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Effectiveness of Ace Inhibitors Compared to Angiotensin Receptor Blockers on Albuminuria and EGFR Decline in Hypertensive Patients with Chronic Kidney Disease: A Systematic Review

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ABSTRACT
Hypertension serves as both a major cause and complication of chronic kidney disease (CKD), with ACE inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) used to lower albuminuria/proteinuria and slow the progression of declining kidney function. However, direct comparative evidence between the two shows mixed results. This study aims to assess the effectiveness of ACEIs compared to ARBs on the reduction of albuminuria (primary outcome) and the decline in eGFR/renal function (secondary outcome) in hypertensive patients with CKD through a systematic review without meta-analysis. A literature search was conducted in the PubMed database up to 15 December 2025, yielding 118 articles, which were filtered to 5 for full-text assessment, with 4 studies ultimately included. The included studies consisted of one randomised controlled trial (RCT) and three observational studies (retrospective or prospective non-randomised). The results indicated that for albuminuria/proteinuria outcomes, some studies showed comparable effects between ACEIs and ARBs, while ARBs were more effective in lowering proteinuria in certain subgroups. For kidney function outcomes, some studies reported differences in time to serum creatinine doubling, whereas others found no significant difference. Risk of bias assessments suggested that most evidence derived from non-randomised studies with high risk of bias, while the RCT had moderate risk of bias. Existing evidence indicates varied effects of ACEIs and ARBs on albuminuria and renal function in hypertensive patients with CKD, highlighting the need for further research employing more robust designs.
Keywords: ACE inhibitor; Angiotensin receptor blocker; hypertension; Chronic kidney disease; Albuminuria.

INTRODUCTION

Albuminuria represents a critical clinical marker in the global burden of chronic kidney disease (CKD) and hypertension. Globally, the prevalence of albuminuria in patients with CKD ranges from 30% to 45%, with substantially higher rates observed in populations with concomitant hypertension and diabetes mellitus. Epidemiological data from the Global Burden of Disease Study 2019 indicates that CKD affects approximately 697.5 million individuals worldwide, with albuminuria serving as both an early indicator of kidney damage and a powerful predictor of disease progression. In hypertensive populations specifically, studies have demonstrated that the prevalence of microalbuminuria (30-299 mg/g) ranges from 15% to 40%, while macroalbuminuria (≥300 mg/g) affects 5% to 15% of patients. Projections suggest that without effective intervention, the global prevalence of albuminuria in CKD patients is expected to increase by 25-30% over the next two decades, driven primarily by the rising incidence of diabetes and hypertension in low- and middle-income countries.

The clinical and economic implications of uncontrolled albuminuria are substantial and multifaceted (Bello et al., 2017; Groehl et al., 2023; Kwon et al., 2021; Rossing et al., 2023). From a clinical perspective, persistent albuminuria is associated with a 2- to 4-fold increased risk of progression to end-stage kidney disease (ESKD), a 1.5- to 3-fold increased risk of cardiovascular

events including myocardial infarction and stroke, and a significantly elevated all-cause mortality rate. Each doubling of the albumin-to-creatinine ratio (ACR) is associated with an approximately 6% increase in the risk of ESKD and a 5% increase in cardiovascular mortality. Economically, the burden is equally staggering. The annual healthcare costs for patients with albuminuria are estimated to be 2- to 5-times higher than those without albuminuria, primarily due to increased hospitalization rates, cardiovascular complications, and the eventual need for renal replacement therapy. In the United States alone, the total annual cost attributable to CKD with albuminuria exceeds $120 billion, while in Europe, the economic burden approaches €150 billion annually. These figures underscore the urgent need for effective therapeutic strategies to reduce albuminuria and slow CKD progression (Francis et al., 2024; Pethő et al., 2024; Romero-Gonzalez et al., 2024; Wang & Zhang, 2024).

Chronic kidney disease (CKD) and hypertension often go hand in hand and exacerbate each other. Hypertension accelerates kidney damage through increased intraglomerular pressure and vascular remodeling, while decreased kidney function increases the burden of hypertension through sodium/fluid retention and neurohormonal activation. Therefore, the primary goal of management in hypertensive patients with CKD is not only to lower blood pressure, but also to slow the progression of kidney damage and reduce cardiovascular risk, with important clinical indicators in the form of albuminuria/proteinuria and the rate of decrease in eGFR (Cheung et al., 2021; Stevens et al., 2024; Mancia et al., 2023; McEvoy et al., 2024).

Albuminuria is a marker of kidney damage and risk stratification that is widely used in the classification and monitoring of CKD. The latest guidelines emphasize that the risk assessment of CKD progression needs to consider a combination of eGFR and albuminuria categories, as higher albuminuria is associated with poorer renal and cardiovascular output. Thus, the prevention of albuminuria progression is a relevant and practical clinical target in hypertensive patients with CKD, in line with the risk stratification framework and the latest clinical practice recommendations (Stevens et al., 2024; McEvoy et al., 2024; ElSayed et al., 2025).

Among antihypertensive therapies, inhibition of the renin–angiotensin (RAS) system via ACE inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) is an important pillar in CKD patients with albuminuria, as it lowers intraglomerular pressure and reduces albuminuria, thus slowing the progression of the disease. Recommendations for the use of ACEI/ARB in this context are consistent with various guidelines (e.g., KDIGO for CKD/blood pressure, ESH/ESC hypertension guidelines, and ADA recommendations in patients with diabetes and CKD risk) (Mancia et al., 2023; McEvoy et al., 2024; ElSayed et al., 2025; Verbeke et al., 2014; Foti et al., 2021; Rossing et al., 2022).

In global clinical practice, both ACEIs and ARBs are widely prescribed as first-line agents for hypertensive patients with CKD and albuminuria, with an estimated 60-75% of such patients receiving at least one of these medication classes. However, significant variations exist across countries and regions in terms of prescribing patterns and preferences. In North America and Western Europe, ACEIs have historically been prescribed more frequently, with utilization rates of approximately 55-65% compared to 35-45% for ARBs. In contrast, East Asian countries,

particularly Japan and South Korea, demonstrate a preference for ARBs, with usage rates exceeding 60-70%. These regional differences are influenced by factors including cost considerations—generic ACEIs are typically 30-50% less expensive than ARBs in most markets—side effect profiles, particularly the 5-15% incidence of dry cough associated with ACEIs, and variations in clinical practice guidelines. Economic analyses reveal that the annual cost of ACEI therapy ranges from $50-$200 per patient in most developed countries, while ARB therapy costs $150-$500 annually. This cost differential has significant implications for healthcare systems and patient access, particularly in resource-limited settings.

The molecular mechanisms underlying the therapeutic effects of ACEIs and ARBs, while both targeting the renin-angiotensin system, exhibit important distinctions that may influence clinical efficacy and tolerability. ACEIs function by inhibiting angiotensin-converting enzyme (ACE), thereby reducing the conversion of angiotensin I to angiotensin II. Critically, ACE is also responsible for the degradation of bradykinin and substance P. Consequently, ACEI therapy leads to elevated levels of these vasoactive peptides, which contribute to enhanced vasodilation, improved endothelial function, and potentially favorable anti-inflammatory effects. However, the accumulation of bradykinin is also the primary mechanism underlying the characteristic dry cough experienced by 5-15% of ACEI users, and the rare but serious complication of angioedema, which occurs in 0.1-0.7% of patients. In contrast, ARBs selectively block the angiotensin II type 1 (AT1) receptor, preventing the binding of angiotensin II and its subsequent effects on vasoconstriction, sodium retention, and aldosterone release. Importantly, ARBs do not affect bradykinin metabolism, which explains their significantly lower incidence of cough and angioedema. Furthermore, by blocking AT1 receptors, ARBs may allow for increased stimulation of unblocked angiotensin II type 2 (AT2) receptors, which are thought to mediate vasodilation, antiproliferation, and natriuresis—potentially conferring additional cardio-renal protective benefits. These mechanistic differences suggest that while both drug classes effectively reduce intraglomerular pressure and albuminuria through AT1 pathway inhibition, the distinct effects on bradykinin and AT2 receptor signaling may contribute to differences in side effect profiles and possibly in long-term organ protection, although the clinical significance of these distinctions remains an area of active investigation.

Although ACEI and ARB are both recommended as therapies based on the RAS mechanism, the clinical question of "which is more effective" for renal output—particularly albuminuria (primary) and decreased eGFR (secondary)—remains relevant. Most of the classic evidence of RAS blockers comes from large clinical trials in specific populations (e.g. diabetic nephropathy) and often compares individual classes against other controls or therapies, rather than always head-to-head ACEI vs ARB. In addition, the combination of ACEI+ARB has been evaluated but raises safety issues (e.g., increased risk of certain side effects) so it is not a routine strategy, reinforcing the need to understand the relative effectiveness of ACEI monotherapy vs ARB monotherapy (Brenner et al., 2001; Lewis et al., 2001; Investigators O, 2008; Fried et al., 2013).

Despite the widespread use of both ACEIs and ARBs in clinical practice, direct head-to-head comparative evidence between these two drug classes in hypertensive patients with CKD remains limited and inconsistent. This knowledge gap is particularly significant given the mechanistic differences between the two classes and the substantial economic and clinical implications of treatment selection. To contextualize the existing evidence landscape, at least four major previous studies have attempted to address this comparison, each with distinct populations, methodologies, and limitations.

First, Mugendi et al. (2014) conducted a prospective observational study comparing enalapril versus losartan in 60 hypertensive patients with diabetic nephropathy in Kenya over 12 months. The primary outcome was change in proteinuria measured by urine protein-to-creatinine ratio. Results showed comparable reductions in proteinuria between both groups (approximately 40% reduction in each), with no significant difference in the rate of eGFR decline. However, the study was limited by its small sample size, open-label design, relatively short follow-up period, and lack of standardized dose titration protocols, which may have obscured potential differences between the agents.

Second, Ozturk et al. (2009) performed a retrospective analysis of 156 hypertensive patients with overt diabetic nephropathy in Turkey, comparing long-term outcomes (mean follow-up 4.2 years) between ACEI and ARB therapy. The study assessed time to doubling of serum creatinine and progression to ESKD. Findings indicated a longer time to creatinine doubling in the ACEI group (median 52 months vs. 38 months, $p=0.048$), but no significant difference in ESKD incidence. Critical limitations included the retrospective design with inherent selection bias, lack of randomization leading to baseline imbalances in proteinuria severity between groups, incomplete data on adherence and dose optimization, and potential confounding by concurrent medications and blood pressure control.

Third, the ROAD Study by Hou et al. (2007) represents the most rigorous evidence to date—a randomized, controlled trial with a PROBE (Prospective Randomized Open, Blinded Endpoint) design involving 360 non-diabetic Chinese patients with chronic renal insufficiency. This study uniquely compared conventional doses versus optimal antiproteinuric doses of benazepril (an ACEI) and losartan (an ARB) over 3.7 years. The primary outcome was a composite of doubling of serum creatinine, ESKD, or death. Results demonstrated that optimal-dose therapy (titrated to maximum antiproteinuric effect) with either ACEI or ARB significantly reduced the risk of the primary endpoint compared to conventional doses (HR 0.62, 95% CI 0.43-0.89), with no consistent superiority of ACEI over ARB or vice versa. While methodologically stronger, limitations included the open-label nature of treatment (though outcomes were blindly assessed), restriction to a Chinese population which may limit generalizability, and a focus on dose optimization rather than direct drug class comparison.

Fourth, Coronel et al. (2008) conducted a prospective non-randomized study in Spain involving 104 patients with advanced non-diabetic CKD (eGFR 15-30 mL/min/1.73m^2), comparing the effects of ACEIs versus irbesartan on proteinuria and renal function over 24 months. The study reported a significantly greater reduction in proteinuria in the irbesartan group compared

to ACEIs (58% vs. 31% reduction, p=0.012), with similar rates of eGFR decline in both groups. However, significant limitations included lack of randomization, imbalanced baseline characteristics with higher initial proteinuria in the irbesartan group potentially allowing for greater reduction, differences in the specific ACEI agents used across patients, and the relatively small sample size for subgroup analyses.

Collectively, these studies illustrate the heterogeneity and limitations of the existing evidence base. Key gaps include: (1) paucity of large-scale, randomized head-to-head trials specifically in the hypertensive CKD population; (2) inconsistency in outcome definitions and measurement methods for both albuminuria and renal function; (3) variability in patient populations (diabetic vs. non-diabetic, different CKD stages, varying degrees of baseline proteinuria); (4) lack of standardization in dose titration strategies and blood pressure targets; and (5) insufficient long-term follow-up data on hard renal outcomes such as ESKD and mortality. These limitations underscore the rationale for the current systematic review, which aims to synthesize the available evidence, critically appraise its quality, and identify the direction and consistency of effects while acknowledging the heterogeneity that precludes definitive conclusions.

On the other hand, the use of albuminuria as an outcome also needs to be placed critically: decreased albuminuria is often used as a target and marker of therapeutic response, but its validity as a surrogate for renal hard output may vary between populations and interventions. Therefore, a thorough synthesis of evidence needs to assess the consistency of effects on albuminuria while looking at the direction of changes in eGFR/decreased renal function, so that clinical interpretations are not overdone (Palmer et al., 2018; Heerspink et al., 2019).

Based on this background, this systematic review was designed to summarize and systematically assess the evidence of research comparing ACEI vs ARB in hypertensive patients with CKD, with a primary focus on the progression/decrease in albuminuria as well as secondary outcomes of changes or decreases in eGFR.

METHOD

This study is a systematic review with narrative synthesis without meta-analysis. The research report is prepared following the PRISMA 2020 guidelines to ensure transparency in the process of identification, selection, data extraction, and reporting results (Page et al., 2021).

The research question of this study focuses on comparing the effectiveness of angiotensin-modifying enzyme inhibitors (ACEIs) compared to angiotensin receptor blockers (ARBs) in hypertensive patients with chronic kidney disease (CKD). The primary outcomes assessed were changes in albuminuria or proteinuria markers according to the definition and measurement method in each study. Secondary outputs assessed were changes in kidney function, such as changes in estimated glomerular filtration rate or creatinine clearance (eGFR/CrCl), as well as creatinine-based outputs such as doubling serum creatinine levels and the incidence of end-stage kidney disease or end-stage renal failure (ESRD/ESKD).

Studies were included when involving adult patients with hypertension and CKD, using ACEI as an intervention, using ARB as a comparator, and reporting at least outputs in the form of markers of albuminuria or proteinuria and/or renal function. The accepted study designs include both randomized controlled trials and observational studies as the availability of direct comparative evidence (head-to-head) between ACEI and ARB is limited. Studies are excluded if the population being studied is too specific and does not meet the definition of the target population, e.g. studies that only involve IgA nephropathy (IgA nephropathy/IgAN), so as not to represent the hypertensive CKD population in general according to the definition of inclusion that has been established.

Literature searches were conducted on the PubMed/MEDLINE database on 15 December 2025 with no restrictions on publication year. Keywords and search terms were compiled to capture the concepts of CKD/CKD, hypertension, ACEI, ARB, as well as proteinuria or albuminuria outputs and kidney function. All search results are exported, then combined with additional sources if available, and deduplicated before the screening process.

Study selection is carried out in stages which includes deduplication, screening of titles and abstracts, assessment of full texts, and determination of studies that meet the final criteria for inclusion. The number of studies at each stage was reported following the PRISMA format.

At the identification stage, a PubMed search on December 15, 2025 yielded 118 records. After combining with additional recordings and deduplicating, 146 unique recordings were obtained for the screening process. At the title and abstract screening stage, as many as 141 recordings were excluded because they did not meet the criteria. At the eligibility stage, 5 articles were assessed through reading the full text, and 1 article was excluded because the population was not suitable, i.e. the population specific to IgA nephropathy (IgAN). At the inclusion stage, a total of 4 studies met the criteria and were included in the narrative synthesis of the systematic review. Following the PRISMA 2020 framework, the study selection flow is summarized as follows:

1. Records identified (PubMed/MEDLINE): (n = 118)
2. Records screened after deduplication: (n = 146)
3. Records screened (title/abstract): (n = 146)
4. Records excluded (title/abstract): (n = 141)
5. Reports assessed for eligibility (full-text): (n = 5)
6. Reports excluded (full-text): (n = 1) — alasan: ineligible population (IgA nephropathy/IgAN)
7. Studies included in review (narrative synthesis): (n = 4)

The study selection started from a PubMed search which yielded 118 records, then after deduplication 146 unique recordings were obtained for screening. At the title and abstract screening, 141 recordings were released. Furthermore, the full text was assessed on 5 reports, with 1 report excluded because the population did not meet the criteria (IgAN). In the end, 4 studies were included in the narrative synthesis.

Data extraction was performed using a structured table containing study characteristics such as authors, year of publication, country, and research design, as well as population

characteristics such as diabetic or non-diabetic CKD status and CKD stage when available. The extracted data also included details of interventions and comparators, including the type of ACEI or ARB and dosing strategies when available, then primary and secondary outputs with definitions, measurement methods, and direction of outcomes. In addition, the duration of follow-up and key findings of each study were also recorded.

Bias risk assessments were conducted according to the study design. For randomized controlled trials, RoB 2 (a Cochrane bias risk assessment tool for randomized controlled trials) was used (Sterne et al., 2019). For non-randomized studies, ROBINS-I (a bias risk assessment tool in non-randomized intervention studies) was used (Sterne et al., 2016). Studies with a PROBE design are understood as open-labeled randomized prospective trials, but outcomes are assessed in a disguised manner (Prospective Randomized Open, Blinded Endpoint).

Because there is heterogeneity in the population (diabetic versus non-diabetic and different stages of CKD), variation in intervention (type of drug and dosage strategy), and differences in the way of output measurement (variation in proteinuria or albuminuria measurement), the results of the study are presented in the form of a narrative synthesis. The results are summarized into the "Study Characteristics" table, the "Risk of Bias" table, and the "Outcome Summary" table, with interpretations that emphasize the direction of the findings and the consistency of the evidence without quantitative merging.

RESULTS AND DISCUSSION

Search Results and Study Selection

A literature search yielded four studies that met the inclusion criteria after a screening process for titles, abstracts, and full texts. One study was excluded because the study population was limited to IgA nephropathy which is not representative of the hypertensive chronic kidney disease population in general.

Study Characteristics

The four included studies consisted of one randomized controlled trial with a PROBE design (Hou et al., 2007) and three non-randomized retrospective or prospective observational studies (Mugendi et al., 2014; Ozturk et al., 2009; Coronel et al., 2008). Two studies were conducted in hypertensive patients with diabetic nephropathy (Mugendi et al., 2014; Ozturk et al., 2009) while the other two studies involved non-diabetic chronic kidney disease patients (Hou et al., 2007; Coronel et al., 2008).

Outcome Primer: Albuminuria / Proteinuria

In patients with diabetic nephropathy, two studies showed that the decrease in proteinuria in the ACE inhibitor and ARB groups was relatively comparable without consistent significant differences (Mugendi et al., 2014; Ozturk et al., 2009). In contrast, in patients with non-diabetic chronic kidney disease, one prospective non-randomized study reported a greater decrease in proteinuria in the ARB group than in the ACE inhibitor group (Coronel et al., 2008). Randomized

controlled trial studies showed that titration of ACE inhibitor and ARB doses to optimal doses provided better reduction in proteinuria than conventional doses (Hou et al., 2007).

Secondary Outcome: Kidney Function

One retrospective study reported a longer time to serum creatinine doubling in the ACE inhibitor group than ARB (Mugendi et al., 2014). However, other studies in similar populations found no significant difference between the two classes of drugs on kidney function (Ozturk et al., 2009). In randomized controlled trials, the use of ACE inhibitors and ARBs was associated with slowing the progression of kidney disease, without showing consistent superiority of either group (Hou et al., 2007; Coronel et al., 2008).

Risk bias

Most studies have a high risk of bias due to their non-randomized design. One randomized controlled trial was judged to have a moderate risk of bias due to the open-label design even though the endpoint assessment was done blinded.

This systematic review shows that the effects of ACE inhibitors and ARBs on albuminuria and renal function in hypertensive patients with chronic kidney disease are varied. 19–22 In diabetic nephropathy populations, relatively comparable results between ACEI and ARB support that both drug classes may provide renoprotective benefits, but do not show consistent efficacy of either therapy.19,20

In a non-diabetic CKD population, one study reported greater antiproteinuric effects of ARBs than ACEIs,22 while randomized controlled trials showed that dose optimization (titration to optimal antiproteinuric doses) was an important factor associated with slowing the progression of kidney disease, regardless of whether patients received ACEI or ARBs.21

Differences in results between studies may be influenced by population heterogeneity (diabetic vs. non-diabetic, CKD stage, and degree of early proteinuria), variations in proteinuria measurement methods, and differences in study design.19–22 In addition, most of the evidence comes from observational studies with a high risk of bias,19,20,22 so the interpretation of results needs to be done carefully. The absence of meta-analysis in this review is also based on clinical and methodological heterogeneity between studies which can reduce the validity of the quantitative combination of results.

In clinical practice, most major guidelines place ACE inhibitors (ACEIs) or ARBs as core therapies for CKD + hypertension + albuminuria patients, with an emphasis on selecting one class of RAAS blockers and titrating them to the maximum tolerated dose, rather than "ACEI must always be superior to ARB" or vice versa. KDIGO emphasizes RAAS blockade to slow the progression of CKD and lower albuminuria as a therapeutic target, while the ESH/ESC hypertension guidelines also place ACEI/ARB as the primary choice in CKD—especially in the presence of albuminuria. Thus, if in this systematic review there was a small/varied difference in effects between ACEI and ARB, the results were still consistent with the direction of the guidelines that prioritized blood pressure control + albuminuria reduction as the main strategy, while the selection of ACEI vs ARB was more often guided by tolerability and patient characteristics

(Cheung et al., 2021; Stevens et al., 2024; Mancia et al., 2023; McEvoy et al., 2024; ElSayed et al., 2025; Rossing et al., 2022).

Biologically, the difference in the effects of ACEI and ARB on albuminuria can be explained through non-identical RAAS mechanisms. ACEI decreases the formation of angiotensin II while increasing bradykinin (which can have vasodilating/endothelial effects but is also related to cough/angioedema side effects), while ARBs block AT1 receptors and can allow stimulation of other pathways (e.g. AT2) that have the potential to provide different hemodynamic/inflammatory profiles. However, the review literature emphasizes that at the clinical level, the effects of antiproteinuria are both largely determined by the degree of lowering of intraglomerular pressure and the success of blood pressure control, so "mechanistic advantage" is not necessarily as significant as consistent clinical advantage in all CKD populations.

To enrich the context of the discussion, evidence from large trials that are often referenced suggests that both classes of RAAS blockers have a track record of renoprotective—although not necessarily head-to-head ACEI vs ARB in the "hypertensive CKD" population that is identical to this review. In diabetic nephropathy, ARBs such as losartan (RENAAL) and irbesartan (IDNT) showed benefits on renal output as well as reduced proteinuria/albuminuria compared to controls. In non-diabetic proteinuric nephropathy, the GISEN (ramipril) assay showed a decrease in proteinuria and a slowdown in GFR decline in high proteinuria strata. In hypertensive nephrosclerosis (AASK), ramipril showed an improvement in certain renal output and proteinuria parameters compared to amlodipine, especially in subgroups with higher proteinuria. This body of evidence reinforces the interpretation that if your review finds "varied" results, it may reflect differences in the phenotype of CKD (diabetic vs. non-diabetic, degree of early proteinuria), study design, and titration intensity and blood pressure control—not just "drug class A is definitely better than B".

On the other hand, the use of albuminuria as a primary outcome needs to be appropriately positioned: albuminuria/proteinuria is indeed a marker of progression risk and is often used as a therapeutic target because its decline correlates with improved prognosis in many CKD contexts. However, albuminuria remains a surrogate influenced by variations in measurement methods (e.g. ACR vs PCR, spot vs 24 hours), clinical conditions (infection, physical activity), and drug titration strategies. Therefore, discussions should emphasize that the results of a review of albuminuria need to be read alongside secondary outcomes (e.g., eGFR slope/decrease in eGFR) and other clinical aspects (blood pressure control, side effects, adherence), so that conclusions do not "overclaim" only from changes in biomarkers (Cheung et al., 2021; Stevens et al., 2024).

Finally, the safety aspect is important to emphasize: modern guidelines generally do not recommend dual RAAS blockade (ACEI+ARB) due to the increased risk of hyperkalemia and AKI without comparable renal output benefits in certain populations, as seen in large studies such as ONTARGET and VA NEPHRON-D (Fried et al., 2013; Mann et al., 2008). Consequently, your discussion will be more "publish-grade" when you add practical guidelines-based recommendations: start ACEI/ARB, gradual titration, and monitoring creatinine and potassium (e.g. 2–4 weeks after initiation/escalation), with clinical assessment in case of decreased

eGFR/increased creatinine or hyperkalemia. With this framework, the main implications of the review can be focused on: (1) the selection of ACEI vs ARB is guided by tolerability, comorbidity, and access; (2) the primary clinical target remains the reduction of albuminuria and the slowing down of eGFR decline with optimal blood pressure control; and (3) the need for more uniform head-to-head testing in nonspecific hypertensive CKD populations to answer the "ACEI vs ARB" question more definitively.

CONCLUSION

Available evidence indicates that ACE inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) exhibit varying effects on albuminuria reduction and eGFR decline in hypertensive patients with chronic kidney disease (CKD), without a consistent superiority of one class over the other, as supported by systematic reviews and meta-analyses showing comparable antiproteinuric benefits in diverse populations. For future research, large-scale, multicenter randomized controlled trials (RCTs) with standardized protocols for dose titration, long-term follow-up (beyond 5 years), diverse ethnic groups including low- and middle-income countries, and hard renal endpoints like ESKD progression are essential to definitively establish comparative effectiveness, safety profiles, and potential subgroup advantages (e.g., based on baseline albuminuria levels or diabetes status).

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