

Association Between Nocturnal Blood Pressure Dipping and Renal Injury: A Systematic Literature Review

Dony Prasetya Nugraha^{1*}, Hery Emria²
Rumah Sakit Pertamina Tarakan, Indonesia¹
Rumah Sakit Pusat Pertamina, Indonesia²
Email: donypn360@gmail.com^{1*}

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ABSTRACT

This systematic literature review synthesized quantitative evidence on the association between nocturnal blood pressure (BP) dipping patterns and renal injury, disease progression, and severity. PubMed was searched using structured search terms related to nocturnal BP, ambulatory blood pressure monitoring (ABPM), and renal outcomes. The search included original quantitative studies published in English between 2015 and 2026. Study eligibility was determined using the Population, Exposure, Comparator, and Outcome (PECO) framework, and the review was reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. Methodological quality was assessed using the QualSyst appraisal tool. [AUTHOR DATA REQUIRED: Specify the exact date of the final PubMed search and clarify the study selection and quality assessment procedures, including the number of reviewers involved.] A total of 596 records were identified, of which 35 primary studies were included in the review. However, discrepancies in the intermediate PRISMA screening counts required verification against the original screening records. Across the included studies, nondipping and, particularly, reverse-dipping (riser) patterns were generally associated with albuminuria, reduced estimated glomerular filtration rate (eGFR), structural kidney injury, chronic kidney disease (CKD) progression, kidney failure, initiation of dialysis, and composite kidney outcomes. Conversely, a greater physiological decline in nocturnal BP was generally associated with more favorable renal outcomes. Nevertheless, the magnitude and independence of these associations varied according to patient population, CKD stage, outcome definitions, dipping thresholds, absolute nighttime BP, 24-hour BP burden, BP variability, and statistical adjustment methods. Overall, the findings suggested that nocturnal BP dipping patterns provided complementary prognostic information for renal risk assessment rather than serving as independent or definitive determinants of renal outcomes. The interpretation of these findings was limited by substantial clinical and methodological heterogeneity, the predominance of observational study designs, the restriction to English-language publications, and the exclusive use of PubMed as the literature database.

INTRODUCTION

Blood pressure (BP) follows a circadian rhythm influenced by variations in autonomic nervous system activity, sleep–wake cycles, vascular tone, renal sodium handling, and hemodynamic demands throughout the 24-hour period.¹ In healthy adults, systolic and diastolic BP generally decrease during sleep. However, some individuals exhibit an attenuated nocturnal decline (nondipping), an increase in nighttime BP (reverse dipping or a riser pattern), or an excessive nocturnal decline (extreme dipping). These circadian BP phenotypes have

attracted considerable clinical attention because conventional office BP measurements cannot adequately characterize nocturnal BP exposure and may consequently overlook important components of cardiovascular and renal risk.

The prevalence of abnormal nocturnal BP patterns represents a substantial public health concern. A 2026 systematic review and meta-analysis involving 449 studies estimated the prevalence of nocturnal hypertension at 45% in the general adult population and 64% among individuals with chronic kidney disease (CKD). The corresponding prevalence estimates for nondipping were 39% and 58%, respectively. These findings emphasize the clinical importance of ambulatory blood pressure monitoring (ABPM), which enables assessment of daytime and nighttime BP, the nighttime-to-daytime BP ratio, and the magnitude of nocturnal BP decline. Unlike conventional office measurements, ABPM provides a more comprehensive characterization of circadian BP patterns and nocturnal hemodynamic exposure.

The kidneys function as both regulators of BP homeostasis and target organs of hypertension-related damage. Impaired sodium excretion, altered fluid balance, sympathetic nervous system activation, vascular dysfunction, diabetes mellitus, obesity, resistant hypertension, and reduced glomerular filtration may contribute to abnormal nocturnal BP patterns.^{5,8,9} Conversely, sustained nocturnal hypertension and disrupted circadian BP regulation may be associated with albuminuria, reduced estimated glomerular filtration rate (eGFR), structural kidney damage, and progressive CKD. This bidirectional relationship complicates the interpretation of observational findings because abnormal nocturnal dipping may reflect preexisting renal dysfunction, contribute to subsequent kidney disease progression, or represent a combination of both mechanisms.

Recent primary studies have highlighted the complexity of the relationship between nocturnal BP dipping and renal outcomes. Park et al. reported that attenuated nocturnal systolic BP dipping was associated with CKD progression in a high-risk CKD cohort.² However, other investigations suggested that absolute nighttime BP levels and overall 24-hour BP burden could attenuate or demonstrate stronger associations with renal outcomes than categorical dipping status.^{7,10} Variations in CKD stage, diabetes status, antihypertensive treatment, definitions of nocturnal dipping, follow-up duration, and renal outcome measures may contribute to inconsistencies across studies, even when ABPM is used as the primary assessment method. These differences underscore the importance of evaluating nocturnal dipping patterns within the broader context of BP exposure and individual clinical characteristics.

Recent systematic reviews and meta-analyses have examined related aspects of nocturnal BP abnormalities, although their objectives and populations have differed. Show et al. (2026) estimated the global prevalence of nocturnal hypertension and nondipping across diverse populations, primarily emphasizing the epidemiological burden of these phenotypes rather than their specific associations with renal prognosis. Berro et al. (2026) reviewed masked and isolated nocturnal hypertension and reported associations with cardiovascular and target-organ damage, including renal impairment and albuminuria. However, their review encompassed multiple organ systems and populations with varying office BP profiles. Similarly, Jan and Dubwa (2026) synthesized evidence concerning cardiovascular risks associated with masked nocturnal hypertension without specifically examining longitudinal kidney disease progression.

Despite these contributions, important gaps remain in the understanding of nocturnal BP dipping patterns and renal outcomes. Existing evidence syntheses have not comprehensively integrated the full spectrum of nocturnal dipping phenotypes with both cross-sectional indicators of renal injury and longitudinal measures of kidney disease progression and severity. Furthermore, comparisons between categorical dipping classifications, continuous measures of nocturnal BP decline, and absolute nighttime BP levels have not been consistently addressed. A kidney-focused systematic review is therefore warranted to distinguish associations with albuminuria and reduced eGFR from those involving CKD progression, kidney failure requiring kidney replacement therapy, dialysis initiation, and composite kidney outcomes. Such a synthesis may also help clarify whether nocturnal dipping patterns provide additional prognostic information beyond absolute nighttime BP and overall BP burden.

The clinical importance of this distinction lies in the potential consequences of interpreting nocturnal BP abnormalities without adequate consideration of confounding factors. Classifying nondipping or reverse dipping as an independent causal determinant of renal injury without accounting for nighttime BP levels, baseline kidney function, BP variability, diabetes mellitus, and antihypertensive treatment may lead to an overestimation of its prognostic significance. Conversely, relying exclusively on office BP measurements may fail to identify individuals with clinically relevant nocturnal hypertension or abnormal circadian BP patterns despite apparently controlled daytime BP. A systematic evaluation of the available evidence is therefore necessary to determine the consistency of associations between ABPM-derived dipping characteristics and renal outcomes and to identify circumstances in which these associations remain uncertain or are influenced by confounding variables.

The contribution of this systematic literature review lies in its kidney-specific synthesis of two interrelated but distinct outcome domains: existing renal injury and subsequent kidney disease progression or severity. The review examines different approaches to characterizing nocturnal BP patterns, including dipper and nondipper classifications, reverse-dipping or riser patterns, and continuous measures of nocturnal BP decline. It also considers whether reported associations persist after adjustment for absolute nighttime BP, 24-hour BP burden, BP variability, baseline renal function, and other relevant clinical characteristics. By integrating these dimensions, the review seeks to complement existing epidemiological, cardiovascular, and broader target-organ damage reviews while providing a more focused understanding of the relationship between circadian BP regulation and renal health.

Accordingly, this systematic literature review aims to synthesize quantitative evidence regarding the association between nocturnal BP dipping patterns and renal injury and to evaluate how these patterns relate to the progression and severity of kidney disease. Two research questions guide the review: (1) What is the association between nocturnal BP dipping patterns and renal injury? and (2) How are nocturnal BP dipping patterns associated with the progression and severity of renal injury? These questions are intended to distinguish evidence concerning existing renal abnormalities from findings related to longitudinal deterioration in kidney function.

The findings of this review are expected to support clinicians and researchers in interpreting ABPM results among individuals with established kidney disease or an increased risk of renal impairment. Specifically, the review seeks to clarify the potential role of nocturnal BP dipping patterns as complementary indicators of renal risk rather than substitutes for

absolute nighttime BP measurements and established kidney disease risk factors. The synthesis may inform clinical risk stratification and guide future research on circadian BP abnormalities and renal outcomes. Nevertheless, the strength and generalizability of its conclusions depend on the methodological quality of the included studies, differences in study populations and outcome definitions, and the comprehensiveness of the literature search. Because the review was restricted to English-language publications indexed in PubMed, potential language and database selection biases should be considered when interpreting the findings.

METHOD

This study employed a systematic literature review (SLR) approach to identify, critically appraise, and synthesize quantitative evidence on the association between nocturnal blood pressure (BP) dipping patterns and renal outcomes.¹¹ The literature search was conducted exclusively in PubMed using structured search terms and Boolean operators related to circadian BP patterns, nocturnal BP, ambulatory blood pressure monitoring (ABPM), dipping phenotypes, and renal injury. The use of a single database was acknowledged as a methodological limitation. [AUTHOR DATA REQUIRED: Specify the exact date of the final PubMed search.] The complete search strategy was documented to ensure reproducibility.

((("Circadian Rhythm"[tiab] OR "circadian blood pressure"[tiab] OR "circadian BP"[tiab] OR "nocturnal blood pressure"[tiab] OR "night-time blood pressure"[tiab] OR "nighttime blood pressure"[tiab] OR "nocturnal BP"[tiab] OR "sleep blood pressure"[tiab] OR "nocturnal dipping"[tiab] OR "blood pressure dipping"[tiab] OR "dipping pattern*"[tiab] OR "dipper*"[tiab] OR "non-dipper*"[tiab] OR "non-dipping"[tiab] OR "reverse dipper*"[tiab] OR "reverse dipping"[tiab] OR "extreme dipper*"[tiab] OR "extreme dipping"[tiab] OR "riser blood pressure"[tiab] OR "rising pattern*"[tiab] OR "ABPM"[tiab]) AND ("acute kidney injury"[tiab] OR "renal insufficiency"[tiab] OR "chronic kidney disease"[tiab] OR "renal injury"[tiab] OR "kidney injury"[tiab] OR "renal damage"[tiab] OR "kidney damage"[tiab] OR "renal dysfunction"[tiab] OR "nephropathy"[tiab] OR "kidney disease"[tiab] OR "CKD"[tiab] OR "AKI"[tiab] OR "renal failure"[tiab] OR "kidney failure"[tiab]))

The study framework was also structured using the PECO approach to clearly define the key components of the review:

Table 1. PECO Criteria

.	Description
P (Population)	Adult human patients (aged ≥ 18 years) with or without comorbidities (such as hypertension, diabetes, or cardiovascular disease).
E (Exposure)	Quantitative assessment of nocturnal or night-time blood pressure dipping status measured strictly via the gold standard of Ambulatory Blood Pressure Monitoring (ABPM), focusing on circadian dipping patterns (non-dipping, reverse-dipping /riser, or extreme-dipping).
C (Comparison)	Direct comparison between different dipping patterns or evaluating nocturnal dipping as a continuous variable.
O (Outcome)	Renal injury outcomes, including: CKD/progression, eGFR, albuminuria/proteinuria, ESRD, AKI.

Source: Authors' review protocol.

Clear inclusion and exclusion criteria also established to ensure the selection of relevant, high-quality, and methodologically appropriate studies aligned with the objectives of this study, including:

Table 2. Inclusion & Exclusion Criteria

Criteria	Inclusion Criteria	Exclusion Criteria
Timeframe	2015-2026	Studies published before 2015.
Focus	Studies evaluating the association, relationship, or correlation between the quantitative classification of nocturnal blood pressure dipping status and renal injury outcomes.	Studies examining only non-renal outcomes, without reporting specific renal injury markers.
Study Type	Original studies (quantitative research designs) that collect and analyze primary data, such as: Prospective cohort studies, Retrospective cohort studies, Cross-sectional studies, Case-control studies, Randomized controlled trials (RCTs), Non-randomized clinical trials, etc	Reviews, SLR, meta-analyses, scoping reviews, editorials, opinion, incomplete conference abstracts, book chapters, case report
Accessibility	Full text	Closed/ restricted article
Language	English.	Non-English articles without professional translations.

Source: Authors' review protocol.

Study selection, data extraction, and disagreement-resolution procedures were conducted as part of the systematic review process. [AUTHOR DATA REQUIRED: Specify the number of independent reviewers involved in title and abstract screening, full-text screening, data extraction, and methodological quality assessment. Describe the procedures used to resolve disagreements and identify the data extraction form and variables collected.] The review's protocol registration status also requires clarification, including the PROSPERO registration number or another relevant registry identifier, if applicable, or confirmation that the review was not prospectively registered.

Methodological quality was assessed using the QualSyst tool for quantitative studies.¹³ [AUTHOR DATA REQUIRED: Specify the scoring criteria, handling of nonapplicable items, calculation of total quality scores, thresholds used to classify methodological quality, and procedures followed by the reviewers.] The assessment criteria should be consistent with the study-level quality ratings presented in the Results section to ensure transparency and reproducibility.

A meta-analysis was not performed because of substantial clinical and methodological heterogeneity among the included studies, particularly regarding study populations, chronic kidney disease (CKD) stages, nocturnal blood pressure dipping definitions, renal outcomes, follow-up durations, and statistical adjustment methods. Therefore, a structured narrative synthesis was conducted to summarize the direction and magnitude of reported associations and study-specific effect estimates without calculating a pooled effect size. The findings were interpreted in conjunction with the methodological quality assessments and heterogeneity identified across the included studies.

RESULTS AND DISCUSSION

Study Selection

The available screening summary reports 596 records identified from PubMed and 35 studies included in the final review. However, the intermediate PRISMA counts do not

reconcile mathematically: 0 records are reported as removed before screening, yet only 429 records are reported as screened, and the same 429 are then reported as reports sought for retrieval despite 167 title/abstract exclusions. Because the disposition of the unaccounted records cannot be reconstructed from the manuscript alone, the selection flow should not be editorially corrected without the original screening database. [AUTHOR DATA REQUIRED: reconcile every PRISMA count from identification through inclusion and update Table 3/flow diagram accordingly.] The 35 included studies in Table 4 should also be cross-checked against the final screening dataset.

Table 3. Study selection process based on the PRISMA 2020 framework

Stage	Description	n
Identification	Records identified from PubMed	596
	Records removed before screening: duplicate records	0
	Records removed before screening: other reasons	0
Screening	Records screened	429
	Records excluded (by title and abstract)	167
	Reports sought for retrieval	429
	Reports not retrieved	8
	Reports assessed for eligibility	421
	Reports excluded: wrong study type	101
	Reports excluded: wrong population	43
	Reports excluded: wrong exposure	128
	Reports excluded: wrong outcome	119
Included	Studies included in review	35

Source: Authors' screening records; PRISMA counts require author reconciliation before publication.

Included Studies

The characteristics of the 35 included studies are presented in Table 4. Sample sizes ranged from 30 to 2,143 participants. The studies were conducted in 11 countries, with the majority from China (10 studies), Japan (5 studies), South Korea (5 studies), and Italy (4 studies); 3 studies were conducted in the United States, 2 each in Poland and India, and 1 each in France, Denmark, Spain, and Turkey.

The study populations differed clinically, including patients with CKD, hypertension, diabetes mellitus, and population-based cohorts. All studies were quantitative primary research; therefore, the QualSyst tool for quantitative studies was used for risk-of-bias assessment. Most of the included evidence consisted of cross-sectional studies (9 studies), prospective cohort studies (8 studies), and retrospective cohort studies (4 studies). Other quantitative designs included prospective observational cohorts, retrospective cross-sectional multicentre studies, propensity score-matched analyses, and randomised or randomised open-label designs, and two further studies used a randomised controlled trial design. Some studies specifically recruited patients with established CKD, including patients across CKD stages G1–G5 and more advanced disease categories, whereas others included patients with hypertension, diabetes mellitus, or both, or used population-based cohorts that did not restrict eligibility to participants with established renal disease. The most frequent comorbidities were hypertension, diabetes mellitus, dyslipidaemia, cardiovascular disease, obesity, and history of smoking.

All studies measured nocturnal BP using ABPM, although the operational definitions were not consistent. In several studies, participants were classified according to the percentage reduction in nocturnal BP relative to daytime BP, commonly using a 10% reduction to separate dipper from non-dipper patterns. Other studies used the nocturnal-to-daytime SBP ratio or analysed nocturnal BP decline as a continuous variable. Some studies categorised participants into extreme-dipper, dipper, non-dipper, and reverse-dipper groups, while others used simpler groupings, e.g., dipper versus non-dipper.

Table 4. Characteristics of the included studies (n = 35)

Study	Country	Study design	Population (n)	Dipping pattern	Main findings
Park et al., 2024	South Korea	Prospective cohort study	CKD G1-G5 with high CVD risk (n = 1,061)	Ext. dipper (≤ 0.8), Dipper (0.8-0.9), Non-dipper (0.9-1.0), Rev. dipper (> 1.0)	Non/reverse dippers have higher CKD progression risk. Emphasizes nocturnal SBP decline importance. Blunted nocturnal BP reduction independently associated with worsening CKD risk. Nighttime aortic SBP/PWV are superior estimators.
Kusunoki et al., 2020	Japan	Cross-sectional study	Essential HTN patients (n = 184)	% day-to-night SBP decrease, Nighttime brachial/aortic SBP	Non-dipping with normotension doesn't predict ESRD. 24-h average BP control is more essential. Nondipping not associated with CV/renal damage in optimal 24h-BP control. Optimal control mitigates nondipping effects. Reverse dipping doubles mortality risk and is associated with CKD/CAN. Strong predictor of reduced survival.
Ida et al., 2019	Japan	Retrospective cohort study	CKD patients with ABPM (n = 1,107)	Dipper ($\geq 10\%$) vs. Non-dipper ($< 10\%$). 4 groups with 24-h BP control.	Bedtime antihypertensives revert non-dippers to dippers and slow eGFR decline. SBP above goal or absence of dipping associated with higher CV/CKD risks. Targeted interventions needed. Non-dipping in normotensive CKD doesn't predict rapid eGFR decline. Overall 24-h SBP control is essential.
Lopez-Sublet et al., 2019	France	Prospective cohort study (cross-sectional analysis of 4th visit)	Healthy cohort (Stanislas) (n = 1,334)	Dipper ($\geq 10\%$ reduction) vs. Non-dipper ($< 10\%$ reduction)	
Chiriaco et al., 2022	Italy	Retrospective cohort study	T1DM/T2DM patients (CHAMPION) (n = 349)	Dipping, Nondipping, Reverse-dipping	
Tiwari et al., 2021	India	Prospective randomized controlled trial	HTN + CKD 3-4, baseline non-dippers (n = 75)	Dipper ($\geq 10\%$) vs. Non-dipper ($< 10\%$)	
Borrelli et al., 2022	Italy	Prospective observational cohort study	HTN + non-dialysis CKD 2-5 (n = 906)	Nocturnal dipping (< 0.9) vs. Nondipping (≥ 0.9)	
Kado et al., 2019	Japan	Retrospective cohort study	CKD G1-G5 with ABPM (n = 676)	Dipper ($\geq 10\%$) vs. Non-dipper ($< 10\%$). 4 clinical categories.	

Study	Country	Study design	Population (n)	Dipping pattern	Main findings
Pluta & Stróżecki, 2025	Poland	Prospective cohort study	CKD 1-3 patients (n = 87)	Dipper+Ext. dipper (D/ED), Non-dipper (ND), Rev. dipper (RD)	Non-dipper and reverse dipper are common in CKD 1-3. Reverse-dipper associated with faster kidney function decline. Subclinical DN
Zhang et al., 2015	China	Retrospective cross-sectional study	T2DM patients stratified by kidney status (n = 99)	Dipper ($\geq 10\%$) vs. Non-dipper ($< 10\%$). SBP/DBP fall continuous.	associated with abnormal circadian BP. Non-dipping increases with early-stage renal impairment.
Hjortkjær et al., 2022	Denmark	Prospective observational cohort study	T1DM patients (n = 654)	Dipper ($\geq 10\%$) vs. Non-dipper ($< 10\%$)	Non-dipping independently associated with increased risk of mortality and kidney disease in T1DM.
Borrelli et al., 2024	Italy	Multicenter prospective study	T2DKD patients (GFR > 25) (n = 83)	Normal dipping (< 0.9) vs. Abnormal dipping (≥ 0.9)	Dapagliflozin 10 mg/day reduces nighttime SBP, albuminuria, and improves circadian BP profile in DKD.
Nakai et al., 2016	Japan	Retrospective cohort study	Mod-severe CKD with 24h ABPM (n = 124)	Extreme dipper, Dipper, Non-dipper, Riser	Riser BP pattern (35%) predicts progression to dialysis. Risk compounded with elevated BNP or DM.
Cho et al., 2021	South Korea	Cross-sectional study	Treated HTN, controlled BP, no advanced CKD (n = 995)	Ext-dipper ($\geq 20\%$), Dipper (10- $< 20\%$), Non-dipper (0- $< 10\%$), Rev-dipper ($< 0\%$)	Insufficient nocturnal SBP dipping associated with higher odds of albuminuria and decreased kidney function.
Hermida et al., 2018	Spain	Prospective, randomized, open-label, blinded endpoint (PROBE) trial	HTN without baseline CKD (n = 2078)	Dipper ($\geq 10\%$) vs. Non-dipper ($< 10\%$)	Bedtime chronotherapy improves BP control and reduces incident CKD risk by 72%.
Jhee et al., 2021	South Korea	Prospective cohort study	CKD 1-5 with proteinuria or eGFR < 60 (n = 751)	Dipper (10-20%), Ext. dipper ($> 20\%$), Non-dipper (0-10%), Rev. dipper ($< 0\%$)	Elevated suPAR levels associated with kidney decline, mainly in reverse dippers.
Cheng et al., 2019	China	Population-based prospective cohort study	Primary HTN adults (n = 1042)	Nighttime BP decline continuous or Low ($\leq 9.42\%$) vs High ($> 9.42\%$)	Blunted nocturnal BP fall predicts renal function decline and rapid kidney injury.
Gupta et al., 2022	India	Cross-sectional study	Normotensive T2DM patients (n = 150)	Dipper (10-20%), Ext. dipper ($> 20\%$), Non-dipper (0-10%), Rev. dipper ($< 0\%$)	Masked/nocturnal HTN strongly associated with early DN and LVH in normotensive T2DM.
Oreschak et al., 2020	USA	Retrospective, cross-sectional study	Heart transplant recipients (> 6 months) (n = 30)	Systolic/Diastolic dipper ($> 10\%$) vs. nondipper ($\leq 10\%$)	Nondipping BP patterns highly prevalent and associated with subclinical kidney damage and diastolic dysfunction.

Study	Country	Study design	Population (n)	Dipping pattern	Main findings
McMullan et al., 2015	USA	Prospective cohort study	African Americans with normal renal function (JHS) (n = 603)	Continuous nocturnal SBP dipping. ABPM phenotypes evaluated.	Blunted nocturnal SBP decline predicts incident CKD and faster eGFR decline in African Americans.
Yu et al., 2023	China	Propensity score-matched cross-sectional study	T2DKD matched with PGD (n = 1002)	Ext. dipper (>20%), Normal dipper (10-20%), Non-dipper (0-10%), Rev. dipper (<0%)	T2DKD patients have higher prevalence of abnormal BP patterns and target-organ damage than PGD patients.
Haruhara et al., 2015	Japan	Retrospective cross-sectional study	CKD with renal biopsy and 24h ABPM (n = 138)	Ext. dipper (4%), Dipper (38%), Non-dipper (46%), Rev. dipper (12%)	Severity of interstitial fibrosis/tubular atrophy independently associated with ABPM abnormalities in CKD.
Rahman et al., 2020	USA	Prospective multicenter cohort study	CKD (eGFR 20-70) in CRIC Study (n = 1,502)	Dipper (37.2%), Nondipper (47.1%), Rev. dipper (15.6%). ABPM Profiles evaluated.	ABPM metrics (masked uncontrolled HTN, reverse dipper) strongly associated with CV outcomes and kidney disease progression.
Lin et al., 2016	China	Cross-sectional study	IgAN patients with normal 24h BP (n = 330)	Dipper (36.4%), Ext. dipper (2.1%), Non-dipper (52.1%), Riser (9.4%)	Abnormal circadian rhythm and nocturnal HTN highly prevalent and associated with renal decline in IgAN.
Zeng et al., 2019	China	Cross-sectional study	Primary HTN patients (n = 198)	Normal rhythm (0-10% decline): 66.7%. Disorder: 33.3%.	Circadian rhythm disorder correlated with target organ damage (cardiac remodeling, vascular injury, renal impairment).
Cha et al., 2020	South Korea	Prospective cohort study	HTN + CKD (APrODiTe-2) (n = 378)	Dipper (22.2%), Non-dipper (31.3%), Rev-dipper (35.0%), Ext-dipper (11.4%)	Higher BP burden associated with faster GFR decline. Restoring dipping pattern improves GFR preservation.
Seçkin et al., 2025	Turkey	Prospective single-center cohort study	Untreated new HTN patients (n = 135)	Dipper ($\geq 10\%$) vs. Non-dipper ($< 10\%$). 50% dippers, 50% non-dippers.	Non-dipper phenotype associated with higher target organ damage (heart disease, nephropathy) and impaired left atrial strain.
Song et al., 2020	China	Cross-sectional study	Non-dialysis CKD patients (n = 823)	Dipper (31.0%), Non-dipper (69.0%). 4 groups based on MBPS and dipping.	MBPS and non-dipping contribute to target organ damage through different pathways (MBPS for renal, non-dipping for LVH).
Cha et al., 2017	South Korea	Prospective cohort study	HTN + CKD patients (n = 378)	Rev-dipper (35.0%), Non-dipper (31.3%), Dipper (22.2%), Ext-dipper (11.4%)	Uncontrolled BP and abnormal dipping highly prevalent. Better BP control associated with slow eGFR progression.

Study	Country	Study design	Population (n)	Dipping pattern	Main findings
Wang et al., 2016	China	Prospective cohort study	Non-dialysis CKD inpatients (n = 588)	Dipper (34.7%), Non-dipper (43.5%), Rev-dipper (18.0%), Ext-dipper (3.7%)	Reverse dipping associated with worse prognosis (renal/CV events, mortality) independent of 24-h SBP.
Carollo et al., 2025	Italy	Cross-sectional study	Essential HTN patients (n = 389)	Inverted dippers, Non-dippers, Dippers, Extreme dippers.	Max SBP slope independently associated with early subclinical renal damage. Blunted diastolic BP dipping predicts faster kidney function deterioration in non/mild-proteinuric CKD.
Kuczera et al., 2018	Poland	Prospective cohort study	CKD 3-4 with mild/no proteinuria (n = 37)	Diurnal SBP/DBP dipping (mmHg). Baseline non-dipper 27%.	Nocturnal HTN, rather than nondipping, is strongly associated with target organ damage.
Wang et al., 2015	China	Cross-sectional study	CKD inpatients (n = 1322)	Normal SBP/dipper (20.7%), Normal SBP/nondipper (19.5%), NSH/dipper (9.8%), NSH/nondipper (50.0%)	24-h DBP ARV predicts rapid renal function decline and ESRD in DKD.
Hu et al., 2026	China	Retrospective cohort study	DKD patients with 24h ABPM (n = 2143)	Nocturnal non-dipping: 54.3%. Dynamic changes in DBPV evaluated.	Abnormal nocturnal dipping and increased RDW strongly associated with early renal damage.
Bi et al., 2022	China	Cross-sectional study	Untreated uncomplicated HTN patients (n = 513)	Dippers (48.0%), Non-dippers (45.0%), Rev-dippers (5.3%), Deep-dippers (1.8%)	

Source: Authors' synthesis of the included studies.

Risk of Bias Assessment

The risk-of-bias judgements for each of the 35 studies across the 11 domains are presented in Table 5, and the proportion of judgements per domain is summarised in Table 6.

Table 5. Risk of bias assessment using QualSyst for quantitative studies

Study	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	Overall I
Park et al. (2024)	+	+	+	+	+	+	+	+	+	+	+	+
Kusunoki et al. (2020)	+	–	+	+	+	+	+	+	+	+	+	+
Ida et al., (2019)	+	+	+	+	+	+	+	+	+	+	+	+
Lopez-Sublet et al. (2019)	+	+	+	+	+	+	+	+	+	+	+	+
Chiriaco et al. (2022)	+	+	+	+	+	+	+	+	+	+	+	+
Tiwari et al. (2021)	+	+	+	+	+	+	+	+	+	+	+	+
Borrelli et al. (2022)	+	+	+	+	+	+	+	+	+	+	+	+
Kado et al. (2019)	+	+	+	+	+	+	+	+	+	+	+	+
Pluta & Stróżecki (2025)	+	+	+	+	+	–	+	+	+	+	+	+
Zhang et al. (2015)	+	–	+	+	+	–	+	+	+	+	+	+
Hjortkjaer et al. (2022)	+	+	+	+	+	+	+	+	+	+	+	+
Borrelli et al. (2024)	+	+	+	+	+	+	+	+	+	+	+	+
Nakai et al. (2016)	+	+	+	+	+	–	+	+	–	+	+	+
Cho et al. (2021)	+	–	+	+	+	+	+	+	+	+	+	+
Hermida et al. (2018)	+	+	+	+	+	+	+	+	+	+	+	+
Jhee et al. (2021)	+	+	+	+	+	+	+	+	+	+	+	+

Study	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	Overall I
Cheng et al. (2019)	+	+	+	+	+	+	+	+	+	+	+	+
Gupta et al. (2022)	+	–	+	+	+	–	+	–	×	+	+	–
Oreschak et al. (2020)	+	–	+	+	+	–	+	+	+	+	+	+
McMullan et al. (2015)	+	+	+	+	+	+	+	+	+	+	+	+
Yu et al. (2023)	+	–	+	+	+	+	+	+	+	+	+	+
Haruhara et al. (2015)	+	–	+	+	+	–	+	+	+	+	+	+
Rahman et al. (2020)	+	+	+	+	+	+	+	+	+	+	+	+
Lin et al. (2016)	+	–	+	+	+	+	+	+	+	+	+	+
Zeng et al. (2019)	+	–	+	+	+	–	+	–	×	+	+	–
Cha et al. (2020)	+	+	+	+	+	–	+	+	+	+	+	+
Seçkin et al. (2025)	+	+	+	+	+	+	+	+	+	+	+	+
Song et al. (2020)	+	–	+	+	+	+	+	+	+	+	+	+
Cha et al. (2017)	+	+	+	+	+	–	+	+	+	+	+	+
Wang et al. (2016)	+	+	+	+	+	+	+	+	+	+	+	+
Carollo et al. (2025)	+	–	+	+	+	–	+	+	+	+	+	+
Kuczera et al. (2018)	+	+	+	+	+	–	+	+	+	+	+	+
Wang et al. (2015)	+	–	+	+	+	+	+	+	+	+	+	+
Hu et al. (2026)	+	+	+	+	+	+	+	+	+	+	+	+
Bi et al. (2022)	+	–	+	+	+	–	+	+	+	+	+	+

Source: Authors' QualSyst assessment of included studies.

+ = low; – = moderate; × = high. D1: question or objective sufficiently described; D2: design evident and appropriate to answer the study question; D3: method of subject selection or source of information/input variables described and appropriate; D4: subject characteristics or input variables sufficiently described; D5: outcome and exposure measures well defined and robust to measurement/misclassification bias; D6: sample size appropriate; D7: analysis described and appropriate; D8: some estimate of variance reported for the main results; D9: controlled for confounding; D10: results reported in sufficient detail; D11: results support the conclusions.

Table 6. Summary of risk of bias judgements per domain (n = 35 studies)

Domain	Item	Low, n (%)	Moderate, n (%)	High, n (%)
D1	Question or objective sufficiently described	35 (100.0%)	0 (0.0%)	0 (0.0%)
D2	Design evident and appropriate to answer the study question	22 (62.9%)	13 (37.1%)	0 (0.0%)
D3	Method of subject selection or source of information/input variables described and appropriate	35 (100.0%)	0 (0.0%)	0 (0.0%)
D4	Subject characteristics or input variables/information sufficiently described	35 (100.0%)	0 (0.0%)	0 (0.0%)
D5	Outcome and exposure measures well defined and robust to measurement/misclassification bias	35 (100.0%)	0 (0.0%)	0 (0.0%)
D6	Sample size appropriate	23 (65.7%)	12 (34.3%)	0 (0.0%)
D7	Analysis described and appropriate	35 (100.0%)	0 (0.0%)	0 (0.0%)
D8	Some estimate of variance reported for the main results/outcomes	33 (94.3%)	2 (5.7%)	0 (0.0%)
D9	Controlled for confounding	32 (91.4%)	1 (2.9%)	2 (5.7%)
D10	Results reported in sufficient detail	35 (100.0%)	0 (0.0%)	0 (0.0%)
D11	Results support the conclusions	35 (100.0%)	0 (0.0%)	0 (0.0%)
Overall	Overall	33 (94.3%)	2 (5.7%)	0 (0.0%)

Source: Authors' summary of the QualSyst assessment.

Most studies were judged to have a low risk of bias across the assessed domains. All studies received a low-risk judgement for D1 (question or objective sufficiently described), D3 (subject selection or source of information appropriately described), D4 (subject characteristics sufficiently described), D5 (outcome and exposure measures adequately defined), D7 (analysis described and appropriate), D10 (results reported in sufficient detail), and D11 (results supporting the conclusions). This suggests that the research questions were generally clearly defined, with suitable descriptions of methodology, sufficient reporting of participant characteristics and outcome measures, and adequate presentation of results.

Some methodological limitations were identified in specific domains. The most frequent areas of concern were D2 (appropriateness of study design) and D6 (appropriateness of sample size), and a small number of studies also received moderate judgements for D8 (reporting of variance estimates). The most prominent concern was D9 (control for confounding), for which Gupta et al. (2022) and Zeng et al. (2019) received high-risk judgements. Overall, 33 of 35 studies were rated as having low risk of bias and 2 studies (Gupta et al., 2022; Zeng et al., 2019) were rated as having moderate risk of bias^{6,14}. No study was classified as having a high overall risk of bias. The overall assessment indicates generally favourable methodological quality, while the identified concerns regarding sample size, study design, variance reporting, and control of confounding should be considered when interpreting the findings.

Association Between Nocturnal Blood Pressure Dipping Patterns and Renal Injury

The included studies demonstrated that abnormal nocturnal BP dipping was generally associated with more severe renal damage, although the magnitude and consistency of the association differed across populations and renal endpoints, as presented in Table 7.

Table 7. The association between nocturnal blood pressure dipping patterns and renal injury (RQ1)

Studies	Renal injury outcome	Main effect estimate
Cho et al. (2021); Chiriaco et al. (2022) ; Bi et al. (2022)	Albuminuria, reduced eGFR, CKD stages III–IV, and early renal damage/microalbuminuria.	Cho et al.: non-dipper OR 1.67 (95% CI 1.20–2.32) for albuminuria and OR 1.98 (1.07–3.08) for reduced eGFR; reverse-dipper OR 1.73 (1.04–2.60) and OR 2.02 (1.06–3.84), respectively. Chiriaco et al.: CKD III–IV prevalence 9% in dippers, 14% in non-dippers, and 22% in reverse dippers (p trend = .039). Bi et al.: nighttime SBP drop rate had the highest feature importance (0.39149) for early renal damage.
McMullan et al. (2015); Carollo et al. (2025)	Incident CKD and subclinical renal damage, including microalbuminuria and mildly-to-moderately reduced GFR.	McMullan et al.: adjusted OR 0.55 (95% CI 0.32–0.96) per 10% greater SBP dipping. Carollo et al.: adjusted OR 0.683 (0.535–0.872) per 1-SD greater SBP decline; $\beta = -0.142$ (p = .003) for log AER.
Hermida et al. (2018)	Incident CKD, incident diminished eGFR, and incident albuminuria.	Adjusted HR 0.28 (95% CI 0.22–0.35) for incident CKD; 0.27 (0.21–0.36) for diminished eGFR; 0.30 (0.18–0.50) for albuminuria.

Studies	Renal injury outcome	Main effect estimate
Zhang et al. (2015); Kusunoki et al. (2020)	Subclinical/early diabetic nephropathy and high/very-high CKD risk.	Zhang et al.: nocturnal SBP fall OR 0.851 (95% CI 0.707–1.023), $p = .086$. Kusunoki et al.: day-to-night SBP decrease declined from 6.9% to 2.1% across increasing CKD risk (p trend $< .01$); adjusted nighttime aortic SBP OR 1.50 (1.02–2.27).
Lopez-Sublet et al. (2019); Gupta et al. (2022); Oreschak et al. (2020)	Reduced eGFR, microalbuminuria, CKD, and early diabetic nephropathy.	Lopez-Sublet et al.: eGFR OR 1.01 (0.69–1.48); albuminuria OR 0.78 (0.43–1.42). Gupta et al.: dipping pattern $p = .193$. Oreschak et al.: systolic non-dipping OR 1.95 (0.12–32.68) for microalbuminuria and OR 0.37 (0.07–1.99) for CKD.
Song et al. (2020); Wang et al. (2015)	Impaired renal function / reduced eGFR < 60 mL/min/1.73 m ² .	Song et al.: adjusted $\beta -0.017$ (95% CI -0.071 to 0.036), $p = .525$. Wang et al.: adjusted OR 1.089 (0.735–1.612), $p = .670$.
Haruhara et al. (2015); Lin et al. (2016); Zeng et al. (2019)	Renal histopathological injury, tubulointerstitial fibrosis, renal ischemia, reduced eGFR, creatinine clearance, and nephropathy.	Haruhara et al.: nighttime hypertension OR 5.31 (1.25–22.5) for IF/TA grade 2; abnormal circadian rhythm increased from ~37% to ~69% with increasing IF/TA severity ($p = .032$). Lin et al.: abnormal circadian rhythm associated with renal tissue injury OR 2.235 (1.082–4.676) and TIF $> 25\%$ OR 2.096. Zeng et al.: creatinine clearance 70.7 vs. 86.1 mL/min and nephropathy 19.7% vs. 3.0% ($p < .0001$).
Yu et al. (2023)	Macroalbuminuria.	Nocturnal hypertension adjusted OR 1.848 (95% CI 1.233–2.771).

Source: Authors' synthesis of included studies for RQ1.

(SMJ et al., 2021) found that non-dippers and reverse-dippers showed higher odds of albuminuria and lower eGFR⁴, and Chiriaco et al. (2022) reported an increasing prevalence of CKD stages III–IV from dipper to non-dipper to reverse-dipper groups¹⁵. Bi et al. (2022) also found that the nocturnal fall rate of SBP was the most significant predictor of early renal damage in their predictive model¹⁶. These findings suggest that the physiological nocturnal BP fall may be blunted or reversed when there is clinically significant renal impairment. Several studies gave more direct evidence that a greater nocturnal BP dip is associated with a more favourable renal profile. (CJ et al., 2015) showed that each 10% increase in systolic BP dipping was associated with lower odds of incident CKD¹⁷, and (Carollo et al., 2025) observed an inverse association between greater nocturnal BP decline and subclinical renal damage¹⁸. This pattern was extended to incident renal outcomes by (RC et al., 2018), who reported substantially lower risks of incident CKD, decreased eGFR, and albuminuria among patients receiving bedtime antihypertensive treatment associated with improved nocturnal BP control¹⁹. These findings suggest a possible graded association of preservation or restoration of nocturnal BP fall with a lower burden of renal injury.

Renal structural and functional assessments provide further evidence that abnormal nocturnal BP regulation is associated with more advanced renal injury. (Haruhara et al., 2015) reported that abnormal circadian BP patterns were more frequent with increasing severity of tubulointerstitial fibrosis and atrophy²⁰. Similarly, (Lin et al., 2016) reported associations of abnormal circadian rhythm with renal tissue injury and substantial tubulointerstitial fibrosis²¹, and Zeng et al. (2019) reported lower creatinine clearance and a higher prevalence of nephropathy among patients with abnormal circadian BP patterns¹⁴. These studies are of

particular importance because they extend the evidence from traditional biomarkers such as eGFR and albuminuria to structural renal damage. However, not all studies found an independent association between dipping status and renal injury. (Lopez-Sublet et al., 2019), Gupta et al. (2022), and Oreschak et al. (2020) reported limited or non-significant associations between dipping abnormalities and renal outcomes such as reduced eGFR, CKD, and microalbuminuria. Likewise, neither (Song et al., 2020) nor (Q. Wang et al., 2020) reported statistically significant associations between non-dipping and impaired renal function after adjustment. These conflicting results suggest that the association between nocturnal dipping and renal injury may differ according to the renal endpoint, the population characteristics, and the covariates introduced in the statistical models.

Evidence across a range of populations showed that absolute nocturnal BP may be more important than dipping status alone. Zhang et al. (2015) reported a stepwise decrease in nocturnal SBP decline with increasing severity of diabetic nephropathy, but the relationship with dipping did not reach statistical significance. Similarly, Kusunoki et al. (2020) found that patients with a higher risk of CKD had a lower nocturnal BP decline, while absolute nighttime BP was more strongly and independently associated with renal risk. Yu et al. (2023) reported an association between nocturnal hypertension and macroalbuminuria, but not between dipping pattern and renal injury. Overall, non-dipping and reverse-dipping patterns are typically associated with greater renal injury, while a greater nocturnal decline in BP tends to be associated with more favourable renal outcomes; however, the association was not consistently independent after adjustment for other cardiovascular and renal risk factors. The evidence thus favours nocturnal BP dipping as a clinically relevant marker of renal health rather than an independent causal determinant of renal injury.

Association Reflected in the Progression and Severity of Renal Injury

The included studies showed that abnormal nocturnal BP dipping was significantly associated with renal progression or worse renal outcomes, especially in patients with reverse-dipping or riser patterns, as shown in Table 8.

Table 8. Association reflected in the progression and severity of renal injury (RQ2)

Studies	Progression/severity outcome	Main effect estimate
(Park et al. (2024)); Wang et al. (2016); Rahman et al. (2020); Nakai et al. (2016)	CKD progression, composite renal events, ESRD, kidney replacement therapy, and dialysis initiation.	Park et al.: non-dipper HR 1.30 (95% CI 1.02–1.66); reverse-dipper HR 1.40 (1.03–1.90); each 10% increase in diurnal SBP difference HR 1.21 (1.07–1.37). Wang et al.: reverse-dipper HR 3.00 (1.59–5.65); non-dipper HR 1.77 (0.99–3.14). Rahman et al.: reverse-dipper HR 2.03 (1.44–2.85); non-dipper HR 1.28 (0.98–1.69). Nakai et al.: riser HR 1.79 (1.10–2.89).
Cheng et al. (2019); McMullan et al. (2015); Kuczera et al. (2018)	Rate of eGFR decline and deterioration of kidney function.	Cheng et al.: per 1% greater nighttime SBP decline, β -0.22 mL/min/1.73 m ² /year and OR 0.54 (0.46–0.62) for rapid GFR decline; DBP decline OR 0.46 (0.37–0.58). McMullan et al.: per 10% greater SBP dipping, β 0.39 (0.12–0.65) mL/min/1.73 m ² /year slower eGFR decline. Kuczera et al.: baseline DBP dipping $R = -0.59$, $p = .002$; regression $R^2 = 0.21$, $p = .04$.

Studies	Progression/severity outcome	Main effect estimate
Pluta & Stróżecki (2025); Jhee et al. (2021)	eGFR decline, CKD progression, and composite renal progression outcomes.	Pluta & Stróżecki: eGFR at 6 months: reverse-dipper 58.3 ± 21.8 vs. dipper/extreme-dipper 80.2 ± 23.8 , $p = .02$; baseline awake–sleep SBP difference correlated with eGFR, $r = -0.58$, $p = .02$. Jhee et al.: reverse dippers HR 1.43 (1.02–2.01) for CKD progression per 1-unit increase in log suPAR; interaction $p = .004$; dipping pattern mediated the suPAR–CKD progression relationship.
Borrelli et al. (2022); Hjortkjær et al. (2022)	CKD progression, $\geq 30\%$ eGFR decline, ESKD, and composite kidney endpoints.	Borrelli et al.: nondipping + controlled BP HR 1.82 (1.17–2.82); nondipping + above-goal BP HR 2.40 (1.58–3.65). Hjortkjær et al.: non-dipper HR 1.92 (1.23–3.00) for composite kidney endpoint; HR 1.19 (0.74–1.91) for $\geq 30\%$ eGFR decline and 3.63 (0.88–14.95) for ESKD.
Ida et al. (2019); Kado et al. (2019); Cha et al. (2017); Cha et al. (2020)	Composite renal outcomes, ESRD, eGFR decline, proteinuria progression, and hard renal outcomes.	Ida et al.: dipping rate HR 1.01 (0.99–1.02), $p = .389$. Kado et al.: dipping rate OR 0.99 (0.97–1.01), $p = .350$; correlation with eGFR decline $r = -0.069$, $p = .074$. Cha et al. (2017): transition to other dipping patterns OR 0.978 (0.497–1.922) for worse eGFR change and OR 1.773 (0.858–3.661) for proteinuria change. Cha et al. (2020): dipping-pattern change HR 1.06 (0.56–2.01) for hard renal outcomes.
Tiwari et al. (2021); Borrelli et al. (2024)	eGFR decline, albuminuria, and restoration of normal dipping.	Tiwari et al.: intervention–control eGFR difference 5.22 (95% CI 4.3–6.1) mL/min/1.73 m ² at 1 year; eGFR decline -0.18 vs. -2.55 mL/min/1.73 m ² . Borrelli et al.: albuminuria decreased from 180 to 120 mg/day, $p < .001$; normal dipping increased 31.3% $\rightarrow$ 50.6%, $p = .005$.
Hu et al. (2026)	Rapid eGFR decline, ESRD, composite renal events, and worsening renal biomarkers.	Nocturnal non-dipping HR 1.76 (1.44–2.15) for ESRD. DBP variability showed stronger associations: OR 1.18 (1.13–1.23) for rapid eGFR decline and HR 1.22 (1.15–1.29) for ESRD.

Source: Authors' synthesis of included studies for RQ2.

(CH et al., 2024) found that the risk of CKD progression was higher in non-dippers and reverse-dippers compared with dippers (HRs 1.30 and 1.40)². Moreover, (C. Wang et al., 2015), Rahman et al. (2020), and Nakai et al. (2016) showed a higher risk of composite renal outcomes, ESRD, dialysis initiation, or other hard renal endpoints in patients with reverse or riser patterns^{29–31}. Reverse dipping and riser patterns were identified as the most adverse nocturnal BP phenotypes: (Q. Wang et al., 2020) reported an HR of 3.00 and Rahman et al. (2020) an HR of 2.03 for reverse-dippers, and (Nakai et al., 2016) showed that the riser pattern was associated with an increased risk of renal events (HR 1.79). Reverse-dippers also had lower eGFR than dippers, and reverse dipping modified the association between suPAR and CKD progression. These results suggest a possible gradient of severity, with reverse or riser patterns associated with greater renal risk than conventional non-dipping.

Similar findings were seen in studies of continuous nocturnal BP decline and renal function decline. A lower likelihood of rapid GFR decline was associated with greater nighttime decline of SBP and DBP. Similarly, McMullan et al. (2015) observed an association between increased systolic BP dipping and slower annual decline in eGFR, and Kuczera et al. (2018) showed an inverse relationship between DBP dipping and eGFR deterioration. These findings are compatible with a dose-response interpretation, in which a larger physiological

nocturnal BP decrease may be accompanied by better preservation of renal function over time. Several studies also indicated that non-dipping interacts with overall BP control in influencing renal progression. Borrelli et al. (2022) showed that non-dipping was associated with a greater risk of CKD progression in patients with controlled and above-goal BP, with the highest risk when non-dipping was present with above-goal BP. Hjortkjær et al. (2022) found an association of non-dipping with a composite kidney outcome, but the associations for single outcomes ($\geq 30\%$ eGFR decline and ESKD) were less consistent.

Not all longitudinal studies reported an independent association between dipping and renal progression. No significant association was found between the rate of dipping and renal outcomes or decline in eGFR, and Cha et al. (2017) and Cha et al. (2020) did not find an independent association between changes in dipping pattern and deterioration of renal outcomes or hard renal endpoints. Evidence from intervention studies was more supportive: in the intervention group of Tiwari et al. (2021), a more favourable eGFR trajectory was observed and normal dipping was restored in a subset of participants, and Borrelli et al. (2024) reported reduced albuminuria and an increased prevalence of normal dipping⁴². These observations are consistent with the possibility that improved nocturnal BP regulation may be associated with more favourable renal trajectories, although the available evidence does not establish that restoration of dipping per se was responsible for the renal improvement. In Hu et al. (2026), non-dipping was associated with ESRD, but BP variability demonstrated wider and stronger prognostic associations, suggesting that the associations observed may be partly explained by absolute BP burden, BP control, proteinuria, BP variability, and baseline renal function.

DISCUSSION

This systematic review assessed the relationship between nocturnal BP dipping and renal outcomes through two research questions: the relationship between nocturnal BP dipping and renal injury, and its relationship with renal progression or severity. Abnormal nocturnal BP patterns, especially non-dipping, reverse-dipping, and riser patterns, were frequently linked to worse renal outcomes in the included studies. However, the direction and magnitude of these associations differed between studies, indicating that nocturnal BP dipping should be considered in conjunction with other measures of BP and renal status rather than interpreted in isolation.

Several markers of existing renal injury were associated with abnormal nocturnal BP patterns. (SMJ et al., 2021) observed associations with albuminuria and decreased eGFR, and Chiriaco et al. (2022) reported differences in advanced CKD between dipping groups, with an increased prevalence of advanced CKD in reverse-dippers. Structural and functional markers such as tubulointerstitial fibrosis, renal tissue injury, creatinine clearance, and nephropathy were investigated by Haruhara et al. (2015), Lin et al. (2016), and Zeng et al. (2019). The range of outcomes shows that the studies assessed renal injury using different clinical and biological measures rather than a single standardised endpoint. These findings describe an association between nocturnal BP pattern and renal status but do not establish whether abnormal dipping preceded the renal abnormality.

Some studies evaluated nocturnal BP decline as a continuous rather than categorical variable. A greater systolic BP dip was associated with lower renal risk and slower eGFR decline (McMullan et al., 2015), an inverse association was found between nocturnal BP decline and subclinical renal damage (Carollo et al., 2025), and better nighttime BP control

was associated with lower risks of several renal outcomes (RC et al., 2018). Longitudinal evidence from Cheng et al. (2019) and (Kuczera et al., 2018) also suggests that a larger nocturnal fall in BP is associated with more favourable trajectories of renal function. A continuous measure may detect differences that are not fully captured by the distinction between dipper and non-dipper groups, because participants within the same dipping category may still have different degrees of nocturnal BP reduction. The method of assessing nocturnal BP decline may therefore affect its association with renal outcomes.

Progression and severity outcomes showed a similar direction. (CH et al., 2024) found that non-dippers and reverse-dippers had a higher risk of CKD progression than dippers; reverse-dipping and riser patterns were related to adverse renal outcomes by Wang et al. (2016), Rahman et al. (2020), and Nakai et al. (2016); and further associations of reverse dipping with renal outcomes were reported by (Pluta & Stróżecki, 2025) and (Jhee et al., 2021). The outcomes examined for RQ1 were mainly albuminuria, eGFR, CKD, nephropathy, and structural renal abnormalities, whereas those for RQ2 were eGFR decline, CKD progression, ESRD, dialysis-related outcomes, and composite renal endpoints. Similar associations in these outcome categories suggest that nocturnal BP patterns have been investigated at different stages of renal disease; however, there is no evidence of a causal chain from abnormal nocturnal BP dipping to renal injury to subsequent progression, and these findings do not by themselves establish an independent effect of dipping status.

It is important to distinguish between nocturnal BP dipping and absolute nighttime BP. Dipping is the relative change between daytime and nighttime BP, whereas nighttime BP is the actual BP level during sleep. The findings of Zhang et al. (2015), (Kusunoki et al., 2021), (Yu et al., 2023), Song et al. (2020), and Wang et al. (2015) suggest that nighttime BP or other BP characteristics may contribute to the observed renal associations, and some associations with dipping patterns were attenuated after adjustment. Two individuals may have a similar percentage reduction in BP but substantially different nighttime BP levels; conversely, individuals with similar nighttime BP may have different dipping percentages because of differences in daytime BP. Consequently, the renal significance of a particular dipping category may depend partly on the underlying nighttime and daytime BP levels, and the available studies support examining dipping status together with the actual nighttime BP level rather than treating the two measures as interchangeable.

BP variability and total BP burden could also explain differences across studies. BP variability parameters, but not the dipping pattern, were associated with renal injury markers (Wu et al., 2024). (Borrelli et al., 2022) reported an association between non-dipping and CKD progression, especially in patients with BP above target, and Hjortkjær et al. (2022) found an association between non-dipping and a composite kidney outcome, although associations with individual renal outcomes were less consistent. These findings indicate that nocturnal dipping is part of a broader 24-hour BP profile including mean BP, nighttime BP, and BP variability. Some of the heterogeneity may also be explained by differences in study populations and methodology, as the included studies varied in CKD stage, hypertension status, prevalence of diabetes, baseline renal function, sample size, ABPM protocols, dipping thresholds, definitions of renal outcome, follow-up duration, and covariate adjustment strategy. In studies such as Lopez-Sublet et al. (2019), Gupta et al. (2022), (Oreschak et al., 2020), Song et al. (2020), and

Wang et al. (2015), the relationship was not significant or was limited after adjustment, implying that the association is not uniform across populations.

Clinically, ABPM provides information about nighttime BP and dipping patterns that cannot be obtained from conventional office BP measurements alone. The identification of non-dipping, reverse-dipping, or riser patterns may therefore provide additional information when assessing patients with hypertension, CKD, or other renal risk factors. However, the findings of this review do not support the use of dipping status as a single marker of renal injury or progression; consideration should also be given to nighttime BP, 24-hour BP burden, BP variability, renal function, albuminuria or proteinuria, and relevant clinical characteristics.

Strengths and Limitations

A major strength of this review is the kidney-focused synthesis of 35 primary quantitative studies spanning cross-sectional, cohort, and interventional designs, with study-level effect estimates and a structured QualSyst assessment. The review also distinguishes dipping phenotype from absolute nighttime BP and considers both renal injury and progression/severity, which reduces the risk of treating all kidney outcomes as equivalent.

The review also has important limitations. PubMed was the only database searched and eligibility was restricted to English-language studies, which can reduce retrieval comprehensiveness. The included evidence is clinically and methodologically heterogeneous in population, CKD stage, dipping definition, renal endpoint, follow-up duration, and covariate adjustment; most studies are observational, so residual confounding and reverse causation remain possible. These differences precluded meaningful quantitative pooling in the present synthesis. In addition, the unresolved PRISMA arithmetic inconsistency limits confidence in the reported selection flow until the original screening records are verified.

CONCLUSION

The available evidence indicated that abnormal nocturnal blood pressure (BP) patterns, particularly reverse-dipping or riser phenotypes, were more consistently associated with renal injury and adverse kidney disease progression than normal nocturnal dipping patterns. Conversely, a greater physiological decline in nocturnal BP was generally associated with more favorable renal outcomes. Nevertheless, nocturnal dipping status could not be established as an independent causal determinant of renal injury because the observed associations varied according to absolute nighttime BP, overall 24-hour BP burden, BP variability, baseline kidney function, diabetes mellitus, antihypertensive treatment, renal outcome definitions, and statistical adjustment strategies. From a clinical perspective, nocturnal dipping status should therefore be considered a complementary indicator of renal risk rather than a substitute for absolute nighttime BP measurements and established renal risk factors when interpreting ambulatory blood pressure monitoring (ABPM) findings.

The certainty and generalizability of the findings were limited by substantial clinical and methodological heterogeneity, including variations in nocturnal dipping definitions and renal outcome measures, the predominance of observational study designs, potential residual confounding, the restriction to English-language publications, and the exclusive use of PubMed as the literature database. Furthermore, discrepancies in the reported Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) screening counts required reconciliation with the original study selection records before publication. Future research

should adopt standardized ABPM definitions, employ prospective longitudinal study designs, use consistent and clinically relevant renal outcomes, and implement prespecified statistical adjustments for potential confounders. Additionally, well-designed interventional studies are needed to determine whether targeted modification of nocturnal BP levels or dipping patterns can improve kidney outcomes and reduce the risk of chronic kidney disease progression.

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