

ORIGINAL ARTICLE

Tibb-based lifestyle education and sleep and blood pressure changes among university employees with hypertension: A single-group pretest–posttest study

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ABSTRACT

Poor sleep may contribute to inadequate blood pressure control. This preliminary single-group pretest–posttest study examined within-participant changes after a four-week multicomponent Tibb-based lifestyle education program emphasizing sleep–wake regulation. Twenty-nine employees with hypertension from one Indonesian Islamic university completed paired assessments. Sleep-related characteristics were assessed using the seven-item Kuesioner Kualitas Tidur (KKT), and clinic blood pressure was measured using an Omron HEM-8712 monitor. The median KKT score increased from 16 (IQR 15–17) to 21 (IQR 21–22), with a median paired change of +5 points (IQR 3–6; Wilcoxon $W = 4.0$; $p < 0.001$; rank-biserial correlation = 0.98). Mean SBP was lower at follow-up (130.00 ± 7.91 vs 142.83 ± 15.05 mmHg; paired difference -12.83 mmHg; 95% CI -16.32 to -9.33 ; $p < 0.001$; Cohen's $d_z = -1.40$), and DBP was also lower (86.07 ± 6.45 vs 96.14 ± 10.86 mmHg; paired difference -10.07 mmHg; 95% CI -13.13 to -7.00 ; $p < 0.001$; Cohen's $d_z = -1.25$). Favorable within-participant changes were observed. However, the uncontrolled design, medication-related confounding, single blood-pressure readings, and incomplete validation of the KKT preclude causal or definitive effectiveness conclusions.

Keywords: hypertension; sleep quality; lifestyle education; Tibb; university employees; pretest–posttest study

Introduction

Hypertension remains a major contributor to cardiovascular morbidity and premature mortality worldwide [1–4]. Alongside pharmacotherapy, blood-pressure management requires sustained attention to modifiable behaviors, including diet, physical activity, stress, and sleep [5,9,22].

Poor sleep may contribute to inadequate blood-pressure control through sustained sympathetic activation, circadian disruption, altered neuroendocrine regulation, and attenuation of the normal nocturnal decline in blood pressure [6,7,17]. These mechanisms are established within contemporary sleep and cardiovascular science and should not be interpreted as biological pathways specific to Tibb.

Tibb organizes health maintenance around six interrelated lifestyle domains: environmental air and breathing, food and drink, movement and rest, sleep and wakefulness, emotional regulation, and elimination and retention [13–16]. In the present study, Tibb served as a culturally contextualized framework for structuring and communicating lifestyle guidance, rather than as an independently established therapeutic mechanism.

University employees may experience workload pressure, irregular schedules, and occupational stress that influence sleep and cardiovascular health [10–12,18]. Sleep-hygiene education and broader lifestyle interventions have been examined in people with hypertension [8,9], but evidence remains limited for workplace programs that use a Tibb framework to organize sleep–wake and related lifestyle education in an Indonesian

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Islamic university. It also remains uncertain whether such a culturally framed, digitally reinforced program is associated with measurable short-term changes in self-reported sleep characteristics and clinic blood pressure.

The program was not designed as a pharmacist-led intervention. Nevertheless, some components may inform future pharmacy-based models that combine sleep-related screening, antihypertensive medication review, adherence assessment, brief lifestyle counseling, digital reinforcement, and referral. Such applicability requires direct testing in pharmacist-led or interprofessional controlled studies [22].

This preliminary study examined within-participant changes in the KKT total score, systolic blood pressure (SBP), and diastolic blood pressure (DBP) after a four-week multicomponent Tibb-based lifestyle education program emphasizing sleep-wake regulation. The study was not designed to establish a Tibb-specific biological mechanism, compare Tibb with conventional sleep education, isolate the contribution of individual program components, or estimate a controlled causal effect.

METHODS

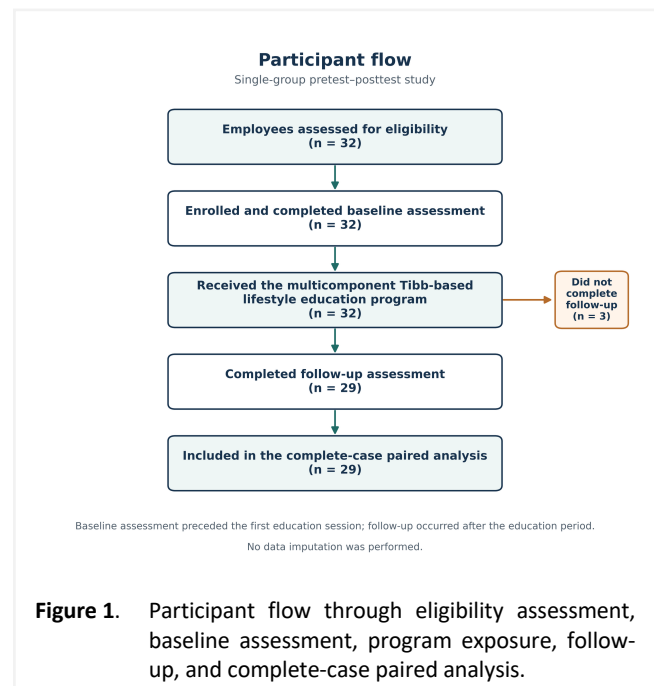
Study design, setting, and participants

This preliminary single-group pretest-posttest study was conducted at one workplace at UIN Syarif Hidayatullah Jakarta, an Indonesian Islamic university. Overall study activities were conducted from November 2024 to February 2025, while the primary baseline and follow-up data collection period was reported as December 2024 to January 2025. All participants received the educational program, and no concurrent control or comparison group was included. The analysis therefore evaluated within-participant changes between baseline and follow-up rather than between-group effects.

The eligible population consisted of 32 employees identified through a preliminary workplace assessment. Total sampling was used. Inclusion criteria were employment at the university, a prior clinical diagnosis of hypertension, current or previous antihypertensive medication use, and willingness to participate. Exclusion criteria were planned employ

ent discontinuation, incomplete outcome data, or

withdrawal. All 32 enrolled participants completed baseline assessment and received the educational program. Three participants could not be contacted at follow-up and therefore did not complete the follow-up questionnaire or blood-pressure assessment. The final complete-case paired analysis included 29 participants; no data imputation was performed. Participant flow is shown in **Figure 1**.



Assessment schedule

Baseline KKT and clinic blood-pressure assessments were completed before the education program. The multicomponent education period lasted four weeks, and follow-up assessments were conducted approximately one month after baseline, following completion of the program. The same outcome instruments and measurement procedures were used at both assessments.

Hypertension eligibility and blood-pressure control classification

Participants were not newly diagnosed during the study. Eligibility required a prior clinical diagnosis and self-reported current or previous antihypertensive medication use. The national threshold of SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg was treated as a diagnostic reference rather than a diagnosis derived from one study visit [3].

For secondary descriptive analysis, the source protocol classified blood-pressure control using age-specific JNC 8 treatment targets: SBP <140 mmHg and DBP <90 mmHg for participants aged <60 years, and SBP <150 mmHg and DBP <90 mmHg for participants aged ≥60 years [23]. These thresholds represented treatment-control status, not diagnostic criteria. Continuous SBP and DBP values and their paired changes were prioritized as the principal blood-pressure outcomes.

Antihypertensive medication profile

Baseline antihypertensive medication use was recorded through participant self-report. Medication use was not systematically reassessed at follow-up. Medication adherence, dose changes, treatment initiation or discontinuation, and regimen stability were not objectively monitored. Antihypertensive treatment was therefore considered an unmeasured potential confounder.

Multicomponent Tibb-based lifestyle education program

The four-week program emphasized sleep-wake regulation but also addressed environmental air and breathing, food and drink, movement and rest, emotional regulation, and elimination and retention. Delivery included educational sessions, breathing practice, face-to-face assessment, webinar instruction, WhatsApp-group reinforcement, infographics, short videos, written guidance, and behavioral reminders. Because the components were delivered as an integrated educational package and component-specific adherence was not objectively quantified, their separate contributions could not be estimated.

Outcome measurements

Sleep-related characteristics were assessed using the Kuesioner Kualitas Tidur (KKT), an Indonesian seven-item self-report questionnaire adapted from selected dimensions of the Pittsburgh Sleep Quality Index and St. Mary's Hospital Sleep Questionnaire [24–26]. The version used in the study assessed sleep latency, nighttime sleep duration, nighttime awakening, morning freshness, sleep depth, sleep satisfaction, and daytime nap duration. Each item was scored from 1 to 4, producing a total score from 7 to 28, with higher scores

indicating more favorable self-reported sleep characteristics.

The source study classified scores of 21–28 as good, 14–20 as less good, and 7–13 as poor. These categories were generated by dividing the possible score range into three equal intervals and were used only for secondary descriptive interpretation. Preliminary local testing in an independent convenience sample of 30 respondents produced statistically significant item-total correlations ranging from 0.406 to 0.699 and a Cronbach's alpha of 0.657. Thus, all seven items showed acceptable item-total association in the local test sample, while internal consistency was modest. Because broader measurement properties, including structural validity, criterion validity, test-retest reliability, measurement error, responsiveness, and a clinically meaningful change threshold, have not been established in employees with hypertension [27], the continuous KKT total score was prioritized and categorical thresholds were not treated as diagnostic cut-offs.

Clinic blood pressure was measured using an Omron HEM-8712 automated upper-arm monitor. A single reading was recorded at baseline and a single reading at follow-up. Before each measurement, participants sat comfortably and remained silent for five minutes. Measurements were obtained with the participant seated in a chair with back support; the measured arm was supported on a table at heart level with the elbow flexed to minimize isometric muscle contraction. The same monitor and documented positioning procedure were used at both assessments. The source documentation did not specify the arm used, cuff-size selection, measurement time of day, or premeasurement restrictions on caffeine intake, smoking, and physical activity. The principal outcomes were within-participant changes in the KKT total score, SBP, and DBP. Categorical KKT and blood-pressure-control status were secondary descriptive outcomes.

Ethics and consent

Ethical approval was obtained from the Ethics Committee of the Faculty of Health Sciences, UIN Syarif Hidayatullah Jakarta (No. Un.01/F.10/KP.01.1/KE.SP/01.08.008/2025). All participants provided informed consent.

Statistical analysis

Participant characteristics and secondary categorical outcomes were summarized using frequencies and percentages. KKT scores were summarized using median and interquartile range, while blood-pressure values were summarized using mean and standard deviation. Distributional assumptions for paired analyses were evaluated using individual difference scores calculated as follow-up minus baseline, rather than evaluating baseline and follow-up distributions separately.

KKT change was analyzed using the Wilcoxon signed-rank test, with matched-pairs rank-biserial correlation as the effect-size measure. Blood-pressure changes were summarized using mean paired differences, standard deviations of paired differences, and 95% confidence intervals. Paired t-tests and Cohen's d_z were used for mean-change reporting; d_z was calculated as the mean paired difference divided by the standard deviation of paired differences. Because SBP differences departed from normality, a Wilcoxon sensitivity analysis was also conducted. All tests were two-sided, with $p < 0.05$ considered statistically significant. Mediation and adjusted regression analyses were not retained because the design and sample size did not support identifiable and stable estimates [21,28–30]. Data cleaning and initial data management were performed using Microsoft Excel, and the original statistical analyses were conducted using IBM SPSS Statistics. The participant-level paired analyses, confidence intervals, and effect-size estimates reported in this revised article were recalculated and independently verified. Software versions were not specified in the available source documentation.

RESULT AND DISCUSSION

Participant characteristics. Twenty-nine participants completed baseline and follow-up assessments. Corrected baseline characteristics are presented in **Table 1**. Age categories were made mutually exclusive and percentages were calculated using 29 as the denominator.

Baseline medication profile. The baseline self-reported antihypertensive medication profile is shown

Table 1. Baseline characteristics of participants

Characteristic	Category	n	%
Age group	18–44 years	7	24.1
	45–59 years	20	69.0
	≥60 years	2	6.9
Sex	Male	21	72.4
	Female	8	27.6
Occupation	Lecturer	12	41.4
	Staff	10	34.5
	Contract/honorary employee	3	10.3
	Leadership position	2	6.9
Temperament	Section head	2	6.9
	Sanguinous	19	65.5
	Phlegmatic	5	17.2
	Bilious	4	13.8
	Melancholic	1	3.4

Note: Percentages were calculated using 29 participants and rounded to one decimal place.

Table 2. Baseline self-reported antihypertensive medication profile

Treatment profile	Medication	n	%
Monotherapy	Amlodipine	13	44.8
	Captopril	3	10.3
	Losartan	2	6.9
	Bisoprolol	2	6.9
	Candesartan	1	3.4
	Ramipril	1	3.4
Combination therapy	Furosemide	1	3.4
	Amlodipine + captopril	2	6.9
	Nifedipine + ramipril	1	3.4
	Bisoprolol + candesartan	1	3.4
	Furosemide + amlodipine	1	3.4
	Total	29	100.0

Note: Medication information was self-reported at baseline. Medication adherence, dose, treatment changes, and regimen stability during follow-up were not systematically assessed.

in **Table 2**. Generic medication names are used throughout. No longitudinal medication-status data were available.

The multicomponent education protocol is summarized in **Table 3**.

Principal paired outcomes. The principal paired results are presented in **Table 4**. **Figure 2** displays the corresponding group-level baseline and follow-up summaries. **Table 4** is the primary source for statistical interpretation because it reports paired changes, confidence intervals, tests, and effect sizes, whereas **Figure 2** facilitates visual assessment of direction and dispersion. KKT is summarized using the median and IQR, while SBP and DBP are summarized using the mean and SD; therefore, comparisons should be made within each panel rather than across outcome scales. The median KKT total score increased from 16 (IQR 15–17) to 21 (IQR 21–22), with a median paired change of +5 points (IQR 3–6). Twenty-seven participants had higher follow-up scores, one showed no change, and one had a lower score. The Wilcoxon signed-rank test yielded $W = 4.0$, $p < 0.001$, with a matched-pairs rank-biserial correlation of 0.98.

Mean SBP was lower at follow-up, with a mean paired difference of -12.83 mmHg (95% CI, -16.32 to -9.33 ; $t(28) = -7.51$; $p < 0.001$; Cohen's $d_z = -1.40$). Because paired SBP differences departed from normality (Shapiro–Wilk $W = 0.919$; $p = 0.029$), a Wilcoxon

sensitivity analysis produced a consistent result ($W = 15.5$; $p < 0.001$; rank-biserial correlation = -0.93). Mean DBP was also lower at follow-up, with a mean paired difference of -10.07 mmHg (95% CI, -13.13 to -7.00 ; $t(28) = -6.73$; $p < 0.001$; Cohen's $d_z = -1.25$).

Secondary descriptive outcomes. Secondary categorical changes are shown in **Table 5**. These categories were not used as the principal basis for inference.

Principal findings and comparison with existing evidence. This preliminary study found higher self-reported KKT scores and lower clinic SBP and DBP values at follow-up than at baseline during the four-week education period. **Table 4** provides the inferential estimates, while **Figure 2** provides a descriptive visual summary of the same outcomes. The sleep-related finding is directionally consistent with evidence that structured sleep-hygiene and behavioral education may improve self-reported sleep outcomes [8,19,20]. The blood-pressure pattern is also compatible with evidence linking sleep, autonomic regulation, circadian physiology, stress, and broader lifestyle behavior with hypertension management [6,7,9]. The magnitude of the paired changes provides a useful signal for designing a future controlled study, but clinical effectiveness cannot

Table 3. Multicomponent Tibb-based lifestyle education program emphasizing sleep–wake regulation

Phase	Content and delivery	Timing	Follow-up support
Session 1	Metabolic syndrome in Tibb and contemporary perspectives; baseline orientation; lecture and discussion.	Week 1; 60 min	Attendance and orientation
Session 2	Blood glucose, body fat, blood pressure, and rhythmic breathing; lecture, discussion, and practice.	Week 1; 150 min	Practical demonstration
Session 3	Tibb lifestyle education emphasizing sleep–wake regulation, behavioral commitment, and self-recording.	Week 3; 150 min	Weekly monitoring
End-of-exposure evaluation	Review of activity, sleep, dietary behavior, stress, and SpO ₂ ; online checklist.	Week 5; 150 min	Checklist not treated as validated adherence outcome
Daily reinforcement	WhatsApp messages, infographics, videos, and written guidance addressing interconnected Tibb lifestyle domains.	Daily for 4 weeks	Adherence encouraged but not objectively quantified

Note: Sleep–wake regulation was the principal educational emphasis, but the program was multicomponent. The contribution of individual components could not be isolated.

Table 4. Baseline-to-follow-up changes in KKT total score and clinic blood pressure among participants with complete paired observations (n = 29)

Outcome	Baseline	Follow-up	Paired change	95% CI	Statistical test	p-value	Effect size
KKT total score	16 (15–17)	21 (21–22)	+5 (3–6)	Not estimated	Wilcoxon signed-rank, W = 4.0	<0.001	Rank-biserial correlation = 0.98
SBP, mmHg	142.83 ± 15.05	130.00 ± 7.91	-12.83 ± 9.19	-16.32 to -9.33	Paired t-test, t(28) = -7.51	<0.001	Cohen's d _z = -1.40
DBP, mmHg	96.14 ± 10.86	86.07 ± 6.45	-10.07 ± 8.06	-13.13 to -7.00	Paired t-test, t(28) = -6.73	<0.001	Cohen's d _z = -1.25

Note: KKT values are presented as median (IQR), whereas SBP and DBP values are presented as mean ± SD. Paired change was calculated as follow-up minus baseline; negative SBP and DBP values therefore indicate lower blood pressure at follow-up. A 95% confidence interval was not estimated for the median KKT change. Because paired SBP differences departed from normality, a Wilcoxon signed-rank sensitivity analysis was performed and produced a result consistent with the paired t-test (W = 15.5; p < 0.001; rank-biserial correlation = -0.93). Effect sizes quantify within-participant changes and should not be interpreted as controlled causal effects. CI, confidence interval; DBP, diastolic blood pressure; IQR, interquartile range; KKT, Kuesioner Kualitas Tidur; SBP, systolic blood pressure; SD, standard deviation.

be inferred because medication-related changes, short-term blood-pressure variability, regression to the mean, and other concurrent influences were not controlled.

Interpretation of the Tibb framework and multicomponent program. The Tibb framework functioned as a culturally contextualized structure for organizing and communicating lifestyle guidance. Physiological explanations involving sympathetic activity, circadian regulation, neuroendocrine function, and nocturnal blood-pressure patterns derive primarily from contemporary sleep and cardiovascular science [6,7,17], rather than from an independently established Tibb-specific biological mechanism. The present findings therefore provide preliminary information about the use of a culturally framed educational model in one university workplace, not evidence of a distinct physiological effect attributable to Tibb.

The program was multicomponent rather than an isolated sleep intervention. Sleep-wake guidance was delivered alongside breathing practice, movement and rest, food and drink, emotional regulation, and other lifestyle domains [13–16]. These components were provided as an integrated educational package, and component-specific exposure, adherence, and behavioral implementation were not objectively quantified. Consequently, neither the sleep-wake

component nor any other individual component can be identified as the active element responsible for the observed pattern.

Confounding and causal interpretation. The blood-pressure findings require particularly cautious interpretation. Baseline medication profiles were available, but medication use was not systematically reassessed at follow-up. Adherence, dose adjustment, treatment initiation or discontinuation, and regimen stability were not documented prospectively. Improved adherence, resumption of previously irregular therapy, or changes in antihypertensive treatment may therefore have contributed to the lower follow-up measurements. In addition, blood pressure was represented by a single clinic reading at each assessment. Although participants rested for five minutes, remained silent, and were positioned consistently according to the documented procedure, a single reading is more susceptible to short-term biological and measurement variability than an average of repeated readings.

Regression to the mean and natural within-person variability are additional plausible explanations. Several participants had elevated baseline values, only one baseline reading was obtained, and no concurrent comparison group was included. The confidence intervals and standardized effect sizes quantify the

precision and magnitude of within-participant change, but they do not estimate a controlled causal effect. Cohen's d_z expresses the mean paired change relative to the variability of participant-level differences [21], whereas rank-biserial correlation summarizes the balance of positive and negative signed ranks. The large standardized changes should therefore be interpreted as descriptive properties of this sample and follow-up period rather than as evidence of program effectiveness.

Potential relevance to pharmacy practice. The relevance to pharmacy practice is prospective rather than demonstrative. Pharmacist-delivered care was not evaluated, and the observed outcomes cannot be attributed to pharmacist involvement. However, the educational structure may inform future pharmacist-led or interprofessional studies that combine sleep-related screening with medication review, adherence assessment, brief lifestyle counseling, digital reinforcement, blood-pressure reassessment, and referral when sleep problems or uncontrolled blood pressure persist [22]. Such models require direct evaluation using controlled designs and prospectively defined implementation outcomes.

Strengths and limitations. Strengths include analysis of participant-level paired data, reporting of paired changes with confidence intervals and effect sizes, a nonparametric sensitivity analysis for SBP, and separation of principal continuous outcomes from

secondary descriptive categories. The KKT also underwent preliminary local testing in an independent sample: all seven item-total correlations were statistically significant and ranged from 0.406 to 0.699, while Cronbach's alpha was 0.657. These findings support item association and modest internal consistency for preliminary use, although they do not constitute comprehensive validation. The study setting, intervention content, medication profile, participant flow, assessment schedule, and documented blood-pressure procedure are reported transparently.

This study has several important limitations. The single-group pretest-posttest design does not permit causal attribution because natural variation, repeated-measurement effects, secular change, and regression to the mean cannot be separated from program-related change. Medication adherence and regimen changes were not monitored prospectively. Blood pressure was based on one clinic reading at each assessment; although five-minute seated rest, silence, back support, and arm support at heart level were documented, cuff-size selection, measurement arm, time of day, and premeasurement restrictions were not recorded. Three of 32 enrolled participants could not be contacted at follow-up, so complete-case analysis may be affected by attrition bias. The KKT demonstrated acceptable item-total associations and modest internal consistency in a preliminary local sample, but comprehensive evidence

Descriptive baseline and follow-up outcomes

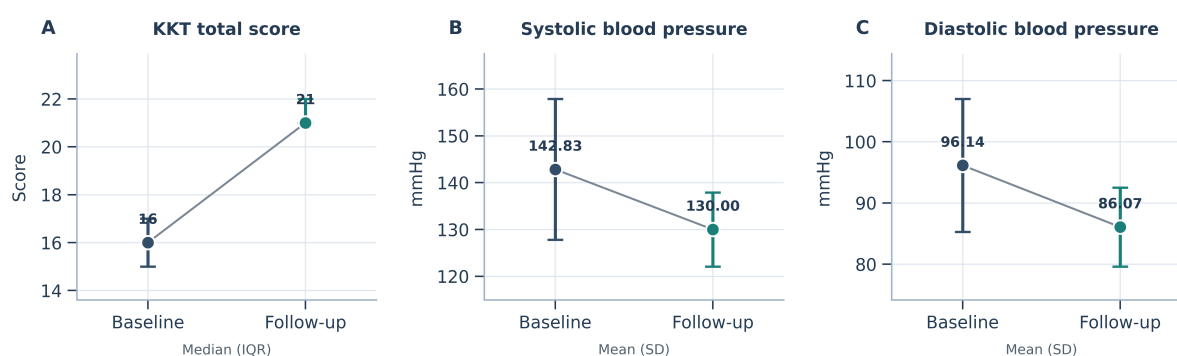


Figure 2. Group-level baseline and follow-up summaries corresponding to the principal outcomes in Table 4. Panel A shows the median KKT total score with IQR; Panels B and C show mean SBP and DBP with SD, respectively. Connecting lines indicate the direction of change in group summaries and do not represent individual participant trajectories. Comparisons should be made within each panel because the outcomes use different scales and summary statistics. Paired changes, 95% confidence intervals, statistical tests, and effect sizes are reported in Table 4.

Table 5. Secondary descriptive changes in sleep-quality and blood-pressure categories

Secondary outcome	Baseline, n (%)	Follow-up, n (%)
Sleep-quality category		
Good	2 (6.9)	22 (75.9)
Less good	23 (79.3)	7 (24.1)
Poor	4 (13.8)	0 (0.0)
Blood-pressure control status		
Controlled	10 (34.5)	18 (62.1)
Uncontrolled	19 (65.5)	11 (37.9)
Total participants	29 (100.0)	29 (100.0)

Note: KKT categories were based on source-study score intervals: good 21–28, less good 14–20, and poor 7–13; these are not clinically validated diagnostic thresholds. Blood-pressure control used the age-specific JNC 8 treatment targets applied in the source protocol and represents treatment-control status, not study-based diagnosis.

for structural validity, test-retest reliability, measurement error, responsiveness, criterion validity, and a clinically meaningful change threshold remains unavailable for employees with hypertension [27]. Component-specific exposure and adherence were not objectively quantified. The small single-institution sample and short follow-up restrict precision, generalizability, and assessment of sustained change. Feasibility was not prespecified [31], and mediation and adjusted regression were not retained because the design and sample did not support identifiable or stable estimates [28–30].

Implications for future research. Future studies should use a controlled design, with random allocation where feasible, and obtain repeated baseline and follow-up blood-pressure readings using a fully prespecified protocol. The protocol should document cuff selection, measurement arm, time of day, premeasurement restrictions, medication exposure, and adherence. Validated sleep instruments, objective or structured adherence measures, longer follow-up, and prespecified

implementation outcomes are also needed. Adequately powered samples with clear temporal measurement are required before mediation or multivariable prediction is examined [28–31].

CONCLUSION

In this preliminary single-group pretest-posttest study, self-reported KKT scores were higher and clinic SBP and DBP values were lower at follow-up among employees with hypertension from one Indonesian Islamic university. The KKT showed statistically significant item-total associations and modest internal consistency in preliminary local testing, supporting use of its continuous total score for exploratory within-participant analysis; however, the categories should not be interpreted as validated diagnostic thresholds. The observed sleep-related and blood-pressure changes are encouraging signals but do not establish effectiveness or causality because no comparison group was included and medication-related confounding, single blood-pressure readings, attrition, and the multicomponent nature of the program remain relevant. Future studies should use controlled and adequately powered designs, repeated standardized blood-pressure readings, validated sleep measures or further KKT validation, prospective medication and adherence monitoring, objective assessment of program exposure, and longer follow-up. Pharmacist-led or interprofessional adaptations should be evaluated directly before implementation conclusions are drawn.

CONFLICT OF INTEREST

The authors declare no conflict of interest regarding this study.

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