International Journal of Pharmacy and Pharmaceutical Sciences

Print ISSN: 2656-0097 | Online ISSN: 0975-1491 Vol 17, Issue 8, 2025

Review Article

OPTIMIZING ANTIHYPERTENSIVE THERAPY IN CHRONIC KIDNEY DISEASE (CKD) PATIENTS: A REVIEW OF DOSE ADJUSTMENTS AND CLINICAL CONSIDERATIONS

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Received: 01 May 2025, Revised and Accepted: 09 Jun 2025

ABSTRACT

Hypertension both causes and is caused by chronic kidney disease (CKD), requiring cautious management strategies to prevent renal and cardiovascular issues. To assure safety and reach a blood pressure (BP) goal of less than 130/80 mmHg, the current study aims to evaluate antihypertensive therapy in participants with chronic kidney disease (CKD), with particular attention to drug selection, combination regimens, dose adjustment, and individual characteristics. Identification of ideal first-line therapy, determination of the best mix of effective combinations, and solutions to issues such as drug-resistant hypertension and harmful drug interactions are among the priority objectives.

Due to their renoprotective and antiproteinuric effects, ACEIs and ARBs are necessary in CKD. CCBs, diuretics, or aldosterone antagonists as part of combination therapy reduce blood pressure. Proteinuria is reduced, and newer drugs, along with sodium limitation, improve treatment efficacy. The present review provides dose modification and appropriate antihypertensive drug options for individuals with chronic kidney disease.

Keywords: Chronic kidney disease (CKD), Hypertension, Blood pressure (BP), Renin-angiotensin-aldosterone system (RAAS), ACE inhibitors (ACEI), Angiotensin receptor blockers (ARB), Calcium channel blockers (CCB), Combination therapy in CKD, Guideline-based treatment, Sympathetic nervous system (SNS)

DOI: https://dx.doi.org/10.22159/ijpps.2025v17i8.54827 Journal homepage: https://innovareacademics.in/journals/index.php/ijpps

INTRODUCTION

Hypertension is a prevalent and significant complication in patients with chronic kidney disease (CKD), affecting approximately 80–90% of those with advanced stages of the condition [1–3]. It is both a consequence and a driving factor in CKD progression, contributing to increased cardiovascular morbidity and mortality as well as accelerated renal function decline [4–10]. Effective management of hypertension in CKD is complex, requiring a careful balance between lowering blood pressure (BP) to reduce cardiovascular and renal risks while avoiding hypotension that may impair renal perfusion.

To provide a comprehensive, evidence-based analysis of antihypertensive therapy optimization in CKD, we conducted a structured literature search across multiple databases including PubMed, Scopus, Springer Link, and Google Scholar. The search strategy used combinations of the following keywords: "chronic kidney disease (CKD)," "hypertension," "renin-angiotensin-aldosterone system (RAAS)," "ACE inhibitors (ACEI)," "angiotensin receptor blockers (ARB)," "diuretics in CKD," "calcium channel blockers (CCB)," "combination therapy in CKD," and "guideline-based treatment." Filters were applied to include only peer-reviewed, English-language studies in human subjects published between 2015 and 2025. Articles were selected if they focused on pharmacological management strategies of hypertension in CKD. Exclusion criteria included editorials, commentaries, case reports, and studies not directly relevant to treatment strategies. This methodology ensured the inclusion of high-quality, clinically relevant studies, allowing for a current and comprehensive review.

Current treatment guidelines recommend RAAS inhibitors (e. g., ACEIs and ARBs) as first-line agents due to their dual benefits in controlling BP and delaying CKD progression. However, new pharmacologic agents such as sodium-glucose cotransporter-2 (SGLT2) inhibitors have shown promising cardiovascular and renal protective effects, broadening the therapeutic landscape [18–20]. Additionally, non-pharmacological interventions-such as sodium intake restriction, weight management, and lifestyle modifications-remain essential components of BP control in CKD patients [18–21].

Obesity, through mechanisms like glomerular hyperfiltration and RAAS activation, can further exacerbate hypertension and kidney damage. Age-related vascular changes also increase the risk of essential hypertension in older adults, compounding the challenge in this population [24–30]. Moreover, the pathophysiology of hypertension in CKD is multifactorial, involving sodium retention, volume overload, sympathetic overactivity, and impaired autoregulation.

Pathophysiology

Chronic Kidney Disease (CKD) is associated with RAAS over activation secondary to decreased blood flow through injured glomeruli, resulting in elevated renin and angiotensin II, which increase blood pressure (BP) [1-3]. Angiotensin II promotes vasoconstriction, sodium retention, and blunts sodium excretion, resulting in hypertension by mechanisms including extracellular volume expansion, vascular stiffness, and sympathetic nervous system (SNS) overactivity. CKD also causes endothelial dysfunction and oxidative stress, further contributing to hypertension [1-3].

Complications such as anaemia therapies, secondary hyperparathyroidism, and calcification of blood vessels worsen hypertension. On the other hand, hypertension worsens CKD progression through arteriolar and glomerular damage, disruption of autoregulation, nephrosclerosis, and GFR reduction [1-3]. The cycle above illustrates how hypertension and CKD are very much interrelated.

Table 1: Antihypertensive drugs in CKD patients: literature review

S. No.	Title	Author	Study type	Study population	Results	Reference
1	Treatment of hypertension in chronic kidney disease	Rigas G. Kalaitzidis *et al.*	Review	Patients with established CKD and/or diabetes with albuminuria.	The most recent guidelines for hypertension recommend a blood pressure (BP) aim of less than 130/80 mmHg. Blood pressure readings over 130/80 mmHg require patients with chronic kidney disease (CKD) to alter their lifestyles and take multiple antihypertensive medications. Recent recommendations state that the first-choice medications must be angiotensin-converting enzyme (ACE) inhibitors. If the ACE inhibitor is not tolerated, angiotensin II receptor blockers (ARBs) ought to be utilized. Non-dihydropyridine CCBs delay the deterioration of kidney function and reliably lower albuminuria. Dihydropyridine CCBs should never be used as a monotherapy in patients with proteinuria CKD; instead, they should always be used in combination with an RAAS blocker. For individuals with chronic renal disease, diuretics constitute the cornerstone of treatment and are used extensively. After treatment with the other primary drugs has failed, all other agents are utilized. It has been advised that individuals with CKD aim for an intensive blood pressure of less than 130/80 mmHg.	[35]
2	Hypertension in chronic kidney disease—treatment standard 2023	Panagiotis I. Georgianos, *et al.*	Review	Patients with chronic kidney disease (CKD)	ACEIs and ARBs continue to be the first-line treatment for hypertension in individuals with chronic kidney disease (CKD), particularly in those with really severe albuminuria. Those who have uncontrolled blood pressure while still on top doses of a dihydropyridine CCB, a diuretic, and a RAS blocker are regarded as having resistant hypertension. The pharmacologic intervention of choice in these patients, added to their standard antihypertensive medication, is spironolactone. Since spironolactone's hyperkalaemia limitation restricts its overall application in resistant hypertension in patients with mid-to-advanced chronic kidney disease, the thiazide-like diuretic chlorthalidone is a substitute therapy for this population of high-risk patients. Chlorthalidone enables one to decrease the risk of hyperkalaemia and administer spironolactone simultaneously. But to prevent side effects like acute renal damage episodes, the spironolactone and chlorthalidone combination needs to be monitored closely. Despite being at various stages of clinical development, newer antihypertensive agents like the dual endothelin receptor antagonist aprocitentan, the aldosterone synthase inhibitor baxdrostat, and the non-steroidal MRA strategy hold more effective blood pressure control features. For one-time interventions in comparison to intensive antihypertensive prescription therapy, regulatory bodies are also likely to approve renal denervation as yet another interventional treatment to be licensed in addition to drugs.	[44]
3.	The effect of antihypertensive drugs on chronic kidney disease: a comprehensive review	Anastasia G. Ptinopoulou, *et al.*	Review.	CKD patients in randomized clinical trials.	If necessary, a CCB or β-blocker might be added to an ACEI or ARB together with a thiazide diuretic or loop diuretic to reach the appropriate blood pressure values. Proteinuria in CKD seems to be further reduced when ACEIs and ARBs are used together. However, this combination has been linked to a considerable risk of acute renal failure and hyperkalaemia. New compounds that may help achieve the best blood pressure control have been discovered through ongoing research. These include the newest RAS blockers, renin inhibitors, and endothelin-1 inhibitors, which work well in combination with ACEIs.	[24]
4.	Management of Hypertension in Chronic Kidney Disease	Dan Pugh, *et al.*	*Review.*	*Patients with CKD and hypertension*	Because of their dual cardioprotective and renoprotective qualities, ACE inhibitors and angiotensin II receptor antagonists (blockers) (ARBs) are especially beneficial for patients with chronic kidney disease (CKD). While ACE inhibitors may be used as first-line treatments for patients with hypertension and non-proteinuric CKD, CCBs and thiazide or thiazide-like diuretics should also be taken into consideration as alternate first-line options in this population.	[32]
5.	Management of Hypertension in CKD: Beyond the Guidelines	Eric Judd, *et al.*	Review.	HTN in patients with CKD, type 2 diabetes mellitus	In CKD, a small dietary sodium restriction can improve the efficacy of antihypertensive drugs such as angiotensin receptor blockers or angiotensin-converting enzyme inhibitors. Crucially, a low salt consumption also enhances the antiproteinuric effects of renin-angiotensin-aldosterone blocking medications and diuretics. The addition of a low-sodium diet to losartan monotherapy boosted the decreases in mean baseline proteinuria in 34 diabetic patients with proteinuria from 30% to 55%. Hydrochlorothiazide and a low-sodium diet together decreased proteinuria by 70% compared to baseline. 47. On the other hand, the ability of renin-angiotensin-aldosterone blockers and diuretics to lower blood pressure and proteinuria is counteracted by a high-salt diet.	[34]
6.	Assessment of achieved clinic and ambulatory blood pressure recordings and outcomes during treatment in hypertensive patients with CKD: a multicentre prospective cohort study	Roberto Minutolo, *et al.*	A Multicentre Prospective Cohort Study	489 consecutive hypertensive patients with CKD (stages 1-5)	Diabetes and prior cardiovascular disease prevalence were 36% and 30%, respectively, and 41% of the group was female. The age range was 64.4±14.2 (SD) years. 16.8%, 22.1%, 14.5%, and 46.6% of the subjects belonged to groups 1-4. Follow-up averaged 5.2 years. Compared with group 1, the risk of the composite cardiovascular event was higher in groups 3 and 4 (HR, 3.17; 95% CI, 1.50-6.69) and 2.83; 95% CI, 1.50-5.34), but not in group 2 (HR, 1.55; 95% CI, 0.75-3.19). Group 2 (HR, 1.24; 95% CI, 0.67-2.27) was not at an increased risk for the composite renal endpoint, but groups 3 (HR, 3.59; 95% CI, 2.05-6.27) and 4 (HR, 2.96; 95% CI, 1.83-4.78) were.	[49]

7.	Hypertension Awareness, Treatment, and Control in Adults With CKD: Results from the Chronic Renal Insufficiency Cohort (CRIC) Study	Paul Muntner*et al.*	Review.	Proteinuria in patients with either diabetic or nondiabetic kidney disease	Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers, which interfere with the renin-angiotensin system, should be used as first-line antihypertensive therapy in patients with proteinuria because they appear to have an antiproteinuric effect that is independent of blood pressure. If blood pressure levels are still not within target, a diuretic should be added to the treatment regimen. To further reduce proteinuria, drugs that have been found to reduce protein excretion, such as aldosterone receptor blockers or nondihydropyridine calcium antagonists, or a combination of an angiotensin-converting enzyme inhibitor and an angiotensin receptor blocker, should be taken into consideration.	[50]
8.	Masked Hypertension and Elevated Night-time Blood Pressure in CKD: Prevalence and Association with Target Organ Damage	Paul E Drawz *et al.*	Cross-sectional study.	Participants with an estimated glomerular filtration rate of 20-70 ml/min/1.73 m^2 were identified from physician offices and review of laboratory databases.	47.8% of the subjects had masked hypertension, 18.8% had sustained hypertension, 4.1% had white-coat hypertension, and 49.3% had controlled blood pressure. Masked hypertension was independently linked with higher proteinuria (+0.9 unit in log2 urine protein), increased left ventricular mass index (+2.52 g/m^2.7), more rapid pulse wave velocity (+0.92 m/s), and lower eGFR (−3.2 ml/min per 1.73 m^2) compared to controlled BP. Subjects with overnight blood pressure<120/70 mmHg did not show a significant reduction in eGFR (−1.4 ml/min per 1.73 m^2), while those with increased blood pressure (−3.6 ml/min per 1.73 m^2) were associated with reduced eGFR. There was a statistically significant interaction between overnight systolic blood pressure and masked hypertension (P = 0.002).	[37]
9.	Antihypertensive therapy prescribing patterns and correlates of blood pressure control among hypertensive patients with chronic kidney disease	Oyunbileg Magvanjav *et al.*	Cross-sectional study.	Electronic health records (EHRs) data from 5658 ambulatory chronic kidney disease (CKD) patients with HTN.	Because they decrease blood pressure and are kidney-protective, ACEIs and ARBs are first-line treatment for patients with hypertensive CKD, especially those with proteinuria. Both thiazide diuretics and BBs are used together; BBs possess cardioprotective benefits in severe chronic kidney disease, whereas thiazide diuretics remain beneficial in combination therapy. While hyperkalaemia risk must be tracked, ACEIs/ARBs are recommended for CKD patients, particularly those with proteinuria or stage ≥3 CKD, by ACC/AHA and KDIGO guidelines. BBs, CCBs, and diuretics are prescribed more often to African Americans than to other races. This highlights the importance of individualized treatment programs and increased clinician knowledge regarding ACEI/ARB therapy in patients with chronic kidney disease.	[40]

Table 2: Guideline-recommended antihypertensive therapy among CKD patients

Hypertension treatment guideline	Initial monotherapy or combination treatment is advised for individuals with chronic kidney disease (CKD)	Reference
KDIGO 2021	• ACEI/ARB in those with very high albuminuria	[44-47]
ESC 2021	• First and second line: ACEI/ARB+CCB/diuretic • Third line: ACEI/ARB+CCB+diuretic	[44-47]
ISH 2020	• ACEI or ARB • CCB or diuretic • Diuretics or CCB • Spironolactone	[44-47]
ESH/ESC 2018	• First and second line: ACEI/ARB+CCB/diuretic • Third line: ACEI/ARB+CCB+diuretic	[44-47]
2017 ACC/AHA2	• ACEI/ARB, especially given proteinuria or eGFR<60 ml/min • Combine drug classes with complementary mechanisms of action (e. g., ACEI/ARB+diuretic [TD], ACEI/ARB+CCB) • Do not combine drugs of the same class (exceptions: diuretic combinations, dihydropyridine CCB with non-dihydropyridine CCB) • Avoid ACEI+ARBa	[37-39]
2012 KDIGO14	• ACEI/ARB, especially if proteinuria is present • ACEI/ARB+diuretic; ACEI/ARB+CCB; Otherb • Avoid ACEI+ARB • Avoid non-dihydropyridine CCB+BB • Avoid dihydropyridine CCB-based combinations that do not include ACEI/ARB	[37-39]
2014 JNC815	• ACEI/ARB-based therapy • Avoid ACEI+ARB	[37-39]
2003 JNC740	• ACEI/ARB, ACEI/ARB+diuretic.	[37-39]

Table 3: Dose adjustments in patients with CKD [48]

Drug class	Drug	Normal dose	Dose adjustment in CKD patients
ACE Inhibitors	Ramipril	2.5 to 20 mg once daily or in two divided doses	• Renal impairment (CrCl 40 ml/min or less): 25% of the usual dose of ramipril is expected to produce full therapeutic levels of ramiprilat • Renal impairment, in hypertension: Initiate at 1.25 mg orally once daily, titrate to effect, MAX 5 mg daily • Renal impairment, in heart failure post myocardial infarction: Initiate at 1.25 mg orally once daily; may increase to 1.25 mg twice daily; titrate to effect, MAX 2.5 mg twice daily
	Captopril	Initially, 25 mg orally 2 or 3 times daily; may increase to 50 mg orally 2 or 3 times daily after 1 to 2 w. Maintenance, 25 to 150 mg orally 2 or 3 times daily; MAX 450 mg/d	• CrCl of 10-50 ml/min: Administer at 75% of normal dose every 12-18 h. • CrCl of<10 ml/min: Administer at 50% of normal dose every 24 h.
	Enalapril	10 to 40 mg/d in single or divided doses.	• Renal impairment, adult (moderate to severe, CrCl 30 ml/min or less) in hypertension: Initial, 2.5 mg orally once daily; MAX 40 mg daily
	Quinapril	Initially, 10 to 20 mg orally once daily; maintenance dose of 20 to 80 mg once daily or in 2 equally divided doses	• Renal impairment (CrCl greater than 60 ml/min) and hypertension: Maximum initial dosage, 10 mg orally once daily; subsequently titrate at 2 w intervals as tolerated • Renal impairment (CrCl 30 to 60 ml/min) and hypertension: Maximum initial dosage, 5 mg orally once daily; subsequently titrate at 2 w intervals as tolerated • Renal impairment (CrCl 10 to 30 ml/min) and hypertension: Maximum initial dosage, 2.5 mg orally once daily; subsequently titrate at 2 w intervals as tolerated
	Perindopril	4 mg orally once daily, MAX 16 mg/d	• Renal impairment (CrCl 30 ml/min or greater): Initial dosage of 2 mg orally daily; MAX dose of 8 mg/d • Renal impairment (CrCl less than 30 ml/min): Use not recommended • Renal impairment (moderate, CrCl 31 to 67 ml/min/1.73m.): 2 mg orally every 24 h • Renal impairment (severe, CrCl 6 to 30 ml/min/1.73m.): 2 mg orally every 48 h
	Benazepril	10 mg orally once daily; maintenance, 20 to 40 mg orally once daily or in 2 equally divided doses. MAX 80MG	• Renal impairment (GFR less than 30 ml/min/1.73 m., or serum creatinine greater than 3 mg/dL) in adults: Initial, 5 mg orally once daily; titrate upward until blood pressure controlled; MAX, 40 mg daily; in GFR less than 15 ml/min, a range of 5 to 20 mg orally daily is suggested • Renal impairment (GFR less than 30 ml/min/1.73 m.) in paediatric patients: Use not recommended
ARB'S	Telmisartan	Initially, 40 mg orally once daily; dosage range, 20 to 80 mg once daily	• No adjustment required.
	Losartan	50 to 100 mg orally once daily or in 2 divided doses (guideline dosage)	• Renal impairment: No adjustment necessary unless patient is also volume depleted (e. g., due to diuretic therapy), then initiate at 25 mg/d; depending on blood pressure response, a 25-mg dose given twice daily may be needed. • Hyperkalaemia: Dosage reduction or discontinuation may be required
	Valsartan	40 to 80 mg orally once daily; target dose, 160 to 320 mg once daily. MAX 320 mg/d.	• Usual dosage range, 80 to 320 mg orally once daily; adjust antihypertensive therapy to target blood pressure of 130/80 mm Hg or lower (guideline dosage) • Addition of a diuretic has a greater effect than dose increases beyond 80 mg (FDA dosage)
	Candesartan	Initially, 16 mg orally once daily or in 2 divided doses; range, 8 to 32 mg once daily or in 2 divided doses	• Renal impairment, adult (moderate to severe, CrCl 15 to 60 ml/min/1.73 m: 8 mg daily may be a sufficient dose • Renal impairment, paediatric (GFR less than 30 ml/min/1.73 m.): Use not recommended
	Irbesartan	150 mg orally once daily; may titrate to MAX 300 mg once daily.	• Renal impairment: No dosage adjustment necessary unless patient is also volume depleted (e. g., due to vigorous diuretic therapy), then initiate at 75 mg orally once daily. • Hemodialysis with intravascular volume or salt depletion: Initiate at 75 mg orally once daily. • Renal function deterioration (clinically significant): Consider withholding or discontinuing therapy.
	Olmesartan	Initially, 20 mg orally once daily when used as monotherapy; after 2 w, may be titrated to a maximum of 40 mg once daily.	• Renal impairment (moderate to marked, CrCl less than 40 ml/min): No initial adjustment required.

Drug class	Drug	Normal dose	Dose adjustment in CKD patients
			• Intravascular volume depletion (eg, use of diuretics, especially with impaired renal function): Consider lower initial doses.
CCB's	Dihydropyridines		
	Amlodipine	Initially, 2.5 mg orally once daily; target dose, 10 mg once daily (guideline dosage). Max dose 10 mg once daily.	• No adjustment required.
	Nifedipine	Initially, 30 or 60 mg orally once daily; generally, titrate over 7 to 14 d; doses greater than 120 mg/d are not recommended (extended-release tablet).	• No specific recommendations are available; the pharmacokinetics of Nifedipine are not significantly affected by the degree of renal impairment; no accumulation in renal failure.
	Felodipine	Initially, 5 mg orally once daily; adjust dose at intervals of not less than 2 w as needed; maintenance, 2.5 to 10 mg once daily.	• No adjustment required.
	Nicardipine	(Immediate-release) Initially, 20 mg orally 3 times daily (Immediate-release) Maintenance, 20 to 40 mg orally 3 times daily; wait at least 3 d before increasing dosage (Sustained-release) Initially, 30 mg orally twice daily (Sustained-release) Maintenance, 30 to 60 mg orally twice daily	• (IV) Renal impairment: Titrate infusion gradually • (Oral, immediate-release) Renal impairment: Initial, 20 mg orally 3 times daily, titrate cautiously • (Oral, sustained-release) Renal impairment: Initial, 30 mg orally twice daily, titrate cautiously
	Isradipine	Initially, 2.5 mg orally 2 times daily alone or in combination with a thiazide diuretic, Max dose 20 mg/d.	• No adjustment required
	Clevidipine	Initially, 1 to 2 mg/hr IV infusion; titration, double the dose at 90-second intervals initially; Maintenance, 4 to 6 mg/hr IV; higher doses up to 32 mg/hour may be required in severe hypertension	• Renal impairment, moderate to severe: Initial, 1 to 2 mg/hr IV infusion
	Cilnidipine	Initial dose 5-10 mg once a day, increase dose up to 20 mg once daily, if necessary.	• No dose adjustment is provided in the manufacturing labelling.
	2)non-Dihydropyridines		
	Verapamil	(Immediate-release) 120 to 360 mg orally daily in 3 divided doses. (Delayed-onset extended-release) 100 to 300 mg orally once daily in the evening (guideline dosage)	• Renal impairment: Initial dose of 100 mg/d may be warranted in some patients; base upward titration on efficacy and safety; multiple doses in patients with renal impairment should be avoided. • (IV Injection) Renal impairment (significant): If repeated IV doses are necessary, give smaller doses and closely monitor PR interval and blood pressure
	Diltiazem	(Extended-release) Initially, 120 to 180 mg orally once daily; target dosage, 360 mg once daily; usual dosage range: 120 to 360 mg orally once daily (guideline dosage). Max dose 540 mg/d.	• No adjustment necessary.
Beta-adrenergic blockers.	Propranolol	(Immediate-release) 80 to 160 mg orally daily in 2 divided doses, up to a dose of 640 mg/d. Extended-release capsule) Initially, 80 mg orally once daily (Extended-release capsule) Maintenance, 120 to 160 mg/d orally up to 640 mg/d	• Renal impairment: Initiate at 80 mg once daily.
	Metoprolol succinate	Initially, 25 to 100 mg orally once daily Dosage titration, adjust dosage at weekly or longer intervals to achieve optimum antihypertensive effect; dosages.	• No adjustment necessary.
	Metoprolol tartrate	Initial dose: 100 mg orally per day in single or divided doses. Maintenance dose: 100 to 450 mg orally per day.	• No dose adjustment is required.
	Atenolol	25 to 100 mg orally daily, 2 divided doses.	• Renal impairment: CrCl 15 to 35 ml/min/1.73 m.: MAX dose 50 mg ORALLY once daily. • Renal impairment: CrCl less than 15 ml/min/1.73 m.: MAX dose 25 mg ORALLY once daily.
Alpha adrenergic blockers	Prazosin	Initially, 1 mg orally 2 or 3 times daily; may titrate slowly up to 20 mg/d in divided doses based upon response; usual maintenance dose, 6 to 15 mg/d in divided doses; some may benefit from doses	• Renal impairment: Initiate at low doses and titrate cautiously.

Drug class	Drug	Normal dose	Dose adjustment in CKD patients
		up to 40 mg/d in divided doses.	
	Terazosin	Initially, 1 mg orally once daily at bedtime; do not exceed the initial dosing regimen, titrate slowly to response; some patients may require 20 mg/d, but doses greater than 20 mg/d do not appear to offer greater efficacy.	• Renal impairment: Impaired renal function had no significant effect on the elimination of terazosin.
	Doxazosin	(Immediate-release) Initially, 1 mg orally once daily; dosage may be doubled as needed based on response; MAX 16 mg/d	• No adjustment provided, use with caution
Alpha+Beta adrenergic blockers	Labetalol	Initially, 100 mg orally twice daily as monotherapy or added to a diuretic regimen, then titrate Maintenance, 200 to 400 mg twice daily. Patients with severe hypertension may require 1200 to 2400 mg/d,	• Renal impairment: Adjustment not required for any degree of renal failure
	Carvedilol	Initially, 6.25 mg orally twice daily; increase if needed to 12.5 mg, then 25 mg twice daily over intervals of 1 to 2 w; MAX dose, 50 mg/d	• Renal impairment: Reduce dosage or discontinue if renal function worsens during up-titration.
Central sympatholytic	Clonidine	Initially, a 0.1 mg/d transdermal patch is applied once every 7 d; if needed, after 1 or 2 w, titrate up by adding another 0.1 mg/d transdermal patch or changing to a larger system.	• Renal impairment: A Lower initial dose may be beneficial
	Methyldopa	500 mg/d in divided doses (Oral) Maintenance, 500 to 2000 mg orally daily in 2 to 4 divided doses; MAX, 3000 mg/d. (IV injection) 250 to 500 mg IV infusion slowly over 30 to 60 min every 6 h; MAX dose, 1000 mg IV every 6 h.	• Renal (mild failure, GFR greater than 50 ml/min): Increase dosage interval to 8 h. • Renal (moderate failure, GFR 10 to 50 ml/min): Increase dosage interval to 8 to 12 h. • Renal (severe failure, GFR less than 10 ml/min): Increase dosage interval to 12 to 24 h.
Arteriolar dilators	Hydralazine	Initially, 25 mg orally 3 times daily, titrated upward; MAX 150 mg/d to avoid drug-induced systemic lupus erythematosus. Hypertensive emergency: Initial, 10 mg slow IV infusion, MAX initial dose 20 mg; repeat every 4 to 6 h as required	• Renal impairment (advanced): Use with caution as for any antihypertensive agent in advanced renal impairment. Dosage reduction of the injection formulation may be required for marked renal damage; adjust to response with frequent blood pressure measurement. For GFR less than 10 ml/min, a dosing interval of 8 to 16 h is suggested
	Minoxidil	Initially, 5 mg orally once daily; titration may increase stepwise to 10, 20, then to 40 mg/d in single or divided doses. Max 100 mg/d. Initially, 2.5 mg orally 2 to 3 times daily, then titrate upward; use with a loop diuretic and beta blocker (guideline dosage).	• Renal impairment: No dosage adjustment is provided in the manufacturer's labeling; However, patients with renal failure and or receiving dialysis may require dosage reduction.
Arteriolar venodilators	Nitroprusside sod. (Hypertensive crisis)	Initial, 0.3 to 0.5 mcg/kg/min IV; titration, increase in increments of 0.5 mcg/kg/min to blood pressure target, MAX 10 mcg/kg/min; keep duration of treatment as short as possible.	• Renal impairment, eGFR less than 30 ml/min: Limit infusion rate to less than 3 mcg/kg/min IV. • Renal impairment, anuric: Limit mean infusion rate to 1 mcg/kg/min IV.
Diuretics	Thiazide		
	Hydrochlorothiazide	(Tablet) Initially, 12.5 to 25 mg orally daily in single or 2 divided doses; target, 25 to 50 mg/d.	• Renal impairment: Use is contraindicated in anuria. The cumulative effects of thiazides may develop in patients with impaired renal function, possibly precipitating azotaemia; use with caution in severe renal disease. Thiazide diuretics become less effective antihypertensive agents at estimated GFR (eGFR) less than 45 ml/min and should be replaced with a loop diuretic if eGFR is less than 30 ml/min • Continuous renal replacement therapy (CRRT): Preferably avoid use. • Progressive renal impairment: Consider withholding or discontinuing therapy.
	Chlorthalidone	Initial, 12.5 mg orally once daily; target dose, 12.5 to 25 mg once daily, may increase to 50 mg once daily if response is insufficient; if additional control is required, may increase to 100 mg once daily or consider adding a second antihypertensive agent.	• Renal impairment (anuria): Use is contraindicated. • Renal impairment (GFR 15 to 59 ml/min): 12.5 to 25 mg orally once daily. • Renal impairment (GFR less than 15 ml/min): Preferably avoid, as it is usually ineffective. • Continuous ambulatory peritoneal dialysis (CAPD): Preferably avoid, as it is usually ineffective • Continuous renal replacement therapy (CRRT): Preferably avoid, as it is usually ineffective. • Hemodialysis: Preferably avoid, as it is usually ineffective.
	Indapamide	Initially, 1.25 mg orally once daily; target, 1.25 to 2.5 mg once daily. If 2.5 mg/d is not effective, may increase to 5 mg orally once daily after 4 w,	• Renal impairment (anuria): Use is contraindicated. • Renal impairment (estimated GFR less than 30 ml/min): Adequate blood pressure response was observed with 2.5 mg daily in a small subset of patients with estimated CrCl of 8 to 27 ml/min;

Drug class	Drug	Normal dose	Dose adjustment in CKD patients
			low-dose indapamide may be useful in some patients with severe renal disease but routine use is not yet recommended. Some thiazide diuretics (indapamide, metolazone, chlorthalidone) may remain effective for blood pressure lowering at a GFR below 30 ml/min/1.73m
	Loop diuretics		
	Furosemide	Usual dosage range, 20 to 80 mg orally daily in 2 divided doses. Concomitant medication, reduce dosage of other antihypertensive agents by at least 50% when furosemide is added to a regimen; further reduction in dosage or discontinuation of other agents may be necessary.	• Renal impairment (anuria): Use is contraindicated. • Renal impairment (moderate to severe) in patients with acute decompensated heart failure: Consider higher initial doses. • Azotemia increasing or development of oliguria in treatment of severe progressive renal disease: Discontinue use.
	Torsemide	Initial 5 mg orally once daily; titration, may increase to 10 mg once daily after 4 to 6 w to allow for optimum antihypertensive effect.	• Renal impairment: Contraindicated in anuria.
	Potassium-sparing agents.		
	Spironolactone	(Tablets) Initially, 25 to 100 mg/d orally in single or divided doses. (Suspension) Initially, 20 to 75 mg orally per d in single or divided doses.	• (Suspension) Renal impairment (estimated GFR [eGFR] 30 to 50 ml/min/1.73m.) in heart failure: Consider initiating at 10 mg/d due to risk for hyperkalaemia. • (Tablet) Renal impairment (eGFR 30 to 50 ml/min/1.73m.) in heart failure: Consider initiating at 25 mg orally every other day due to the risk of hyperkalaemia. • Diarrhea causing dehydration or loop diuretic therapy interruption: Consider temporary withholding of spironolactone due to the risk of hyperkalaemia or worsening renal function.
	Eplerenone	Initially, 50 mg orally once daily; allow 4 w to achieve optimum antihypertensive effect; may increase to MAX 50 mg twice daily for inadequate response.	• Renal impairment, CrCl 30 ml/min or less: Contraindicated. • Diarrhea causing dehydration or loop diuretic therapy interruption: Consider temporary withholding of eplerenone due to the risk of hyperkalaemia or worsening renal function.
	Amiloride	Initially, 5 mg orally once daily with food; may increase to 10 mg daily as needed; if persistent hypokalaemia occurs, may increase to 15 to 20 mg once daily.	• Renal impairment (anuria, acute or chronic renal insufficiency, and evidence of diabetic nephropathy): Use is contraindicated. • Renal impairment (BUN greater than 30 mg/dL or serum creatinine greater than 1.5 mg/dL): Avoid use without careful, frequent, and continuing monitoring of serum electrolytes, creatinine, and BUN levels. • Renal impairment (GFR 15 to 59 ml/min): 2.5 mg orally once daily or 5 mg orally every 48 h. • Renal impairment (GFR less than 15 ml/min): Preferably avoid due to risk for hyperkalaemia and cardiac irregularities. • Continuous ambulatory peritoneal dialysis (CAPD): Initial, 2.5 to 5 mg orally 3 times weekly, increasing to 5 to 10 mg once daily if necessary, and monitor carefully. (Recommendation based on limited data) • Continuous renal replacement therapy (CRRT): Preferably avoid due to risk for hyperkalaemia and cardiac irregularities. • Hemodialysis: Preferably avoid due to risk for hyperkalaemia and cardiac irregularities.

RESULTS

This review illustrates that ACE inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) are efficacious first-line therapy for blood pressure management in proteinuria patients with chronic kidney disease (CKD). ACEI/ARB+TD or CCB monotherapy showed improved blood pressure control compared with monotherapy. The combination of ACEI and ARB resulted in a higher risk of AKI and hyperkalaemia. Diuretics helped in CKD with volume-overload, while non-dihydropyridine CCBs were withheld from beta-blockers to prevent cardiac risks. Low blood pressure thresholds (<130/80 mmHg) lowered renal and cardiovascular endpoints, according to KDIGO, ACC/AHA, and JNC. Personalized care as per the stage of CKD and comorbidities must be used to get the best possible treatment.

DISCUSSION

Hypertension plays a key role in the onset of chronic kidney disease (CKD) and cardiovascular disease. In CKD patients, reduction of cardiovascular risk and slowing renal progression are contingent on optimal blood pressure (BP) control. Initial therapy is always recommended by global guidelines to employ renin-angiotensin system (RAS) inhibitors, including angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor blockers (ARBs), particularly when proteinuria exists [11-17].

The current research supports the intervention of ACEIs/ARBs in the control of CKD, especially in combination with TDs or CCBs for augmented BP lowering [4-10, 29]. Importantly, dihydropyridine CCBs are recommended in place of non-dihydropyridine CCBs because of their renal safety [1-3, 16]. The findings also support the established practice of the avoidance of dual RAS blockade (ACEI+ARB) because of the associated risk of hyperkalaemia, hypotension, and declining renal function [23].

Although guideline suggestions offer a formalized strategy, patient-specific factors-e. g., CKD stage, electrolyte disturbances, and comorbidities—require judicious dose adjustment. ACEIs/ARBs need close surveillance of serum creatinine and potassium levels, especially in severe CKD. Diuretics are frequently needed for volume management but need to be used with caution to avoid electrolyte imbalances. Beta-blockers, while not first-line, can also be useful in patients with concomitant heart failure or ischemic heart disease [18-20].

In general, the results validate existing hypertension treatment strategies in CKD but highlight the need for individualized treatment regimens according to disease severity, risk factors, and response to therapy [18-20, 22].

CONCLUSION

This study highlights the importance of guideline-directed therapy in managing hypertension among CKD patients, with ACEIs/ARBs remaining the cornerstone of treatment, particularly in proteinuric CKD. Combination therapy with diuretics or CCBs provides additional BP control while minimising adverse effects. However, treatment strategies must be individualized based on CKD stage, electrolyte status, and comorbidities. Optimized BP management in CKD is essential to slowing disease progression and reducing cardiovascular risk, emphasizing the need for careful drug selection and dose adjustments.

FUNDING

Nil

AUTHORS CONTRIBUTIONS

All authors contributed to the conception and design of the review article. Mr. Tejas Parkhe conducted the literature search, data analysis, and drafted the initial manuscript. Ms. Kanchan Shelke critically reviewed the literature, contributed to the writing of specific sections, and provided expertise in the methodological framework. Dr. Dhanraj Shinde supervised the project, revised the manuscript for intellectual content, and ensured the accuracy and coherence of the final version. All authors read and approved the final manuscript.

CONFLICT OF INTERESTS

Declared none

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