Hypertension

REVIEW

Hypertension in Patients With End-Stage Kidney Disease Requiring Dialysis: Bridging the Divide Between Evidence and Practice

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ABSTRACT: Hypertension is a highly prevalent and modifiable risk factor, affecting >80% of patients undergoing dialysis. Its pathophysiology is complex and differs from that of the general population, driven by factors such as volume overload, arterial stiffness, overactivation of the sympathetic and renin-angiotensin-aldosterone systems, and endothelial dysfunction. Achieving optimal blood pressure and volume control is central to dialysis care, with significant implications for cardiovascular outcomes and patient quality of life. Despite its importance, evidence guiding hypertension management in this population remains limited. Furthermore, reliable, objective methods to assess extracellular volume are lacking. This review examines current approaches to the assessment and management of hypertension in maintenance hemodialysis, summarizing existing evidence, clinical guidelines, and ongoing challenges in blood pressure and volume control.

Key Words: blood pressure ■ dialysis ■ hypertension ■ kidney ■ renal insufficiency ■ renin-angiotensin system

Kidney disease affects 1 of 7 adults in the United States, and over 500 000 individuals develop kidney failure and require dialysis. Cardiovascular disease (CVD) accounts for 40% to 50% of deaths in this population.[1] There is an incredibly high disease burden of hypertension in these patients, with the prevalence ranging between 72% and 90%.[1–3] Hypertension is one of the most important and modifiable risk factors for CVD in this population,[4,5] yet there remain many challenges in the diagnosis and management of hypertension in patients with kidney failure. Here, we review the current state of evidence and the persistent gaps in knowledge in hypertension in the care of patients with kidney failure.

CKD AND HYPERTENSION: INTRINSICALLY RELATED RISK FACTORS FOR CVD

Both hypertension and chronic kidney disease (CKD) are intrinsically related risk factors for CVD. CKD itself is an established risk factor for CVD events and mortality, independent of traditional cardiovascular risk factors such as hypertension.[6] All major forms of CVD occur more frequently among individuals with CKD, including atherosclerotic disease, acute myocardial infarction, heart failure, and stroke.[7,8] For example, among older adults, the prevalence of CVD is approximately twice as high in those with CKD compared with those without, and survival following major cardiovascular events is significantly lower in patients with CKD.[9] Both CKD and hypertension independently have substantial implications on cardiovascular morbidity and mortality, and their coexistence further amplifies overall risk.[9–12] The intersection of these 2 conditions underscores the increased risk for patients with kidney disease and hypertension.

PATHOPHYSIOLOGY OF HYPERTENSION IN PATIENTS WITH KIDNEY FAILURE

As kidney disease progresses to kidney failure requiring dialysis, blood pressure (BP) control becomes increasingly challenging. Multiple interrelated factors contribute to the complexity of hypertension management in

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Nonstandard Abbreviations and Acronyms

ABPM	ambulatory blood pressure monitoring
ACHIEVE	Aldosterone Blockade for Health Improvement Evaluation in End-Stage Renal Disease
ALCHEMIST	Aldosterone Antagonist Chronic Hemodialysis Interventional Survival Trial
BID	Blood Pressure in Dialysis
BP	blood pressure
BVM	blood volume monitoring
CKD	chronic kidney disease
CVD	cardiovascular disease
DISO	Dialysate Individualized Sodium
DRIP	Dry-Weight Reduction in Hypertensive Hemodialysis Patients
FOSIDIAL	Fosinopril in Dialysis
GLP-1 RA	GLP-1 receptor agonist
LUS	lung ultrasound
LUST	Lung Water by Ultrasound-Guided Treatment in Hemodialysis Patients
SBP	systolic blood pressure

patients with kidney failure, including chronic volume overload, increased arterial stiffness, endothelial dysfunction, heightened sympathetic nervous system activity, and upregulation of the renin-angiotensin-aldosterone system.[13] Moreover, the kidney's ability to regulate plasma volume and sodium excess through normal mechanisms is progressively impaired, further exacerbating hypertension and its cardiovascular consequences.[14] Due to the unique pathophysiologic conditions, BP management in other populations cannot be readily applied to patients on dialysis.[14]

Vascular Injury, Arterial Stiffness, and Endothelial Damage

CKD promotes a state of progressive vascular injury, resulting in both structural remodeling and functional impairment of the vasculature that exacerbates difficulties in BP control.[15] Vascular calcification, alterations in the intrinsic properties of the vessel wall, enlargement of the carotid artery wall, and intimal-media layer remodeling are all reported in CKD.[16] These functional and structural changes in the arterial wall heighten arterial stiffness, thereby increasing aortic systolic and pulse pressures, contributing to increased afterload and accelerated pulse-wave velocity.[17] High prevalence of vascular calcification and medial arterial calcification increases arterial stiffness, reflected by elevated pulse-wave velocity, and leads to higher systolic BP (SBP), left ventricular afterload, and poor end-organ perfusion. Endothelial dysfunction and impaired arterial compliance further reduce the elastic capacity of the large arteries.[18] The resulting hemodynamic burden promotes left ventricular hypertrophy, exacerbates volume overload, and further worsens hypertension in patients on dialysis[19] (Figure 1).

Neurohormonal Dysregulation, Inflammation, and Uremic Toxins

Sympathetic nervous system activation and renin-angiotensin-aldosterone system upregulation markedly increase systemic vascular resistance and BP in patients with kidney failure. As kidney function declines, these neurohormonal disturbances become more pronounced, resulting in persistently heightened vascular tone and sustained hypertension.[20]

Patients with CKD have elevated proinflammatory cytokines and increased uremic vascular toxins normally excreted by the kidneys.[21] Dialysis itself can activate the immune system and exacerbate chronic inflammation, which, in turn, promotes arterial stiffening.[22] This persistent inflammatory state contributes to progressive structural remodeling of the vasculature and further increases in arterial stiffness.[16]

Volume Overload

Chronic fluid overload remains a central pathophysiologic mechanism contributing to hypertension in dialysis patients. Volume overload leads to increased cardiac output and heightened systemic vascular resistance.[23] Persistent volume expansion contributes not only to elevated BP but also to left ventricular hypertrophy, arterial stiffness, and a higher risk of adverse cardiovascular outcomes. Given the loss of normal physiological processes to regulate fluid overload in the body, excess extracellular volume is highly prevalent among individuals receiving hemodialysis. Volume overload is, therefore, primarily driven by factors such as excessive interdialytic weight gain, high dietary sodium intake, and insufficient fluid removal during treatment sessions.[23]

Dialysis-Related Factors

The impact of the dialysis procedure confers a unique challenge in BP management for these patients. During dialysis, ultrafiltration-induced sodium and volume shifts can cause increased activation of the sympathetic nervous system and renin-angiotensin-aldosterone system that aggravate the already maladaptive responses to volume overload and arterial stiffness.[24] In addition, the use of erythropoietin-stimulating agents may further contribute to vasoconstriction.

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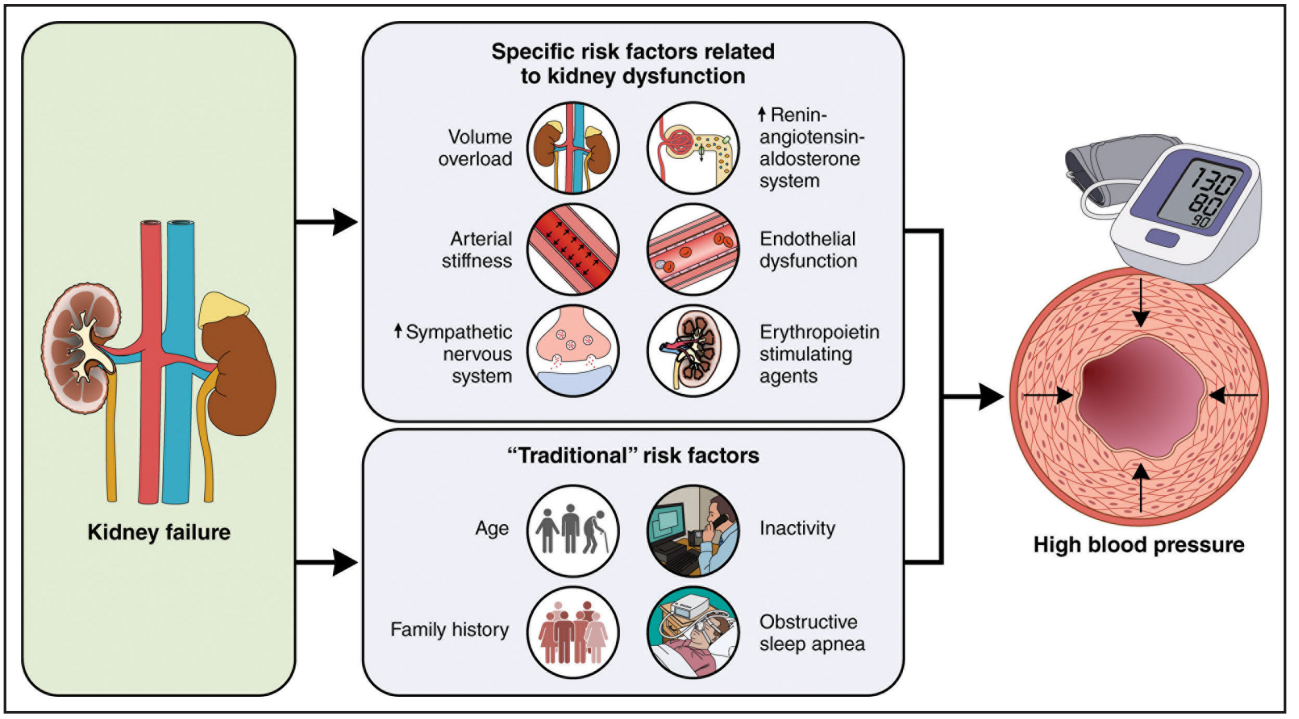

Figure 1. Pathophysiology of hypertension in patients receiving hemodialysis.[14]

CURRENT EVIDENCE AND GUIDELINES FOR BP TARGETS IN PATIENTS TREATED WITH DIALYSIS

There have been limited clinical trials to test the effectiveness of treating different BP targets in patients treated with hemodialysis. For example, the BID (BP in Dialysis) was a pilot trial of 126 participants, which randomized participants to either an intensive predialysis SBP goal of 110 to 140 versus 155 to 165 mm Hg. The primary objective of the study was to evaluate the feasibility of intensive BP interventions to inform larger randomized controlled trials. The BID study demonstrated that intensified BP treatment is feasible in the dialysis population with separation between the treatment groups.[20] However, there was a trend toward higher rates of vascular access thrombosis in the intensive treatment group.

The limited data to guide management of hypertension in patients treated with hemodialysis are reflected in clinical practice guidelines. While guidelines provide recommendations for patients with CKD, they notably omit specific recommendations for individuals receiving maintenance dialysis. The recently published 2025 American College of Cardiology/American Heart Association Hypertension Guidelines define target BP levels for the general population and in those with mild-to-moderate CKD,[25] recommending a BP target of <130/80 mm Hg.[25] The 2021 Kidney Diseases: Improving Global Outcomes Guidelines recommend a goal of SBP <120 mm Hg using standardized BP measurement techniques in nondialysis requiring patients with CKD, but BP targets for patients treated with dialysis are absent.[26,27] These omissions underscore a critical unmet need for dialysis-specific evidence and consensus guidelines to guide BP measurement and goal in patients undergoing dialysis.

MEASURING BP IN DIALYSIS PATIENTS: TIMING AND SETTING MATTER

The setting and timing of BP measurement in relation to dialysis are important in evaluating BP trends in patients treated with hemodialysis as there is substantial variability in BP measurements taken before dialysis (predialysis BP), during dialysis, after dialysis, or on a nondialysis day.[28] Studies have demonstrated poor correlation between dialysis and nondialysis day BPs.[29] During dialysis, there are large hemodynamic fluctuations that contribute to the significant differences in BP measured around the dialysis procedure compared with BPs measured on a nondialysis day. In practice, management of hypertension is most often based on predialysis and postdialysis BP measurements obtained in the dialysis unit.

However, observational studies utilizing peridialysis BPs demonstrate either no association or a U- or J-shaped relationship between mortality and BP.[30–32] This has led to heterogeneity and confusion on how best to manage BP in patients treated with hemodialysis. The nadir of the U-shape is a predialysis SBP of 140 to 160 mm Hg, above thresholds seen in other populations. Moreover, these observational studies have demonstrated that predialysis SBP<140 mm Hg is paradoxically associated with a higher risk of mortality and CVD, which

has contributed to permissive hypertension. The reason for this U-shape paradox is unclear; hypotheses have included confounding due to malnutrition, widened pulse pressure due to arterial stiffness, inflammation, heart failure, and effects of volume accumulation. However, studies that have investigated these hypotheses have not identified a clear explanation for the U-shaped association between predialysis SBP and clinical outcomes.

However, data have shown that in the same patients in whom the U-shaped paradox is observed, when BP is measured on a nondialysis day, there is a strong linear and positive association with risk of death and CVD events (Figure 2), more consistent with data from other populations.[33,34] These studies suggest that perhaps focus should be shifted to measuring nondialysis BPs to guide clinical management as overreliance on dialysis-unit BP measurements may lead to overtreatment or undertreatment of BP. Collectively, these findings suggest that BP targets in patients treated with hemodialysis may be better informed by out-of-dialysis BP measurements and

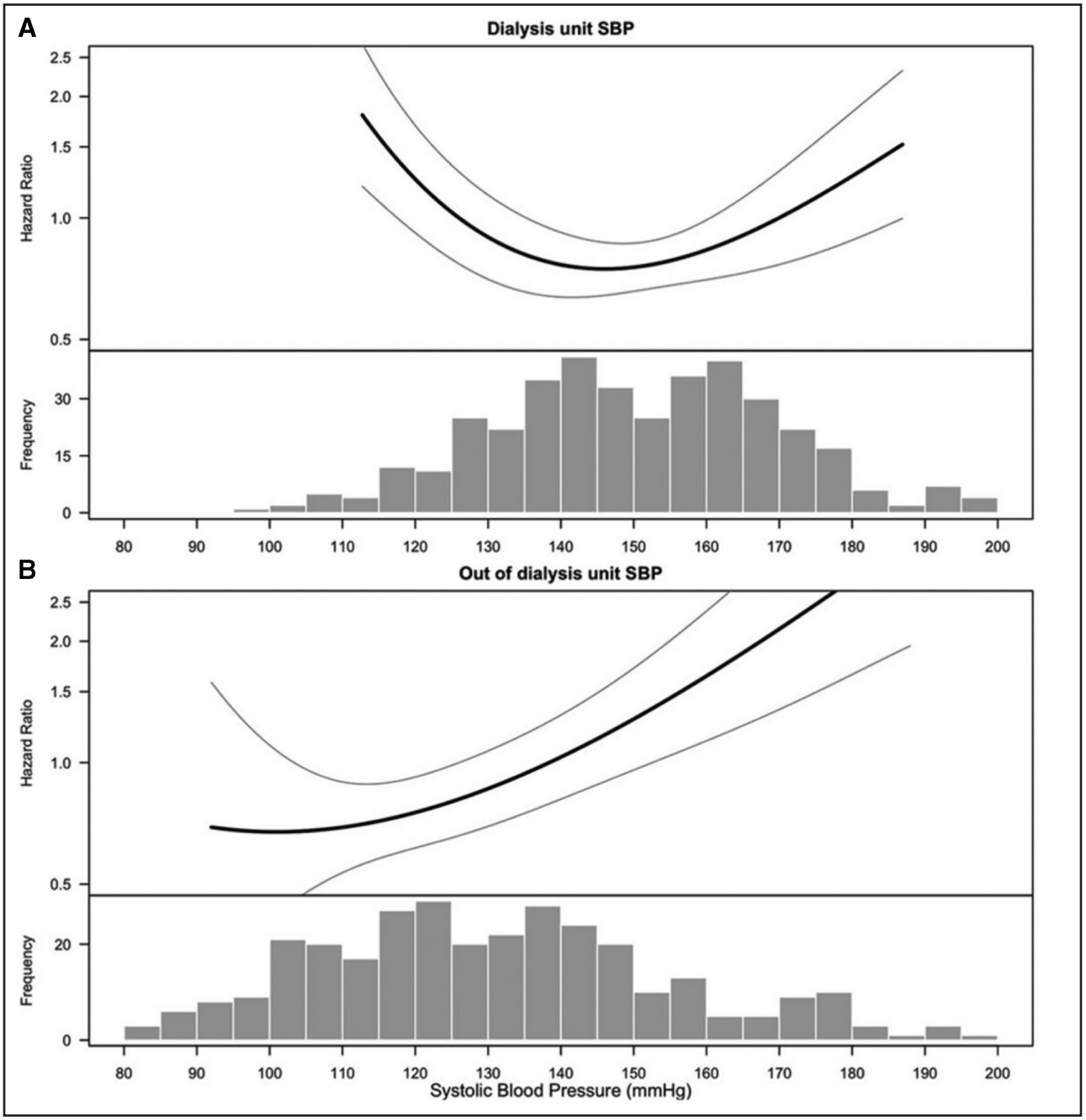

Figure 2. Associations of systolic blood pressure (SBP) with mortality.
The smooth spline estimates the hazard ratio of all-cause mortality according to SBP (mm Hg) at maintenance hemodialysis by dialysis-unit measurements (**A**) and at maintenance hemodialysis by out-of-dialysis-unit measurements (**B**). All analyses are adjusted for age, sex, race/ethnicity, tobacco use, body mass index, diabetes, and history of cardiovascular disease.[33]

should be individualized, considering cardiovascular risk such as vascular calcifications and other patient-specific factors, rather than relying solely on peridialysis BP measurements.

NONDIALYSIS DAY BP: A BETTER APPROACH TO BP MANAGEMENT?

Ambulatory BP monitoring (ABPM) is considered the gold-standard method for hypertension evaluation in the general population. In patients treated with hemodialysis, ABPM is typically performed over a 44-hour interval from the end of the dialysis session to the next dialysis session, given the considerable variability in BPs during this interval. Compared with peridialytic BPs, 44-hour ABPM has had more consistent and strong associations with cardiovascular events and mortality.[29,35,36]

Given the burden of ABPM, there has been increasing interest in home BP measurement as an alternative. Data have shown strong correlations between 44-hour ABPM and home BP measures performed several times a week on nondialysis patients. Studies have also demonstrated strong and linear associations between home BP and outcomes in patients treated with dialysis.[37–39]

It should be recognized that there are many challenges with ABPM and home BPs in this population. First, ABPM and home BP measurement are not integrated into the usual hemodialysis workflow and present practical challenges for patients.[40] In addition, dialysis access restrictions, including prior accesses, scarring, and central stenoses, may interfere with the accuracy of the oscillometric devices. The timing of out-of-unit BPs in relation to the dialysis procedure can also impact the interpretation of home BPs, especially in patients with high intradialytic weight gain. Finally, patient limitations including adherence, comorbid conditions, and physical limitations present further challenges in obtaining accurate BP measurements.

However, a recent 2-site clinical trial of patients treated with hemodialysis demonstrated the feasibility of treating home BPs with high adherence rates and reassuring safety signals (unique identifier: NCT03459807). A larger clinical trial is underway (the HOME-BP trial [Home Blood Pressure Trial DOPPS: Dialysis Outcomes and Practice Patterns Study]; unique identifier: NCT05159999) is underway to test the efficacy of treating home-BP (versus predialysis SBP) on clinical outcomes in patients treated with hemodialysis.

TREATMENT OF HYPERTENSION IN PATIENTS TREATED WITH HEMODIALYSIS: VOLUME MANAGEMENT

Volume overload is a critical contributor to hypertension in patients treated with hemodialysis, but accurately assessing volume status is difficult. Many patients appear euvolemic despite fluid excess and conventional indicators such as physical examination findings or interdialytic weight fail reflect true body fluid volume, complicating BP management.[41]

Assessment of Dry Weight in Dialysis

Dry weight is estimated using both subjective and objective measures. Each dialysis session's ultrafiltration is determined based on predialysis weight, target weight, and clinical signs and symptoms of volume overload. Excessive ultrafiltration, either from challenging or attempting to reach the target weight, can cause intradialytic hypotension and symptoms of orthostasis, dizziness, headache, muscle cramping, fatigue, and syncope and is associated with poor clinical outcomes including CVD, mortality, cerebral hypoperfusion, and loss of residual kidney function.[42–45]

When ultrafiltration rates exceed the ability of the interstitial space to refill the intravascular compartment, intradialytic hypovolemia and hypotension may occur, impairing organ perfusion. In DOPPS (n=15 536), ultrafiltration rate >10 mL/h per kg was associated with worse outcomes.[46] Post hoc analysis of the HEMO study (hemodialysis trial), ultrafiltration rate >13 mL/h per kg was independently associated with higher all-cause and cardiovascular mortality, while rates of 10 to 13 mL/h per kg conferred a modestly increased risk compared with ≤10 mL/h per kg.[47]

The DRIP study (Dry-Weight Reduction in Hypertensive Hemodialysis Patients) evaluated whether systematic volume reduction improves BP in hemodialysis patients.[48] Among 150 patients, 100 were assigned to additional ultrafiltration and had the dry weight probed without longer dialysis duration, while 50 controls received standard physician visits. At 8 weeks, target weight was reduced by 1 kg, and systolic BP changed by −6.6 mm Hg from baseline (95% CI, −12.2 to −1.0 mm Hg; *P*=0.021), suggesting that dry weight reduction can lower BP as effectively as adding another antihypertensive drug.[48]

Tools for Estimating Volume Status

Dry weight estimation is imprecise, and there are no objective gold standards for assessing volume status. New technologies can help guide clinicians by providing objective measures and estimates of volume status. Although these tools are not yet widely available in dialysis units, they hold promise for improving the detection and management of volume overload. Table 1 presents commonly used techniques for evaluating volume status among patients on dialysis, along with their major advantages and disadvantages.

Point-of-Care Lung Ultrasound

Zoccali et al[49] identified interstitial lung tissue as an extravascular compartment that reflects total extracellular volume. Lung ultrasound (LUS) can visualize

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Table 1. Volume Assessment Tools and Applicability in Patients Treated With Dialysis

	Point-of-care ultrasound	Bioimpedance spectroscopy	Blood volume monitoring
Method	Ultrasound to assess volume markers such as IVC diameter and lung ultrasound comets (B-lines) as a marker of extracellular volume	Measures resistance to an electrical current to estimate TBW, ECW, and ICW	Measures relative changes in hematocrit during a dialysis session, often via an optical or ultrasonic sensor in the extracorporeal circuit
Application	Real-time assessment of extracellular volume	Assesses overall fluid status and body composition to help determine target dry weight in the interdialytic period	Real-time monitoring during dialysis to adjust the ultrafiltration rate and prevent intradialytic hypotension
Strengths	Noninvasive, real-time assessment of extracellular volume on specific congestion sites (lungs and IVC). Data supporting severe congestion as a marker for cardiac mortality	Noninvasive, quick, inexpensive, and easy to perform. Provides objective, quantitative data on fluid compartments	Provides continuous, real-time feedback during HD. Can inform fluid removal to limit intradialytic hypotension
Weaknesses	Operator-dependent. Requires adequate training and standard protocols for accurate and reproducible results. Accuracy of the assessment depends on the operator's skill and the interpretation of B-lines and IVC diameter. Semiquantitative, no standardized cutoffs. The presence of congestion does not necessarily indicate cardiogenic origin. Certain lung pathologies can affect interpretation.	Interpretation can be challenging. May not be accurate for single measurements in individuals, requires trends. Results can be affected by patient condition (such as obesity). Optimal parameters and target values need standardization. Requires device calibration and proper modeling. The presence of a vascular fistula and abdominal fluid can affect the accuracy of compartmental measurements.	Only provides relative changes, not absolute volume. Only used during the HD session. Only compatible with proprietary dialysis machines. Reliability decreases during dialysis sessions with low ultrafiltration rates.
Strength of evidence and outcomes in dialysis patients	Some supportive data for short-term outcomes only	Some, but not all, data support improved blood pressure control and potentially reduce mortality and hospitalization when used for guided management.	Limited

ECW indicates extracellular water; HD, hemodialysis; ICW, intracellular water; IVC, inferior vena cava; and TBW, total body water.

excess extravascular fluid, informing ultrafiltration during dialysis.[49] In a multicenter cohort of 392 hemodialysis patients, LUS was used to assess lung congestion and evaluate whether LUS may predict death and cardiac events beyond classical methods. Moderate-to-severe lung congestion was observed in 45% of participants, while severe congestion was present in 14%. Participants with severe congestion had a 4.2-fold higher risk of death (hazard ratio [HR], 4.20 [95% CI, 2.45–7.23]) and a 3.2-fold higher risk of cardiac events (HR, 3.20 [95% CI, 1.75–5.88]) compared with those with mild or no congestion.[49]

Although LUS has shown promise in assessing volume status, evidence regarding its impact on clinical outcomes remains limited. The LUST trial (Lung Water by Ultrasound-Guided Treatment in Hemodialysis Patients) was an international multicenter randomized controlled trial that compared usual care to an LUS-guided treatment strategy. Lung congestion was relieved in 78% of the intervention group ($P<0.001$ versus control). However, the composite end point (all-cause death, nonfatal myocardial infarction, and decompensated heart failure) did not significantly differ between the 2 study arms (HR, 0.88 [95% CI, 0.63–1.24]).[50]

Whole Body Bioimpedance Spectroscopy

Bioimpedance spectroscopy is a noninvasive method to quantitatively measure the volume of fluid in body compartments. Because the cell membrane conducts differently at low versus high frequencies, the potential difference across the tissue undergoes what is called a phase shift. The overall measurement is known as the impedance because it depends on frequency. By combining measurements from different segments of the body (arm, leg, and trunk) with derived constants, extracellular water and intracellular water can be calculated.

Kim et al[51] demonstrated a higher incidence of mortality in volume overload in participants, as measured by bioimpedance (odds ratio, 2.57 [95% CI, 1.08–6.13]; $P=0.033$). A meta-analysis demonstrated that volume overload as determined by bioimpedance was an independent predictor of all-cause mortality and hospitalization in dialysis populations.[52] Several studies have evaluated the use of bioimpedance to determine ultrafiltration goals and dry weight in hemodialysis and peritoneal dialysis patients. In 3 randomized control trials, the use of bioimpedance to guide treatment had no impact on mortality.[53–55] In addition, studies evaluating bioimpedance as part of a standardized protocol for assessing fluid status have not demonstrated improvements in all-cause hospitalization or hospital-free survival.[51,54] Moreover, bioimpedance-guided management appeared to have a favorable effect on BP control, as evidenced by a reduction in antihypertensive medication use in the randomized study group.[54,55] In a prospective study of 65 hemodialysis patients, bioimpedance was used to adjust dry weight at baseline, and 1 and 3 months. At baseline, over 50% of patients were hypertensive, and 9.5% of patients had an increase in total body water but were normotensive. After 6 months, participants demonstrated a reduction in BP, and 78.5% were normotensive (mean SBP, 128±16 mm Hg; $P<0.0001$; compared with baseline).[56]

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Blood Volume Monitoring and Dry Weight

The use of blood volume monitoring (BVM) began in the early 1990s when the CRIT-LINE (Continuous Real-Time Intravascular Volume Monitoring) technology was developed by Fresenius Medical Care, Bad Homburg, Germany. BVM, originally developed to measure real-time hematocrit changes during dialysis via an arterial line monitor, reflects shifts in blood density as ultrafiltration removes fluid. A flat BVM curve indicates plasma refill matches or exceeds fluid removal.

Several studies have utilized BVM to guide ultrafiltration goals on dialysis. In a randomized cross-sectional observation study of 169 participants on hemodialysis, 43% of patients were noted to be BVM wet (flat curve), and 27 of these patients had intradialytic signs or symptoms of hypotension. While most studies of BVM have focused on reducing intradialytic hypotension and improving dialysis tolerability, a 6-month randomized controlled trial evaluated its use in SBP management. Overall, the study observed a statistically significant reduction in home SBP over the study period (P=0.005). However, there was no significant difference in BP reduction between the BVM and standard treatment groups. In the BVM group, SBP decreased from 147.8±21.7 to 139.8±16.2 mm Hg, whereas, in the standard treatment group, SBP declined from 141.9±19.2 to 135.2±9.9 mm Hg.

Other Strategies to Mitigate Volume Overload

Sodium Intake

In observational studies, sodium-restricted diets have been shown to reduce interdialytic weight gain, require fewer antihypertensive medications, and reduce left ventricular hypertrophy.[57–59] In a meta-analysis of randomized controlled trials with 91 patients on dialysis, either hemodialysis or peritoneal dialysis, dietary sodium restriction was associated with a reduction in SBP of 8.4 mm Hg and a 4.4-mm Hg reduction in diastolic BP.[60] In a crossover study involving 15 patients, dialysis conditions including dry weight were held constant, and sodium restriction counseling was provided. Daily sodium intake decreased from 4.2 g/d at baseline to 2.9 g/d following the intervention. SBP declined from 138.7 to 131.8 mm Hg with interdialytic weight gain decreasing from 2.26 to 1.87 kg.[61] As such, dietary counseling to dialysis patients should include limitation of dietary sodium.

Sodium in Dialysate

Sodium dialysate concentration is a dialysis parameter that can be modified by the nephrologist and is most often matched with the patient's predialysis serum sodium. Frequently, increased sodium dialysate concentrations have been used to reduce dialysis-associated symptoms and intradialytic hypotension.[62,63] In these situations, higher sodium dialysate results in net diffusive movement of sodium into the patient, resulting in higher interdialytic weight gain and increased postdialysis thirst.[54] The DISO trial (Dialysate Individualized Sodium) was a prospective interventional study designed to evaluate the impact of individualized dialysate sodium prescriptions on weight gain, BP, and intradialytic complications.[64] In this trial, 40 maintenance hemodialysis patients initially prescribed a fixed dialysate sodium concentration of 140 mEq/L for 12 sessions, followed by 12 sessions with dialysate sodium concentrations aligned with their serum sodium levels. Both predialysis and postdialysis SBPs were on average 4 mm Hg lower during the individualized phase compared with the fixed phase (P=0.008). Larger randomized clinical trials are warranted to further evaluate strategies for optimizing dialysate sodium prescriptions in maintenance hemodialysis patients.

TREATMENT OF HYPERTENSION IN PATIENTS TREATED WITH HEMODIALYSIS: ANTIHYPERTENSIVE MEDICATIONS AND PROCEDURES

Unfortunately, there are limited data to guide a specific antihypertensive medication approach in patients treated with hemodialysis. A meta-analysis of 8 randomized controlled trials involving dialysis patients found that antihypertensive treatment with medications was linked to a statistically significant reduction in cardiovascular events and all-cause mortality.[65] Although it was not possible to determine whether the observed benefits were due to BP reduction itself or to the effects of specific antihypertensive drug classes, the trials included a variety of medications: 3 used angiotensin receptor blockers, 2 used angiotensin-converting enzyme inhibitors, 2 used β-blockers, and 1 used a calcium channel blocker. There was also notable heterogeneity in participant selection across the studies, including differences in the inclusion of patients with preexisting cardiac conditions, which makes interpreting and comparing the results across these trials particularly challenging. In the following, we review the evidence of specific BP medication classes, highlighting unique considerations in hemodialysis patients such as dualizability. Table 2 summarizes key randomized controlled trials and the antihypertensive regimens evaluated, highlighting the limited evidence base and paucity of studies addressing antihypertensive therapies in maintenance dialysis populations.

Angiotensin-Converting Enzyme Inhibitor/ Angiotensin Receptor Blocker

Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are first-line BP agents in CKD to

Table 2. Summary of Antihypertensive Pharmacotherapies and Key Clinical Trials in Patients Treated With Dialysis

Study name/type, year	Medication(s)	N and population	Main findings/notes
ACE inhibitors/ARBs			
FOSIDIAL, 2006	Fosinopril vs placebo	397 dialysis patients with left ventricular hypertrophy	Underpowered; no clear CV benefit; many patients are normotensive at baseline.
RCT, 2006	Candesartan vs placebo	80 dialysis patients without cardiac disorders	Reduced CV events vs control, despite similar BP between arms
HDPAL trial, 2014	Lisinopril vs atenolol	200 dialysis patients	Atenolol showed greater CV benefit than lisinopril.
β-Blockers			
RCT, 2003	Carvedilol vs placebo	114 dialysis patients with dilated cardiomyopathy	Reduced CV risk and all-cause mortality with carvedilol in the population
Calcium channel blockers			
RCT, 2008	Amlodipine	251 dialysis patients with hypertension	Amlodipine showed a nonsignificant mortality reduction.
Mineralocorticoid receptor antagonists			
ALCHEMIST, 2025	Spironolactone vs placebo	644 dialysis patients	Spironolactone did not reduce major CV events. Trial follow-up stopped early due to lack of funding. High rates of hyperkalemia
ACHIEVE trial, 2025	Spironolactone 25 mg vs placebo	2538 dialysis patients	Stopped early for futility; no CV or HF benefit

ACE indicates angiotensin-converting enzyme; ACHIEVE, Aldosterone Blockade for Health Improvement Evaluation in End-Stage Renal Disease; ALCHEMIST, Aldosterone Antagonist Chronic Hemodialysis Interventional Survival Trial; ARB, angiotensin receptor blocker; BP, blood pressure; CV, cardiovascular; FOSIDIAL, Fosinopril in Dialysis; HF, heart failure; HDPAL, Hemodialysis Patients Randomized to Alternate-Day Dialysis Pilot Study; and RCT, randomized controlled trial.

reduce the rate of CKD progression and are well tolerated in patients on dialysis. In general, angiotensin-converting enzyme inhibitors are removed with dialysis, but angiotensin receptor blockers are not.

Despite strong data in other populations, the cardiovascular benefit of renin-angiotensin-aldosterone system inhibition has not been consistently observed in the dialysis population. The FOSIDIAL study (Fosinopril in Dialysis) enrolled 397 patients on dialysis with left ventricular hypertrophy but was underpowered due to a lower than anticipated event rate, with 40% of patients normotensive at baseline.[66] In a small randomized controlled trial, the risk of cardiovascular events was significantly reduced in individuals assigned to take candesartan despite similar follow-up BP between the trial arms.[67] Lee et al[68] conducted a large-scale population-based cohort study of dialysis patients and demonstrated a lower cardiovascular death and all-cause mortality than among the nonuser group. The HDPAL trial (Hemodialysis Patients Randomized to Alternate-Day Dialysis Pilot Study) of 200 dialysis patients with hypertension randomized to lisinopril versus atenolol suggested greater cardiovascular benefit with atenolol.[69]

β-Blockers

Patients on dialysis are at a high risk of arrhythmia and ischemic cardiovascular events, underscoring the potential benefit of the use of β-blockers for cardioprotective effects. In a retrospective cohort study of hemodialysis and peritoneal dialysis patients without a prior diagnosis of heart failure, β-blocker use was significantly associated with a reduced incidence of new-onset heart failure and cardiac death.[70] Cice et al[71] randomized hemodialysis patients with reduced left ventricular ejection fraction to carvedilol or placebo and demonstrated a reduction in cardiovascular risk and all-cause mortality. Metoprolol and atenolol have high dialyzability, while carvedilol and labetalol are not readily dialyzed off.

Calcium Channel Blockers

Calcium channel blockers are beneficial because they are long-acting and are not dialyzed off. There are some data supporting their use for cardiovascular risk reduction, with amlodipine showing a nonsignificant reduction in mortality, but the numbers are small.[72]

Mineralocorticoid Receptor Antagonist

Mineralocorticoid receptor antagonists, such as eplerenone and spironolactone, are cornerstone therapy in resistant hypertension and heart failure, but their use in hemodialysis patients has been debated, with concern for the risk of hyperkalemia. Previously, a meta-analysis that included 1630 patients across 16 randomized controlled trials showed a significant improvement in the left ventricular ejection fraction, left ventricular mass index, and SBP in hemodialysis patients and improved the long-term survival rate without significantly increasing the serum potassium level.[73] However, ALCHEMIST (Aldosterone Antagonist Chronic Hemodialysis Interventional Survival Trial), a large placebo-controlled study, demonstrated that spironolactone did not reduce the incidence of major cardiovascular events.[74] The updated meta-analysis shows that mineralocorticoid receptor antagonist did not reduce all-cause or cardiovascular mortality. Another recent placebo-controlled study, the ACHIEVE study (Aldosterone Blockade for Health

Improvement Evaluation in End-Stage Renal Disease), was stopped early due to futility because spironolactone did not reduce cardiovascular mortality and hospitalization due to heart failure compared with placebo.[75] An important limitation of the study is that spironolactone 25 mg daily was the only dose studied. Given these data, the use of a mineralocorticoid receptor antagonist in patients on dialysis is not supported for cardiac benefit.

Aldosterone Synthase Inhibitor

Aldosterone excess contributes to hypertension through salt-retention, with growing evidence that aldosterone has harmful effects on the cardiorenal system through promoting inflammation, fibrosis, and vascular stiffness and remodeling.[76,77] Novel aldosterone synthase inhibitors show promise in BP reduction in resistant hypertension, but these trials do not include patients with advanced CKD or on dialysis.[78–82] In addition, to date, the trial end points do not include cardiovascular events and mortality as the primary outcome. As of now, there are no recruiting studies evaluating aldosterone synthase inhibitors among patients on dialysis.

SGLT2 Inhibitor

While SGLT2 (Sodium–Glucose Cotransporter 2) inhibitors are approved by the Food and Drug Administration in advanced CKD regardless of diabetes status, the safety and efficacy of these medications in end-stage kidney disease (ESKD) have not been established although several trials are ongoing. Preclinical and clinical data have shown promising results for use of SGLT2 inhibitors in the dialysis population regardless of diabetes status due to their pleiotropic effects.[83,84] Benefits include direct cardiovascular effects, preserving remaining kidney function, alleviation of anemia, and improving nutritional status and potentially blood sugars in patients with diabetes.[84,85] A small number of patients in the DAPA-CKD study (Dapagliflozin and Prevention of Adverse Outcomes in Chronic Kidney Disease) continued dapagliflozin or placebo after estimated glomerular filtration rate <15 mL/min, and no significant differences in adverse events between the 2 groups were seen.[86] Although end point findings should be interpreted with caution, the dapagliflozin group had fewer deaths compared with the placebo group. Several ongoing clinical trials evaluating the effects of SGLT2 inhibitors in the dialysis population are registered in ClinicalTrials.gov, including RENAL LIFECYCLE (unique identifier: NCT05374291), DAPA-HD (Dapagliflozin in Hemodialysis [investigational/early-phase work studying SGLT2 inhibitors in HD patients]; unique identifier: NCT05179668), and the DARE-ESKD-2 trial (Dapagliflozin in Renal Endpoints in End-Stage Kidney Disease – Phase 2 [trial within the DARE-ESKD program]; unique identifier: NCT05685394). RENAL LIFECYCLE is a large, randomized, placebo-controlled clinical trial currently enrolling patients with advanced CKD, on dialysis, and kidney transplant (estimated enrollment n=1500), evaluating dapagliflozin and placebo.

Neprilysin Inhibition

Sacubitril/valsartan has demonstrated benefits in improving cardiovascular outcomes among patients with heart failure and in slowing the progression of CKD.[87] A meta-analysis of 19 studies including 1597 patients with both hypertension and ESKD on dialysis found that treatment with sacubitril/valsartan was associated with significant reductions in BP, with a mean decrease of −11.09 mm Hg in SBP (95% CI, −14.51 to −7.66). Compared with conventional therapy, sacubitril/valsartan also lowered the risk of cardiovascular hospitalization (risk ratio, 0.63 [95% CI, 0.44–0.90]).[88] Although not statistically significant, there was a trend toward reduction in all-cause mortality.[88,89]

Incretin Therapy

GLP-1 (Glucagon-Like Peptide-1) RAs (GLP-1 receptor agonists) have been shown in the general population and in patients with early stages of CKD to modestly reduce BP.[90,91] However, evidence supporting a direct role for GLP-1 RAs in BP control specifically among patients with ESKD on dialysis remains limited. A meta-analysis with 8 studies (5 trials and 3 cohort studies) consisting of 27 639 patients evaluated the safety and efficacy of GLP-1 RA in patients with type 2 diabetes and advanced CKD (stage 5 or ESKD). While a reduction in mean blood glucose and weight loss was observed, no difference was observed in 1-year mortality, and there was no decrease in SBP.[92] BP reduction with GLP-1 RAs is thought to be mediated primarily through weight loss, but also other pathways such as natriuresis, and reductions in sympathetic activity.[93,94]

It should be noted that numerous observational studies have demonstrated a paradoxical association between higher BMI and improved survival in patients on dialysis.[95] The reasons for this paradox are unclear, but possible contributing factors include the possible protective effects of better nutrition or increased muscle mass. This phenomenon may complicate the interpretation of intentional weight loss as a therapeutic strategy in this population, particularly when weight reduction may include loss of lean body mass. Therefore, clinical trials are needed to test the efficacy of GLP-1 RA on important outcomes including BP and mortality in patients treated with dialysis.

Renal Denervation

Renal sympathetic efferent and afferent nerves may play a role in the pathophysiology of hypertension in

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patients on dialysis because, even in patients who are anuric, the kidneys may remain sympathetically active. Renal denervation has been evaluated in several pilot-sized intervention studies in dialysis patients with resistant hypertension. Five case studies have demonstrated that the procedure is feasible and possibly effective in patients with ESRD, even in patients with narrow renal artery lumen and atrophic kidneys.[96–100] Although renal denervation has been studied in select patients with ESRD, large multicenter trials assessing its effects in broader ESRD populations have not yet been conducted.

FUTURE DIRECTIONS AND CONCLUSIONS

There is an urgency to mitigate the disproportionate and premature risk of CVD and death in patients with kidney failure treated with hemodialysis. Hypertension is highly prevalent among patients receiving dialysis and is a strong and modifiable risk factor for CVD and death. Yet, hypertension remains challenging to manage due to its multifactorial pathophysiology and the unique hemodynamic conditions of this population. There is a critical need for more research to guide BP management in this vulnerable population. Greater understanding of the unique mechanistic insults that drive hypertension may help identify novel therapeutic targets. Observational evidence increasingly supports the value of home BP monitoring over predialysis measurements, but randomized controlled trials are needed to establish key questions around timing and setting of BP measures, as well as BP targets in this population. Optimal BP control requires a multifaceted approach with volume management as the cornerstone of BP treatment and greater study of novel methods of volume status monitoring are needed. In addition, most studies of hypertension treatment have excluded patients treated with dialysis. Well-designed clinical trials are required to evaluate the effectiveness and safety of interventions such as lifestyle modifications, pharmacological therapies, and interventions to improve cardiovascular outcomes. These trials should be designed not only to assess efficacy and safety but also to generate the evidence needed to establish dialysis-specific BP management guidelines in this population. Given the profound impact of BP on outcomes in dialysis patients, accelerating research in this field is not just important; it is imperative to save lives and transform care.

ARTICLE INFORMATION

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