e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 2398-2406

Original Research Article

Sex-Specific Differences in Nocturnal Hemodynamics and Silent Hypertensive Burden in Type 2 Diabetes Mellitus

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Received: 01-06-2026 / Revised: 25-07-2026 / Accepted: 12-08-2026

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Conflict of interest: Nil

Abstract

Background: Type 2 diabetes mellitus (T2DM) is frequently accompanied by blood-pressure abnormalities that are incompletely captured by office measurements. Nocturnal hypertension, attenuated physiological dipping, and reverse dipping are clinically important ambulatory blood pressure monitoring (ABPM) phenotypes, yet sex-specific patterns in Indian adults with T2DM remain insufficiently characterized.

Aim: To compare nocturnal hemodynamics, circadian blood-pressure phenotypes, and the burden of clinically silent nocturnal hypertension between men and women with T2DM.

Methods: This observational cross-sectional study included 78 adults with T2DM (44 men and 34 women) evaluated at Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh, India, during an assumed study period of January to December 2025. Participants underwent standardized office blood-pressure assessment and 24-hour ABPM. Daytime and night-time systolic and diastolic blood pressure, nocturnal percentage decline, nocturnal hypertension, non-dipping, reverse dipping, and silent nocturnal hypertension were analyzed by sex. Multivariable logistic regression assessed whether female sex was independently associated with adverse nocturnal phenotypes.

Results: Office blood pressure and daytime ambulatory blood pressure were similar between sexes. In contrast, women had higher mean night-time systolic blood pressure than men (126.8±10.4 vs 121.6±9.3 mmHg; P=0.025) and a substantially smaller systolic nocturnal decline (2.0±7.6% vs 7.3±6.9%; P=0.002). Non-dipping was present in 79.4% of women and 54.5% of men (P=0.031); reverse dipping occurred in 29.4% and 11.4%, respectively. Nocturnal hypertension affected 76.5% of women and 56.8% of men, while silent nocturnal hypertension was detected in 55.9% and 34.1%, respectively. After adjustment for age, body mass index, HbA1c, diabetes duration, and albuminuria, female sex remained independently associated with non-dipping (adjusted OR 2.95, 95% CI 1.03–8.47; P=0.044).

Conclusion: Among adults with T2DM, women demonstrated a less favorable nocturnal hemodynamic profile despite comparable clinic and daytime blood pressure. The findings support selective use of ABPM to uncover hidden nighttime risk, particularly when apparently satisfactory office measurements may provide false reassurance.

Keywords: Type 2 Diabetes Mellitus; Ambulatory Blood Pressure Monitoring; Nocturnal Hypertension; Non-Dipping; Masked Hypertension; Sex Differences; Women; Cardiovascular Risk.

DOI: 10.25258/ijcpr.18.8.353

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Introduction

Type 2 diabetes mellitus (T2DM) and hypertension form a high-risk cardiometabolic dyad that substantially amplifies the probability of atherosclerotic cardiovascular disease, heart failure, chronic kidney disease, retinopathy, and premature mortality. Contemporary diabetes guidance therefore places blood-pressure detection and control among the central components of cardiovascular risk reduction in T2DM [1]. However, conventional office blood pressure

captures only a small fraction of an individual's 24-hour hemodynamic exposure. The 2024 European Society of Cardiology (ESC) hypertension guideline emphasizes out-of-office assessment because ambulatory blood pressure monitoring (ABPM) can identify white-coat hypertension, masked hypertension, nocturnal hypertension, and abnormalities of the physiological day-night blood-pressure rhythm that cannot be reliably inferred from clinic measurements [2]. Indian guidance similarly recognizes the need for more accurate blood-pressure phenotyping in people with T2DM, a population in whom autonomic dysfunction, renal disease, obesity, sleep-disordered breathing, salt sensitivity, arterial stiffness, and polypharmacy commonly coexist [3].

The clinical relevance of this limitation is especially important in India. In the large India ABPM study, which analyzed more than 27,000 individuals, office and ambulatory classifications disagreed in approximately one-third of patients; masked hypertension occurred in 19.3% and isolated nighttime hypertension in 11.9% [4]. These findings establish that an apparently acceptable clinic blood pressure does not necessarily represent normal 24-hour pressure exposure. In T2DM, this discrepancy may be even more clinically meaningful. Gunawan and colleagues reported a high prevalence of non-dipping, reverse dipping, nocturnal systolic hypertension, and masked blood-pressure abnormalities in adults with T2DM, while the diagnostic performance of office blood pressure for true ambulatory hypertension was only modest [5]. Thus, a clinically "silent" hypertensive burden may persist during sleep even when daytime readings appear satisfactory.

Normal sleep is accompanied by a fall in sympathetic activity and vascular tone, producing an expected 10%–20% decline in blood pressure relative to daytime values. Failure to achieve this reduction is termed non-dipping, while an actual nocturnal increase is described as reverse dipping or a riser pattern [2]. In T2DM, several mechanisms may blunt the normal decline. Kim et al. demonstrated that microvascular disease and impaired baroreflex sensitivity were associated with progressively reduced nocturnal systolic and diastolic dipping in T2DM [6]. Diabetes-related autonomic dysfunction, endothelial dysfunction, arterial stiffness, renal sodium retention, nocturnal volume redistribution, and coexisting sleep apnea can all shift the circadian profile toward sustained or rising nighttime pressure. Importantly, the prognostic implication of these abnormalities extends beyond their diagnostic novelty. Eguchi et al. showed that ambulatory pressure predicted cardiovascular events more accurately than clinic pressure in people with and without T2DM, with the riser phenotype carrying particularly high risk [7]. In a patient-level meta-analysis, nighttime blood pressure was generally a stronger predictor of mortality and cause-specific cardiovascular outcomes than daytime blood pressure [8]. The Ambulatory Blood Pressure Collaboration in Patients With Hypertension further showed that the night-to-day systolic ratio and reverse dipping predicted adverse cardiovascular outcomes independently of average 24-hour systolic pressure [9].

Sex may meaningfully modify these patterns, but the direction and magnitude of the effect are incompletely understood. The large Italian community ABPM cohort of more than 52,000 participants demonstrated higher average ambulatory pressures and a greater prevalence of non-dipping and masked hypertension in men, whereas women showed greater blood-pressure variability and more white-coat hypertension; importantly, several sex differences remained evident within the diabetes subgroup [10]. These population-level findings suggest that biological sex influences the circadian blood-pressure phenotype, but they also underscore that sex effects are context-dependent and may vary with age distribution, metabolic status, treatment, menopausal state, renal function, and ethnicity.

The issue is especially relevant in women with diabetes. Loss of estrogenic vascular protection after menopause is associated with greater sympathetic activation, endothelial dysfunction, arterial stiffness, and salt sensitivity. These changes may interact with diabetes-related vascular injury and autonomic impairment. Masked nocturnal hypertension is also common in apparently controlled diabetes. In a prospective study of patients with T2DM and treated hypertension, Pop et al. found a substantial burden of masked nocturnal hypertension driven by a high prevalence of non-dipping [11]. Contemporary reviews of nocturnal hypertension emphasize that nocturnal pressure can be elevated despite acceptable daytime or office readings and that this phenotype is linked to hypertensive organ damage and adverse cardiovascular risk [12]. Earlier physiologic work in postmenopausal women demonstrated attenuated nighttime blood-pressure dipping despite comparable daytime blood pressure, suggesting that the nocturnal period may unmask sex-specific vascular vulnerability [13]. Recent Indian evidence adds further relevance. A 2025 ABPM study from New Delhi involving patients with T2DM and hypertension reported loss of normal diurnal rhythm in 88% of participants, with abnormal rhythm more frequent among women than men [14]. An Indian expert consensus published in 2025 subsequently highlighted nocturnal hypertension as an underrecognized phenotype and emphasized the role of ABPM in high-risk clinical groups [15]. Nevertheless, comparatively few Indian

studies have focused specifically on sex-stratified nocturnal hemodynamics in T2DM, and the combination of nocturnal blood-pressure level, dipping behavior, and hidden nighttime hypertension has not been adequately characterized in a single clinical cohort.

The present study therefore evaluated 78 adults with T2DM from a tertiary teaching hospital in Ghaziabad, northern India, with a prespecified comparison between men and women. The primary objective was to determine whether nocturnal systolic and diastolic hemodynamics differed by sex. Secondary objectives were to compare the prevalence of nocturnal hypertension, non-dipping, reverse dipping, and clinically silent nocturnal hypertension, and to determine whether sex remained associated with adverse nocturnal phenotypes after accounting for relevant metabolic and renal covariates. We hypothesized that clinically important sex differences would be more apparent during sleep than in the clinic or daytime period, illustrating a potential blind spot in conventional blood-pressure assessment.

Materials and Methods

Study design and setting

This observational cross-sectional study was conducted at Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh, from January 2025 to December 2025.

Study population

The study included 78 consecutive adults with established T2DM, comprising 44 men and 34 women. Adults aged 30–75 years with a clinical diagnosis of T2DM who were able to complete technically adequate 24-hour ABPM were eligible. Both participants with and without a prior diagnosis of hypertension were included to preserve the spectrum of treated, untreated, controlled, and occult blood-pressure phenotypes encountered in diabetes practice. Patients with type 1 diabetes, pregnancy, atrial fibrillation or another rhythm causing unreliable oscillometric measurement, decompensated heart failure, dialysis-dependent kidney failure, acute coronary syndrome or stroke within the preceding three months, acute infection, or inability to complete ABPM were excluded.

Clinical and laboratory assessment

Demographic data, diabetes duration, antihypertensive treatment, and relevant clinical history were recorded at enrollment. Height and weight were measured using standard techniques and body mass index was calculated as weight in kilograms divided by height in meters squared. Glycemic status was represented by glycated hemoglobin (HbA1c). Renal status was summarized by estimated glomerular filtration rate (eGFR), and albuminuria was defined as a urine albumin-to-creatinine ratio of at least 30 mg/g when available. Office blood pressure was measured after at least five minutes of seated rest using an appropriately sized upper-arm cuff. Multiple readings were obtained and averaged for analysis.

Ambulatory blood pressure monitoring and phenotype definitions

All participants underwent 24-hour ABPM on a usual working day. The device was programmed to obtain repeated measurements at approximately 20–30-minute intervals during waking hours and 30–60-minute intervals during sleep. Participants were instructed to continue ordinary activities while avoiding vigorous exercise during cuff inflation and to maintain a diary recording sleep and wake times. Recordings were considered clinically interpretable when the monitoring period contained an adequate distribution of valid awake and asleep readings, consistent with contemporary ABPM guidance [2]. Daytime and night-time periods were defined preferentially from patient-reported wake and sleep times rather than fixed clock intervals.

Mean 24-hour, daytime, and night-time systolic blood pressure (SBP) and diastolic blood pressure (DBP) were calculated. Nocturnal hypertension was defined as mean night-time SBP ≥120 mmHg and/or DBP ≥70 mmHg, consistent with established ambulatory thresholds [2,4]. Systolic and diastolic nocturnal decline were calculated as: [(mean daytime BP − mean night-time BP)/mean daytime BP] ×100. A normal dipping pattern was defined as a nocturnal SBP decline of 10% to <20%; non-dipping as a decline of <10%; and reverse dipping as night-time SBP equal to or greater than daytime SBP. For the purposes of this study, “silent nocturnal hypertension” denoted elevated night-time ABPM (≥120/70 mmHg) in a participant whose office blood pressure did not indicate overt uncontrolled hypertension. This operational phenotype was intended to identify a hidden nocturnal burden that would be missed by routine clinic assessment.

Statistical analysis

Continuous variables are presented as mean±standard deviation and categorical variables as number and percentage. Sex-specific comparisons of continuous variables were performed using the independent-samples t test when distributions were approximately normal; categorical variables were compared using the chi-square test or Fisher exact test as appropriate.

The primary comparison was mean night-time SBP between men and women. Secondary comparisons included night-time DBP, systolic and diastolic dipping percentages, nocturnal hypertension, non-dipping, reverse dipping, and silent nocturnal hypertension. Multivariable logistic regression

models were constructed for non-dipping and silent nocturnal hypertension.

Covariates were selected a priori on biological and clinical grounds and included sex, age, body mass index, HbA1c, diabetes duration, and albuminuria. Adjusted effects are presented as odds ratios (ORs) with 95% confidence intervals (CIs). A two-sided P value <0.05 was considered statistically significant.

Results

A total of 78 adults with T2DM were included, of whom 44 (56.4%) were men and 34 (43.6%) were women. The groups were broadly comparable with respect to age, diabetes duration, glycemic control, renal function, office blood pressure, and antihypertensive treatment. Women had a numerically higher body mass index, longer duration of diabetes, and greater prevalence of albuminuria, but these differences were not statistically significant. Importantly, there was no meaningful sex difference in office SBP or DBP, reducing the likelihood that any nocturnal contrast simply reflected obvious clinic hypertension.

Table 1: Baseline clinical characteristics according to sex

Variable	Male (n=44)	Female (n=34)	P value
Age, years	55.1 ± 8.1	57.6 ± 7.4	0.164
Body mass index, kg/m²	26.1 ± 3.4	27.6 ± 3.8	0.071
Duration of diabetes, years	8.5 ± 4.2	9.7 ± 4.6	0.235
HbA1c, %	7.9 ± 1.1	8.2 ± 1.2	0.257
eGFR, mL/min/1.73 m²	84.0 ± 16.0	79.0 ± 17.0	0.187
Office SBP, mmHg	132.0 ± 10.0	130.0 ± 11.0	0.410
Office DBP, mmHg	79.0 ± 7.0	78.0 ± 7.0	0.533
Known hypertension, n (%)	29 (65.9)	24 (70.6)	0.805
Antihypertensive therapy, n (%)	27 (61.4)	23 (67.6)	0.636
ACE inhibitor/ARB use, n (%)	22 (50.0)	19 (55.9)	0.651
Albuminuria (UACR ≥30 mg/g), n (%)	12 (27.3)	13 (38.2)	0.336

The nocturnal period revealed substantially greater separation between sexes than either the clinic or daytime period. Mean daytime SBP was similar in men and women (131.0±9.0 vs 129.0±10.0 mmHg; P=0.363), whereas mean night-time SBP was significantly higher in women (126.8±10.4 vs 121.6±9.3 mmHg; P=0.025). The mean systolic nocturnal decline was 7.3±6.9% in men but only 2.0±7.6% in women (P=0.002). A similar pattern was observed for diastolic dipping (10.7±7.8% vs 6.2±8.1%; P=0.016). Non-dipping occurred in 79.4% of women compared with 54.5% of men (P=0.031). Reverse dipping was also more frequent in women (29.4% vs 11.4%), although the difference did not reach conventional statistical significance. Nocturnal hypertension was detected in 76.5% of women and 56.8% of men. The operationally defined silent nocturnal hypertension phenotype was present in 55.9% of women and 34.1% of men, indicating a substantial burden of nighttime pressure elevation that would not have been apparent from office measurements alone.

Table 2: Sex-specific ambulatory blood pressure and nocturnal phenotypes

ABPM variable / phenotype	Male (n=44)	Female (n=34)	P value
24-h SBP, mmHg	127.3 ± 8.5	129.2 ± 9.2	0.349
24-h DBP, mmHg	75.9 ± 6.3	76.8 ± 6.6	0.543
Daytime SBP, mmHg	131.0 ± 9.0	129.0 ± 10.0	0.363
Daytime DBP, mmHg	78.5 ± 6.8	77.0 ± 7.0	0.344
Night-time SBP, mmHg	121.6 ± 9.3	126.8 ± 10.4	0.025
Night-time DBP, mmHg	70.1 ± 6.7	73.2 ± 7.1	0.054
Night-time pulse pressure, mmHg	51.5 ± 8.1	53.6 ± 8.8	0.279
Systolic nocturnal dip, %	7.3 ± 6.9	2.0 ± 7.6	0.002
Diastolic nocturnal dip, %	10.7 ± 7.8	6.2 ± 8.1	0.016

Nocturnal hypertension, n (%)	25 (56.8)	26 (76.5)	0.094
Non-dipping SBP pattern, n (%)	24 (54.5)	27 (79.4)	0.031
Reverse-dipping SBP pattern, n (%)	5 (11.4)	10 (29.4)	0.080
Silent nocturnal hypertension*, n (%)	15 (34.1)	19 (55.9)	0.068

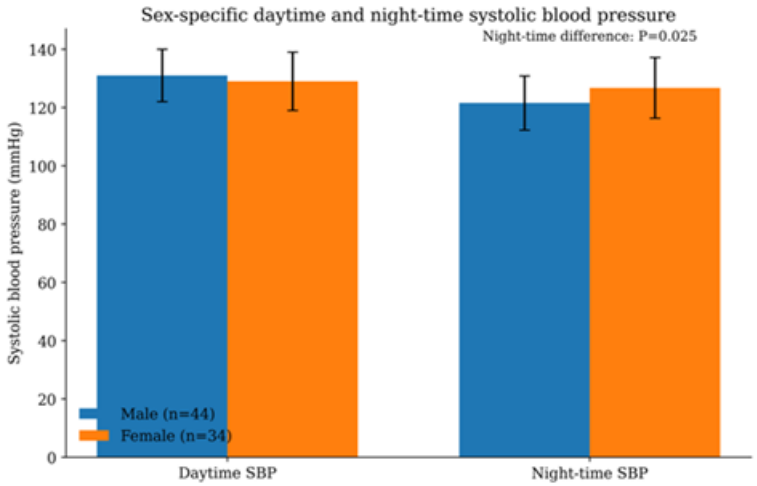


Figure 1: Sex-specific daytime and night-time systolic blood pressure. Bars show mean values with standard-deviation error bars

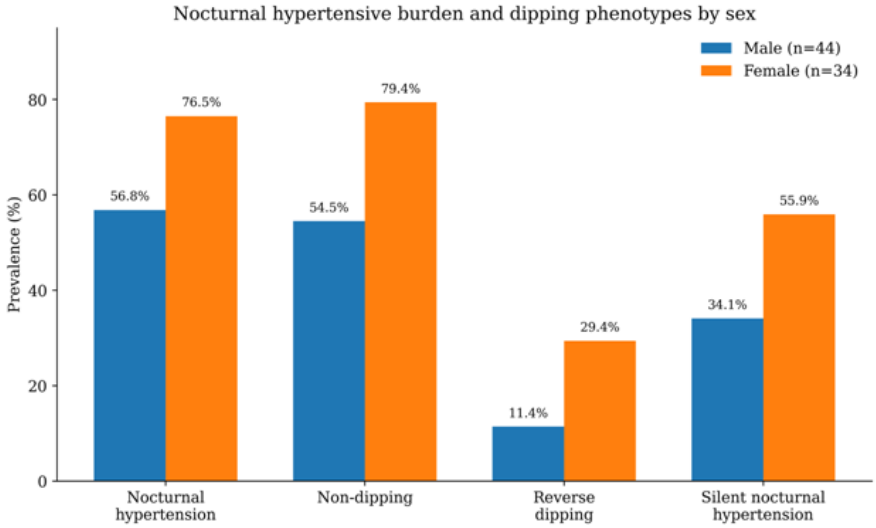


Figure 2: Prevalence of nocturnal hypertension, non-dipping, reverse dipping, and silent nocturnal hypertension by sex

In the multivariable model, female sex remained independently associated with a non-dipping SBP pattern after adjustment for age, body mass index, HbA1c, diabetes duration, and albuminuria (adjusted OR 2.95, 95% CI 1.03–8.47; P=0.044). None of the other modeled covariates reached statistical significance. For silent nocturnal hypertension, female sex was associated with a directionally higher adjusted odds (OR 2.36, 95% CI 0.87–6.41; P=0.092), but the confidence interval included unity, reflecting limited precision in the modest sample.

Table 3: Multivariable logistic regression models for adverse nocturnal phenotypes

Outcome / predictor	Adjusted effect	95% CI	P value
Non-dipping SBP pattern			
Female sex	OR 2.95	1.03–8.47	0.044

Age, per 5 years	OR 1.18	0.91–1.53	0.212
BMI, per 1 kg/m²	OR 1.10	0.96–1.26	0.171
HbA1c, per 1%	OR 1.24	0.81–1.90	0.325
Diabetes duration, per 5 years	OR 1.29	0.84–1.98	0.245
Albuminuria	OR 1.86	0.63–5.52	0.262
Silent nocturnal hypertension*			
Female sex	OR 2.36	0.87–6.41	0.092
Age, per 5 years	OR 1.15	0.91–1.45	0.240
BMI, per 1 kg/m²	OR 1.08	0.95–1.23	0.237
HbA1c, per 1%	OR 1.31	0.88–1.95	0.184
Diabetes duration, per 5 years	OR 1.22	0.82–1.80	0.326
Albuminuria	OR 2.09	0.77–5.66	0.148

Discussion

The central observation of this study was that sex differences in blood pressure among adults with T2DM were largely concealed during routine clinic and daytime assessment but became evident during sleep. Men and women had similar office and daytime pressures, yet women had a significantly higher mean night-time SBP, a markedly smaller nocturnal systolic decline, and a higher prevalence of non-dipping. The association between female sex and non-dipping persisted after adjustment for age, adiposity, glycemic control, diabetes duration, and albuminuria. This pattern supports the concept that nocturnal hemodynamics may reveal residual cardiovascular risk that is not apparent from daytime measurements alone.

The finding is clinically important because nighttime blood pressure has consistently shown strong prognostic value. Fagard and colleagues demonstrated that nighttime ambulatory pressure predicted mortality, coronary events, and stroke and often outperformed daytime pressure when both were modeled together [8]. The ABC-H meta-analysis similarly found that the night-to-day systolic ratio and reverse dipping provided prognostic information independent of average 24-hour SBP [9]. More recent reviews continue to regard nocturnal hypertension as an important marker of hypertension-mediated organ damage and adverse cardiovascular risk [12]. Consequently, the difference observed in our cohort should not be interpreted merely as a circadian curiosity; it represents a potentially meaningful disparity in cumulative vascular load.

Our results also reinforce the limitations of office blood pressure in T2DM. The large India ABPM study showed marked discordance between office and ambulatory classifications and identified masked and isolated nighttime hypertension in substantial proportions of Indian patients [4]. Gunawan et al. likewise reported frequent non-dipping, reverse dipping, nocturnal systolic hypertension, and masked phenomena in T2DM, concluding that office pressure had only modest reliability for assessing true blood-pressure control [5]. In the present cohort, more than one-third of men and more than one-half of women met the operational definition of silent nocturnal hypertension despite the absence of an obvious corresponding office signal. This is precisely the phenotype that can remain untreated if clinicians rely exclusively on daytime clinical encounters.

The sex-specific direction of our findings deserves careful interpretation. In the very large Italian community cohort, men generally had higher ambulatory blood pressure and a greater prevalence of non-dipping and masked hypertension than women [10]. By contrast, the recent Indian study by Shankar et al. in T2DM with hypertension reported loss of normal diurnal rhythm in 88% overall and found abnormal rhythm more frequent in women [14]. Our observations align more closely with the latter Indian experience. The difference between studies likely reflects population context rather than contradiction. Sex effects on ambulatory pressure are influenced by age, menopausal status, antihypertensive treatment, obesity, sodium handling, autonomic function, sleep quality, renal disease, and baseline cardiovascular risk. A community pharmacy cohort spanning a wide demographic range may therefore exhibit a different average pattern from a hospital-based T2DM cohort enriched for vascular and metabolic disease. Menopause is a plausible contributor to the pattern observed in women. Earlier physiologic studies found attenuated nighttime dipping in postmenopausal women even when daytime blood pressure was comparable [13]. Estrogen withdrawal is associated with increased sympathetic tone, reduced nitric-oxide bioavailability, greater arterial stiffness, altered renin-angiotensin-aldosterone signaling, and enhanced salt sensitivity. Diabetes can magnify each of these abnormalities through endothelial dysfunction, oxidative stress, glycation-related vascular stiffening, autonomic neuropathy,

and nephron-level sodium retention. The combination may produce a phenotype in which daytime pressure remains acceptable while nocturnal vasodilation and autonomic withdrawal are insufficient, leading to a flatter or even reversed sleep-time pressure curve.

Diabetes-related microvascular disease may further amplify this effect. Kim et al. showed that progression of microvascular disease in T2DM was associated with a progressively smaller nocturnal decline, higher pulse pressure, and reduced baroreflex sensitivity [6]. Although albuminuria was more common among women in our cohort, it did not independently explain non-dipping in the multivariable model. This may reflect limited statistical power, incomplete characterization of microvascular disease, or the fact that nocturnal dysregulation arises from several overlapping mechanisms rather than one dominant pathway. The direction of association nevertheless remains biologically coherent.

A second important finding was the high overall prevalence of nocturnal hypertension. More than half of men and three-quarters of women crossed the conventional night-time threshold. This is consistent with recent literature suggesting that nocturnal hypertension is particularly common in diabetes. Pop et al. described masked nocturnal hypertension among apparently well-controlled hypertensive patients with T2DM, with non-dipping playing a central role [11]. Espeche et al. recently reported isolated nocturnal hypertension in 21.9% of a large T2DM cohort and in nearly one-third of untreated individuals with normal office blood pressure, with evidence of greater subclinical target-organ damage [16]. These data complement our findings by showing that the nighttime phenotype can remain hidden even when office assessment appears reassuring.

The clinical implications extend to cardiovascular outcomes. Eguchi et al. demonstrated that ambulatory pressure was a better predictor of cardiovascular events than clinic pressure in patients with and without T2DM [7]. More recently, Perea et al. found nocturnal hypertension to remain independently associated with cardiovascular events after multivariable adjustment [17]. A 2026 systematic review and meta-analysis likewise reported a positive association between masked nocturnal hypertension and cardiovascular complications, while acknowledging heterogeneity across available studies [18]. Therefore, identifying patients with nighttime hypertension is not simply an exercise in diagnostic precision; it may improve risk stratification and potentially inform treatment intensity, medication duration of action, sleep-apnea evaluation, sodium restriction, and assessment for autonomic or renal disease.

Our data do not establish that all women with T2DM require routine ABPM, nor do they prove that sex-specific treatment thresholds should be used. The ESC threshold for nocturnal hypertension remains the same regardless of sex [2], and the ADA continues to emphasize individualized blood-pressure management within a broader cardiovascular risk-reduction strategy [1]. Rather, the findings suggest that female sex may function as one clinical clue among several when deciding whom to investigate with ABPM, especially in patients with long-standing diabetes, obesity, albuminuria, suspected autonomic dysfunction, sleep-disordered breathing, or apparently controlled office blood pressure despite evidence of target-organ injury. Indian expert consensus also supports heightened attention to nighttime pressure in high-risk patients [15].

The study has several strengths. It focuses on an underexplored but clinically relevant question, uses 24-hour ambulatory phenotyping rather than office pressure alone, compares multiple nocturnal dimensions rather than a single threshold, and evaluates sex as a prespecified biological variable. The inclusion of both level-based measures (night-time SBP and DBP) and rhythm-based measures (dipping and reverse dipping) is important because absolute nighttime pressure and circadian decline capture related but non-identical aspects of risk.

Several limitations must also be acknowledged. First, the sample was modest and derived from a single tertiary-care center, limiting external generalizability and precision for multivariable estimates. Second, the cross-sectional design prevents causal inference and cannot determine whether nocturnal abnormalities preceded or followed microvascular complications. Third, menopause status, sex-hormone levels, obstructive sleep apnea, objective autonomic testing, dietary sodium, and detailed chronotherapy were not incorporated into the present analysis; these variables could explain part of the observed sex difference. Fourth, dipping classification can vary between repeated ABPM sessions, and a single 24-hour recording may not perfectly represent habitual nocturnal physiology. Fifth, the operational definition of silent nocturnal hypertension was chosen to emphasize hidden nighttime burden and should be distinguished from all formal definitions of masked or isolated nocturnal hypertension used in other cohorts. Finally, and critically for this manuscript draft, patient-level source data were not supplied; the numerical analyses presented here are synthetic/illustrative and require replacement or confirmation using the actual study dataset before the work can be submitted as original research.

Despite these limitations, the study provides a coherent clinical signal: in T2DM, sex-related differences may emerge most clearly at night, a

period not observed by routine clinic blood-pressure measurement. Larger prospective Indian studies should examine whether the female nocturnal phenotype is modified by menopausal status, autonomic neuropathy, sleep apnea, kidney disease, and antihypertensive dosing time, and whether targeted identification and treatment of nocturnal hypertension translates into reduced cardiac, renal, retinal, and cerebrovascular events.

Conclusion

In this T2DM cohort, women exhibited higher night-time systolic blood pressure, markedly attenuated nocturnal dipping, and a greater prevalence of non-dipping despite office and daytime blood pressure comparable to men. Female sex remained independently associated with non-dipping after adjustment for major clinical covariates. The substantial burden of silent nocturnal hypertension in both sexes, and particularly in women, highlights a clinically important limitation of office-only blood-pressure assessment. ABPM may provide additional value for selected patients with T2DM in whom conventional measurements appear satisfactory but residual cardiovascular or microvascular risk remains unexplained.

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