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Research Article

The effects of chlorthalidone and furosemide on serum creatinine in Thai patients with stage 4 and 5 chronic kidney disease: Retrospective cohort study.

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ABSTRACT

Chlorthalidone has been shown to lower blood pressure in CKD stages 4–5 but may raise serum creatinine (SCr), particularly when combined with furosemide. Data in Asian populations remain limited. This retrospective cohort study, conducted at Ramathibodi Hospital, included patients with stage 4-5 CKD who were recruited between April 1, 2021 and December 31, 2022 and followed until January 31, 2024. From 904 screened patients, 30 participants receiving chlorthalidone were enrolled and categorized into three groups: (1) chlorthalidone with unchanged furosemide dose (n=8), (2) chlorthalidone with reduced or discontinued furosemide (n=8), and (3) chlorthalidone without furosemide (n=14). The primary endpoint was an increase in SCr >25% from baseline. Secondary outcomes included changes in estimated glomerular filtration rate (eGFR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and adverse events, followed for up to 18 months. At baseline, mean eGFR was 22 ± 7 mL/min/1.73 m² and mean SCr was 2.6 ± 1.4 mg/dL. Group 1 showed the largest rise in SCr, with a 168.8% median increase (from 3.57 to 6.37 mg/dL) at 18 months and 100% (4/4) experiencing an SCr rise >25%. Group 2 maintained stable renal function with median SCr decreasing slightly from 1.79 to 1.73 mg/dL Group 3 had a mild upward median SCr from 1.94 to 2.41 mg/dL with 75% experiencing an SCr increase >25% in both groups. Adverse events occurred in 88%, 63%, and 86% of patients in Groups 1, 2, and 3, respectively. Concomitant chlorthalidone with unchanged furosemide accelerated renal function decline, whereas reducing or discontinuing furosemide appeared more protective. Careful adjustment of diuretic regimens may mitigate risk when combining these agents. However, larger prospective studies are warranted.

Keywords:
Chlorthalidone; Furosemide; Advance chronic kidney disease; Serum creatinine

1. INTRODUCTION

Hypertension and chronic kidney disease (CKD) are closely linked, with hypertension both contributing to CKD progression and arising as a complication of it. Effective blood pressure (BP) management is therefore essential in this population. A range of antihypertensive agents are used in CKD, including angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), beta-blockers, calcium channel blockers (CCBs), and diuretics such as thiazides, chlorthalidone, and indapamide. Antihypertensive therapy was generally initiated when blood pressure (BP) reached ≥140/90 mmHg, with treatment goals individualized according to age, comorbidities, CKD stage, and albuminuria status [1]. In patients with significant proteinuria (≥300 mg/day or ≥300 mg/g creatinine), ACE inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) were recommended as first-line therapy because of their proven renoprotective effects [1-2] For those with lower levels

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of proteinuria, target BP was typically 130–139/70–79 mmHg [1]. Recent evidence shifted toward more intensive BP control. The SPRINT trial demonstrated cardiovascular benefits with lowering systolic blood pressure (SBP) to ≤120 mmHg, and this approach was endorsed by the 2021 KDIGO guidelines in appropriately selected patients [3-4]. When BP remained uncontrolled with two agents, the addition of a thiazide-like diuretic was often considered. Combination therapy with both an ACEI and an ARB, however, should be avoided because of increased risks of hyperkalemia, acute kidney injury, and hypotension [2]. According to the 2020 ISH guidelines, thiazide-like diuretics (e.g., chlorthalidone, indapamide) were preferred over traditional thiazides, particularly in patients with eGFR <30 mL/min/1.73 m² or those with volume overload [5]. Meanwhile, the 2012 KDIGO guidelines suggested loop diuretics for advanced CKD, either alone or in combination with thiazides, to improve volume control [2]. However, loop diuretics must be used cautiously as they can cause electrolyte disturbances, including hypokalemia and hypomagnesemia [6]. Recent evidence suggested that chlorthalidone might remain effective in patients with advanced CKD, even when the eGFR was <30 mL/min/1.73 m². In a 12-week randomized trial, chlorthalidone achieved significant reductions in blood pressure, although mild adverse events such as hypokalemia, hyponatremia, and dizziness were reported more frequently. Notably, chlorthalidone use was linked to increases in serum creatinine (SCr), particularly in patients who were also receiving furosemide, raising concern for a possible additive or synergistic effect that might accelerate renal function decline. These findings suggested that adjusting furosemide dosage could help mitigate this risk, while lower doses of chlorthalidone appear to preserve most of its BP-lowering benefit [7].

Nonetheless, previous studies have been limited by underrepresentation of Asian populations and short study durations. To address this gap, the present study investigated the impact of chlorthalidone and furosemide co-administration on SCr levels in Thai patients with stage 4–5 CKD, using a retrospective cohort design at Ramathibodi Hospital for 18 months.

2. MATERIALS AND METHODS

2.1. Study design and patient population

This retrospective cohort study included patients with stage 4-5 CKD who received chlorthalidone, either with or without furosemide, during the recruitment period between April 1, 2021, and December 31, 2022, were screened for eligibility. Included patients were then followed up for 18 months after their first chlorthalidone prescription. The overall study period, including data collection and follow-up, spanned from April 1, 2021, to January 31, 2024. This study protocol was reviewed and approved by the Human Research Ethics Committee, Faculty of Medicine Ramathibodi Hospital, Mahidol University (no. MURA 2022/386).

The inclusion criteria were patients aged 18 years and older, diagnosed with CKD stage 4 or 5, and received chlorthalidone at least once from the outpatient pharmacy, Ramathibodi Hospital. We excluded patients who had been kidney transplant recipients, were receiving peritoneal dialysis, or hemodialysis, intermittent (thrice weekly). Participants were classified into three groups:

1. Chlorthalidone + unchanged furosemide dose
2. Chlorthalidone + reduced or discontinued furosemide dose
3. Chlorthalidone without furosemide

2.2. Data collection

Patients were collected data as three categories.

1. Medication data: Information on chlorthalidone and furosemide use was obtained from the SAP NetWeaver system to classify patients into the three study groups.

2. SCr: SCr values were extracted from electronic medical records of patients who had received chlorthalidone at least once. Patients were identified during the recruitment period between April 1, 2021, and December 31, 2022. Follow-up SCr values were recorded at 3, 6, 9, 12, 15, and 18 months after the first chlorthalidone prescription. Consequently, follow-up data were available through January 31, 2024. Measurements within ±1 month of each time point were accepted.

3. Additional laboratory data: Urinary albumin-to-creatinine ratio, blood urea nitrogen (BUN), estimated glomerular filtration rate (eGFR, CKD-EPI equation), electrolytes, uric acid, and glucose levels were also collected during the study period.

2.3. Trial outcomes

The primary outcome was an increase in SCr of more than 25% from baseline. Secondary outcomes included longitudinal trends in eGFR, SBP, diastolic blood pressure (DBP), treatment retention rates, and the incidence of adverse events (AEs) and serious adverse events (SAEs). Serious Adverse event included serum potassium < 2.5 mEq/L and Scr increased ≥50 percent from baseline.

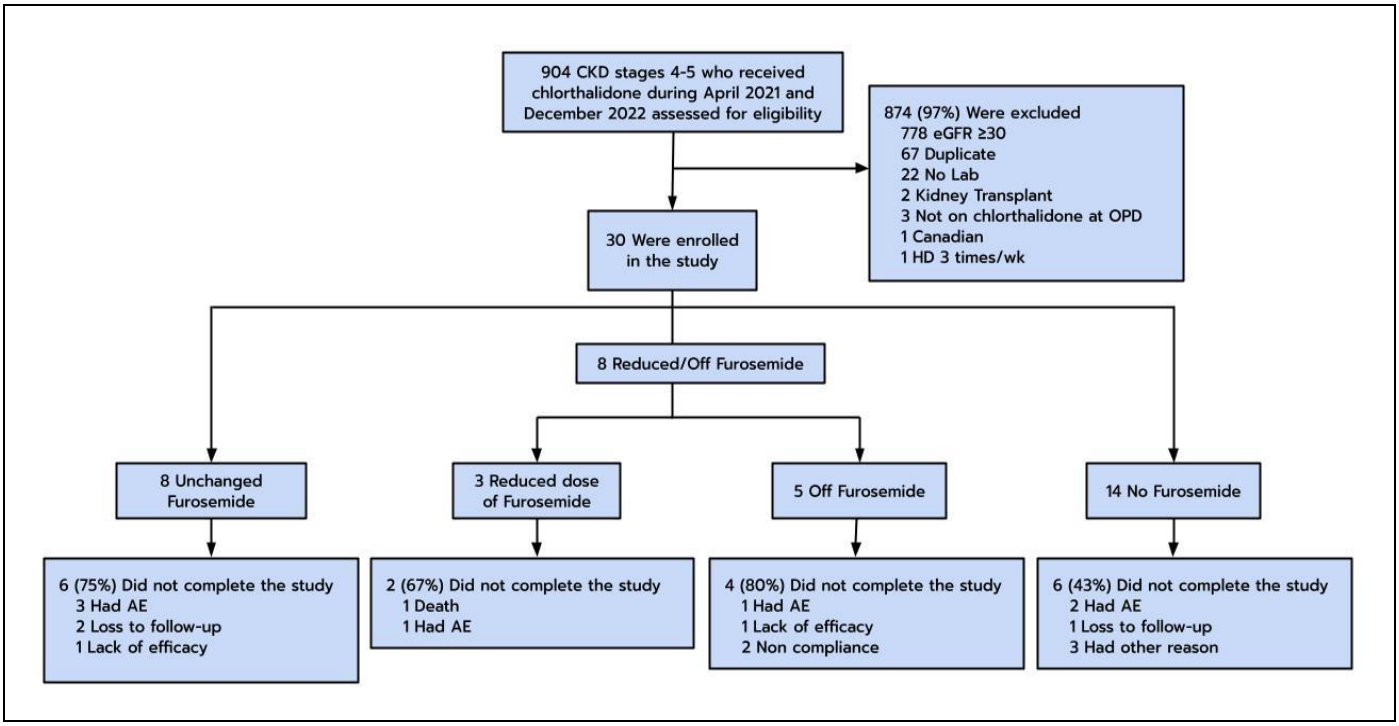

Figure 1. The patient recruitment Flow

2.4. Statistical analysis

Descriptive statistics were used to summarize patient characteristics and outcomes. Due to the limited sample size in each subgroup, formal normality testing was not performed as it lacks statistical power in small cohorts. Consequently, data were considered non-normally distributed. Continuous variables (SCr, eGFR, SBP, DBP) were reported as means ± standard deviations or medians with interquartile ranges (IQR), while categorical variables (SCr increase >25%, AEs, SAEs) were presented as counts and percentages. No inferential statistical hypothesis testing was conducted. Trends in SCr, eGFR, BP, and AEs were plotted for three groups over the study period. To analyze trends in the primary endpoint (SCr increase >25%), a cumulative analysis was performed. patients were censored upon reaching the endpoint to prevent overestimation, and their events were carried forward. Treatment retention rates were calculated as the percentage of patients remaining in the study group at each follow-up interval.

3. RESULTS

3.1. Patients and treatments

A total of 904 patients who received chlorthalidone were screened for eligibility during the recruitment period. Of these, 30 patients met the study criteria, while 874 patients were excluded: 778 patients had eGFR ≥30 ml/min/1.73m², 67 patients were duplicates, 22 patients lacked baseline SCr data, 3 patients did not receive chlorthalidone, 2 patients had kidney transplants, 1 was Canadian, and 1 was on thrice-weekly hemodialysis (Figure 1).

Participants were divided into three groups. Group 1 (n=8) was a chlorthalidone with unchanged furosemide doses. Group 2 (n=8) received chlorthalidone with reduced or discontinued furosemide. Group 3 (n=14) was chlorthalidone without furosemide. Baseline characteristics of these patients are summarized in Table 1, presenting an average age of 78 years, eGFR of 22 ± 7.0 mL/min/1.73 m², SCr of 2.6 ± 1.4 mg/dL, a mean daily chlorthalidone dose of 17.9 mg, and 30% had diabetes.

Treatment retention rates declined over the study period. At 18 months, twelve patients (40% of the enrolled cohort) remained on the assigned therapy. Retention rates for Groups 1, 2, and 3 were 25% (2/8), 25% (2/8), and 57% (8/14), respectively. Specifically, in Group 1, retention dropped to 50% by month 3 and remained stable until month 12 before declining further. Group 2 showed a gradual decline to 37.5% by month 12. In contrast, Group 3 maintained a higher retention rate (>70%) throughout the first 15 months.

3.2. Primary outcome

The prevalence of patients experiencing a >25% increase in SCr over baseline at each time point is shown in Figure 2. Due to the descriptive nature of analysis and varying numbers of patients available at each follow-up time point, results are presented as observed trends without statistical inference. At 3 months,

Table 1. Baseline demographic and clinical characteristics of patients

Characteristic	Group1 (N=8)	Group2 (N=8)	Group3 (N=14)
Age-yr	75.9±7.6	83.8±8.2	75.9±10
Male sex-no. (%)	3 (38)	1 (13)	2 (14)
Medical history-no. (%)			
Diabetes mellitus	5 (63)	7 (88)	9 (64)
Coronary artery disease	4 (50)	3 (38)	0 (0)
Hospitalization for heart failure	2 (25)	2 (25)	1 (7)
Gout	0 (0)	0 (0)	3 (21)
Stroke	0 (0)	2 (25)	1 (7)
Myocardial infarction	2 (25)	3 (38)	1 (7)
Percutaneous coronary revascularization	3 (38)	2 (25)	1 (7)
Coronary-artery bypass graft	0 (0)	1 (13)	0 (0)
Peripheral vascular bypass	0 (0)	0 (0)	0 (0)
others	0 (0)	0 (0)	0 (0)
Current smoking-no. (%)	0 (0)	0 (0)	0 (0)
Height-cm	156.3±7.9	156.1±7.3	155.1±7.7
Weight-kg	68.2±9.3	62±15.5	56.9±11.2
BMI	27.8±2	25.3±5.4	23.8±4.1
SBP-mmHg	164.8±17.8	149.8±26.3	158.4±9.3
DBP-mmHg	76.5±6.9	65±9.3	73.2±9.6
PR-bpm	65.6±9.3	65.5±8.6	74.6±14.5
Urinary albumin-to-creatinine ratio category-no. (%)			
<30	0 (0)	0 (0)	0 (0)
30 to <300	0 (0)	0 (0)	2 (25)
≥300	1 (13)	1 (13)	1 (7)
no Lab	7 (88)	7 (88)	11 (79)
eGFR-ml/min/1.73m^2	19.3±10.3	22.8±6.5	23±4.8
SCr-mg/dL	3.6±2.4	2.2±0.6	2.2±0.5
chlorthalidone dose (N)			
at baseline	17.2±6.5 (8)	14.6±5.1 (8)	17.9±6.4 (14)
at 3 months	15.6±6.3 (4)	14.6±5.1 (6)	17.3±6.3 (13)
at 6 months	15.6±6.3 (4)	16.7±6.5 (6)	17.7±6.4 (12)
at 9 months	18.8±7.2 (4)	17.2±9.4 (4)	18.2±6.5 (11)
at 12 months	21.9±6.3 (4)	18.8±10.8 (3)	19.3±6.5 (11)
at 15 months	20.8±7.2 (3)	18.8±10.8 (3)	18.8±6.6 (10)
at 18 months	25±0 (2)	25±0 (2)	17.2±6.5 (8)

no patients (0/4) in Group 1 had an SCr rise >25%, compared with 16.7% (1/6) in Group 2 and 7.7% (1/13) in Group 3. By 6 months, the proportions increased to 25% (1/4), 16.7% (1/6), and 16.7% (2/12), respectively, and at 9 months to 25% (1/4), 20% (1/5), and 27.3% (3/11). At 12 months, 75% (3/4) of patients in Groups 1 and 2 and 36.4% (4/11) in Group 3 showed an SCr increase >25%. This trend continued at 15 months (75% (3/4), 75% (3/4), and 60% (6/10)) and 18 months (100% (4/4), 75% (3/4), and 75% (6/8)).

Considering a change of SCr level over 18 months of study, Group 1 showed a pronounced elevation in median SCr, rising from 2.37 mg/dL (IQR 3.17) to 6.37 mg/dL (IQR 2.57), corresponding to a 168.8% increase. Group 2 exhibited stability, with baseline SCr of 1.79 mg/dL (IQR 0.35) and 1.73 mg/dL (IQR 0.37) at 18 months. Group 3 demonstrated a gradual increase, from 1.94 mg/dL (IQR 0.81) to 2.41 mg/dL (IQR 1.54). These median SCr levels altered consistence with the changes in mean and SD values. Group 1 showed a mean ± SD of SCr, rising from 3.57 ± 2.40 mg/dL to 6.37 ± 3.63 mg/dL. Group 2 exhibited a minor fluctuation, with baseline SCr of 2.00 ± 0.48 mg/dL and 1.85 ± 0.38 mg/dL at 18 months. Group 3 demonstrated a gradual rise from 2.20 ± 0.54 mg/dL to 3.52 ± 2.39 mg/dL as presented in Figure 3.

Over the 18-month follow-up (Figure 4), Group 1 demonstrated a mean ± SD of SBP declined

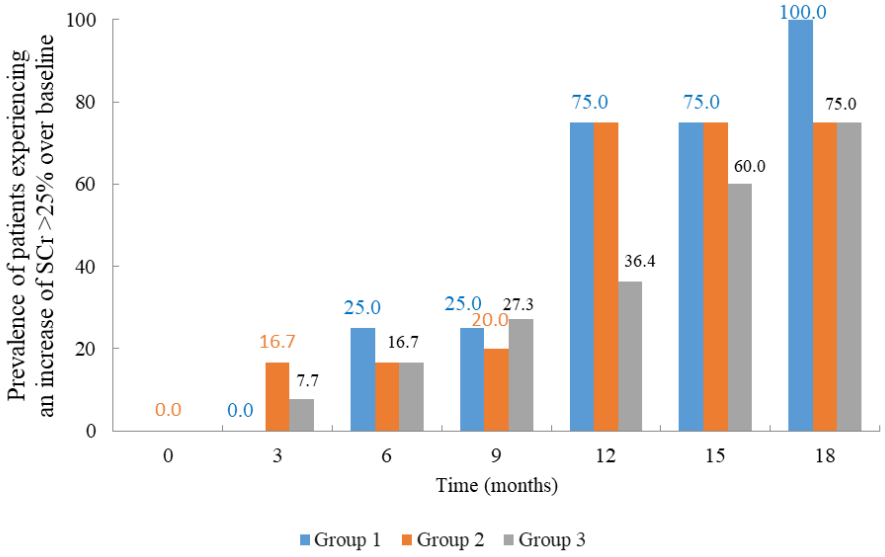

Figure 2. Trends in the prevalence of serum creatinine (SCr) increased >25% from baseline over 18 months in the trial groups

from 163.0 ± 18.1 mmHg at baseline to 137.5 ± 16.8 mmHg at 6 months, followed by subsequent fluctuations, reaching 145.5 ± 9.2 mmHg at 18 months. Group 2 presented variability of mean ± SD of SBP, with an early reduction from 149.8 ± 26.3 to 133.2 ± 15.7 mmHg at 3 months, intermittent oscillations, and a final value of 138.3 ± 32.3 mmHg. Group 3 showed a more consistent decline from 158.4 ± 9.3 to 144.7 ± 24.8 mmHg, with only minor fluctuations. DBP analyses revealed reductions in Group 1 from 75.6 ± 7.3 to 70.5 ± 12.0 mmHg. Group 2 increased from 65.0 ± 9.3 to 73.2 ± 8.0 mmHg at 6 months, before decreasing to 68.7 ± 4.0 mmHg at 18 months. Group 3 demonstrated a gradual decline from 73.2 ± 9.6 to 67.7 ± 16.4 mmHg, with minimal variability.

Over the 18-month observation period (Figure 5), Group 1 exhibited eGFR variability, declining from 19.3 ± 10.3 ml/min/1.73m² at baseline to 18.1 ± 9.5 at 3 months, rising to 21.6 ± 10.4 at 6 months, and falling markedly to 5.3 ± 0.0 by 18 months. Group 2 demonstrated an initial reduction from 22.8 ± 6.5 to 20.9 ± 7.6 at 3 months, followed by an increase to 28.07 ± 7.1 at 18 months. Group 3 showed a progressive reduction from 22.99 ± 4.8 at baseline to 17.1 ± 7.8 at 18 months, accompanied by minor fluctuations.

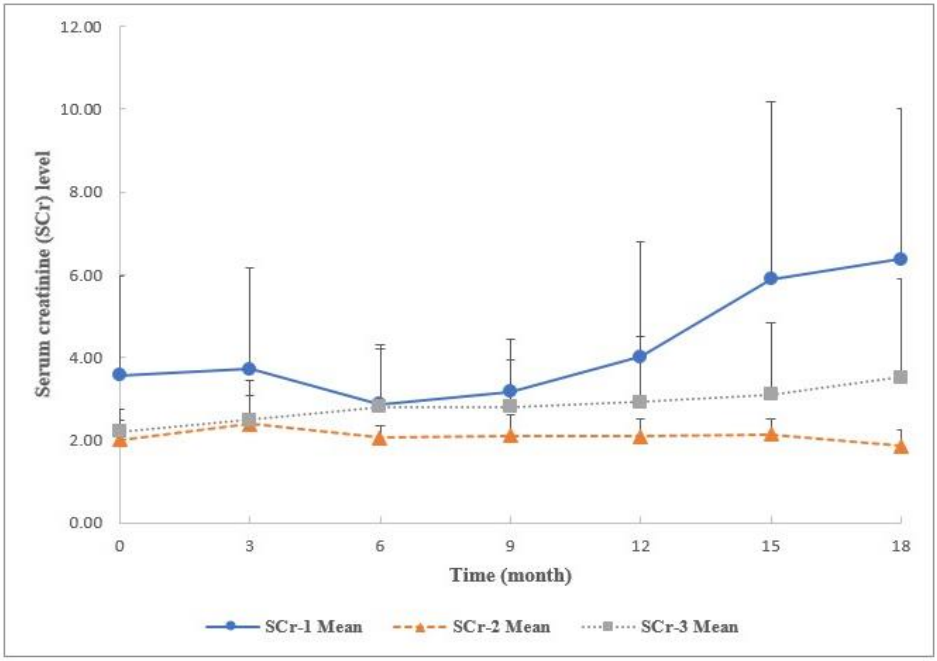

Figure 3. Mean ± SD of serum creatinine (SCr) over 18 months in the trial groups

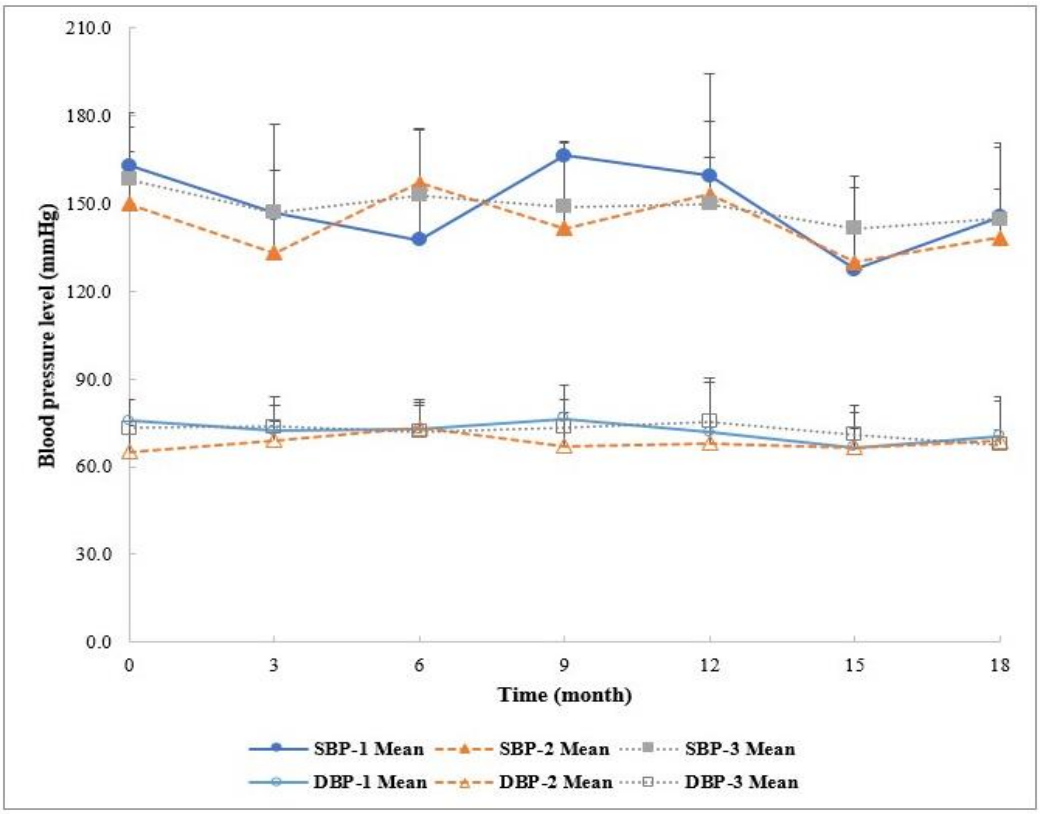

Figure 4. Mean ± SD of systolic blood pressure (SBP) and diastolic blood pressure (DBP) trends over 18 months in the trial groups

3.3 Adverse events

AEs occurred in most patients: 88% in Group 1, 63% in Group 2, and 86% in Group 3 (Table 2). The most frequent events were electrolyte disturbances, hyperuricemia (≥6.8 mg/dL), and increases in serum creatinine. Mild hypokalemia (3 - <3.5 mEq/L) was reported in 13% of patients in Groups 1 and 2 and 21% in Group 3, while moderate hypokalemia (2.5 - <3 mEq/L) occurred in one patient each in Groups 1 and 3.

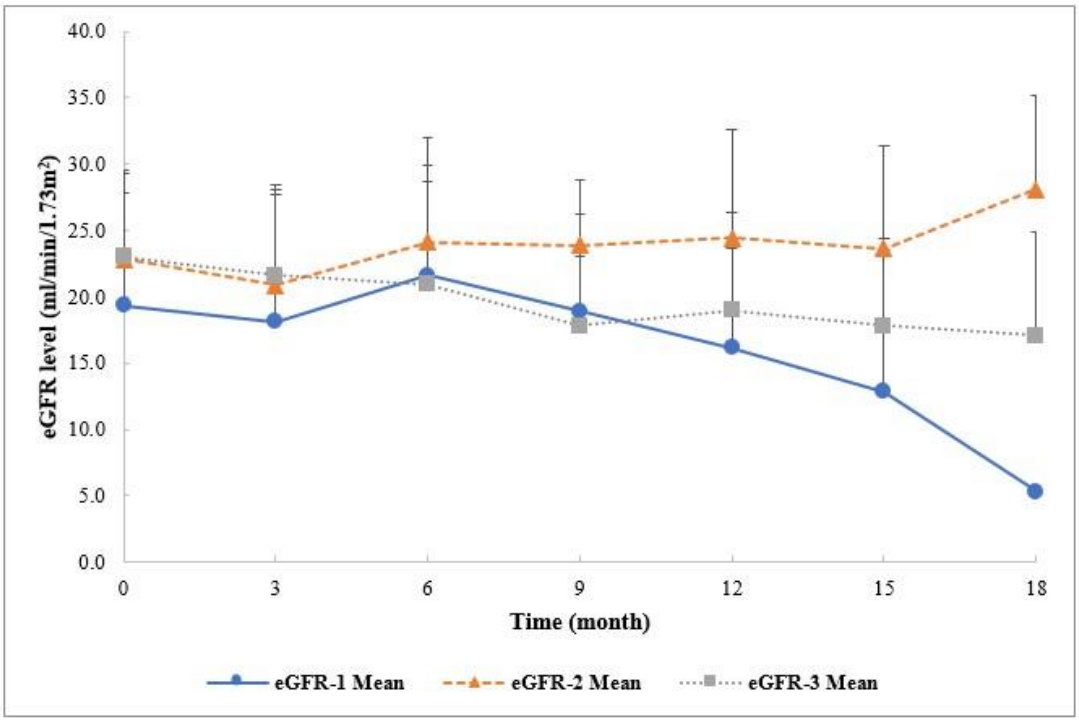

Figure 5. Estimated GFR (eGFR) trends over 18 months in the trial groups

Table 2. Adverse events and serious adverse events during the study period

Event	Group1		Group2		Group3	
	no. of patients with event (%)	no. of events	no. of patients with event (%)	no. of events	no. of patients with event (%)	no. of events
Adverse events						
Total	7 (88)	23	5 (63)	25	12 (86)	51
Hypokalemia (total)	2 (25)	2	1 (13)	1	3 (21)	4
mild(3-3.4)	1 (13)	1	1 (13)	1	3 (21)	3
moderate(2.5-2.9)	1 (13)	1	0 (0)	0	1 (7)	1
Hypomagnesemia	2 (25)	2	1 (13)	1	1 (7)	2
Hyponatremia	3 (38)	4	2 (25)	2	1 (7)	1
Hypercalcemia	0 (0)	0	1 (13)	1	1 (13)	1
Hyperglycemia	0 (0)	0	0 (0)	0	0 (0)	0
Hyperuricemia	5 (63)	7	3 (38)	9	9 (64)	22
Hyperkalemia	1 (13)	1	1 (13)	3	2 (14)	3
Increase in SCr(>25-<50%)	4 (50)	6	3 (38)	7	5 (36)	10
Nocturia	0 (0)	0	1 (13)	1	0 (0)	0
Serious adverse events						
Severe hypokalemia(<2.5)	0 (0)	0	0 (0)	0	0 (0)	0
AKI(>=50%)	1 (13)	1	1 (13)	0	2 (14)	8

No cases of severe hypokalemia (<2.5 mEq/L) were observed. Hyponatremia (<135 mEq/L) was more common in Group 1 (38%), compared with Group 2 (25%) and Group 3 (7%). Hypomagnesemia (<1.8 mg/dL) was detected in 25%, 13%, and 7% of patients in Groups 1, 2, and 3, respectively. Hyperuricemia was prevalent across groups, affecting 63% of Group 1, 38% of Group 2, and 64% of Group 3. Other events, such as hypercalcemia (>10.5 mg/dL) and nocturia, were rare, and no cases of hyperglycemia (both in diabetic and nondiabetic patients) were recorded. Trends over time showed that overall AE rates increased steadily, reaching 100% in Group 3 by 6 months. SAEs were most prominent in Group 1, rising from 12.5% at 3 months to 40% at 12 months and persisting thereafter. Group 2 reported no SAEs throughout follow-up, while Group 3 showed fluctuating rates, peaking at 23.1% at 6 months before declining (Figure 6).

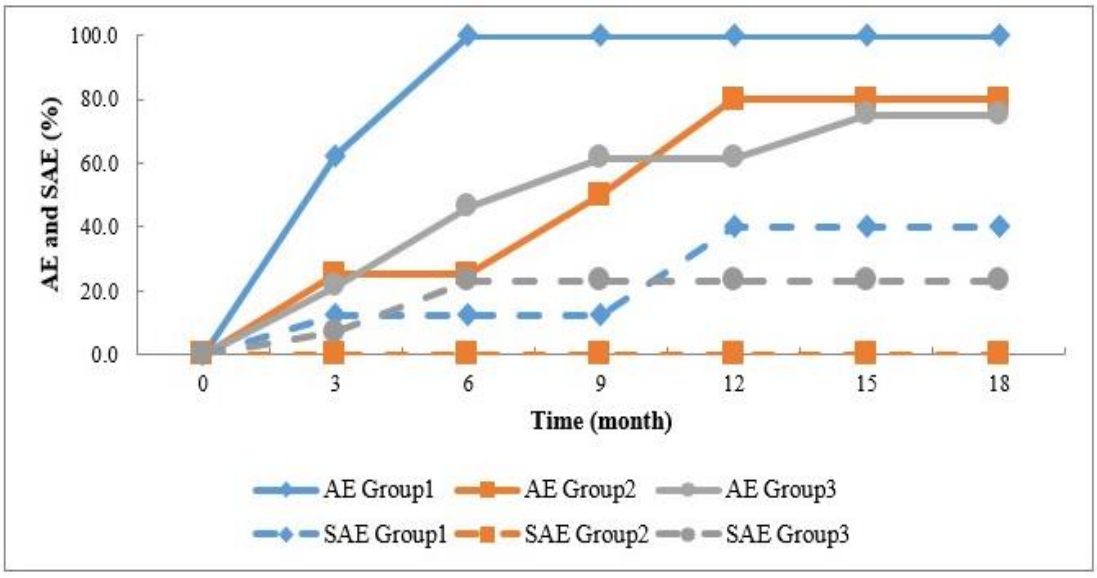

Figure 6. Percentage of adverse event (AE) and severe Adverse event (SAE) over 18 months in the trial groups

4. DISCUSSION

This study investigated the trends of renal outcomes in patients with advanced CKD receiving chlorthalidone with or without furosemide over 18 months. The rationale creation started with a previous randomized controlled trial reported a significant blood pressure reduction with chlorthalidone over 12 weeks, but also suggested a higher risk of SCr increase >25% in patients concurrently receiving furosemide comparing with patients not receiving furosemide (59% vs. 21%) [7]. However, the durability and longer-term implications of these short-term risks, particularly in Asian populations, remained unclear. Our descriptive trends suggest these risks may persist over 18 months in the Thai cohort. Crucially, our findings align with a recent large retrospective cohort study in Taiwan [8], which reported that loop diuretic use in stage 3-5 CKD patients was associated with higher risks of hospitalization for acute renal failure, end-stage renal disease on dialysis, and mortality compared to thiazide monotherapy. The consistency between the trends observed in our group 1 (chlorthalidone with unchanged furosemide) and the outcomes reported in the Taiwanese cohort supports the concern that diuretic combinations without careful modulation may be detrimental to preserving residual renal function in Asian patients, potentially indicating the risk of rapid CKD progression, and greater AE and SAE rates. These findings suggested that continued co-administration without furosemide dose adjustment accelerated a renal function decline and increased adverse event risk, as mentioned in the study of Agarwal R and colleagues [7]. In contrast, Group 2 (chlorthalidone with reduced or discontinued furosemide) demonstrated stable SCr trends, fewer patients with >25% increases, slightly eGFR elevation, and consistently lower SAE rates, indicating that dose adjustment may mitigate risk. Group 3 (chlorthalidone monotherapy) showed some variability in renal outcomes but generally lower prevalence of SCr elevation, slightly eGFR reduction, and fewer AEs than Group 1, which parallels the safer profile of thiazide diuretics noted in the Taiwanese study. Although not a primary endpoint, SBP trends showed reductions in all groups, with slightly greater reductions in Groups 2 and 3 compared to Group 1. This suggested that chlorthalidone could provide blood pressure control even with furosemide dose adjustment.

However, this study has several limitations. First, baseline differences in eGFR across treatment groups may have influenced outcomes, as patients on furosemide generally had lower baseline eGFR than those not receiving it. Second, the retrospective cohort observational design, small sample size, and low completion rates further limit the ability to perform inferential statistics and interpretation of observed trends. Finally, the reliance on surrogate endpoints such as SCr and albuminuria, while useful, may not capture the full spectrum of patient-centered outcomes. Future studies with larger cohorts, randomized controlled designs, and appropriate adjustment for baseline differences are needed to confirm these findings and minimize confounding.

5. CONCLUSIONS

This retrospective cohort study evaluated the impact of chlorthalidone therapy, with or without furosemide, in Thai patients with stage 4–5 CKD over 18 months. The findings revealed that co-administration of chlorthalidone and furosemide without dose adjustment showed a trend of progressive rise in SCr and a higher frequency of >25% SCr elevations. In contrast, patients in whom the furosemide dose was adjusted or discontinued demonstrated more stable renal function, while chlorthalidone monotherapy resulted in only a moderate increase in SCr. In clinical practice, the observed trends suggest the importance of reassessing the furosemide dose after chlorthalidone initiation. Individualized adjustment of furosemide, together with close renal function monitoring, may help balance blood pressure control and renal tolerance in patients with advanced CKD.

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Author contribution

TC: study conceptualization and design, methodology, data analysis and interpretation, writing, revision.
PT: study conceptualization and design, methodology, data analysis, and interpretation.
WN: study conceptualization and design, methodology, data analysis and interpretation, article structuring, writing, revision, approval, and supervision.

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Conflict of interest

None to declare.

Ethics approval

The study protocol was approved by the Human Research Ethics Committee, Faculty of Medicine

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