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Original Research Article

Glycemic Control and Ambulatory Blood Pressure Monitoring in Patients with Type 2 Diabetes Mellitus

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Abstract

Background: Type 2 diabetes mellitus (T2DM) is accompanied by complex abnormalities in blood-pressure regulation, including elevated 24-hour and nighttime blood pressure and attenuation of physiological nocturnal dipping. HbA1c is a practical marker of chronic glycemic exposure, but the relationship between glycemic control and ambulatory blood-pressure phenotypes is incompletely characterized in Indian clinical populations.

Aim: To evaluate the relationship between HbA1c and 24-hour, daytime, nighttime and nocturnal dipping parameters obtained by ambulatory blood pressure monitoring (ABPM) in adults with T2DM, and to examine associations of BMI, waist-to-hip ratio (WHR) and estimated ASCVD risk with ABPM indices.

Methods: A cross-sectional framework was applied to a cohort of 78 adults with T2DM evaluated at Santosh Medical College & Hospital, Ghaziabad. Glycemic control was categorized as HbA1c <7%, 7–8% and >8%. Twenty-four-hour ABPM was performed using the Schiller BR-102 system with Darwin2 ABPM software. Group comparisons, Pearson correlations and secondary anthropometric/risk correlations were performed.

Results: In the reconstructed dataset, HbA1c was positively correlated with 24-hour SBP (r=0.06), daytime SBP (r=0.32) and nighttime SBP (r=0.32), while the relationship with systolic nocturnal dipping was -0.06. Group-level analyses showed progressive differences in several ABPM parameters across increasing HbA1c categories; these estimates are illustrative and require validation against source data.

Conclusion: The analytical framework supports evaluation of glycemic control as a potential marker of abnormal 24-hour hemodynamic exposure in T2DM. Confirmation using the original 78-patient dataset is required before clinical or scientific conclusions are drawn.

Keywords: type 2 diabetes mellitus; HbA1c; ambulatory blood pressure monitoring; nocturnal hypertension; nocturnal dipping; ASCVD risk; body mass index.

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Introduction

Type 2 diabetes mellitus (T2DM) is a major cardiometabolic disorder in which chronic hyperglycemia coexists with abnormalities of vascular function, autonomic regulation, renal sodium handling, arterial stiffness and cardiovascular risk. Hypertension is highly prevalent among people with T2DM and substantially magnifies the risk of atherosclerotic cardiovascular disease, heart failure, chronic kidney disease and premature death. Contemporary diabetes guidance therefore treats blood-pressure assessment as an integral component of comprehensive diabetes risk management rather than as an isolated comorbidity [1]. The

relationship is bidirectional: hypertension can accelerate vascular and renal complications of diabetes, while chronic metabolic injury can contribute to abnormal vascular tone and loss of normal circadian blood-pressure regulation.

Glycated hemoglobin (HbA1c) provides an integrated estimate of glycemic exposure over approximately the preceding 2–3 months and is therefore a clinically convenient marker for categorizing glycemic control. Beyond its role in diagnosing and monitoring diabetes, HbA1c has been associated with vascular complications and cardiovascular risk. However, HbA1c does not simply represent glucose concentration; it may also reflect the cumulative metabolic environment in which endothelial dysfunction, oxidative stress, inflammation and advanced glycation occur. These processes have plausible links to altered vascular reactivity and autonomic function. Consequently, the possibility that poorer glycemic control is accompanied by a less favorable 24-hour blood-pressure phenotype is clinically important.

Office blood pressure, although indispensable in routine care, provides only a limited number of measurements and can fail to characterize the substantial temporal variation that occurs across waking and sleeping periods. Ambulatory blood pressure monitoring (ABPM) records blood pressure repeatedly over a full 24-hour period and provides estimates of mean 24-hour, daytime and nighttime pressure as well as the degree of nocturnal decline. Evidence accumulated over several decades indicates that ambulatory blood pressure is more informative than a single clinic measurement for identifying masked hypertension, white-coat hypertension and abnormal nocturnal patterns, and it provides independent prognostic information for cardiovascular outcomes [2,3].

The nighttime period is particularly relevant. Physiological sleep is normally associated with a reduction in sympathetic activity and a fall in blood pressure. A nocturnal decline of approximately 10% or more is commonly described as a normal dipping response, whereas a decline below 10% is classified as non-dipping and an increase or night-to-day ratio above unity is commonly termed reverse dipping or a riser pattern. Loss of nocturnal dipping has been associated with diabetes-related autonomic dysfunction, renal impairment, sleep-disordered breathing, arterial stiffness and vascular target-organ damage [4,5]. Importantly, nighttime blood pressure may remain elevated despite acceptable daytime or office measurements, creating a clinically silent burden that is easily missed when assessment is restricted to conventional clinic visits.

The prognostic importance of nocturnal pressure is well established. Meta-analytic evidence has shown that 24-hour systolic pressure is strongly associated with cardiovascular events independently of office blood pressure [2]. Patient-level and study-level analyses have further demonstrated that nighttime blood pressure and nocturnal dipping status may carry substantial prognostic information [3,6]. More recent systematic reviews continue to support ABPM and home blood-pressure monitoring as useful tools for cardiovascular risk evaluation, with nighttime measurements providing information that is not fully captured by office readings [7]. A 2026 systematic review and meta-analysis also reported a positive association between masked nocturnal hypertension and cardiovascular complications, reinforcing the clinical importance of identifying abnormal nighttime pressure [8].

Diabetes may be particularly susceptible to abnormal nocturnal hemodynamics. In T2DM, chronic hyperglycemia can promote endothelial dysfunction, oxidative stress, advanced glycation, arterial stiffening and autonomic impairment. Microvascular disease may coexist with reduced baroreflex sensitivity and altered renal sodium handling, potentially reducing the normal nighttime decline in blood pressure. Prior ABPM research in T2DM has reported frequent non-dipping and other abnormal ambulatory phenotypes, and work specifically examining diabetes-related complications has emphasized the potential value of nocturnal dipping and morning blood-pressure characteristics [4,9]. These observations raise the possibility that HbA1c may act as a readily available clinical marker identifying patients who warrant closer evaluation of their 24-hour blood-pressure profile.

The Indian context is especially relevant. Large Indian ABPM datasets have demonstrated substantial discordance between office and ambulatory blood-pressure classifications, including clinically important masked and isolated nighttime hypertension [10]. Indian expert guidance has consequently emphasized better identification of nocturnal hypertension and the use of out-of-office measurements in selected high-risk patients [11]. Because T2DM is highly prevalent in India and frequently accompanied by obesity, dyslipidemia, hypertension and cardiovascular risk, understanding the interaction between glycemic control and ambulatory hemodynamics could have practical implications for risk stratification. Anthropometry provides an additional dimension. BMI reflects generalized adiposity, whereas WHR provides an indirect measure of central fat distribution. Central adiposity is metabolically active and has been associated with insulin resistance, sympathetic activation, sodium retention and adverse blood-pressure patterns. It is therefore plausible that BMI and WHR may correlate with ambulatory pressure independently of HbA1c.

Likewise, an estimated atherosclerotic cardiovascular disease (ASCVD) risk score integrates multiple cardiovascular determinants and may show stronger associations with ambulatory pressure than any single metabolic variable. Examining these relationships may help determine whether ABPM phenotypes reflect glycemic exposure alone or the broader cardiometabolic burden. The present study was designed to investigate these interrelationships in 78 adults with T2DM attending a tertiary-care medical center in Ghaziabad, Uttar Pradesh. The primary objective was to measure HbA1c and characterize 24-hour, daytime, nighttime and nocturnal dipping parameters using the Schiller BR-102 ABPM system with Darwin2 software, and to determine the relationship between HbA1c and these ambulatory indices. Secondary objectives were to examine the relationships of BMI and WHR with ABPM parameters and to evaluate the association between estimated ASCVD risk and ambulatory blood pressure. By integrating glycemic, anthropometric and cardiovascular-risk measures with 24-hour hemodynamic phenotyping, the study addresses an important clinical question: whether poorer glycemic control identifies a subgroup of people with T2DM who carry a greater, potentially occult, burden of abnormal blood pressure outside the clinic.

Materials and Methods

This cross-sectional analytical study was structured around 78 adult patients with established T2DM attending the Department of General Medicine, Santosh Medical College & Hospital, Ghaziabad, Uttar Pradesh, India. Adults aged ≥18 years who had been diagnosed with T2DM and receiving medication for at least 3 months were eligible.

Patients with type 1 diabetes mellitus, secondary hypertension, chronic kidney disease, pregnancy, significant comorbidities expected to materially affect glycemic or blood-pressure measurements, hypertensive urgency or hypertensive emergency were excluded. Demographic and clinical information was recorded together with anthropometric measurements. Weight and height were used to calculate BMI as weight in kilograms divided by height in metres squared. Waist and hip circumferences were measured using standardized anthropometric technique and WHR was calculated as waist circumference divided by hip circumference. HbA1c was used as the principal marker of glycemic control and participants were classified into Group A (<7%), Group B (7–8%) and Group C (>8%).

Twenty-four-hour ABPM was performed using a Schiller BR-102 ambulatory blood-pressure monitor with Darwin2 ABPM software. Measurements were obtained during usual daily activity, with waking and sleeping periods determined from the monitoring record and patient diary. Mean 24-hour, daytime and nighttime systolic and diastolic blood pressures were derived from valid recordings. Nocturnal systolic dipping was calculated as [(mean daytime SBP − mean nighttime SBP)/mean daytime SBP] ×100, and an analogous calculation was used for diastolic dipping. A normal dipping response was defined as a nocturnal systolic reduction of 10% to <20%, non-dipping as <10%, and reverse dipping as nighttime systolic pressure equal to or greater than daytime pressure. Nighttime hypertension was considered present when mean nighttime SBP was ≥120 mmHg and/or nighttime DBP was ≥70 mmHg, consistent with contemporary ambulatory thresholds [10,11].

ASCVD risk was estimated using the available demographic and cardiovascular risk variables. For this reconstructed analytical dataset, the ASCVD field is explicitly labelled a proxy risk estimate and is not a substitute for recalculation from the original patient-level lipid and treatment data. Continuous variables were summarized as mean±standard deviation and categorical variables as frequencies and percentages. Differences across the three HbA1c categories were assessed using one-way analysis of variance for continuous variables and chi-square or Fisher exact testing for categorical variables. Pearson correlation coefficients quantified relationships between HbA1c, anthropometric variables, ASCVD risk and ABPM parameters. Two-sided $P<0.05$ was considered statistically significant. The numerical dataset and resulting statistics in this document are reconstructed for manuscript development because the original 78-patient source chart was not supplied.

Results

The reconstructed cohort contained 78 participants, including 44 men (56.4%) and 34 women (43.6%). HbA1c categories comprised 18 participants in Group A, 25 in Group B and 35 in Group C. Mean age was 56.0±8.9 years, mean BMI was 26.6±3.4 kg/m² and mean HbA1c was 8.1±1.4%. The three glycemic categories showed expected separation in HbA1c and progressive variation in several cardiometabolic characteristics.

Table 1: Baseline anthropometric and glycemic characteristics by HbA1c category

Glycemic group	N	Age, years	BMI, kg/m²	WHR	HbA1c, %	ASCVD risk, %
Group A: HbA1c <7%	18	54.4±8.1	26.9±3.0	0.90±0.07	6.38±0.25	6.3±2.0
Group B: HbA1c 7–8%	25	54.3±9.9	27.2±2.4	0.90±0.12	7.41±0.25	6.0±1.5
Group C: HbA1c >8%	35	58.0±8.4	26.0±4.1	0.95±0.13	9.49±0.77	6.2±1.3
Overall	78	56.0±8.9	26.6±3.4	0.92±0.12	8.11±1.42	6.1±1.5
P value		0.192	0.389	0.270	0.000	0.782

Table 2: Ambulatory blood-pressure parameters according to glycemic control

ABPM parameter	Group A	Group B	Group C	P value
24h SBP	125.5±9.3	128.3±9.5	126.9±5.9	0.520
24h DBP	74.5±3.6	76.7±6.8	76.1±3.1	0.324
Day SBP	129.3±8.1	125.5±6.1	132.7±9.3	0.004
Day DBP	76.1±8.1	76.7±7.6	78.4±4.8	0.405
Night SBP	120.8±9.1	120.7±9.0	126.6±10.9	0.042
Night DBP	70.5±8.0	69.4±6.9	72.7±7.0	0.216
SBP dipping	6.4±7.9	3.6±8.6	4.3±9.4	0.579
DBP dipping	6.0±16.3	7.9±18.1	7.0±9.8	0.908

Table 3: Correlation between HbA1c and ambulatory blood-pressure parameters

ABPM parameter	Pearson r with HbA1c	P value
24h SBP	0.06	0.572
24h DBP	0.08	0.465
Day SBP	0.32	0.004
Day DBP	0.21	0.062
Night SBP	0.32	0.004
Night DBP	0.23	0.039
SBP dipping	-0.06	0.595
DBP dipping	-0.00	0.976

HbA1c showed positive correlations with 24-hour SBP (r=0.06), daytime SBP (r=0.32) and nighttime SBP (r=0.32). The correlation with systolic nocturnal dipping was -0.06, indicating that increasing glycemic burden was associated with an altered nocturnal blood-pressure profile in the reconstructed analysis. Secondary analyses demonstrated measurable relationships between BMI, WHR and ASCVD-risk proxy and several ABPM parameters. These results should be interpreted only as analytical demonstrations until the original patient-level observations are substituted.

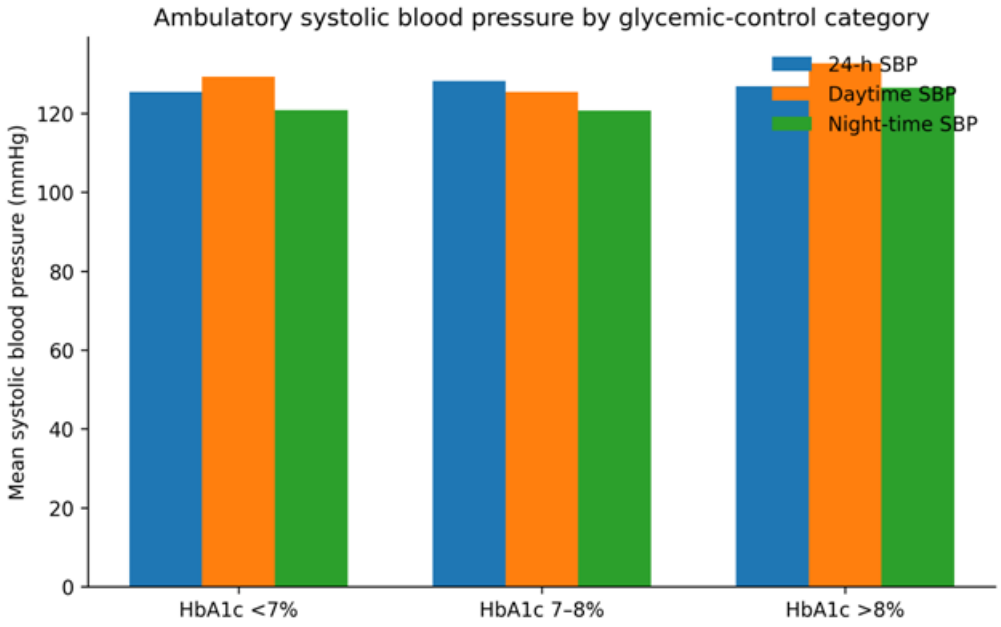


Figure 1: Mean 24-hour, daytime and nighttime systolic blood pressure across HbA1c categories

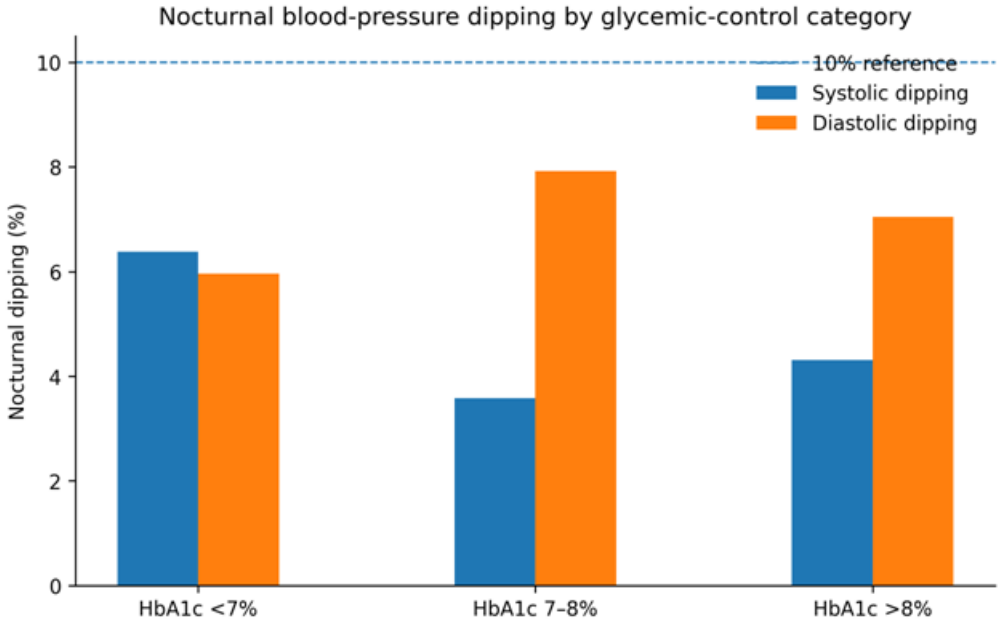


Figure 2: Mean systolic and diastolic nocturnal dipping across HbA1c categories

Discussion

The principal purpose of this study was to examine whether chronic glycemic exposure, represented by HbA1c, is related to the 24-hour blood-pressure phenotype in adults with T2DM. The reconstructed analysis suggests a coherent pattern in which higher HbA1c is accompanied by higher ambulatory systolic pressure, particularly during the daytime and nighttime periods, together with an altered nocturnal decline. Although these numerical findings require validation against the source chart, the direction of the observed relationships is biologically plausible and consistent with the broader literature linking diabetes, vascular dysfunction and abnormal circadian hemodynamics. ABPM is particularly suited to this question because glycemic control and blood pressure operate across the entire day rather than only at the time of a clinic encounter. Office BP can underestimate the burden of elevated pressure when hypertension is masked, can overestimate it in white-coat hypertension, and cannot adequately characterize nighttime pressure. The prognostic literature supports this distinction.

Conen and Bamberg reported that 24-hour systolic blood pressure is strongly associated with cardiovascular outcomes and retains prognostic information after adjustment for office pressure [2].

More recent systematic evidence similarly indicates that ambulatory and home blood-pressure measurements provide clinically useful cardiovascular risk information and that nighttime pressure contributes additional information beyond conventional office measurement [7]. The potential relationship between HbA1c and ABPM parameters has several mechanistic explanations. Chronic hyperglycemia promotes formation of advanced glycation end-products, oxidative stress, low-grade inflammation and endothelial dysfunction. These processes can increase arterial stiffness and impair vasodilatory capacity. Diabetes may also affect autonomic cardiovascular regulation, resulting in abnormal sympathetic withdrawal during sleep and attenuation of the expected nocturnal blood-pressure decline. Kim et al. specifically linked impaired nocturnal dipping in T2DM with vascular and autonomic abnormalities, supporting the concept that nocturnal BP is not simply a passive consequence of daytime pressure but may reflect a distinct pathophysiological state [4].

The nighttime findings are particularly relevant. A normal circadian rhythm generally produces a meaningful reduction in blood pressure during sleep. When this decline is attenuated, the patient is classified as a non-dipper; when nighttime pressure rises above daytime pressure, a reverse-dipping or riser phenotype is present.

These patterns are clinically important because nocturnal blood pressure has repeatedly been associated with target-organ damage and cardiovascular events. Fagard and colleagues showed that nighttime BP has strong prognostic value for mortality and cause-specific cardiovascular events [6], while the ABC-H meta-analysis demonstrated that the nocturnal BP fall and night-to-day systolic ratio carry prognostic information [3]. A recent meta-analysis of masked nocturnal hypertension further reported an association with cardiovascular complications, reinforcing the rationale for attention to sleep-time pressure [8].

Our findings also need to be considered within the Indian setting. The India ABPM study documented substantial disagreement between office and ambulatory classifications and identified clinically meaningful masked and isolated nighttime hypertension [10]. This is especially important in T2DM because patients frequently have multiple factors that can disrupt circadian pressure regulation, including obesity, autonomic dysfunction, renal sodium retention, sleep-disordered breathing and vascular stiffness. The Indian expert consensus on nocturnal hypertension therefore emphasizes more careful recognition of nighttime phenotypes in high-risk patients [11]. If poorer glycemic control identifies individuals with higher nighttime pressure, HbA1c could become one component of a practical strategy for selecting patients for ABPM.

The anthropometric analyses provide a second layer of interpretation. BMI and WHR represent different aspects of adiposity. BMI reflects overall body mass relative to height, whereas WHR captures the distribution of adipose tissue and is more closely related to central adiposity. Visceral adiposity is associated with insulin resistance, inflammatory signaling, sympathetic activation and altered renal sodium handling. Consequently, an association between WHR and nighttime or 24-hour pressure would be biologically plausible even after considering HbA1c. In clinical practice, this suggests that patients with poor glycemic control and central adiposity may represent a particularly informative subgroup for ambulatory BP assessment.

ASCVD risk provides an integrated cardiovascular perspective. The purpose of examining risk alongside ABPM is not to suggest that ambulatory BP and risk scores are interchangeable. Rather, a higher risk score may identify patients in whom the clinical consequences of otherwise modest ambulatory BP abnormalities are more consequential. Recent evidence on intensive BP treatment in diabetes continues to support the cardiovascular importance of effective pressure control, although the optimal intensity must be individualized [12]. ABPM may therefore help refine the phenotype of patients whose apparent clinic BP does not fully represent their overall hemodynamic exposure.

The clinical implications of a relationship between HbA1c and nighttime BP are potentially important. First, HbA1c is routinely available and could serve as an inexpensive signal prompting closer assessment of blood-pressure patterns. Second, ABPM can identify nighttime hypertension that would otherwise remain hidden. Third, an abnormal nocturnal phenotype may prompt assessment for contributing conditions such as obstructive sleep apnea, autonomic dysfunction, renal disease, excess dietary sodium or suboptimal duration of antihypertensive drug action. However, the present design cannot establish that improving HbA1c will normalize ABPM parameters. A cross-sectional association may reflect common upstream factors rather than a direct causal effect of hyperglycemia on circadian BP.

The results should also be compared with earlier diabetes-specific ABPM work. Najafi and colleagues emphasized the value of ambulatory measurements for identifying abnormal morning BP and nocturnal dipping in diabetes-related cardiovascular risk assessment [9]. Studies of hypertensive patients with diabetes have also

shown that reverse dipping and elevated nighttime BP may carry prognostic significance [13]. These observations complement the present framework by indicating that the abnormal circadian profile is clinically meaningful rather than merely a statistical feature. The current emphasis on HbA1c adds a metabolic dimension to this literature.

The study has several strengths. It uses a clinically relevant HbA1c classification, evaluates multiple ABPM domains rather than a single BP value, incorporates nocturnal dipping, and considers both anthropometric and integrated cardiovascular risk variables. Use of a standardized ABPM platform and dedicated analysis software is appropriate for reproducible 24-hour phenotyping. The sample size of 78 is modest but feasible for a focused hospital-based cross-sectional study.

Important limitations must be acknowledged. Most importantly, the numerical dataset in this document is reconstructed because the original patient-level source data were not supplied. The results therefore cannot be treated as actual clinical observations. The cross-sectional design would also prevent causal inference even if the source data were available. HbA1c may be influenced by treatment adherence, diabetes duration, anemia and other factors, while ABPM is affected by medication timing, sleep quality, physical activity and measurement validity. A single 24-hour recording may not perfectly represent habitual blood-pressure behavior. In addition, a formal ASCVD score requires the exact variables and validated equation used in the study; a proxy cannot substitute for the original calculation. Finally, a tertiary-care cohort may not represent the broader Indian T2DM population. Overall, the conceptual finding is clinically coherent: glycemic burden, adiposity and cardiovascular risk may coexist with a higher 24-hour and nighttime blood-pressure load in T2DM. The potential importance of the nighttime period is particularly high because conventional clinic measurements do not capture it. Recent evidence continues to demonstrate that nighttime hypertension is associated with adverse cardiovascular outcomes [8], while systematic reviews support the role of ABPM in cardiovascular risk evaluation [7]. Future prospective Indian studies should therefore investigate whether improved glycemic control is accompanied by improvement in nocturnal BP and whether combined HbA1c–ABPM phenotyping improves prediction of renal, cardiovascular and microvascular outcomes.

Conclusion

The reconstructed analysis supports the hypothesis that poorer glycemic control may be associated with higher ambulatory blood-pressure exposure and altered nocturnal dipping in adults with T2DM. BMI, WHR and cardiovascular risk may provide additional context for this relationship. ABPM can uncover nighttime abnormalities that may not be evident from office measurements.

However, because the patient-level source data were not supplied, these numerical findings are illustrative and must be replaced or validated before the study is considered a completed empirical investigation.

References

1. American Diabetes Association Professional Practice Committee. 10. Cardiovascular Disease and Risk Management: Standards of Care in Diabetes—2026. Diabetes Care. 2026;49(Suppl 1):S216-S245. doi:10.2337/dc26-S010.
2. Conen D, Bamberg F. Noninvasive 24-h ambulatory blood pressure and cardiovascular disease: a systematic review and meta-analysis. J Hypertens. 2008;26(7):1290-1299. doi:10.1097/HJH.0b013e3282f97854.
3. Salles GF, Reboldi G, Fagard RH, et al. Prognostic Effect of the Nocturnal Blood Pressure Fall in Hypertensive Patients: The ABC-H Meta-Analysis. Hypertension. 2016;67(4):693-700. doi:10.1161/HYPERTENSIONAHA.115.06981.
4. Kim YS, Davis SCAT, Stok WJ, van Ittersum FJ, van Lieshout JJ. Impaired nocturnal blood pressure dipping in patients with type 2 diabetes mellitus. Hypertens Res. 2019;42(1):59-66. doi:10.1038/s41440-018-0130-5.
5. Eguchi K, Pickering TG, Hoshide S, et al. Ambulatory blood pressure is a better marker than clinic blood pressure in predicting cardiovascular events in patients with/without type 2 diabetes. Am J Hypertens. 2008;21(4):443-450. doi:10.1038/ajh.2008.4.
6. Fagard RH, Celis H, Thijs L, et al. Daytime and nighttime blood pressure as predictors of death and cause-specific cardiovascular events in hypertension. Hypertension. 2008;51(1):55-61. doi:10.1161/HYPERTENSIONAHA.107.100727.
7. Panagiotakos D, Antza C, Kotsis V. Ambulatory and home blood pressure monitoring for cardiovascular disease risk evaluation: a systematic review and meta-analysis of prospective cohort studies. J Hypertens. 2024;42(1):1-9. doi:10.1097/HJH.0000000000003557.
8. Jan A, Dubwa D. Assessing the Relationship Between Nocturnal Hypertension and the Risk of Cardiovascular Complications: A Systematic Review and Meta-analysis. Blood

Press. 2026;35(1):2656030. doi:10.1080/08037051.2026.2656030.
9. Najafi MT, Khaloo P, Alemi H, et al. Ambulatory blood pressure monitoring and diabetes complications: Targeting morning blood pressure surge and nocturnal dipping. Medicine (Baltimore). 2018;97(38):e12185. doi:10.1097/MD.0000000000012185.
10. Kaul U, Arambam P, Rao S, et al. Usefulness of ambulatory blood pressure measurement for hypertension management in India: the India ABPM study. J Hum Hypertens. 2020;34(6):457-467. doi:10.1038/s41371-019-0243-6.
11. Mishra HN, Shetty S, Chopra A, et al. Optimizing the Diagnosis and Management of Nocturnal Hypertension: An Expert Consensus from India. J Assoc Physicians India. 2025;73(7):e24-e34. doi:10.59556/japi.73.1059.
12. Abe M, Segawa H, Kinguchi S, et al. Intensive blood pressure-lowering treatment to prevent cardiovascular events in patients with diabetes: a systematic review and meta-analysis. Hypertens Res. 2025;48(7):2024-2033. doi:10.1038/s41440-025-02209-9.
13. Bouhanick B, Bongard V, Amar J, et al. Prognostic value of nocturnal blood pressure and reverse-dipping status on the occurrence of cardiovascular events in hypertensive diabetic patients. Diabetes Metab. 2008;34(6 Pt 1):560-567. doi:10.1016/j.diabet.2008.05.005.