

Original Research Article

EVOLUTION OF 24-HOUR AMBULATORY BLOOD PRESSURE VARIABILITY AND DIPPING STATUS PATTERNS IN ACUTE ISCHEMIC STROKE: A PROSPECTIVE OBSERVATIONAL STUDY FROM NORTHERN INDIA

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ABSTRACT

Background: Acute ischemic stroke (AIS) induces complex hemodynamic shifts, including altered blood pressure variability (BPV) and disrupted circadian blood pressure rhythms. This study evaluates the evolution of 24-hour ambulatory blood pressure parameters, specifically BPV and nocturnal dipping status, from admission to discharge in an AIS cohort from Northern India.

Materials and Methods: A prospective observational study was conducted following STROBE guidelines. Patients presenting with AIS underwent 24-hour Ambulatory Blood Pressure Monitoring (ABPM) at admission and prior to discharge. Mean systolic (SBP) and diastolic blood pressures (DBP), BPV (calculated as the standard deviation of SBP), and nocturnal dipping patterns were recorded and analyzed.

Results: The cohort demonstrated a significant reduction in overall blood pressure during hospitalization. Mean 24-hour SBP decreased from 137.19 ± 9.10 mmHg at admission to 132.50 ± 8.13 mmHg at discharge, and mean 24-hour DBP decreased from 71.56 ± 5.94 mmHg to 67.50 ± 5.51 mmHg. A marked reduction in BPV was observed, with the SD of SBP declining sharply from 12.55 ± 0.61 mmHg to 6.97 ± 0.61 mmHg. However, circadian rhythm impairment persisted; 90.5% of patients were classified as non-dippers at admission, and 84.1% remained non-dippers at discharge, showing no statistically significant improvement ($p = 0.18$).

Conclusion: Standard inpatient stroke management effectively stabilizes mean blood pressure and significantly reduces pathological BPV. However, mechanisms regulating nocturnal blood pressure dipping remain profoundly impaired during the acute-to-subacute phase. This persistent non-dipping status suggests prolonged central autonomic dysfunction despite overall hemodynamic stabilization, emphasizing the need for extended autonomic monitoring and potential chronotherapeutic interventions.

Keywords: Acute Ischemic Stroke, Ambulatory Blood Pressure Monitoring, Blood Pressure Variability, Circadian Rhythm, Dipping Status, Autonomic Nervous System.

INTRODUCTION

Acute ischemic stroke (AIS) is a major cause of morbidity and mortality globally, with a rapidly increasing burden in Northern India.^[1,2] The acute

phase of AIS is frequently accompanied by a vigorous hemodynamic response. While transient elevations in systemic blood pressure are common and often self-limiting, the dynamic fluctuations—termed blood pressure variability (BPV)—and the

loss of the physiological nocturnal blood pressure drop carry profound prognostic implications.^[3,4]

The autonomic nervous system, primarily regulated by the central autonomic network (including the insular cortex and hypothalamus), is severely disrupted during cerebral ischemia.^[5,6] This dysautonomia manifests hemodynamically as sympathetic overactivity, impaired baroreflex sensitivity, and disruption of the normal circadian rhythm.^[7] High short-term BPV is increasingly recognized as an independent predictor of hemorrhagic transformation, cerebral edema, and poor functional outcomes in stroke.^[8] Furthermore, the loss of normal nocturnal blood pressure dipping (defined as a >10% drop in nighttime compared to daytime blood pressure) indicates persistent sympathetic drive and is a known risk factor for recurrent cerebrovascular events.^[9,10]

While the acute management of blood pressure in stroke is well protocolized, the temporal evolution of these specific autonomic indices—BPV and dipping status—throughout the inpatient stay remains underexplored in South Asian populations.^[11] This study aims to prospectively evaluate the evolution of 24-hour ABPM parameters from admission to discharge in an AIS cohort, testing the hypothesis that while mean pressures may stabilize, deeper markers of autonomic function may exhibit delayed recovery.

MATERIALS AND METHODS

Study Design and Setting This was a prospective, observational cohort study conducted at a tertiary care center in Northern India. The study methodology adhered strictly to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.^[12] The protocol was approved by the Institutional Ethics Committee, and the study was conducted in accordance with the Declaration of Helsinki.^[13] Written informed consent was obtained from all patients or their legal surrogates.

Participants Adult patients (≤75 years) admitted with a radiologically confirmed (Non-Contrast Computed Tomography) diagnosis of acute ischemic stroke within 24–48 hours of symptom onset were enrolled. Exclusion criteria included hemorrhagic stroke, transient ischemic attack (TIA), end-stage renal disease, severe pre-existing autonomic neuropathy, or clinical instability preventing 24-hour ABPM application (e.g., requirement of invasive mechanical ventilation).

Ambulatory Blood Pressure Monitoring (ABPM) Protocol

Non-invasive 24-hour ABPM was performed twice for each patient: initially upon admission (acute phase) and subsequently prior to hospital discharge (subacute phase, typically day 5–7). The ABPM devices were calibrated and fitted to the non-paretic arm according to the European Society of Hypertension guidelines.^[14] Measurements were recorded every 30 minutes during the day (06:00 to 22:00) and every 60 minutes during the night (22:00 to 06:00).^[15]

The following parameters were extracted:

- 24-hour mean Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP).
- Blood Pressure Variability (BPV), quantified as the standard deviation (SD) of the 24-hour SBP.^[16]
- Dipping Status: Defined by the ratio of nighttime to daytime mean SBP. Patients with a nighttime SBP reduction of ≥10% were classified as "dippers," while those with <10% reduction, or an increase in nighttime BP, were classified as "non-dippers".^[17]

Statistical Analysis: Data were expressed as mean ± standard deviation (SD) for continuous variables and as frequencies/percentages for categorical variables.^[18] The paired Student's t-test was utilized to compare continuous ABPM parameters (mean SBP, mean DBP, and SD of SBP) between admission and discharge. McNemar's test was used to evaluate changes in the proportion of non-dippers over the two time points. A two-tailed p-value of <0.05 was considered statistically significant.

RESULTS

Evolution of Mean Blood Pressure and Variability Throughout the hospitalization period, patients exhibited a marked and statistically significant improvement in overall hemodynamic burden. The 24-hour mean SBP demonstrated a significant reduction from 137.19 ± 9.10 mmHg at admission to 132.50 ± 8.13 mmHg at discharge (p < 0.05). Similarly, the 24-hour mean DBP decreased significantly from 71.56 ± 5.94 mmHg at the time of admission to 67.50 ± 5.51 mmHg prior to discharge (p < 0.05).

A particularly profound improvement was observed in blood pressure variability. The SD of the systolic blood pressure, a surrogate marker for short-term BPV and acute sympathetic lability, declined sharply from 12.55 ± 0.61 mmHg to 6.97 ± 0.61 mmHg.

Table 1: Changes in 24-Hour ABPM Parameters from Admission to Discharge

Parameter	Admission (Mean ± SD)	Discharge (Mean ± SD)	Trend
24h Mean SBP (mmHg)	137.19 ± 9.10	132.50 ± 8.13	Significant Reduction
24h Mean DBP (mmHg)	71.56 ± 5.94	67.50 ± 5.51	Significant Reduction
BP Variability (SD of SBP)	12.55 ± 0.61	6.97 ± 0.61	Marked Decrease

Dipping Status and Circadian Rhythm Despite the highly significant reductions in mean blood

pressures and variability, the physiological circadian rhythm remained severely perturbed. At admission,

a vast majority of the cohort—90.5%—exhibited a non-dipping or reverse-dipping pattern. At the time of discharge, despite clinical improvement and mean BP stabilization, 84.1% of the cohort remained classified as non-dippers. This slight reduction did not achieve statistical significance ($p = 0.18$), indicating a persistent impairment of nocturnal blood pressure regulation.

DISCUSSION

This prospective observational study yields a crucial physiological dichotomy in the short-term recovery of patients with acute ischemic stroke: while standard inpatient management rapidly normalizes mean arterial pressures and dampens pathological blood pressure variability, it fails to restore the circadian nocturnal dipping rhythm within the acute-to-subacute window.

The significant reduction in 24-hour SBP (137.19 to 132.50 mmHg) and DBP (71.56 to 67.50 mmHg) aligns with the natural history of post-stroke hemodynamics. Blood pressure typically peaks within the first 24 hours of stroke onset—driven by stress, pain, increased intracranial pressure, and a reactive Cushing's response—and gradually tapers over subsequent days.^[19]

More notably, our data demonstrate a sharp decline in BPV, with the SD of SBP dropping from 12.55 to 6.97 mmHg. High early BPV is a hallmark of acute autonomic failure. In the acute phase, damage to central autonomic pathways or heightened systemic stress severely blunts the cardiovagal baroreflex sensitivity.^[6,16] The rapid improvement in BPV suggests that the extreme, highly labile sympathetic surges seen immediately post-infarct are heavily attenuated by supportive inpatient care, bed rest, and the resolution of acute physiological stress.^[8]

However, the central finding of this study is the stubborn persistence of non-dipping status. With 90.5% non-dippers at admission and 84.1% at discharge ($p = 0.18$), it is evident that the mechanisms regulating nocturnal blood pressure decline remain impaired long after mean blood pressures have stabilized. Normal dipping is regulated by a complex interplay involving the withdrawal of sympathetic tone during sleep, pineal melatonin secretion, and the intrinsic circadian clock of the suprachiasmatic nucleus.^[7,20]

The persistence of non-dipping suggests a prolonged state of central autonomic dysfunction. The ischemic insult likely causes a sustained disruption of the pathways connecting the cortex, hypothalamus, and brainstem, preventing the normal nocturnal reduction in sympathetic efferent activity.^[5,10] This finding is highly significant clinically. Persistent non-dipping subjects the cerebral microvasculature to sustained nocturnal shear stress and is a robust independent predictor of recurrent stroke and poor cognitive recovery.^[9]

These findings imply that relying solely on casual or mean daytime blood pressure measurements at discharge is fundamentally insufficient. The underlying autonomic architecture remains fragile. There is a compelling need for integrating ABPM into routine post-stroke care to identify these persistent non-dippers, who may specifically benefit from chronotherapy (the administration of anti-hypertensive medications at bedtime) to manually restore the diurnal hemodynamic profile.^[17,20]

Limitations: The sample reflects a specific demographic in Northern India, which may limit broader generalizability. The study evaluated short-term trends up to hospital discharge; longitudinal follow-up is required to determine exactly when—or if—the circadian dipping rhythm naturally restores itself in this population.

CONCLUSION

In acute ischemic stroke, standard inpatient care effectively stabilizes mean blood pressure parameters and drastically curtails excessive short-term blood pressure variability. However, the autonomic mechanisms responsible for nocturnal blood pressure dipping remain significantly impaired during the short-term hospitalization period. This persistent non-dipping state reveals an ongoing, silent autonomic dysfunction despite apparent hemodynamic stability, highlighting the vital role of continuous ambulatory monitoring and the potential necessity for targeted chronotherapy in early stroke rehabilitation.

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