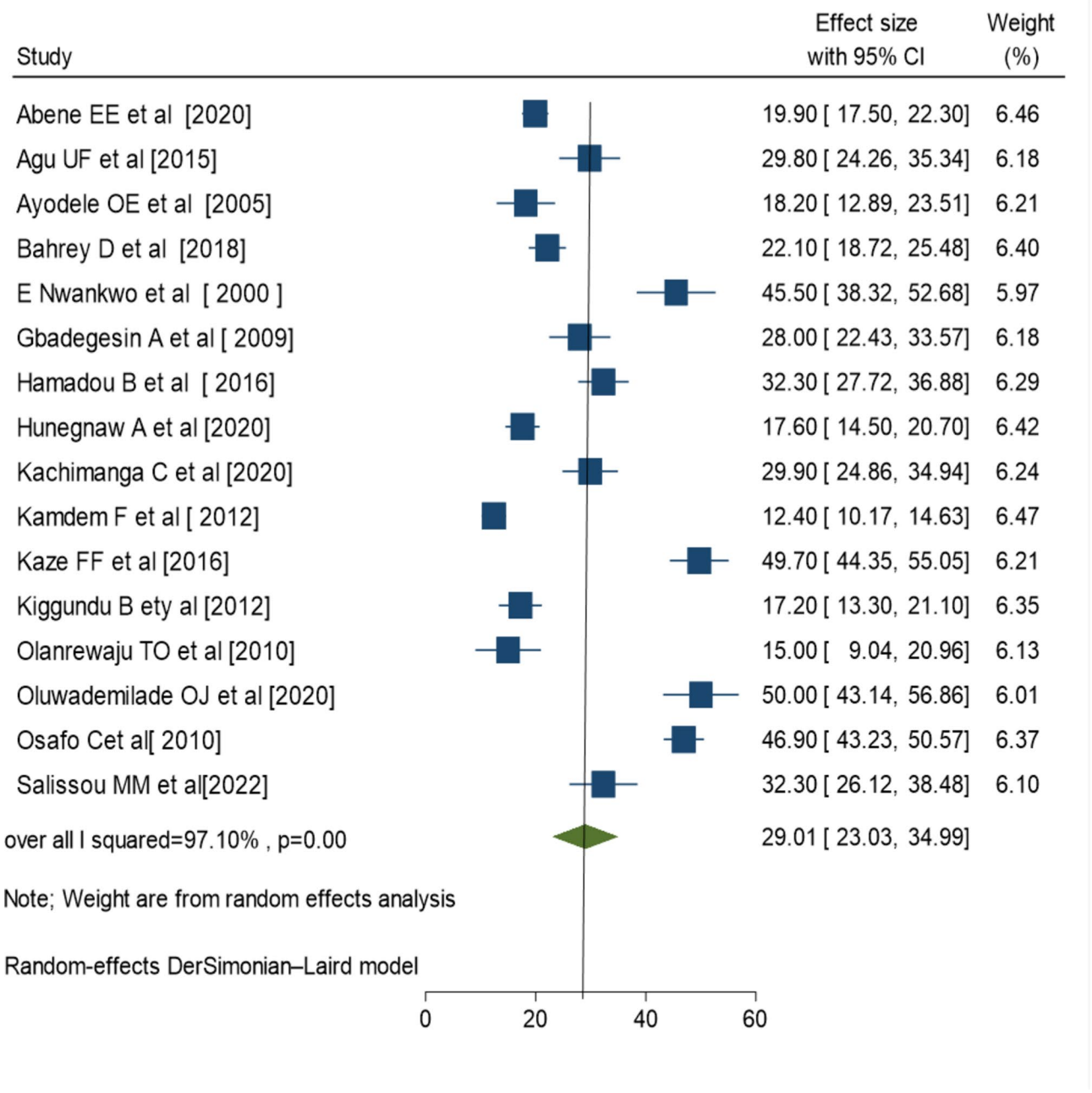

Fig. 2 Forest plot showed pooled prevalence CKD among hypertension 2024

Data extraction

The relevant data were extracted by three authors after screening eligible studies. Any inconsistency between the two authors was managed by discussion and other invited reviewers. For each included study, the authors' names, publication year, study region, study setting, study design, sample size, definition of kidney disease, GFR equation/formula used, and prevalence and factors were extracted on a Microsoft Excel spreadsheet.

Quality assessment

The Joanna Briggs Institute (JBI) critical appraisal checklists were used to determine the quality of the original studies (https://jbi.global/critical-appraisal-tools). Two independent reviewers critically appraised all the eligible studies and scored them for the validity of their results. The category was done for each observational article study and was assigned a score of 1 (Yes) or 0 (No) for each domain, and these domain scores were added to give an overall study quality score. According to this,

Table 1 Characteristics of included studies CKD among hypertension Sub-Saharan Africa

ID	Author[Year]	Region	study design	Sample size	Diagnosis criteria	equation	Prev.	quality
1	Abene EE et al. [2020] [39]	West Africa	CS	1063	e-GFR) < 60 ml/min/1.73 m2	MDRD	19.90%	low risk
2	Agu UF et al. [2016] [40]	West Africa	CS	262	e-GFR) < 60 ml/min/1.73 m2	MDRD	29.80%	low risk
3	Ayodele OE et al. [2005] [41]	West Africa	CS	203	not stated	Not stated	18.20%	low risk
4	Bahrey D et al. [2018] [42]	East Africa	CS	578	e-GFR) < 60 ml/min/1.73 m2	Cockcroft–Gault.	22.10%	low risk
5	E Nwankwo et al. [2000] [43]	West Africa	CS	185	elevated serum creatinine	serum creatinine	45.50%	low risk
6	Gbadegesin A et al. [2019] [44]	West Africa	CS	250	e-GFR) < 60 ml/min/1.73 m2	MDRD	28%	low risk
7	Hamadou B et al. [2017] [45]	Middle Africa	CS	400	e-GFR) < 60 ml/min/1.73 m2	CKD-EPI	32.30%	low risk
8	Hunegnaw A et al. [2021] [46]	East Africa	CS	581	e-GFR) < 60 ml/min/1.73 m2	Cockcroft–Gault	17.60%	low risk
9	Kachimanga C et al. [2020] [47]	West Africa	CS	317	e-GFR) < 60 ml/min/1.73 m2	CKD-EPI	29.90%	low risk
10	Kamdem F et al. [2017] [48]	Middle Africa	CS	839	e-GFR) < 60 ml/min/1.73 m2	MDRD	12.40%	low risk
11	Kaze FF et al. [2016] [49]	Middle Africa	CS	336	e-GFR) < 60 ml/min/1.73 m2	MDRD	49.70%	low risk
12	Kiggundu B et al. [2017] [50]	East Africa	CS	360	e-GFR) < 60 ml/min/1.73 m2	MDRD	17.20%	low risk
13	Olanrewaju TO et al. [2010] [37]	West Africa	CS	138	Hematuria	Hematuria	15%	low risk
14	Oluwademilade OJ et al. [2020] [38]	West Africa	CS	204	e-GFR) < 60 ml/min/1.73 m2	MDRD	50%	low risk
15	Osafo C et al [2010] [51]	West Africa	CS	712	e-GFR) < 60 ml/min/1.73 m2	MDRD)	46.90%	low risk
16	Salissou MM et al [2022] [52]	East Africa	CS	220	e-GFR) < 60 ml/min/1.73 m2	Cockcroft Gault	32.30%	low risk

from the total of 16 studies, 3 studies scored 8, 11 studies scored 7, and the rest, 2 studies scored 6 [32].

Statistical analysis

The extracted Excel data was exported to STATA version17 software for analysis. The pooled prevalence of chronic kidney disease among hypertensive adults and its factors was estimated by using a random effect model using the DerSimonian-Laird model weight [33]. The pooled effect size (the pooled prevalence) with a 95% CI was generated and presented using a forest plot. The total variation across studies due to heterogeneity was assessed using I^2 statistics [34], which estimates the percentage of total variation across studies due to true between-study differences rather than chance, with I^2 values of 25, 50, and 75% representing low, medium, and high heterogeneity, respectively [35]. Subgroup analysis was used to explore the source of heterogeneity. The study region, sample size, and publication year were considered for subgroup analysis. Sensitivity analysis was also performed to assess the effect of a single study on the pooled effect. Publication bias was assessed by visually inspecting funnel plots and objectively using statistics to determine small study effects [36].

Result

Study selection and identification

A total of studies were initiated from different electronic databases through different approaches of searching. Of the total studies retrieved from PubMed (490), Medline [8], Science Direct [4], Web Science [5], Hinari [10], and Google Scholar (520), Open Goggle [7]. From the total searched article on different electronic databases, only 140, then 109 records were excluded after reading the title and abstract; finally, 16 studies were included after full text reading. (Fig. 1)

Characteristics of included studies

This included 16 studies from different regions of sub-Saharan Africa. The sample size for each study ranges from 138 to 1063. The prevalence of CKD among the eligible in this study in patients with hypertension was obtained from various regions in sub-Saharan Africa. The prevalence of CKD among hypertension in each study varied from 15% [37] to 50% [38]. All 16 studies were cross-sectional studies where seven studies were from Nigeria and three were from Cameroon. Two were from Ethiopia, one from Uganda, one from Ghana, one from Zimbabwe, and one from Sierra Leone (Table 1).

Chronic Kidney Disease: This meta-analysis study showed the overall pooled prevalence of chronic kidney

Table 2 Summary of prevalence of CKD among hypertension in Sub-Saharan Africa

Variables		Included study	Prevalence (95%)	Heterogeneity (I^2,*p*-value)
By region	West Africa	9	31.37(22.76,39.98)	$I^2 = 96.67\%$, $p < 0.00$
	Central Africa	3	31.37(9.13,53.60)	$I^2 = 98.95\%$, $p < 0.00$
	East Africa	4	21.77(16.6,26.94)	$I^2 = 85.66\%$, $p < 0.00$
By year	< 2015	7	26.09 (16.67,37.52)	$I^2 = 98.05\%$, $p < 0.00$
	≥ 2015	9	31.24(24.37, 38.11)	$I^2 = 95.91\%$, $p < 0.00$

disease among hypertension was 29.01% (95% CI: 23.03–34.99, I^2_97.10%) in sub-Saharan Africa. (Fig. 2)

Investigation of Heterogeneity: The percentage of I^2 statistics of the forest plot indicates a marked heterogeneity among the included studies ($I^2 = 97.10\%$, $P = 00$) (Fig. 2). Sensitivity and subgroup analysis were performed to manage heterogeneity.

Subgroup analysis

Subgroup analysis was done based on study year and region. The pooled prevalence of CKD among hypertension studies conducted before the year 2015 was 26.09% (95% CI: 14.67, 37.52; $I^2 = 98.05\%$, $P < 0.001$, which was lower than studies conducted after 2015 [31.24 (95% CI: 24.37, 38.11; $I^2 = 97.08\%$, $P < 0.001$)]. Even though subgroup analysis was done by study year and region, I^2 is still high. This implies that the heterogeneity in the prevalence of CKD among patients with hypertension is high. This may be due to study area, study period, study design, and also due to differences in statistical methodology. (Table 2)

Sensitivity analysis

Sensitivity analysis was conducted to detect the influence of a particular study on the overall meta-analysis. The forest plot showed that the estimate from a single study is closer to the combined estimate, which implied the absence of a single study effect on the overall pooled estimate. (Fig. 3)

Publication Bias

The presence of publication bias was assessed graphically and statistically using. Egger's test. There is asymmetry of the graph (Fig. 4), and Egger's test evidenced that small study effect (P-value = 0.014). Trim and fill analysis was done to handle this publication bias (Fig. 5).

Factors associated with CKD among hypertension

Diabetic comorbidity is around about 3 times more likely to develop CKD than non-diabetic patients and 3 fold more likely to develop CKD in overweight among hypertension patients (Table 3).

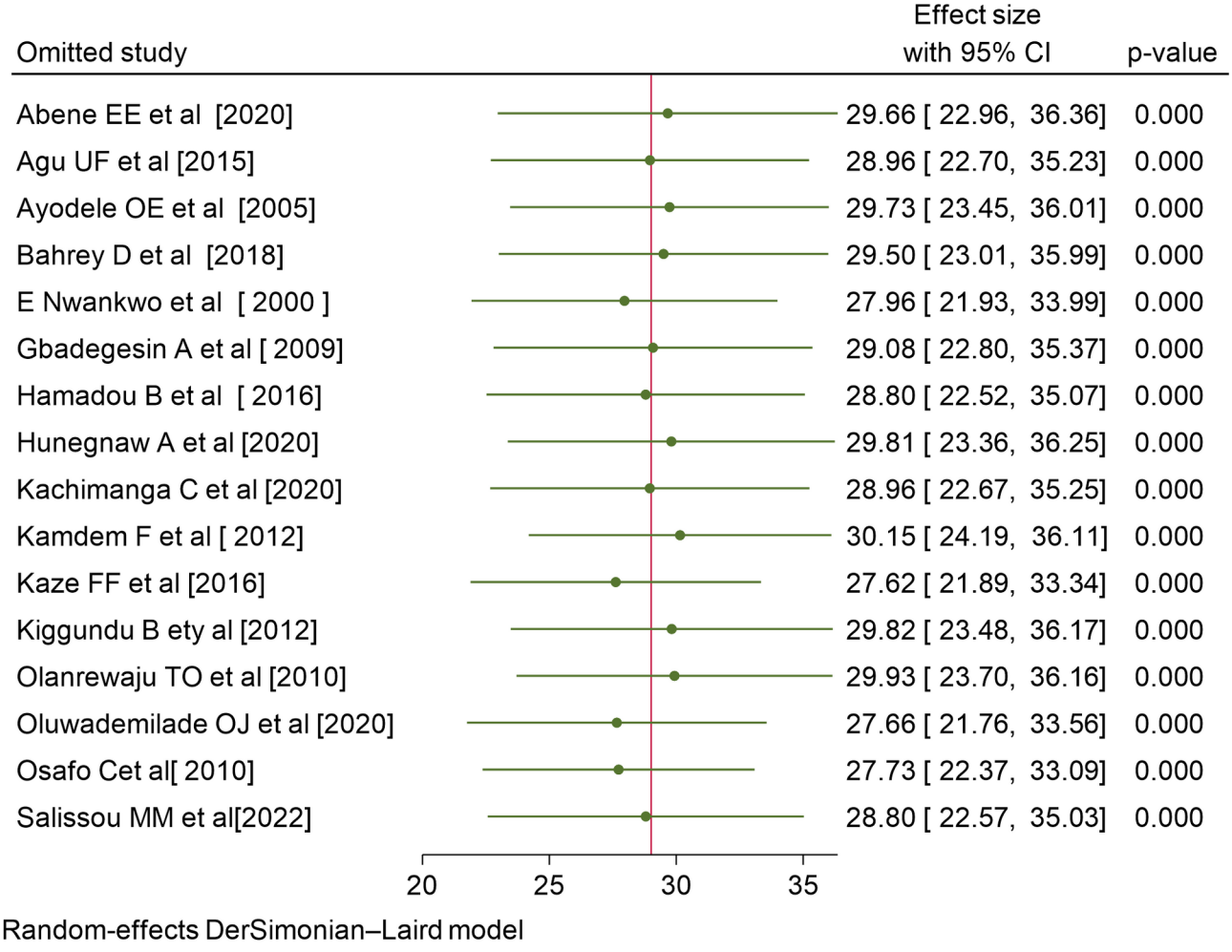

Fig. 3 Sensitivity analysis of 16 studies

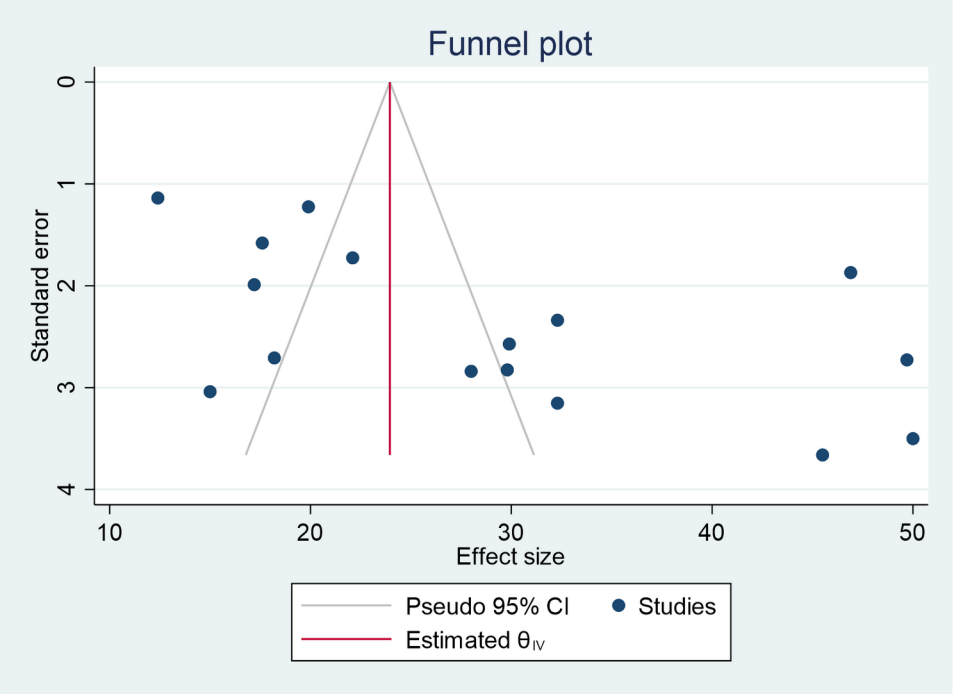

Fig. 4 Funnel plot to test publication bias of the 16 studies

Discussion

This is the first meta-analysis study conducted to determine the pooled prevalence of CKD among patients with hypertension in sub-Saharan Africa. The findings provide unique insights about the magnitude of CKD among hypertensive patients across different regional parts of sub-Saharan Africa as a whole. Chronic kidney disease is becoming a foremost public health problem globally and an important contributor to the overall non-communicable disease burden [14]. Previous primary findings indicated that the magnitude of CKD among hypertension in Sub-Saharan Africa ranges from 15 (9.04–20.96) to 50 (43.14–56.86). In this meta-analysis study, the pooled prevalence of chronic kidney disease among hypertension in Sub-Saharan Africa was 29.01 (23.03–34.99), $I^2 = 97.10\%$, $P < 00$, which was in line with the overall global pooled prevalence of CKD among hypertension [28], while it was greater than the pooled prevalence of West Africa [53].

It is higher than the global prevalence (11-13%) [54] from the general population and Sub-Saharan Africa (13.9%) in the general population [55]. This discrepancy may be due to sample size, demographics, or comorbidities; different definitions used to determine kidney failure, healthcare quality, infrastructure, treatment and follow-up adherence, clinical characteristics, awareness, and healthy habits may contribute. This review also showed hypertensive patients who were greater than 60 years old were more than two times more likely to develop CKD. Similarly, a study conducted in Brazil revealed that people who were greater than 65 years old were 2.68 times more likely to develop CKD [56, 57]. This may be explained by the fact that as age increases, physiological decline in kidney function occurs due to decreased renal mass and number of nephrons due to the presence of comorbidity and impaired immunity and alterations in renal blood flow and glomerular filtration [58]. Other systematic reviews and meta-analyses revealed that the median prevalence of CKD was 7.2% and varied from 23.4 to 35.8% in persons aged 30 years or older and aged 64 years or older, respectively [59]. This is due to aging being associated with kidney structural changes and functional decline [60, 61]. As age increases, kidney mass declines; arteriosclerosis, thickening of the glomerular basement membrane and functional nephrons also decrease kidney cortical thickness. Along with this, kidney blood flow is reduced, and glomerular filtration declines, which hastens the progression of chronic kidney disease.

Accordingly, this review of uncontrolled blood pressure leads to a more than six times greater likelihood of developing CKD. Likewise, other studies revealed hypertension is a determinant cause of chronic kidney disease risk, increasing systolic blood pressure above 120 mmHg; each 10 mmHg increase in baseline and time-varying systolic BP was associated with a 6% increase in the risk of developing CKD [62]. From 1990 to 2019, DALY numbers caused by CKD secondary to hypertension increased by 125.2% in the world [23]. Increased blood pressure was associated with higher ESRD risk, starting at systolic

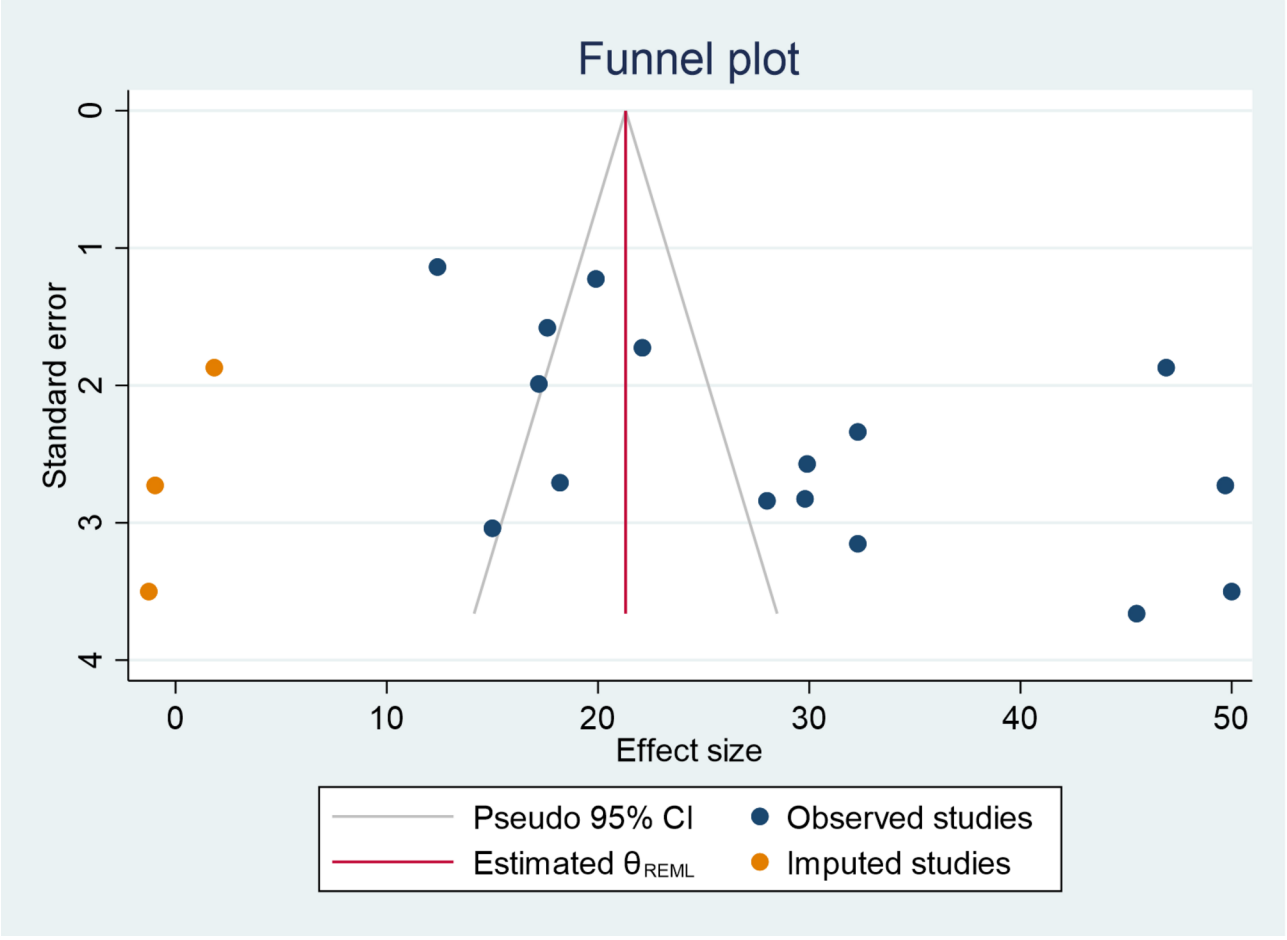

Fig. 5 Trim and fill analysis funnel plot for CKD among hypertension

blood pressure of 140 mm Hg or higher. The pathophysiology of hypertension in CKD is complex and is a sequela of multiple factors, including reduced nephron mass, increased sodium retention and extracellular volume

Table 3 Factors associated with CKD among hypertension

Variable	Category	OR (95% CI)	Heterogeneity (I2, *P*-value)
Age	< 60 years	1	
	≥ 60 years	2.36(1.02–3.71)	$I^2 = 99.11\%, P < 00$
Sex	Male	1	
	Female	1.31(0.41–3.03)	$I^2 = 97.52\%, P < 00$
Blood pressure	Controlled	1	
	Uncontrolled	6.57(2.44–10.71)	$I^2 = 97.38\%, P < 00$
Comorbidity (DM)	No	1	
	Yes	3.27(1.65–4.89)	$I^2 = 95.79\%, P < 00$
Weight	Normal	1	
	Overweight	2.75(1.04–4.46)	$I^2 = 98.22\%, P < 00$
Dyslipidemia	No	1	
	Yes	6.57(0.57–18.72)	$I^2 = 99.67\%, P < 00$
Proteinuria	No	1	
	Yes	4.64(4.09–5.18)	$I^2 = 0.00\%, P < 00$
Antihypertensive	No	1	
	Yes	1.17(0.20–2.54)	$I^2 = 90.6\%, P < 00$

*note the age category was based on the previous finding [38, 42]

expansion, sympathetic nervous system over activity, activation of hormones including the renin-angiotensin-aldosterone system, and endothelial dysfunction [63, 64]. The major pathways involved in the progression of inflammation are oxidative stress, lipo-toxicity, and fibrosis leading to glomerular sclerosis, tubular atrophy, and interstitial fibrosis [65, 66]. The progression of kidney lesions of the vascular or glomerular compartment gives rise to inflammation with progressive reduction of the glomerular filtration surface and loss of nephrons [67].

The finding also showed hypertension with diabetic comorbidity had a risk of about three times developing CKD. Similarly, studies showed hypertension and diabetic comorbidity had a synergetic effect on the progression of CKD [68]. Type II diabetes and hypertension together account for more than 75% of ESRD [69]. Diabetes mellitus and hypertension have synergistic effects to promote kidney dysfunction, albuminuria, oxidative stress, and glomerular injury [70, 71]. Similarly, studies revealed the joint effect of hypertension and diabetes was significantly larger than the sum of their independent impact on CK [72]. This may be due to activation of the renin–angiotensin–aldosterone system, mechanical stretch, oxidative stress, endoplasmic reticulum stress, mitochondrial dysfunction, and apoptosis contributing to diabetic-hypertensive nephropathy. Otherwise, the other study demonstrated no synergic or multiplicative and no additive interaction effect between diabetes and hypertension on the development of CKD [73, 74].

In this meta-analysis, the overweight leads are around three times more likely to develop CKD compared to the normal weight. Similar studies showed that obesity (BMI > 30) among men and morbid obesity (BMI > 35) among women at any time during their lifetime was linked to three- to four-fold risk increases [75, 76]. Increases in the rates of CKD have paralleled increases in overweight and obesity [77, 78]. High body fat increases inflammatory cytokine production. This activates the renin-angiotensin-aldosterone system and enhances the risk of chronic kidney disease [79]. This is explained by physical compression of the kidneys by fat in and around the kidneys; obesity causes vasoconstriction and salt and water retention, activation of sympathetic nervous system activity, inflammation, hemodynamic abnormalities, metabolic disorders, and change in hormone synthesis.

This study also showed that people with dyslipidemia have a high risk of developing CKD. Lipid abnormalities have been associated with the development and progression of kidney disease [80, 81]. Likewise, increased triglycerides and total cholesterol are independently associated with an increased likelihood of deterioration of the estimated glomerular filtration rate and development of chronic kidney disease [82]. This may be due to cholesterol plaque, which can also clog the renal arteries and cut off blood flow to the kidneys and damage glomerular podocytes, resulting in loss of kidney function. This meta-analysis study also revealed that increased proteinuria is the other risk of developing CKD. Similarly, evidence shows proteinuria is a powerful and independent risk factor for incidents of kidney disease [83, 84]. Other studies explained that proteinuria is identified as an important marker and risk factor for the progression of CKD, even though the mechanism of action in the progress of CKD is still unclear; mesangial toxicity and inflammatory cytokines are some of the proposed mechanisms [85–87]. Another study also revealed consistent experimental evidence that supports the crucial role of proteinuria in accelerating kidney disease progression to end-stage kidney failure through multiple pathways, including induction of tubular chemokine expression and complement activation leading to inflammatory cell infiltration in the interstitial and sustained fibro-genesis [88, 89]. Untreated proteinuria is strongly associated with progressive damage to kidney function and kidney failure [90, 91]. Proteinuria is both a biomarker of chronic kidney disease (CKD) and also a driver of CKD progression [87, 89, 91–93]. Proteinuria accelerate kidney disease progression to end-stage renal failure through multiple pathways, including induction of tubular chemokine expression and complement activation, toxic effect on renal tissue, inflammatory cell infiltration, and sustained fibrogenesis.

Conclusion and recommendation

Hypertension is one of the major causes of chronic kidney disease. Most patients living with hypertension develop CKD over time in sub-Saharan Africa. The highest prevalence of CKD among hypertension was observed in West Africa and Middle Africa. Age, blood pressure, comorbidity with diabetes mellitus, overweight, and proteinuria were significantly associated with developing CKD among hypertension patients. It is recommended that further study be done for researchers to determine all aspects of the burden of CKD in hypertensive patients, and recommendations be provided for policymakers, different task holders, and the Ministry of Health to organize community-based mass screening for hypertension, prepare guidelines, and supply treatment adequately, and recommendations for healthcare providers to link hypertensive patients to NCD centers, consider urine analysis like proteinuria evaluation during follow-up, strictly follow-up with hypertensive patients with comorbidity, and educate the patient about lifestyle modification such as weight reduction.

Limitations of the study

This systematic review and meta-analysis have limitations that include studies that were not equally distributed in

sub-Saharan Africa, in which there was more than one study in some countries and no study at all in the majority of countries in this region. In addition, the primary studies used different diagnostic criteria to diagnose CKD, which lack uniformity.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-025-22828-8.

Supplementary Material 1: The Joanna Briggs Institute (JBI) critical appraisal checklists 2: PRISMA checklist for meta-analysis of the prevalence of CKD among Hypertension patients in Sub-Saharan Africa.

Supplementary Material 2

Supplementary Material 3

Author contributions

GK, TM and AK participated in conception and design of the study, EG, KS, AA and GK Participated in on methodology, GB, ME, AT and TA participated in Analysis, GK, FD, TD and GA participated manuscript preparation and YE, YA and BB involved in reviewing the manuscrpit.

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Data availability

The data is available in the main manuscript and attached as supplementary files.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that there was no commercial or financial conflict.

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