



REAL-WORLD IMPLEMENTATION OF KDIGO BLOOD-PRESSURE TARGETS AND CARDIORENAL PHARMACOTHERAPY ACROSS CHRONIC KIDNEY DISEASE STAGES: A PROSPECTIVE CROSS-SECTIONAL AUDIT

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ABSTRACT

Aim: To describe blood-pressure target attainment, in-hospital cardioresnal prescribing, and recorded cardiovascular conditions across chronic kidney disease stages, including dialysis.

Methods: This single-centre, prospective, cross-sectional observational study included 130 adults with chronic kidney disease and at least one prespecified cardiovascular condition, admitted between January and July 2025 (88 not on dialysis; 42 on dialysis). Final systolic blood pressure below 120 mmHg (the Kidney Disease: Improving Global Outcomes target for people not on dialysis) and below 140 mmHg was compared by dialysis status and disease stage. Cross-sectional associations with recorded cardiovascular conditions were explored using Firth penalised-likelihood logistic regression.

Results: Final systolic pressure was below 120 mmHg in 33.1% of patients (non-dialysis 37.5% versus dialysis 23.8%; difference -13.7 percentage points, 95% confidence interval -28.4 to +3.8) and below 140 mmHg in 72.3% (80.7% versus 54.8%; adjusted odds ratio 0.32, 95% confidence interval 0.14-0.71). No patient received an angiotensin-converting enzyme inhibitor together with an angiotensin receptor blocker (0/130; 95% confidence interval 0.0-2.8%), and no sodium-glucose cotransporter-2 inhibitor was prescribed. Left ventricular hypertrophy was associated with arrhythmias. Associations of diabetes or smoking with myocardial infarction were imprecise, and smoking and peripheral artery disease showed complete separation. Dyslipidemia was inversely associated with composite cardiovascular events, which should not be read as protective.

Conclusion: Systolic pressure below 140 mmHg was recorded less often among dialysis patients, and neither dual renin-angiotensin-aldosterone system blockade nor inpatient sodium-glucose cotransporter-2 inhibitor use was observed. Findings are cross-sectional and hypothesis-generating.

KEYWORDS: Chronic Kidney Disease, Blood Pressure, Dialysis, Cardiovascular Disease, Cross-Sectional Study.

Introduction

Chronic kidney disease (CKD) represents a significant worldwide health concern and is linked to increased cardiovascular incidents, hospital admissions, and early death[1,2]. This heightened risk illustrates the cardiorenal syndrome, wherein traditional cardiovascular risk factors merge with CKD-related mechanisms like volume overload, neuro-hormonal activation, and endothelial dysfunction[3-5]. Reduced kidney function is also independently associated with vascular calcification, oxidative stress, and adverse cardiac remodeling, compounding cardiovascular risk beyond traditional factors alone[6-8].

Blood-pressure management is central to cardiovascular risk reduction in CKD[9]. The Kidney Disease: Improving Global Outcomes (KDIGO) guideline recommends a standardized office systolic blood pressure (SBP) target of <120 mmHg, when tolerated, in adults with high blood pressure (BP) and CKD who are not receiving dialysis; the recommendation does not extend to people on dialysis or with a kidney transplant[10,11]. This approach was inspired by the Systolic Blood Pressure Intervention Trial (SPRINT) [12,13], and has since been supported by the ESPRIT trial, which compared SBP targets of <120 and <140 mmHg in high-risk hypertensive adults[14]. Attaining rigorous blood pressure regulation entails a significant treatment burden, and intensive-control studies in chronic kidney disease cohorts have demonstrated inconsistent impacts on mortality and disease advancement[15,16]. Dialysis patients exhibit added complexity: home and interdialytic blood pressure patterns significantly diverge from office measurements and independently forecast outcomes[17,18]. Consensus guidance likewise highlights the practical difficulty of applying standard BP targets to dialysis populations given this haemodynamic variability[19-21]. Randomised evidence on dialysis-specific targets is limited to pilot trials[22].

Two further considerations frame this study. First, kidney-protective prescribing now extends beyond RAAS blockade: the KDIGO 2024 CKD guideline recommends sodium-glucose cotransporter-2 (SGLT2) inhibitors for adults with CKD and an eGFR of at least 20 mL/min/1.73 m² who have substantial albuminuria or heart failure, on the basis of the DAPA-CKD and EMPA-KIDNEY trials, but its scope excludes dialysis patients[11,23,24]. Second, the Indian setting differs from trial populations: reported CKD prevalence ranges from under 1% to 17% across surveys[25-27], and registry data indicate that many patients present late, with nearly half of those with a recorded stage already at stage 5[28].

Two practice-oriented inquiries remain inadequately addressed by trial data and are pertinent to standard CKD management. Initially, the inhibition of the renin-angiotensin-aldosterone system (RAAS) is a crucial approach in specific chronic kidney disease (CKD) cohorts; however, the simultaneous use of angiotensin-converting enzyme

(ACE) inhibitors and angiotensin receptor blockers (ARBs) is typically discouraged due to significant trials revealing heightened renal and hyperkalemic complications without adequate supplementary clinical advantages[29,30]. The implementation of this safety lesson in daily prescribing practices is not regularly assessed. Secondly, individuals undergoing dialysis exhibit unique hemodynamic difficulties in managing blood pressure, and the extent to which actual blood pressure achievement varies significantly between dialysis and non-dialysis chronic kidney disease at both stringent and practical thresholds remains inadequately defined beyond clinical trial settings. Observational medication associations more broadly require caution because additional agents may be preferentially prescribed to patients with more difficult-to-control BP[31].

This study addresses these gaps in a single-centre inpatient CKD population. It describes the use of RAAS monotherapy and the absence of dual blockade during admission, and it measures the difference in SBP-target attainment between dialysis and non-dialysis chronic kidney disease at both the KDIGO threshold and a conventional threshold, with effect sizes and adjusted estimates. It additionally reports pharmacotherapy patterns and recorded cardiovascular conditions stage by stage across CKD stages 1 to 5, and employs predetermined Firth logistic-regression models with profile-likelihood confidence intervals to explore cross-sectional associations of recorded cardiovascular conditions amid restricted events and scant data[32-34].

Materials and Methods

Design and Participants

This was a single-centre, prospective, cross-sectional observational study of 130 unique adult inpatients with CKD and complete data for the primary variables analysed. Clinical information was collected prospectively during each admission from contemporaneous clinical records, with one observation per patient and no post-discharge follow-up. Accordingly, cardiovascular conditions were analysed as prevalent recorded conditions rather than incident outcomes, and no incidence or time-to-event analysis was performed. Data included demographics, CKD stage, dialysis status, cardiovascular risk factors, blood-pressure readings, inpatient medication records, and recorded cardiovascular conditions. The manuscript was prepared in accordance with the STROBE statement for cross-sectional studies[35].

Setting, Period, and Eligibility

The study was conducted at a tertiary care teaching hospital (Srinivas Institute of Medical Sciences and Research Centre, Mangaluru, Karnataka, India) and included patients admitted between 5 January and 30 July 2025 (median length of stay 4 days, interquartile range 3–6, range 1–19). Inclusion criteria were age ≥18 years, a documented diagnosis of CKD stage 1–5 (including maintenance dialysis), and at least one of the prespecified cardiovascular conditions: ischaemic heart

disease, myocardial infarction, heart failure, stroke/transient ischaemic attack, arrhythmia, or peripheral artery disease.

Ethics

The study was approved by the Institutional Ethics Committee of Srinivas Institute of Medical Sciences and Research Centre (reference number SIEC/SIMSRC/PG06/02/2025; approval date: 04 January 2025) and was conducted in accordance with the Declaration of Helsinki. Written informed consent for the use of clinical data in the study was obtained prospectively from each patient or the appropriate consenting representative, as applicable. Identifiers were removed from the analysis dataset, and clinical information was handled confidentially.

Variables and Measures

Final BP was the last inpatient BP recorded in the daily-vitals chart (median 4 readings per patient, range 1 to 7). Because the KDIGO target is expressed as a systolic pressure, the primary targets were final SBP <120 mmHg (the KDIGO 2021 target for adults with CKD not on dialysis, reported for dialysis patients for descriptive comparison only) and final SBP <140 mmHg (a conventional, historical threshold). Stricter definitions requiring both systolic and diastolic values (<120/80 and <140/90 mmHg) were examined as sensitivity analyses. Blood pressure was measured with a traditional mercury sphygmomanometer. Exact values were not always discernible in routine practice, so readings were recorded to the nearest discernible value; posture and timing relative to dialysis were not recorded. CKD stage (1, 2, 3a, 3b, 4, or 5) was taken from the pre-established diagnosis documented in the patient file, not from creatinine measured during the admission. eGFR was recalculated with the CKD-EPI 2021 creatinine equation from the first and last inpatient serum creatinine, age, and sex; because admission creatinine reflects acute illness and may include acute-on-chronic injury, the eGFR-derived stage was used only as a sensitivity measure. Dialysis status denoted maintenance dialysis (thrice weekly, n=30; twice weekly, n=12), and all dialysis patients were in stage 5. Recorded cardiovascular conditions were myocardial infarction (MI), heart failure, stroke/transient ischemic attack (TIA), arrhythmias, ischaemic heart disease (IHD), peripheral artery disease (PAD), and composite major adverse cardiovascular events (MACE), defined as the recording of MI, heart failure, or stroke/TIA. Because at least one of the six prespecified cardiovascular conditions was an inclusion criterion, every included patient had at least one such condition documented. Left ventricular hypertrophy (LVH) was coded when the echocardiography report mentioned it. Medication markers were derived from inpatient prescription records, which cover the admission only, and included ACE-inhibitor and ARB use, RAAS monotherapy (ACE inhibitor or ARB, not both), dual ACE-inhibitor-ARB exposure, calcium-channel blocker (CCB), beta-blocker (including alpha-beta blockers), loop diuretic, mineralocorticoid-receptor antagonist

(spironolactone), statin, antiplatelet agent, and the number of antihypertensive classes (ACE inhibitor, ARB, beta-blocker, CCB, diuretic, and alpha-adrenergic agents (agonists or blockers such as prazosin); diuretics given for volume control were counted, and noradrenaline was excluded as a vasopressor rather than an antihypertensive). The highest serum potassium during admission was used to identify hyperkalaemia (>5.5 mmol/L).

Statistical Analysis

Continuous variables are presented as mean $\pm$ standard deviation and categorical variables as n (%). Welch t-tests and Pearson chi-square tests were used as appropriate, with the Fisher exact test where expected counts were small. Differences between dialysis and non-dialysis patients are given in percentage points with Newcombe hybrid-score 95% confidence intervals. Stage-wise comparisons used permutation chi-square tests (10,000 permutations) or one-way analysis of variance across the six recorded stages, with a linear-by-linear trend test reported descriptively. Holm-Bonferroni-adjusted P values are reported alongside raw P values to control the family-wise error rate within the comparison family stated in each table footnote; findings significant only at the raw level are interpreted as hypothesis-generating. Target attainment in dialysis versus non-dialysis patients was additionally modelled with Firth logistic regression adjusted for age, sex, diabetes, and LVH, and with the number of antihypertensive classes added; recorded stage was not adjusted for because dialysis occurred only in stage 5. Firth penalized-likelihood logistic regression was also used for six prespecified models of recorded cardiovascular conditions because several conditions had limited event counts (19 to 65 events) and one model (PAD with smoking) showed complete separation. Firth regression reduces small-sample bias, and 95% confidence intervals were obtained by profile penalized likelihood rather than the Wald method, which is more robust for sparse-data models[32-34]; P values are penalized likelihood-ratio P values, consistent with the profile intervals. Models were adjusted as follows: heart failure (hypertension, LVH, age, sex, dialysis); MI (diabetes, smoking, age, sex, dyslipidemia); stroke/TIA (LVH, diabetes, age, sex); arrhythmias (LVH, age, sex, dialysis); PAD (smoking, diabetes, age, sex); and composite MACE (diabetes, dyslipidemia, age, sex, smoking, LVH, dialysis). Age was entered per 10-year increment. Because all variables were recorded at one time point, odds ratios describe cross-sectional associations of prevalent conditions and imply no temporal order; reverse causation is possible. Model discrimination was summarized with the in-sample (apparent) concordance (C) statistic, which is descriptive only. Two-sided P < 0.05 was considered statistically significant. All analyses were performed in Python 3.12 (NumPy, SciPy, and pandas) using a custom Firth-regression implementation that maximizes the penalized log-likelihood numerically. The implementation was validated by confirming that the

modified-score equations were satisfied at each solution (maximum absolute penalized score $<2 \times 10^{-9}$) and by an independent modified-score (Heinze–Schemper) implementation, which reproduced all coefficients, profile-likelihood limits, and likelihood-ratio P values.

Results and Discussion

Figure 1 summarises the principal blood-pressure findings and in-hospital pharmacotherapy patterns in this sample of 130 patients with CKD.

Mean age was 61.6 ± 12.4 years, and roughly two-thirds of participants were male, with a somewhat higher proportion of men among dialysis patients than non-dialysis patients. Approximately one in three participants were maintained on dialysis. CKD stage 5 accounted for the largest single group, followed in descending order by stages 3a, 3b, 4, 1, and 2. Hypertension was near-universal across the sample, and diabetes mellitus, dyslipidemia, smoking history, and left ventricular hypertrophy (LVH) were all common findings

(Table 1). Dyslipidemia was more prevalent among dialysis patients than non-dialysis patients at the raw significance level ($p=0.011$), though this association did not remain significant after Holm correction for the eight comparisons tested (Holm-adjusted $p=0.091$); no other baseline characteristic differed significantly by dialysis status.

Because stage was taken from the pre-established diagnosis in the patient file, it need not agree with an eGFR calculated from admission creatinine, which reflects the patient’s acute condition. Agreement between the two was 53 of 130 (40.8%), and 22 of the 23 patients with a documented stage of 1 or 2 had an eGFR below $60 \text{ mL/min/1.73 m}^2$ on both the first and last inpatient creatinine, which is consistent with acute-on-chronic injury during the admission. The median first-admission eGFR was $16.5 \text{ mL/min/1.73 m}^2$ (interquartile range 8.3–30.6). The documented stage was therefore retained as the primary stage variable, and the eGFR-derived stage is presented as a sensitivity analysis (Supplementary Table S1).

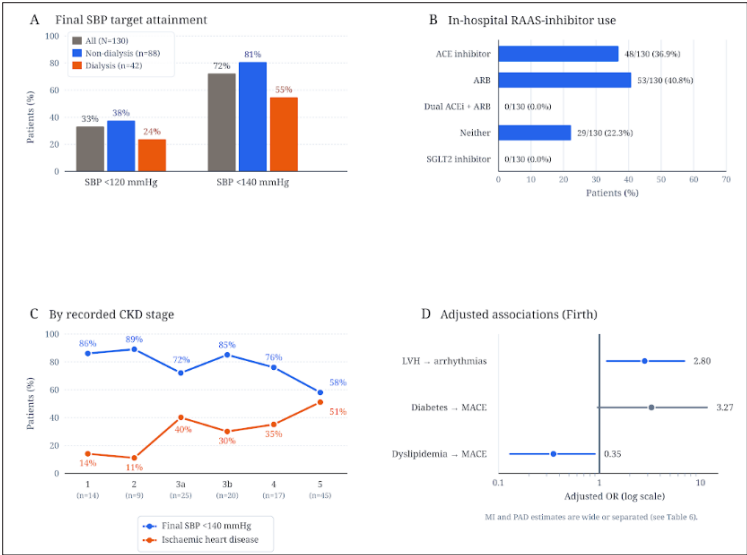


Figure 1. Graphical summary of key findings: final SBP target attainment (<120 mmHg overall; <140 mmHg by dialysis status), in-hospital RAAS-prescribing pattern with zero dual ACE-inhibitor–ARB exposure and no SGLT2-inhibitor use, stage-wise trends, and three cross-sectional Firth-adjusted associations (LVH with arrhythmias, diabetes with composite MACE, dyslipidemia with composite MACE). The wide or separated MI and PAD estimates are not displayed

Table 1: Baseline characteristics by dialysis status (N=130)

Characteristic	Overall (N=130)	Non-dialysis (n=88)	Dialysis (n=42)	P value	Holm-adjust-ed P
Age, years, mean±SD	61.6±12.4	62.7±11.8	59.3±13.3	0.172	1.000
Male sex, n (%)	83 (63.8)	54 (61.4)	29 (69.0)	0.394	1.000
Hypertension, n (%)	118 (90.8)	79 (89.8)	39 (92.9)	0.570	1.000
Diabetes, n (%)	69 (53.1)	47 (53.4)	22 (52.4)	0.913	1.000
Dyslipidemia, n (%)	104 (80.0)	65 (73.9)	39 (92.9)	0.011*	0.091
Smoking history, n (%)	60 (46.2)	41 (46.6)	19 (45.2)	0.885	1.000
Family history of CVD, n (%)	21 (16.2)	14 (15.9)	7 (16.7)	0.913	1.000
LVH, n (%)	51 (39.2)	37 (42.0)	14 (33.3)	0.341	1.000

CVD, cardiovascular disease; LVH, left ventricular hypertrophy; SD, standard deviation. *Raw $P<0.05$; Holm-adjusted P values control the family-wise error rate across the eight comparisons in this table.

Final SBP was below 120 mmHg, the KDIGO target for adults with CKD who are not on dialysis, in 43 of 130 patients (33.1%). Attainment was numerically lower among dialysis patients (23.8%) than non-dialysis patients (37.5%; difference -13.7 percentage points, 95% CI -28.4 to +3.8), but the difference did not reach statistical significance ($p=0.121$, Holm-adjusted $p=0.242$), and because KDIGO does not apply this target to dialysis, the comparison is descriptive. Final SBP was below 140 mmHg in 94 patients (72.3%) and was significantly less frequent among dialysis patients (54.8%) than non-dialysis patients (80.7%; difference -25.9 percentage points, 95% CI -42.3 to -9.0; $p=0.002$, Holm-adjusted $p=0.006$) (Table 2, Figure 2). Mean final SBP was 136.4 ± 21.1 mmHg in dialysis and

124.6 ± 12.8 mmHg in non-dialysis patients (difference 11.9 mmHg, 95% CI 4.8 to 18.9). The difference at the 140 mmHg threshold persisted after adjustment for age, sex, diabetes, and LVH (adjusted OR 0.32, 95% CI 0.14–0.71; $P=0.005$) and after further adjustment for the number of antihypertensive classes (OR 0.31, 95% CI 0.14–0.70; $P=0.005$), whereas the difference at the 120 mmHg threshold remained non-significant (adjusted OR 0.60, 95% CI 0.25–1.37; $P=0.231$) (Table 3). With stricter definitions requiring both systolic and diastolic values, 32.3% reached <120/80 mmHg and 63.8% reached <140/90 mmHg; for <140/90 mmHg the dialysis difference was 50.0% versus 70.5% (adjusted OR 0.47, 95% CI 0.22–1.02; $P=0.056$).

Table 2: Final SBP target attainment and in-hospital pharmacotherapy markers, by dialysis status

Variable	Overall (N=130)	Non-dialysis (n=88)	Dialysis (n=42)	Difference (95% CI)	P value	Holm-adjusted P
A. Primary comparisons						
Final SBP <120 mmHg†, n (%)	43 (33.1)	33 (37.5)	10 (23.8)	-13.7 (-28.4 to +3.8)	0.121	0.242
Final SBP <140 mmHg, n (%)	94 (72.3)	71 (80.7)	23 (54.8)	-25.9 (-42.3 to -9.0)	0.002	0.006*
RAAS monotherapy (ACEi or ARB, not both), n (%)	101 (77.7)	69 (78.4)	32 (76.2)	-2.2 (-18.7 to +11.9)	0.776	0.776
B. Sensitivity definitions and additional markers (exploratory)						
Final BP <120/80 mmHg (SBP and DBP), n (%)	42 (32.3)	32 (36.4)	10 (23.8)	-12.6 (-27.2 to +4.8)	0.152	1.000
Final BP <140/90 mmHg (SBP and DBP), n (%)	83 (63.8)	62 (70.5)	21 (50.0)	-20.5 (-37.2 to -2.7)	0.023	0.209
Final SBP, mmHg, mean±SD	128.4±16.8	124.6±12.8	136.4±21.1	+11.9 (+4.8 to +18.9)	0.001	0.016*
ACE inhibitor, n (%)	48 (36.9)	35 (39.8)	13 (31.0)	-8.8 (-24.6 to +9.0)	0.330	1.000
ARB, n (%)	53 (40.8)	34 (38.6)	19 (45.2)	+6.6 (-10.9 to +24.2)	0.474	1.000
Dual ACEi + ARB, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	—	—	—
Calcium-channel blocker, n (%)	40 (30.8)	20 (22.7)	20 (47.6)	+24.9 (+7.6 to +41.4)	0.004	0.040*
Beta-blocker (incl. alpha-beta), n (%)	103 (79.2)	70 (79.5)	33 (78.6)	-1.0 (-17.1 to +12.7)	0.898	1.000
Loop diuretic, n (%)	87 (66.9)	67 (76.1)	20 (47.6)	-28.5 (-44.7 to -10.8)	0.001	0.015*
MRA (spironolactone), n (%)	2 (1.5)	1 (1.1)	1 (2.4)	+1.2 (-4.1 to +11.2)	0.543	1.000
SGLT2 inhibitor, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	—	—	—
Statin, n (%)	67 (51.5)	45 (51.1)	22 (52.4)	+1.2 (-16.6 to +18.8)	0.894	1.000
Antiplatelet agent, n (%)	66 (50.8)	48 (54.5)	18 (42.9)	-11.7 (-28.7 to +6.5)	0.213	1.000
Antihypertensive classes, mean±SD	2.63±1.01	2.64±0.98	2.62±1.08	-0.02 (-0.41 to +0.38)	0.930	1.000

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BP, blood pressure; CI, confidence interval; DBP, diastolic blood pressure; MRA, mineralocorticoid-receptor antagonist; RAAS, renin-angiotensin-aldosterone system; SBP, systolic blood pressure; SGLT2, sodium-glucose cotransporter-2. Differences are dialysis minus non-dialysis; percentage points with Newcombe hybrid-score 95% CI for proportions, and mean difference with Welch 95% CI for continuous measures. P values are Pearson chi-square or Welch t-test (Fisher exact test for MRA). Holm-adjusted P values are computed within Panel A (three primary comparisons) and within Panel B (twelve exploratory comparisons). *Raw and Holm-adjusted $P<0.05$. †KDIGO's SBP <120 mmHg target applies to adults not on dialysis; values in dialysis patients are descriptive. Medication data are in-hospital prescriptions only.

Table 3: Association of dialysis with final BP-target attainment: Firth penalized logistic regression (odds ratio <1 indicates lower odds of reaching target on dialysis)

Outcome	Model	OR (95% CI) for dialysis	P value
Final SBP <140 mmHg (94 events)	Crude	0.29 (0.13–0.65)	0.002
Final SBP <140 mmHg (94 events)	Adjusted for age, sex, diabetes, LVH	0.32 (0.14–0.71)	0.005
Final SBP <140 mmHg (94 events)	Adjusted + number of antihypertensive classes	0.31 (0.14–0.70)	0.005
Final SBP <120 mmHg (all patients; descriptive†) (43 events)	Crude	0.54 (0.23–1.19)	0.125
Final SBP <120 mmHg (all patients; descriptive†) (43 events)	Adjusted for age, sex, diabetes, LVH	0.60 (0.25–1.37)	0.231
Final SBP <120 mmHg (all patients; descriptive†) (43 events)	Adjusted + number of antihypertensive classes	0.60 (0.25–1.37)	0.233
Final BP <140/90 mmHg (sensitivity) (83 events)	Crude	0.42 (0.20–0.90)	0.025
Final BP <140/90 mmHg (sensitivity) (83 events)	Adjusted for age, sex, diabetes, LVH	0.47 (0.22–1.02)	0.056
Final BP <140/90 mmHg (sensitivity) (83 events)	Adjusted + number of antihypertensive classes	0.47 (0.22–1.01)	0.053

CI, confidence interval; LVH, left ventricular hypertrophy; OR, odds ratio. Profile-likelihood 95% CI and likelihood-ratio P values; N=130. Recorded CKD stage was not adjusted for because dialysis occurred only in stage 5. †KDIGO's SBP <120 mmHg target applies to adults not on dialysis.

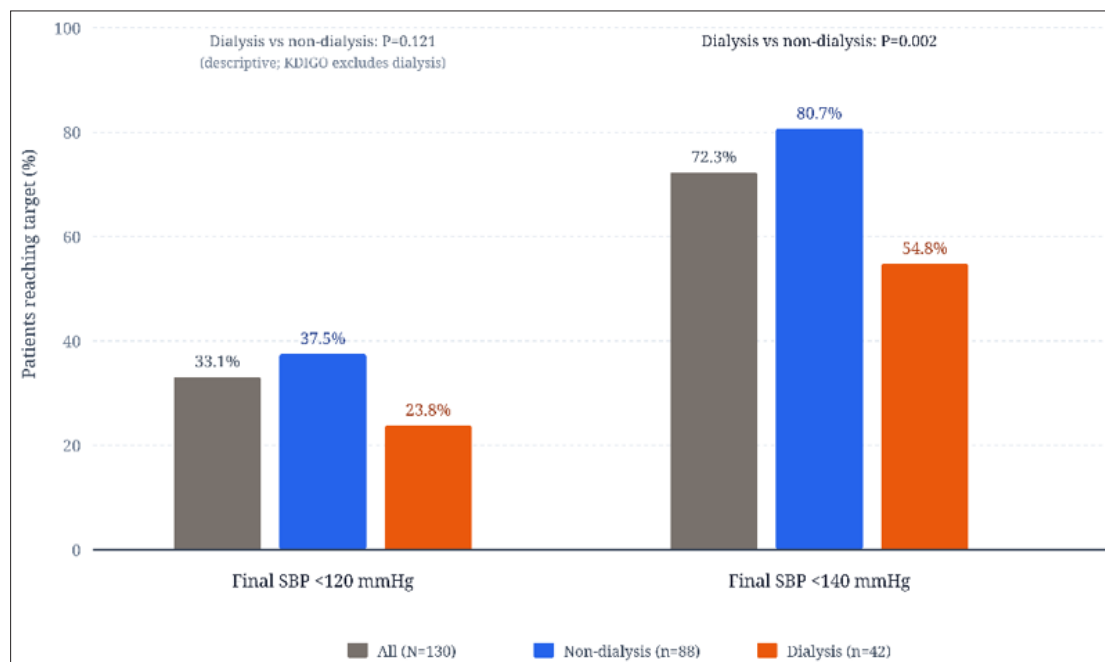


Figure 2. Final systolic blood-pressure target attainment among patients with and without dialysis. Final SBP <120 mmHg was reached by 33.1% overall, 37.5% of non-dialysis patients, and 23.8% of dialysis patients (P=0.121). Final SBP <140 mmHg was reached by 72.3% overall and was significantly less frequent in dialysis than non-dialysis CKD (54.8% vs 80.7%; P=0.002)

RAAS monotherapy was recorded in 101 of 130 patients (77.7%; 95% CI 69.8–84.0%), with ACE inhibitors in 36.9% and ARBs in 40.8%. No patient had documented concurrent ACE-inhibitor and ARB exposure during admission (0/130; exact 95% CI 0.0–2.8%; 0/101 among patients on any RAAS agent, 0.0–3.6%). This is a descriptive in-hospital count without a comparator group; it is compatible with, but cannot establish, adherence to the established safety evidence against dual RAAS blockade in outpatient practice[29,30]. Hyperkalaemia (serum potassium >5.5 mmol/L) was recorded in 14 of 100 patients on a RAAS agent (14.0%) and 4 of 29 patients not on one (13.8%; Fisher exact $P=1.00$). Beyond RAAS blockade, CCBs were prescribed to 30.8% of patients (47.6% of dialysis versus 22.7% of non-dialysis patients, $p=0.004$), loop diuretics to 66.9% (47.6% versus 76.1%, $p=0.001$), statins to 51.5%, antiplatelet agents to 50.8%, spironolactone to 1.5%, and SGLT2 inhibitors to none (Table 2). CCB use was not associated with attainment of SBP <120 mmHg (30.0% versus 34.4%; $p=0.62$), although CCB-treated patients were, on average, receiving a significantly greater total number of antihypertensive classes (3.5 versus 2.3; $p<0.001$). Because CCB is itself one of the counted classes, this difference is partly arithmetic (excluding CCB, 2.5 versus 2.3 classes; $p=0.13$); to the extent that it persists, it is consistent with confounding by indication, whereby additional agents tend to be added preferentially in patients whose blood pressure has proved more difficult to control[31].

Stage-wise findings are shown in Tables 4 and 5. Final

SBP <140 mmHg was recorded in 85.7% of stage 1 and 57.8% of stage 5 patients (linear trend $P=0.016$, unadjusted; across-stage $P=0.109$), whereas attainment of SBP <120 mmHg ranged from 22.2% to 50.0% without a clear gradient ($P=0.620$). RAAS monotherapy was similar across stages (70.6% to 88.9%; $P=0.929$) and dual blockade was absent at every stage. CCB use was most frequent in stages 5 (44.4%) and 4 (41.2%) ($P=0.024$; Holm-adjusted $P=0.612$), loop-diuretic use was lowest in stage 5 (51.1%) and next-lowest in stage 3b (55.0%) ($P=0.017$; Holm-adjusted $P=0.455$), no SGLT2-inhibitor use was recorded at any stage, and statin use ranged from 28.6% to 68.0%. Dyslipidemia became more common at higher stages (42.9% in stage 1 to 93.3% in stage 5; $P=0.002$; Holm-adjusted $P=0.062$). Results by the eGFR-derived stage were directionally similar (Supplementary Table S1).

Composite MACE were the most frequently recorded cardiovascular condition (65 patients, 50.0%), followed by IHD (36.9%), heart failure (25.4%), arrhythmias (20.0%), stroke or TIA (18.5%), MI (16.9%), and PAD (14.6%) (Table 5, Figure 3). Every patient had at least one condition recorded (99 patients had one, 22 had two, 7 had three, and 2 had four). None of these conditions differed significantly across CKD stage once corrected for multiple testing (Holm-adjusted $P=1.000$ for each), although IHD was recorded in 14.3% of stage 1 and 51.1% of stage 5 patients (across-stage $P=0.075$; linear trend $P=0.006$, unadjusted) and MI in 7.1% and 24.4% (linear trend $P=0.037$, unadjusted), gradients that should be regarded as hypothesis-generating.

Table 4: Baseline characteristics, final SBP target attainment, and in-hospital pharmacotherapy by recorded CKD stage

Characteristic	Stage 1	Stage 2	Stage 3a	Stage 3b	Stage 4	Stage 5	Overall	P across stages	P trend	Holm-adjusted P
Baseline characteristics										
Patients, n	14	9	25	20	17	45	130	—	—	—
Age, years, mean±SD	63.1±10.8	56.9±16.5	63.6±13.8	64.5±10.9	61.3±8.8	59.8±13.0	61.6±12.4	0.522	—	1.000
Male sex, n (%)	5 (35.7)	6 (66.7)	15 (60.0)	14 (70.0)	12 (70.6)	31 (68.9)	83 (63.8)	0.302	0.056	1.000
On dialysis, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	42 (93.3)	42 (32.3)	<0.001	<0.001	0.003
Hypertension, n (%)	12 (85.7)	7 (77.8)	22 (88.0)	20 (100.0)	15 (88.2)	42 (93.3)	118 (90.8)	0.424	0.216	1.000
Diabetes, n (%)	5 (35.7)	4 (44.4)	11 (44.0)	11 (55.0)	13 (76.5)	25 (55.6)	69 (53.1)	0.248	0.076	1.000
Dyslipidemia, n (%)	6 (42.9)	8 (88.9)	20 (80.0)	15 (75.0)	13 (76.5)	42 (93.3)	104 (80.0)	0.002	<0.001	0.062
Smoking history, n (%)	7 (50.0)	2 (22.2)	11 (44.0)	9 (45.0)	9 (52.9)	22 (48.9)	60 (46.2)	0.771	0.485	1.000
Final SBP target attainment										
Final SBP <120 mmHg†, n (%)	5 (35.7)	2 (22.2)	8 (32.0)	10 (50.0)	5 (29.4)	13 (28.9)	43 (33.1)	0.620	0.723	1.000
Final SBP <140 mmHg, n (%)	12 (85.7)	8 (88.9)	18 (72.0)	17 (85.0)	13 (76.5)	26 (57.8)	94 (72.3)	0.109	0.016	1.000

Characteristic	Stage 1	Stage 2	Stage 3a	Stage 3b	Stage 4	Stage 5	Overall	P across stages	P trend	Holm-adjusted P
In-hospital pharmacotherapy										
RAAS monotherapy, n (%)	11 (78.6)	8 (88.9)	20 (80.0)	16 (80.0)	12 (70.6)	34 (75.6)	101 (77.7)	0.929	0.457	1.000
ACE inhibitor, n (%)	7 (50.0)	3 (33.3)	10 (40.0)	9 (45.0)	6 (35.3)	13 (28.9)	48 (36.9)	0.715	0.166	1.000
ARB, n (%)	4 (28.6)	5 (55.6)	10 (40.0)	7 (35.0)	6 (35.3)	21 (46.7)	53 (40.8)	0.740	0.467	1.000
Dual ACEi + ARB, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	—	—	—
CCB, n (%)	2 (14.3)	0 (0.0)	8 (32.0)	3 (15.0)	7 (41.2)	20 (44.4)	40 (30.8)	0.024	0.003	0.612
Beta-blocker, n (%)	9 (64.3)	8 (88.9)	21 (84.0)	15 (75.0)	16 (94.1)	34 (75.6)	103 (79.2)	0.351	0.748	1.000
Loop diuretic, n (%)	11 (78.6)	8 (88.9)	19 (76.0)	11 (55.0)	15 (88.2)	23 (51.1)	87 (66.9)	0.017	0.018	0.455
MRA, n (%)	0 (0.0)	0 (0.0)	1 (4.0)	0 (0.0)	0 (0.0)	1 (2.2)	2 (1.5)	1.000	0.785	1.000
SGLT2 inhibitor, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	—	—	—
Statin, n (%)	4 (28.6)	5 (55.6)	17 (68.0)	9 (45.0)	7 (41.2)	25 (55.6)	67 (51.5)	0.215	0.498	1.000
Antiplatelet, n (%)	7 (50.0)	3 (33.3)	17 (68.0)	11 (55.0)	8 (47.1)	20 (44.4)	66 (50.8)	0.429	0.466	1.000
Antihypertensive classes, mean±SD	2.43±0.94	2.67±0.50	2.84±1.07	2.30±1.13	2.94±0.90	2.60±1.05	2.63±1.01	0.372	—	1.000

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium-channel blocker; CKD, chronic kidney disease; MRA, mineralocorticoid-receptor antagonist; RAAS, renin-angiotensin-aldosterone system; SBP, systolic blood pressure; SD, standard deviation; SGLT2, sodium-glucose cotransporter-2. Values are n (%) unless stated. P across stages: permutation chi-square test (10,000 permutations) or one-way ANOVA; P trend: linear-by-linear test (descriptive, unadjusted). Holm-adjusted P values apply to the across-stage P values over all 28 stage-wise comparisons in Tables 4 and 5. Stage cells are small (n=9 to 45); results are descriptive. †KDIGO's SBP <120 mmHg target applies to adults not on dialysis. Medication data are in-hospital prescriptions only.

Table 5: Recorded cardiovascular conditions and their distribution across recorded CKD stage

Condition	Stage 1	Stage 2	Stage 3a	Stage 3b	Stage 4	Stage 5	Overall	P across stages	P trend	Holm-adjusted P
LVH on echocardiography, n (%)	3 (21.4)	4 (44.4)	9 (36.0)	7 (35.0)	11 (64.7)	17 (37.8)	51 (39.2)	0.236	0.280	1.000
Composite MACE, n (%)	7 (50.0)	6 (66.7)	9 (36.0)	11 (55.0)	10 (58.8)	22 (48.9)	65 (50.0)	0.610	0.919	1.000
Ischaemic heart disease, n (%)	2 (14.3)	1 (11.1)	10 (40.0)	6 (30.0)	6 (35.3)	23 (51.1)	48 (36.9)	0.075	0.006	1.000
Heart failure, n (%)	5 (35.7)	2 (22.2)	5 (20.0)	8 (40.0)	5 (29.4)	8 (17.8)	33 (25.4)	0.431	0.316	1.000
Myocardial infarction, n (%)	1 (7.1)	1 (11.1)	2 (8.0)	3 (15.0)	4 (23.5)	11 (24.4)	22 (16.9)	0.424	0.037	1.000
Arrhythmias, n (%)	4 (28.6)	1 (11.1)	4 (16.0)	5 (25.0)	3 (17.6)	9 (20.0)	26 (20.0)	0.892	0.859	1.000
Stroke/TIA, n (%)	3 (21.4)	4 (44.4)	2 (8.0)	4 (20.0)	2 (11.8)	9 (20.0)	24 (18.5)	0.265	0.689	1.000
Peripheral artery disease, n (%)	1 (7.1)	1 (11.1)	3 (12.0)	2 (10.0)	2 (11.8)	10 (22.2)	19 (14.6)	0.668	0.119	1.000

CKD, chronic kidney disease; LVH, left ventricular hypertrophy; MACE, major adverse cardiovascular events (MI, heart failure, or stroke/TIA); TIA, transient ischaemic attack. Values are n (%) of patients with the condition recorded (prevalent, not incident). P across stages: permutation chi-square test (10,000 permutations); P trend: linear-by-linear test (descriptive, unadjusted); Holm-adjusted P values over the 28 stage-wise comparisons in Tables 4 and 5.

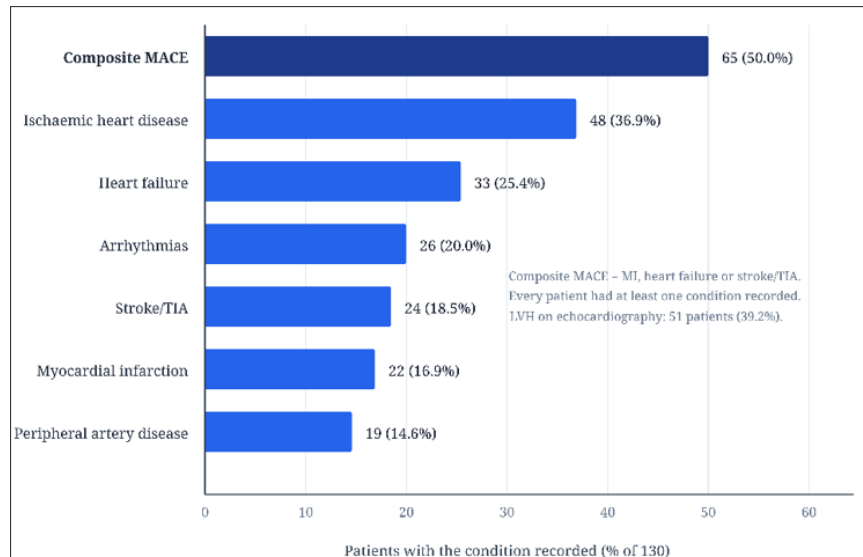


Figure 3. Distribution of recorded cardiovascular conditions and left ventricular hypertrophy in the 130-patient study sample. LVH was present in 51 patients (39.2%). Composite MACE were recorded in 65 (50.0%) patients; IHD was recorded in 48 (36.9%). Every patient had at least one condition recorded

Table 6: Cross-sectional associations of recorded cardiovascular conditions: Firth penalized logistic regression with profile-likelihood 95% CI

Outcome (events)	Exposure	Prevalence, exposed vs unexposed, n/N (%)	Adjusted OR (95% CI)	P value	C-statistic
Heart failure (33)	Hypertension	30/118 (25.4%) vs 3/12 (25.0%)	0.93 (0.27–3.97)	0.914	0.59
	LVH	16/51 (31.4%) vs 17/79 (21.5%)	1.60 (0.71–3.58)	0.252	
Stroke/TIA (24)	LVH	13/51 (25.5%) vs 11/79 (13.9%)	2.30 (0.60–12.77)	0.241	0.66
	Diabetes mellitus	15/69 (21.7%) vs 9/61 (14.8%)	0.94 (0.16–3.85)	0.934	
Arrhythmias (26)	LVH	16/51 (31.4%) vs 10/79 (12.7%)	2.80 (1.18–6.92)*	0.020	0.68
Composite MACE (65)	Diabetes mellitus	42/69 (60.9%) vs 23/61 (37.7%)	3.27 (0.96–11.63)	0.058	0.68
	Dyslipidemia	48/104 (46.2%) vs 17/26 (65.4%)	0.35 (0.13–0.90)*	0.030	
MI (22)	Diabetes mellitus	21/69 (30.4%) vs 1/61 (1.6%)	10.80 (2.34–105.25)*	0.001	0.89
	Smoking history	20/60 (33.3%) vs 2/70 (2.9%)	7.95 (2.17–43.09)*	0.001	
PAD (19)	Smoking history	19/60 (31.7%) vs 0/70 (0.0%)	Not reported†	<0.001†	0.89
	Diabetes mellitus	15/69 (21.7%) vs 4/61 (6.6%)	1.63 (0.48–6.31)	0.437	

CI, confidence interval; LVH, left ventricular hypertrophy; MACE, major adverse cardiovascular events; MI, myocardial infarction; OR, odds ratio; PAD, peripheral artery disease; TIA, transient ischaemic attack. Conditions are prevalent (recorded), not incident. Models were adjusted as described in Materials and Methods; P values are penalized likelihood-ratio P values. * $P < 0.05$. C-statistic is in-sample (apparent) and descriptive. †Complete separation (no events in the unexposed group): the adjusted OR is not interpretable and is not reported; P is the crude Fisher exact P. MI estimates are imprecise (4.4 events per variable). Full model output, including all covariates, is in Supplementary Table S2.

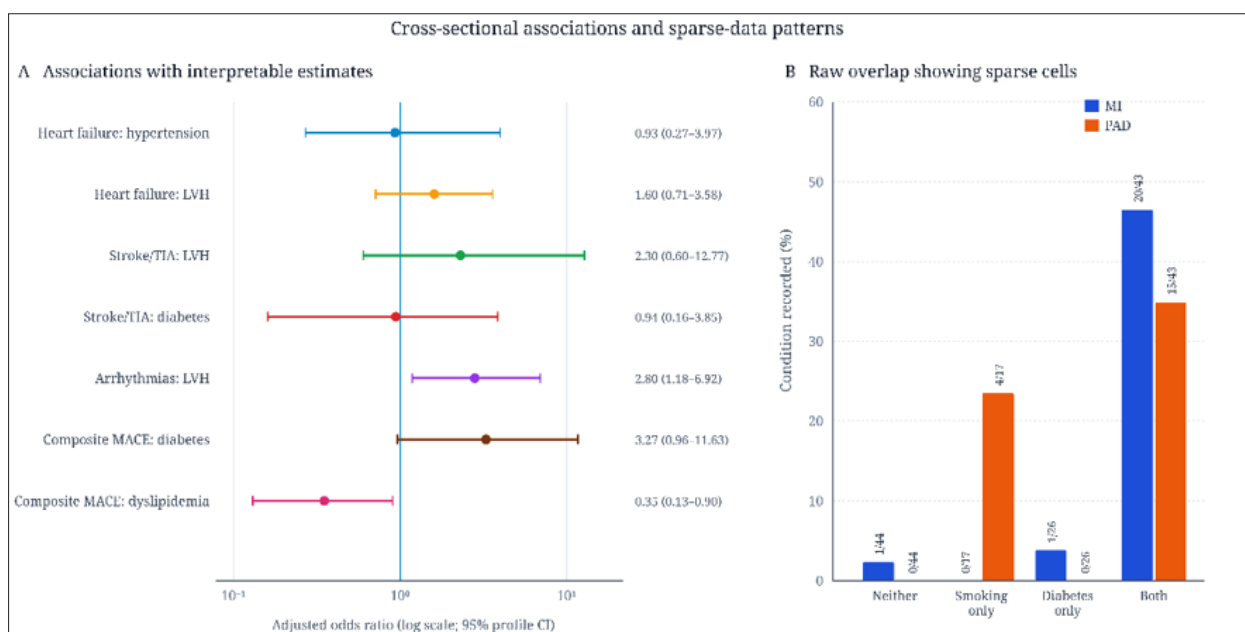


Figure 4. Panel A: adjusted associations between selected clinical factors and recorded cardiovascular conditions from Firth penalized logistic regression, with profile-likelihood 95% confidence intervals on a logarithmic scale. MI and PAD odds ratios are not plotted because of sparse-data instability; the selected MI estimates remain in Table 6 with explicit caution, while the PAD–smoking estimate is not reported because of complete separation. The reference line indicates an odds ratio of 1.0. Panel B: crude prevalence of MI and PAD by diabetes and smoking status; 20 of 22 MI cases and 15 of 19 PAD cases occurred among the 43 patients with both diabetes and smoking history, and no non-smoker had PAD

Prespecified Firth models (Table 6, Figure 4) showed that LVH was associated with recorded arrhythmias (adjusted OR 2.80, 95% CI 1.18–6.92; $P=0.020$). Diabetes (adjusted OR 10.80, 95% CI 2.34–105.25; $P=0.001$) and smoking history (7.95, 2.17–43.09; $P=0.001$) were associated with MI, but the intervals are very wide because MI was recorded in only 1 of the 61 patients without diabetes and 2 of the 70 non-smokers. Dyslipidemia showed an inverse association with composite MACE (adjusted OR 0.35, 95% CI 0.13–0.90; $P=0.030$), and the association of diabetes with composite MACE had a confidence interval that included 1 (3.27, 0.96–11.63; $P=0.058$). Neither hypertension nor LVH was associated with heart failure, and no covariate reached statistical significance in the stroke/TIA model. All 19 PAD patients were smokers, and none of the 70 non-smokers had PAD. With a zero cell this is complete separation, so the Firth odds ratio for smoking is a penalisation artefact rather than an effect size; it is not reported, and the crude count is shown instead (Fisher exact $P<0.001$; Table 6). Diabetes was not significantly associated with PAD (1.63, 0.48–6.31; $P=0.437$). Full output is in Supplementary Table S2.

These findings should be read as a descriptive snapshot rather than as evidence of care quality or prognosis. On the medication-safety side, no dual ACE-inhibitor–ARB exposure was recorded during admission (0/130; upper 95% bound 2.8%) in a setting where 77.7% of patients received a single RAAS agent, which is consistent with the trial-based advice against dual blockade[29,30]. The observation rests on a zero count with no comparator and covers inpatient

prescriptions only, so it does not show that these safety lessons have been translated into routine outpatient prescribing; hyperkalaemia was recorded at similar frequency with and without RAAS exposure. On the blood-pressure side, a smaller proportion of dialysis patients had a final SBP below 140 mmHg, and the difference persisted after adjustment for age, sex, diabetes, LVH, and antihypertensive class count. Because the comparison rests on a single non-standardised in-hospital reading, and dialysis readings reflect volume status and session timing, we describe this as lower recorded attainment rather than a treatment gap and do not infer inadequate treatment.

In dialysis patients, volume status and the timing of measurement relative to the dialysis session are established determinants of recorded blood pressure[19–21], and haemodynamic instability including intradialytic hypotension further complicates interpretation of any single reading[36,37]. The only randomised comparison of SBP targets in haemodialysis to date was a pilot feasibility trial[22], and trial evidence for SBP targets below 120 mmHg was generated largely outside dialysis populations[12,14]. KDIGO's SBP <120 mmHg target explicitly applies to adults not on dialysis[10,11], so the <120 mmHg comparison in dialysis patients is descriptive only. Individualized evaluation is consistent with KDIGO guidance and with evidence from intensive blood-pressure-control trials more broadly[10,38].

The higher antihypertensive class counts among CCB-treated patients, with no difference in attainment of SBP <120 mmHg, are partly arithmetic, since CCB contributes one class

to the count, and are otherwise compatible with confounding by indication: additional agents are typically added for patients whose blood pressure has proved resistant to initial therapy[31]. CCB use was also twice as common in dialysis as in non-dialysis patients and may therefore contribute to the dialysis comparison, which cannot be resolved with the present data.

Diabetes mellitus was not significantly associated with prevalent composite MACE after adjustment (adjusted OR 3.27, 95% CI 0.96–11.63; 60.9% versus 37.7% crude), and its association with MI (10.80, 2.34–105.25) is consistent with the substantially elevated vascular risk that diabetes confers in CKD[4–7], and with its well-documented interaction with declining kidney function in determining cardiovascular and renal outcomes[39]. Chronic hyperglycaemia accelerates vascular injury through the accumulation of advanced glycation end-products, activation of protein kinase C, and increased generation of reactive oxygen species, together promoting endothelial dysfunction and accelerated atherogenesis. Tobacco exposure operates through a parallel but distinct pathway, triggering endothelial dysfunction, oxidative stress, and impaired nitric oxide bioavailability that collectively accelerate intimal injury and plaque formation. MI and PAD clustered in patients with diabetes and a smoking history: 21 of 22 MI patients had diabetes, 20 of 22 were smokers, and 20 had both, while all 19 PAD patients were smokers and 15 of 19 also had diabetes. No non-smoker had PAD, so no adjusted estimate for smoking could be given for it, and the MI estimates are imprecise. Overlap this close may partly reflect how conditions were documented, and verification against source charts is advisable.

LVH, associated with arrhythmias in this sample (recorded in 31.4% of patients with LVH versus 12.7% without), is a well-established marker of adverse cardiac remodelling in CKD, mechanistically linked to fibroblast growth factor 23-mediated cardiac hypertrophy [6,40] and to structural cardiac abnormalities that predict adverse outcomes in hypertensive CKD populations more broadly[41,42]. Subclinical structural change of this kind is detectable even before overt heart failure develops, and its progression tracks closely with long-term prognosis in dialysis cohorts specifically[43–45]; reduced kidney function is, independently, itself a recognized risk factor for incident heart failure[46]. In a cross-sectional design, however, direction cannot be established: LVH and arrhythmias may arise together from the same underlying cardiac disease. The inverse association of dyslipidemia with composite MACE (adjusted OR 0.35, 95% CI 0.13–0.90) should not be read as protective. Every patient had at least one recorded cardiovascular condition, so patients without MACE were necessarily those with IHD, arrhythmias, or PAD, and 56 of these 65 patients had dyslipidemia; the association may reflect this selection, reverse causation (dyslipidemia diagnosed or treated after an event), or chance.

The PAD model showed complete separation for smoking, so that estimate is not reported. The MI model is estimable

but imprecise (4.4 events per variable; the interval for diabetes spans 2.34 to 105.25), and the in-sample C-statistics of the MI and PAD models (0.89 for both) are optimistic; the C-statistics of the remaining models were modest (0.59 to 0.68). Estimates from all models remain imprecise (for example, the interval for LVH and arrhythmias spans 1.18 to 6.92) and require replication.

No patient received an SGLT2 inhibitor during admission, although 53.1% had diabetes and 25.4% had recorded heart failure. KDIGO 2024 recommends SGLT2 inhibitors for adults with CKD and an eGFR of at least 20 mL/min/1.73 m² who have substantial albuminuria or heart failure[11,23, 24], but albuminuria data were not part of the dataset, the median first-admission eGFR was 16.5 mL/min/1.73 m², and many patients were on dialysis; moreover, inpatient records seldom capture the start of long-term therapy. The finding therefore cannot be read as under-prescribing. The sample was also advanced (34.6% in stage 5; 32.3% on dialysis), which mirrors the late presentation reported in Indian registries[25,28] but limits generalisability to community-based, earlier-stage CKD.

Several limitations warrant consideration. First, the design is cross-sectional: conditions are prevalent records without post-discharge follow-up, no incidence or time-to-event analysis is possible, associations carry no temporal information, and reverse causation is likely. Second, blood pressure is the last reading from a non-standardised inpatient chart: posture, timing relative to dialysis, and ambulatory or home readings were not available, and 35 patients (26.9%) had a final reading of exactly 115/75 mmHg, a rounded value. Because readings from the mercury sphygmomanometer were recorded to the nearest discernible value, this imprecision may have contributed to repeated values and should be considered when interpreting target attainment. Third, stage rests on the pre-established diagnosis in the patient file, whose date, method, and baseline eGFR were not available, and it agreed with the stage derived from admission creatinine in only 53 of 130 patients (admission values probably reflecting acute illness); dialysis and stage 5 are collinear so stage could not be adjusted for in the dialysis models, and stage-specific cells are small (9 to 45 patients). Fourth, medication data cover the admission only (median 4 days), without dose, adherence, or outpatient information, and the absence of dual blockade is a zero count without a comparator. Fifth, every patient had at least one recorded cardiovascular condition, so prevalence cannot be generalised to CKD patients without one, a condition not documented in the file was treated as absent, and albuminuria data were unavailable. Sixth, the modest single-centre sample, limited event counts, and many comparisons produced wide intervals, several stage-wise findings are hypothesis-generating only, and the close overlap of MI and PAD with diabetes and smoking, with no PAD in non-smokers, should be verified against source charts. External validation in independent, multicentre, prospective cohorts is needed before these

findings are used to guide practice. Despite these limitations, this study describes real-world blood-pressure and pharmacotherapy patterns across the recorded CKD stages in this selected inpatient sample.

Conclusion

In this single-centre, prospective cross-sectional study of 130 inpatients with CKD, no dual ACE-inhibitor-ARB prescription was recorded during admission (0/130; 95% CI 0.0–2.8%), and no patient received an SGLT2 inhibitor. A final SBP below 140 mmHg was recorded less often among dialysis patients, a difference that persisted after adjustment, whereas the <120 mmHg KDIGO target, which does not apply to dialysis, was met by about one in three patients overall. LVH was associated with recorded arrhythmias; exploratory associations between diabetes or smoking and MI were imprecise, and dyslipidemia showed an inverse association with composite MACE that should not be interpreted as protective. However, the cross-sectional design, small event counts, a sample in which every patient had a recorded cardiovascular condition, and complete separation for smoking and PAD mean that these associations are hypothesis-generating. These findings support standardised blood-pressure measurement in dialysis units, continued vigilance around RAAS-inhibitor prescribing, and prospective multicentre studies with follow-up.

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Data Availability

The individual-level patient data are not publicly available because data sharing is not permitted under the applicable confidentiality and institutional restrictions.

Use of AI-Assisted Tools

QuillBot was used for language editing/paraphrasing. The authors reviewed the manuscript and take responsibility for the final content. QuillBot was not used to generate or analyse study data. Gemini canvas is used to generate the high quality images/figures for the manuscript.

Publication Status

This manuscript has not been published previously and is not under consideration by another journal.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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Supplementary Table S1. Baseline characteristics, blood-pressure target attainment, pharmacotherapy, and recorded cardiovascular conditions by eGFR-derived CKD stage (sensitivity analysis)

Variable	Stage 1	Stage 2	Stage 3a	Stage 3b	Stage 4	Stage 5	Overall	P across stages (permutation chi-square / ANOVA)	P trend (linear-by-linear)	Holm-adjusted P
N patients	1	4	11	17	38	59	130	—	—	—
Age, mean±SD	-	65.8±16.0	60.9±15.2	57.5±14.1	64.5±9.8	60.6±12.6	61.6±12.4	0.315	—	1
Male, n (%)	1 (100.0)	3 (75.0)	10 (90.9)	11 (64.7)	20 (52.6)	38 (64.4)	83 (63.8)	0.264	0.177	1
On dialysis, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	6 (15.8)	36 (61.0)	42 (32.3)	<0.001	<0.001	0.003
Hypertension, n (%)	1 (100.0)	3 (75.0)	10 (90.9)	13 (76.5)	35 (92.1)	56 (94.9)	118 (90.8)	0.227	0.095	1
Diabetes, n (%)	1 (100.0)	1 (25.0)	4 (36.4)	11 (64.7)	20 (52.6)	32 (54.2)	69 (53.1)	0.517	0.557	1
Dyslipidemia, n (%)	1 (100.0)	4 (100.0)	8 (72.7)	11 (64.7)	30 (78.9)	50 (84.7)	104 (80.0)	0.449	0.473	1
Smoking history, n (%)	1 (100.0)	2 (50.0)	5 (45.5)	6 (35.3)	18 (47.4)	28 (47.5)	60 (46.2)	0.914	0.981	1
LVH on echo, n (%)	0 (0.0)	1 (25.0)	4 (36.4)	9 (52.9)	15 (39.5)	22 (37.3)	51 (39.2)	0.836	0.947	1
Final SBP <120, n (%)	1 (100.0)	1 (25.0)	5 (45.5)	6 (35.3)	12 (31.6)	18 (30.5)	43 (33.1)	0.728	0.309	1
Final SBP <140, n (%)	1 (100.0)	3 (75.0)	9 (81.8)	14 (82.4)	28 (73.7)	39 (66.1)	94 (72.3)	0.744	0.132	1
Any ACEi/ARB, n (%)	1 (100.0)	3 (75.0)	8 (72.7)	15 (88.2)	31 (81.6)	43 (72.9)	101 (77.7)	0.761	0.456	1
RAAS monotherapy, n (%)	1 (100.0)	3 (75.0)	8 (72.7)	15 (88.2)	31 (81.6)	43 (72.9)	101 (77.7)	0.757	0.456	1
Dual ACEi+ARB, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	—	—	—
ACE inhibitor, n (%)	1 (100.0)	2 (50.0)	5 (45.5)	5 (29.4)	17 (44.7)	18 (30.5)	48 (36.9)	0.453	0.183	1
ARB, n (%)	0 (0.0)	1 (25.0)	3 (27.3)	10 (58.8)	14 (36.8)	25 (42.4)	53 (40.8)	0.493	0.501	1
CCB, n (%)	0 (0.0)	1 (25.0)	1 (9.1)	4 (23.5)	13 (34.2)	21 (35.6)	40 (30.8)	0.552	0.076	1
Beta-blocker, n (%)	1 (100.0)	4 (100.0)	9 (81.8)	13 (76.5)	32 (84.2)	44 (74.6)	103 (79.2)	0.745	0.249	1
Loop diuretic, n (%)	0 (0.0)	4 (100.0)	10 (90.9)	11 (64.7)	25 (65.8)	37 (62.7)	87 (66.9)	0.186	0.162	1
MRA, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (3.4)	2 (1.5)	0.728	0.233	1
SGLT2i, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	—	—	—
Statin, n (%)	1 (100.0)	3 (75.0)	5 (45.5)	7 (41.2)	19 (50.0)	32 (54.2)	67 (51.5)	0.763	0.993	1
Antiplatelet, n (%)	1 (100.0)	3 (75.0)	5 (45.5)	8 (47.1)	21 (55.3)	28 (47.5)	66 (50.8)	0.817	0.447	1
Composite MACE (prevalent), n (%)	0 (0.0)	1 (25.0)	4 (36.4)	8 (47.1)	17 (44.7)	35 (59.3)	65 (50.0)	0.382	0.033	1
IHD, n (%)	0 (0.0)	2 (50.0)	5 (45.5)	4 (23.5)	14 (36.8)	23 (39.0)	48 (36.9)	0.811	0.812	1
Heart failure, n (%)	0 (0.0)	0 (0.0)	3 (27.3)	6 (35.3)	11 (28.9)	13 (22.0)	33 (25.4)	0.688	0.998	1
MI, n (%)	0 (0.0)	0 (0.0)	2 (18.2)	1 (5.9)	4 (10.5)	15 (25.4)	22 (16.9)	0.275	0.059	1
Arrhythmia, n (%)	0 (0.0)	2 (50.0)	2 (18.2)	6 (35.3)	6 (15.8)	10 (16.9)	26 (20.0)	0.341	0.198	1
Stroke/TIA, n (%)	0 (0.0)	1 (25.0)	1 (9.1)	2 (11.8)	6 (15.8)	14 (23.7)	24 (18.5)	0.729	0.221	1
PAD, n (%)	1 (100.0)	0 (0.0)	0 (0.0)	2 (11.8)	6 (15.8)	10 (16.9)	19 (14.6)	0.138	0.465	1
Antihypertensive classes, mean±SD	2.00	3.00±0.82	2.55±0.69	2.59±1.12	2.76±1.10	2.56±1.00	2.63±1.01	0.858	—	1

Note: eGFR-derived stages were calculated from the first-admission creatinine using the CKD-EPI 2021 equation. This is a sensitivity analysis only because acute inpatient creatinine may not represent baseline kidney function. P trend values are descriptive; Holm-adjusted P values apply to the specified across-stage comparison family.

Supplementary Table S2. Full prespecified Firth logistic-regression model output for recorded cardiovascular conditions

Condition (events)	Variable	Adjusted OR (95% profile CI)	Penalized likelihood-ratio P	Apparent C-statistic	Events per variable
Heart failure (33)	Hypertension	0.93 (0.27–3.97)	0.914	0.59	6.6
Heart failure (33)	LVH	1.60 (0.71–3.58)	0.252	0.59	6.6
Heart failure (33)	Age per 10-year increase	1.06 (0.77–1.49)	0.712	0.59	6.6
Heart failure (33)	Male	0.95 (0.42–2.25)	0.914	0.59	6.6
Heart failure (33)	Dialysis	0.66 (0.26–1.55)	0.347	0.59	6.6
Myocardial infarction (22)	Diabetes	10.80 (2.34–105.25)	0.001	0.89	4.4
Myocardial infarction (22)	Smoking	7.95 (2.17–43.09)	0.001	0.89	4.4
Myocardial infarction (22)	Age per 10-year increase	0.65 (0.39–1.01)	0.057	0.89	4.4
Myocardial infarction (22)	Male	0.43 (0.12–1.55)	0.197	0.89	4.4
Myocardial infarction (22)	Dyslipidemia	2.54 (0.46–26.20)	0.304	0.89	4.4
Stroke/TIA (24)	LVH	2.30 (0.60–12.77)	0.241	0.66	6.0
Stroke/TIA (24)	Diabetes	0.94 (0.16–3.85)	0.934	0.66	6.0
Stroke/TIA (24)	Age per 10-year increase	1.26 (0.87–1.89)	0.237	0.66	6.0
Stroke/TIA (24)	Male	0.58 (0.23–1.48)	0.251	0.66	6.0
Arrhythmias (26)	LVH	2.80 (1.18–6.92)	0.020	0.68	6.5
Arrhythmias (26)	Age per 10-year increase	0.92 (0.65–1.32)	0.659	0.68	6.5
Arrhythmias (26)	Male	1.33 (0.51–3.69)	0.561	0.68	6.5
Arrhythmias (26)	Dialysis	0.79 (0.29–2.00)	0.629	0.68	6.5
PAD (19)	Smoking	Not reported—complete separation	Crude Fisher <0.001	0.89	4.8
PAD (19)	Diabetes	1.63 (0.48–6.31)	0.437	0.89	4.8
PAD (19)	Age per 10-year increase	1.40 (0.86–2.46)	0.183	0.89	4.8
PAD (19)	Male	3.79 (0.99–18.28)	0.053	0.89	4.8
Composite MACE (65)	Diabetes	3.27 (0.96–11.63)	0.058	0.68	9.3
Composite MACE (65)	Dyslipidemia	0.35 (0.13–0.90)	0.030	0.68	9.3
Composite MACE (65)	Age per 10-year increase	1.14 (0.85–1.54)	0.369	0.68	9.3
Composite MACE (65)	Male	0.75 (0.34–1.63)	0.470	0.68	9.3
Composite MACE (65)	Smoking	0.84 (0.37–1.88)	0.670	0.68	9.3
Composite MACE (65)	LVH	1.05 (0.33–3.34)	0.933	0.68	9.3
Composite MACE (65)	Dialysis	1.14 (0.52–2.52)	0.741	0.68	9.3

Note: P values are penalized likelihood-ratio P values, except the PAD–smoking entry, for which no adjusted odds ratio is reported because all recorded PAD cases occurred among smokers and no PAD case occurred among non-smokers; the displayed P value is the crude Fisher exact result. Apparent C-statistics are in-sample and should not be interpreted as validated predictive performance. Wide intervals and sparse cells make several estimates exploratory.