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Effectiveness of ACE Inhibitor Compared with Angiotensin Receptor Blockers in Reducing Chronic Renal Deterioration

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Abstract

Background: Chronic kidney disease (CKD) is a progressive disorder characterized by gradual deterioration of renal function and is associated with increased cardiovascular morbidity and mortality. Pharmacological inhibition of the renin–angiotensin system remains an important component of CKD management, particularly in patients with hypertension and albuminuria. **Objective:** To compare the effectiveness of ACE inhibitors and ARBs in preserving renal function and slowing the progression of CKD among adult patients. **Methodology:** A comparative clinical study was conducted among adults diagnosed with CKD who were receiving either an ACE inhibitor or an ARB as part of their treatment. Participants were divided into two groups according to their prescribed therapy. Baseline demographic and clinical characteristics were documented, including age, sex, blood pressure, diabetes status, serum creatinine, estimated glomerular filtration rate (eGFR), and urinary protein or albumin excretion. Patients were followed for changes in renal function, blood pressure, proteinuria, and progression of CKD. The outcomes of the two treatment groups were compared using appropriate statistical methods. **Results:** Patients receiving ACE inhibitor therapy demonstrated favorable stabilization of renal function during follow-up, while those receiving ARB therapy also showed significant preservation of kidney function. Both treatment groups experienced reductions in blood pressure and urinary protein excretion. **Conclusion:** ACE inhibitors and ARBs appear to be effective therapeutic options for slowing CKD progression and preserving renal function, particularly among patients with hypertension and proteinuria. **Keywords:** Chronic kidney disease, ACE inhibitors, angiotensin receptor blockers, renal protection, eGFR, proteinuria, hypertension, kidney function, renin–angiotensin system.

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Introduction

Chronic kidney disease (CKD) is a major global health problem characterized by persistent abnormalities in kidney structure or function and is associated with progressive loss of renal function, cardiovascular complications, and premature mortality [1]. The clinical course of CKD varies considerably among patients, but hypertension, diabetes, albuminuria, and activation of the renin–angiotensin–aldosterone system (RAAS) are important contributors to ongoing renal injury [2]. Consequently, effective control of blood pressure and reduction of albuminuria are central objectives in strategies designed to delay CKD progression.

Angiotensin-converting enzyme inhibitors (ACE inhibitors or ACEIs) and angiotensin II receptor blockers (ARBs) are among the most widely used pharmacological therapies for patients with CKD, particularly those with hypertension and increased urinary albumin excretion. By interfering with the RAAS, these medications reduce systemic and intraglomerular pressure and can decrease proteinuria, thereby providing effects that extend beyond conventional blood-pressure reduction [3]. Current KDIGO recommendations support the use of either an ACEI or an ARB in patients with CKD and moderately to severely increased albuminuria, with or without diabetes, depending on the clinical context [4].

The renoprotective effects of RAAS inhibition have been demonstrated in numerous clinical trials. ACE inhibitors have been shown to reduce proteinuria and delay deterioration of renal function in several populations with proteinuric kidney disease [5]. Similarly, ARBs have demonstrated beneficial effects on albuminuria and renal outcomes, particularly among patients with diabetic kidney disease [6]. These observations have established RAAS blockade as an important component of modern CKD management.

Despite their common therapeutic target, ACEIs and ARBs differ in their pharmacological mechanisms. ACEIs inhibit the conversion of angiotensin I to angiotensin II and also influence bradykinin metabolism, whereas ARBs selectively block the angiotensin II type 1 receptor. These differences may contribute to variations in adverse effects, treatment tolerance, and potentially clinical outcomes [7]. ACEIs are more frequently associated with cough and, rarely, angioedema, while ARBs are generally less likely to cause these effects and are commonly used when ACEI intolerance occurs [8].

The comparative renal efficacy of ACEIs and ARBs remains an area of clinical interest. A large Bayesian network meta-analysis involving 119 randomized trials and more than 64,000 participants with CKD found that both ACEIs and ARBs reduced the risk of kidney failure compared with placebo, although ACEIs demonstrated a numerically greater reduction in kidney-failure risk [9]. Another network meta-analysis involving patients with non-dialysis CKD stages 3–5 reported favorable renal and cardiovascular outcomes with ACEI monotherapy and suggested potentially

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greater overall protective effects compared with ARBs, although differences between individual agents and patient populations must be interpreted carefully [10].

Evidence from patients with diabetes also supports the use of either class in appropriate patients. The American Diabetes Association and KDIGO recommend ACEIs or ARBs for individuals with diabetes, hypertension, and albuminuria, particularly when albuminuria is substantially increased [11]. Importantly, these medications should not be combined routinely. Dual ACEI and ARB therapy has been associated with increased risks of hyperkalemia, acute kidney injury, and hypotension without providing sufficient long-term benefit to justify combined treatment [11,12].

Monitoring is essential when initiating or increasing ACEI or ARB therapy. Serum creatinine and potassium levels should be assessed following treatment initiation or dose escalation, because RAAS blockade can produce an initial change in renal function and may increase serum potassium. KDIGO recommends continuing ACEI or ARB therapy unless serum creatinine rises by more than 30% within four weeks of initiation or dose escalation, while hyperkalemia should often be managed through appropriate measures rather than immediate discontinuation of treatment [4].

Although both ACEIs and ARBs are established therapies for CKD, uncertainty remains regarding whether one class provides superior long-term protection against renal function decline, kidney failure, and cardiovascular events. Differences in underlying kidney disease, diabetes status, albuminuria, baseline eGFR, medication tolerance, and treatment adherence may influence comparative outcomes. Moreover, evidence from direct head-to-head comparisons does not always produce the same conclusions as indirect comparisons and network meta-analyses [9,10].

Therefore, the present study aims to comparatively evaluate the effectiveness of ACE inhibitors and ARBs in slowing CKD progression and preserving renal function. Particular attention is given to changes in estimated glomerular filtration rate (eGFR), serum creatinine, proteinuria, and blood pressure during follow-up. The findings may help clarify the comparative clinical utility of these two major RAAS-blocking strategies and support individualized pharmacological management of patients with chronic kidney disease.

Methodology

A comparative observational study was conducted to evaluate the effectiveness of angiotensin-converting enzyme (ACE) inhibitors versus angiotensin receptor blockers (ARBs) in slowing chronic renal deterioration. Adult patients diagnosed with chronic kidney disease (CKD) and receiving stable treatment with either an ACE inhibitor or an ARB were enrolled. Patients were divided into two groups according to their prescribed renin–angiotensin system

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blockade: the ACE inhibitor group and the ARB group. Individuals with acute kidney injury, end-stage renal disease requiring dialysis, renal transplantation, or incomplete clinical records were excluded.

Baseline demographic and clinical characteristics, including age, sex, duration of CKD, blood pressure, diabetes status, and baseline estimated glomerular filtration rate (eGFR), were recorded. Laboratory parameters included serum creatinine, urinary protein or albumin excretion, and serum potassium. Patients were followed for a predefined period, with renal function assessed at regular intervals. The primary outcome was the change in eGFR over the follow-up period, while secondary outcomes included changes in serum creatinine, proteinuria, progression to advanced CKD, and treatment-related adverse effects. Statistical comparisons between groups were performed using appropriate parametric or non-parametric tests. Multivariable analysis was used to account for relevant confounding factors, and a p-value <0.05 was considered statistically significant.

Results

A total of 200 patients with chronic kidney disease were included in the analysis, with 100 patients receiving ACE inhibitors and 100 receiving angiotensin receptor blockers (ARBs). The two treatment groups were broadly comparable regarding age, sex, diabetes status, baseline blood pressure, and initial estimated glomerular filtration rate (eGFR). During the follow-up period, both groups demonstrated a decline in renal function; however, the magnitude of deterioration differed between the treatment groups.

Patients receiving ACE inhibitors showed a comparatively slower reduction in eGFR than those receiving ARBs. Mean serum creatinine increased in both groups, but the increase was less pronounced among patients treated with ACE inhibitors. A greater reduction in urinary protein excretion was also observed in the ACE inhibitor group, suggesting a stronger antiproteinuric effect. The proportion of patients progressing to a more advanced stage of CKD was lower among ACE inhibitor users.

Treatment-related adverse effects were observed in both groups. Hyperkalemia and symptomatic hypotension occurred at similar frequencies, while cough was reported more commonly among patients receiving ACE inhibitors. Overall, ACE inhibition was associated with more favorable renal outcomes, particularly with respect to preservation of eGFR and reduction of proteinuria.

Parameter	ACE Inhibitor Group (n=100)	ARB Group (n=100)	p-value
Mean age (years)	56.8 ± 10.4	57.3 ± 9.8	0.72
Baseline eGFR (mL/min/1.73 m²)	48.6 ± 12.1	47.9 ± 11.8	0.68
Final eGFR	43.2 ± 12.4	40.1 ± 11.9	0.04
eGFR decline	5.4 ± 2.8	7.8 ± 3.1	<0.001

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Parameter	ACE Inhibitor Group (n=100)	ARB Group (n=100)	p-value
Baseline proteinuria	1.42 ± 0.61	1.39 ± 0.58	0.74
Final proteinuria	0.91 ± 0.47	1.08 ± 0.51	0.02
Serum creatinine increase	0.18 ± 0.12	0.26 ± 0.15	<0.001

The findings indicate that ACE inhibitor therapy was associated with a significantly smaller decline in eGFR compared with ARB therapy. The reduction in proteinuria was also significantly greater in the ACE inhibitor group. These results suggest that ACE inhibition may provide a modest advantage in preserving renal function and reducing markers of chronic renal injury.

Clinical Outcome	ACE Inhibitors n (%)	ARBs n (%)	p-value
CKD progression	18 (18.0)	29 (29.0)	0.06
Hyperkalemia	9 (9.0)	8 (8.0)	0.80
Symptomatic hypotension	6 (6.0)	7 (7.0)	0.77
Treatment discontinuation	7 (7.0)	6 (6.0)	0.77
Persistent proteinuria	21 (21.0)	34 (34.0)	0.04

In adjusted analysis, treatment with an ACE inhibitor remained associated with a slower decline in renal function after accounting for age, diabetes, baseline eGFR, blood pressure, and baseline proteinuria. The observed difference supports the potential role of ACE inhibition in delaying chronic renal deterioration, although the overall difference in CKD progression did not reach conventional statistical significance.

Discussion

The present study compared the effectiveness of angiotensin-converting enzyme inhibitors (ACE inhibitors) and angiotensin receptor blockers (ARBs) in slowing chronic renal deterioration. The findings demonstrated that patients receiving ACE inhibitors experienced a smaller decline in estimated glomerular filtration rate (eGFR), a lower increase in serum creatinine, and a greater reduction in proteinuria than those treated with ARBs. These findings suggest that ACE inhibition may provide a modest renal protective advantage in patients with chronic kidney disease (CKD), particularly among individuals with significant proteinuria.

The renin–angiotensin system plays a central role in the progression of CKD. Persistent activation of this system contributes to glomerular hypertension, protein leakage, inflammation, and progressive renal fibrosis. ACE inhibitors and ARBs reduce intraglomerular pressure and albuminuria through inhibition of angiotensin II–mediated effects. Current KDIGO recommendations support the use of either an ACE inhibitor or an ARB in patients with CKD and moderately to severely increased albuminuria, with treatment generally continued at the highest tolerated dose. [1]

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The greater reduction in proteinuria observed in the ACE inhibitor group is clinically important because persistent albuminuria is an established marker of renal injury and an important predictor of CKD progression. Previous evidence has demonstrated that renin–angiotensin system inhibition reduces the risk of kidney failure compared with placebo or other active antihypertensive therapies. A large Bayesian network meta-analysis involving 119 randomized trials and more than 64,000 participants found that both ACE inhibitors and ARBs reduced the risk of kidney failure, although ACE inhibitors showed a potentially greater protective effect in several outcomes. [2]

Our finding of a slower eGFR decline with ACE inhibitor therapy is also consistent with previous comparative evidence. A network meta-analysis of randomized clinical trials in patients with nondialysis CKD stages 3–5 reported that ACE inhibitors demonstrated favorable effects on kidney and cardiovascular outcomes and were associated with higher overall treatment benefits compared with several alternative antihypertensive strategies. However, the same analysis found increased risks of hyperkalemia and cough with ACE inhibitor therapy. [3]

The present study also showed that CKD progression occurred less frequently among ACE inhibitor users, although the difference between groups did not reach conventional statistical significance. This may indicate that the relatively limited sample size and follow-up duration reduced the ability to detect differences in hard renal outcomes. Previous research indicates that the renal benefits of renin–angiotensin system blockade are most evident when treatment is maintained over longer periods and particularly in patients with albuminuric CKD.

Safety findings in our study were generally comparable between treatment groups. Hyperkalemia and symptomatic hypotension occurred at similar frequencies, while cough was more frequently observed among ACE inhibitor users. This observation agrees with previous evidence showing that ACE inhibitors are associated with a higher incidence of cough than other antihypertensive therapies. [3] Current KDIGO guidance recommends monitoring blood pressure, serum creatinine, and serum potassium within 2–4 weeks after starting or increasing renin–angiotensin system inhibitors. Hyperkalemia can often be managed without immediately discontinuing treatment. [1]

Importantly, ACE inhibitors and ARBs should not routinely be combined in CKD. Although dual blockade can produce additional reductions in proteinuria, current guidelines recommend avoiding the combination because the potential risks, including hyperkalemia and acute kidney injury, outweigh the additional benefits. [1] The present comparison therefore supports selecting either ACE inhibition or ARB therapy rather than combining the two.

The results should nevertheless be interpreted in light of several limitations. The study design was observational, which introduces the possibility of selection bias and residual confounding. Differences in diabetes control, baseline albuminuria, blood-pressure control, medication adherence, and concomitant therapies could also influence renal outcomes. In addition, the relatively limited sample size may have reduced statistical power for detecting differences in CKD progression. Larger prospective randomized studies with longer follow-up are therefore required to

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determine whether the observed advantage of ACE inhibitors translates into meaningful reductions in kidney failure and mortality. Overall, the findings support the established role of renin–angiotensin system inhibition in CKD and suggest that ACE inhibitors may offer a modest advantage over ARBs in preserving renal function and reducing proteinuria. Nevertheless, treatment selection should remain individualized according to albuminuria, blood pressure, comorbidities, tolerability, potassium concentration, and other clinical factors rather than assuming that one drug class is universally superior.

Conclusion

ACE inhibitor therapy demonstrated a favorable effect on the preservation of renal function compared with angiotensin receptor blockade. Patients receiving ACE inhibitors showed a slower decline in eGFR, a smaller rise in serum creatinine, and greater reduction in proteinuria. Although both treatment approaches provided renal protection, ACE inhibition may offer a modest advantage in delaying chronic renal deterioration, particularly in patients with proteinuric CKD. Treatment should nevertheless be individualized according to renal function, albuminuria, blood pressure, potassium levels, comorbidities, and medication tolerability. Larger, long-term prospective studies are recommended to confirm these findings and determine their effect on progression to kidney failure.

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