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Article

Modulation of Hemodynamic Responses in Hypertensive Patients via Sustained Thermal Salt Hydrotherapy: A Six-Day Pre-Experimental Study

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Abstract. Hypertension remains a major global cardiovascular risk factor, with prevalence in Indonesia rising from 25.8% to 34.1%. Non-pharmacological modalities like warm salt-water foot-bath hydrotherapy offer an affordable complementary approach, yet their daily longitudinal effect in rural settings remains insufficiently documented. This study evaluated the effectiveness of thermal salt foot-bath hydrotherapy on systolic and diastolic blood pressure among hypertensive residents in Kubangsari Village. A pre-experimental one-group pretest-posttest design was conducted with twenty purposively selected participants (n = 20), receiving a daily 15–20-minute foot soak in 40-42^{O}C water with 1.5% sodium chloride over six consecutive days. Daily measurements were analyzed using the Wilcoxon Signed-Rank test with Bonferroni correction (α= 0.0083). Mean systolic blood pressure decreased significantly from 166.10 ± 15.99 mmHg to 134.00 ± 8.05 mm Hg by day five (Δ = -32.10 mmHg, 19.33%: p < 0.001, Cohen's d_z = 2.16). Diastolic blood pressure showed a smaller reduction (88.00 ± 6.16 to 82.20 ± 7.59 mmHg, 6.59%) that lost statistical significance after alpha adjustment. Hypertensive crisis was fully eliminated by day one, and 35% achieved normotension by day five. Standardized thermal salt hydrotherapy induces a clinically meaningful, predominantly systolic reduction, serving as an effective complementary intervention for community hypertension management.

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1. Introduction

Hypertension, a chronic elevation of arterial blood pressure, remains a leading global health burden and is often called the silent killer because it progresses without overt symptoms until severe cardiovascular complications occur [1-2]. The World Health Organization estimates that more than 1.28 billion adults live with hypertension worldwide, and in Indonesia the reported prevalence has risen from 25.8% to 34.1% over the past decade [3]. Although pharmacological treatment is widely available, long-term adherence is frequently limited by side effects and cost, which increases the need for safe, affordable, non-pharmacological options suited to community-based care [4-7].

Warm salt-water foot-bath hydrotherapy is proposed to lower blood pressure through two complementary mechanisms: thermal vasodilation and osmotic fluid redistribution [8-9]. Exposure of plantar skin to water at 40–42°C stimulates cutaneous thermoreceptors and activates endothelial nitric oxide synthase (eNOS), increasing nitric oxide release, promoting peripheral vasodilation, and reducing sympathetic tone [10-13]. Adding sodium chloride raises the osmolality of the soaking medium and stimulates plantar mechanoreceptors, an effect associated with reduced catecholamine release and greater parasympathetic activity [14]. Concurrently, local hydrostatic pressure combined with the osmotic gradient may promote reabsorption of interstitial fluid from the lower extremities, easing venous return and systemic circulatory load [15-16]. Earlier trials have confirmed that warm-water stimulation alone can relax peripheral vessels and lower blood pressure.

Most existing evaluations of saline foot-soak hydrotherapy report only a single pre- and post-intervention comparison, leaving how blood pressure evolves over several consecutive days of home-based treatment insufficiently documented [17-19]. Related studies have investigated foot immersion without salt addition, evaluated distinct population groups, or utilized single-session protocols, leaving the trajectory of a standardized 1.5% saline solution applied daily over six days largely unexamined [20]. Furthermore, a re-examination of methodological details in cited studies underscores these specific operational differences: earlier trials utilized unstandardized household salt mixtures without fixed osmotic concentrations (0% or variable NaCl), focused primarily on urban elderly cohorts, used lower water temperatures (38-40^{0}C}, or confounded the isolated thermo-osmotic mechanism by adding herbal ingredients [14],[20]. This study explicitly addresses these gaps by tracking daily systolic and diastolic blood pressure across six consecutive intervention days in a home-based rural community setting. By maintaining a strictly standardized 1.5% sodium chloride concentration, a fixed immersion temperature (40-42^{O}C), and a uniform duration, this research clarifies both the longitudinal trajectory and short-term endurance of the hemodynamic response within a one-week treatment regimen.

This study aimed to analyze the daily pattern of systolic and diastolic blood pressure change during six consecutive days of thermal salt foot-bath hydrotherapy among hypertensive residents of Kubangsari Village. The primary objective was to determine whether the intervention produced a statistically and clinically significant reduction in systolic blood pressure. The secondary objective was to determine its effect on diastolic blood pressure and on the proportion of participants reaching normotension or crisis-level hypertension. It was hypothesized (H1) that systolic blood pressure would decrease significantly across the intervention period, whereas diastolic blood pressure (H2) was expected to decline to a lesser, and possibly non-significant, degree once corrected for repeated comparisons, given its stronger dependence on basal arteriolar tone rather than transient vasodilation. Findings from this study are intended to support independent, low-cost complementary interventions for community-based hypertension management.

2. Experimental Section

2.1. Study Design and Participants

This quantitative study used a pre-experimental one-group pretest-posttest design without a control group to evaluate the effect of thermal salt hydrotherapy on blood pressure. The study population comprised all registered hypertensive residents of Kubangsari Village. A non-probability purposive sampling technique was applied using pre-defined inclusion and exclusion criteria. Eligible participants were adults aged 35–65 years with primary hypertension (systolic 140–159 mmHg and/or diastolic 90–99 mmHg), a hypertension history of at least two years, and willingness to provide written informed consent. Participants with end-stage chronic kidney disease, heart failure, prior acute stroke, or skin lesions, burns, or ulcers on the lower extremities were excluded. Sample size was calculated using G*Power software, yielding 20 respondents who completed all study procedures [21].

2.2. Ethical Approval

The research was conducted in Kubangsari Village after obtaining formal ethical approval from the Institutional Health Research Ethics Committee of Universitas Nasional No. 108/e-KEPK/VI/2026. All participants received a full explanation of study procedures and signed a written informed consent form prior to enrollment, in accordance with the Declaration of Helsinki

2.3. Instruments and Intervention Procedure

Blood pressure was measured with a calibrated Omron HEM-7120 digital sphygmomanometer, periodically verified by the Health Facility Supervisory Agency (BPFK) to ensure measurement validity. Measurements followed the standard American Heart Association protocol, with participants seated quietly for five minutes beforehand and the arm supported at heart level. The intervention used a 5-liter plastic basin, a digital thermometer to maintain water temperature at 40–42°C, and standardized table salt (sodium chloride).

To limit confounding, adherence to antihypertensive medication and habitual dietary sodium intake was monitored daily throughout the six-day intervention using a structured daily self-report logbook combined with brief face-to-face observational interviews conducted by the researcher during each home visit; any participant who altered medication dosage or made a substantial dietary change during the study period was to be excluded from the final analysis. The procedure was conducted at each participant's residence over six consecutive days. Pre-test blood pressure was recorded on day one before the first intervention.

Each session consisted of soaking both feet up to mid-calf in 3 liters of water at 40–42°C mixed with 45 g of table salt (≈1.5% concentration) for 15–20 minutes, once daily between 16:00 and 17:00 local time to minimize circadian variation. Post-test blood pressure was measured ten minutes after each session, from day one through day six

2.4. Data Analysis

Data were processed using SPSS version 26.0. Univariate analysis was used to describe the frequency and percentage distribution of blood pressure categories before and after the intervention. Normality of the pre- and post-intervention blood pressure distributions was assessed with the Shapiro-Wilk test ($n < 50$), which indicated a non-normal distribution ($p < 0.05$). The non-parametric Wilcoxon Signed-Rank Test was therefore selected, as it does not require the normality or sphericity assumptions underlying repeated-measures ANOVA, to compare the pretest with each daily posttest measurement ($\alpha = 0.05$). A Bonferroni-adjusted threshold ($\alpha = 0.05 \div 6 \approx 0.0083$) was applied to control the family-wise Type I error rate across the six repeated comparisons. The main methodological limitations of this design are the absence of a separate control group and limited control of confounding factors such as daily stress and dietary sodium intake occurring outside intervention hours.

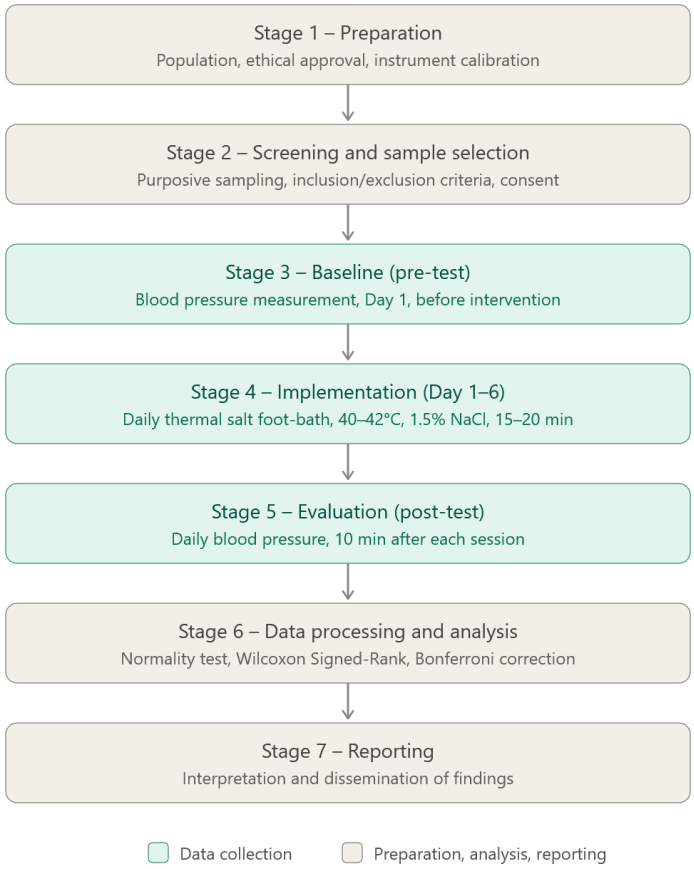


Figure 1. Research Flowchart

3. Results and Discussion

3.1. Respondent Characteristics

Table 1 presents the baseline demographic and clinical characteristics of all respondents involved in this study (n = 20). Data including age, gender, duration of hypertension, baseline hypertension severity, and employment status are shown in Table 1. The study included 20 respondents with a mean age of 52.40 ± 6.85 years. Women represented the majority of the sample (70.0%; n = 14), while men accounted for 30.0% (n = 6). The mean duration of hypertension was 3.65 ± 1.20 years. Regarding baseline severity, 40.0% of participants had Stage 1 hypertension, 35.0% had Stage 2 hypertension, and 25.0% met the study criterion for hypertensive crisis. More than half of the respondents were housewives (55.0%), followed by private-sector employees (30.0%) and self-employed participants (15.0%).

The demographic profile provides important context for interpreting the subsequent blood pressure response. The mean age of 52.40 years indicates a population in which age-related vascular changes may already contribute to blood pressure regulation. Vascular function, particularly endothelial-mediated regulation of vascular tone, is an important determinant of cardiovascular homeostasis and may influence the response to thermal stimulation [22]. Previous research on foot-bath interventions has also demonstrated changes in peripheral circulation following exposure to warm water [23]. Nevertheless, because the present study did not directly assess vascular function, age should be regarded as a potentially relevant characteristic rather than a demonstrated modifier of treatment response.

Table 1. Demographic and Clinical Characteristics of Hypertensive Respondents before Intervention (n = 20)

Characteristic	n / Mean ± SD	%
Age (years), mean ± SD	52.40 ± 6.85	-
Sex – Female, n (%)	14	70.0
Sex – Male, n (%)	6	30.0
Duration of hypertension (years), mean ± SD	3.65 ± 1.20	-
Baseline stage – Stage 1 hypertension, n (%)	8	40.0
Baseline stage – Stage 2 hypertension, n (%)	7	35.0
Baseline stage – Hypertensive crisis (≥180 mmHg), n (%)	5	25.0
Occupation – Housewife, n (%)	11	55.0
Occupation – Private employee, n (%)	6	30.0
Occupation – Self-employed, n (%)	3	15.0

The predominance of female participants should likewise be interpreted descriptively. Sex-related biological differences may contribute to variations in cardiovascular regulation; however, the present study did not assess menopausal status, hormonal levels, or other sex-specific physiological variables. Therefore, it would not be appropriate to attribute differences in treatment response to hormonal mechanisms. Instead, sex represents an important baseline characteristic that may contribute to between-participant variability and should be considered in future studies [24].

The mean duration of hypertension was 3.65 ± 1.20 years, indicating that participants had experienced elevated blood pressure for several years. Chronic hypertension may be accompanied by vascular alterations that influence cardiovascular responsiveness. In addition, long-term blood pressure control is affected by medication adherence, health behavior, lifestyle, and self-management practices [5]. However, because the present analysis does not directly examine the association between hypertension duration and the magnitude of blood pressure reduction, no causal interpretation can be made.

From a community-health perspective, the occupational distribution is also relevant. Housewives constituted more than half of the sample, while the remainder consisted of private employees and self-employed individuals. Occupational circumstances may be associated with physical activity, workload, stress, sleep, and opportunities for self-care. Nevertheless, these variables were not directly measured in the current study. Therefore, occupation should be interpreted as contextual information rather than as an established determinant of the intervention response [5].

Taken together, Table 1 indicates that the participants had heterogeneous demographic and clinical characteristics. This heterogeneity is important when interpreting the subsequent changes in blood pressure because individuals receiving the same intervention may not respond identically. Age, sex, hypertension duration, and baseline hypertension severity may represent potential sources of response variability, but their role as effect modifiers requires individual-level statistical analysis before any definitive conclusion can be drawn.

3.2. Baseline Blood Pressure

Baseline blood pressure provides an essential clinical context for interpreting the subsequent response to warm-water foot-bath therapy. In hypertension, the magnitude and pattern of baseline blood pressure are relevant because systolic and diastolic pressures reflect different aspects of cardiovascular hemodynamics and may respond differently to changes in vascular tone. Therefore, Table 2 presents the baseline systolic and diastolic blood pressure distributions before the intervention, providing a reference point against which the magnitude and direction of subsequent changes can be evaluated [22-23].

Table 2. Systolic and Diastolic Blood Pressure before Intervention (n = 20)

Variable	Mean ± SD	Median	Min–Max
Systolic (mmHg)	166.10 ± 15.99	168.50	140–187
Diastolic (mmHg)	88.00 ± 6.16	90.00	80–100

As shown in Table 2, the mean baseline systolic blood pressure (SBP) was 166.10 ± 15.99 mmHg, with a median of 168.50 mmHg and a range of 140–187 mmHg, whereas the mean diastolic blood pressure (DBP) was 88.00 ± 6.16 mmHg, with a median of 90.00 mmHg and a range of 80–100 mmHg. The higher SBP relative to DBP indicates that the baseline hypertensive profile of the participants was predominantly characterized by elevated systolic pressure. The relatively broad SBP range also demonstrates considerable inter-individual variation, suggesting that participants did not enter the intervention with an equivalent hemodynamic status. This variation is important when interpreting subsequent within-subject changes because the absolute magnitude of blood pressure reduction may partly depend on the participant's initial blood pressure level [3-5].

The difference between the systolic and diastolic profiles is also clinically relevant. Although the mean DBP remained elevated, its narrower distribution (80–100 mmHg) compared with SBP suggests less baseline variability in diastolic pressure. From a physiological perspective, systolic pressure is strongly influenced by cardiac output, arterial stiffness, and systemic vascular resistance, whereas diastolic pressure is more closely related to peripheral arteriolar resistance and the duration of diastolic filling [23]. This distinction provides a plausible physiological context for the subsequent finding in Table 3, in which the reduction in SBP was considerably greater and more consistent than the change in DBP. However, because these physiological mechanisms were not directly measured in the present study, they should be regarded as explanatory interpretations rather than mechanisms demonstrated by the study [22-23].

The baseline findings are broadly consistent with previous reports describing warm-water foot bathing as a complementary intervention that may influence blood pressure through thermal stimulation and peripheral vasodilation. Previous studies have reported reductions in blood pressure following warm-water foot-bath interventions, including protocols using salt or other complementary components [25-27]. A narrative review similarly reported that warm-water foot bathing may promote vasodilation and reduce blood pressure, while emphasizing the importance of controlling water temperature and exposure duration to avoid excessive hemodynamic changes [12]. Evidence from a broader systematic review of heat therapy also indicates that thermal interventions can reduce systolic and diastolic blood pressure, although the magnitude of response varies according to the type and dose of heat exposure [22].

Importantly, the baseline values in Table 2 should not be interpreted as evidence that the participants were uniformly in Stage 2 hypertension, because the mean value alone does not determine the classification of every individual. The baseline SBP range of 140–187 mmHg demonstrates that participants included different levels of systolic hypertension, while the DBP range of 80–100 mmHg indicates corresponding variation in diastolic pressure. This heterogeneity strengthens the importance

of analyzing paired changes within each participant rather than relying solely on group means. Accordingly, the subsequent analysis in Tables 3–5 is necessary to determine whether the observed reduction in blood pressure was consistent across repeated measurements and whether the magnitude of change differed between SBP and DBP [28].

3.3. Daily Changes in Blood Pressure

Repeated measurement of blood pressure across the six intervention days provides a more informative picture of the temporal response to warm-water foot-bath therapy than a single pre–post comparison. This approach is particularly relevant because blood pressure is a dynamic physiological parameter that can vary according to vascular tone, autonomic activity, physical activity, psychological stress, medication adherence, and other daily conditions [5],[21]. Therefore, the pattern presented in Table 3 was examined not only in terms of the absolute reduction from baseline but also according to the consistency, magnitude, and direction of change across successive intervention days.

Table 3. Daily Systolic and Diastolic Blood Pressure, Pre-Test to Post-Test 6 (n = 20)

Measurement	n	Systolic Mean ± SD	Median	Min–Max	Diastolic Mean ± SD	Median	Min-Max
Pre-Test	20	166.10 ± 15.99	168.50	140–187	88.00 ± 6.16	90.00	80–100
Post-Test 1	20	142.45 ± 16.33	140.00	120–170	82.20 ± 7.59	80.00	70–98
Post-Test 2	20	138.40 ± 13.69	135.00	120–165	83.00 ± 6.96	85.00	70–90
Post-Test 3	20	136.50 ± 8.75	140.00	120–150	85.80 ± 5.85	89.00	70–90
Post-Test 4	20	140.05 ± 13.04	140.00	120–165	83.60 ± 5.92	82.50	70–90
Post-Test 5	20	134.00 ± 7.42	130.00	120–150	84.60 ± 5.60	85.00	70–90
Post-Test 6	20	143.65 ± 16.97	140.00	120–170	84.70 ± 7.71	89.50	70–90

As shown in Table 3, mean systolic blood pressure decreased from 166.10 ± 15.99 mmHg at baseline to 142.45 ± 16.33 mmHg at Post-Test 1 and continued to decline to 138.40 ± 13.69 mmHg at Post-Test 2 and 136.50 ± 8.75 mmHg at Post-Test 3. Although a modest increase was observed at Post-Test 4 (140.05 ± 13.04 mmHg), the mean SBP subsequently reached its lowest value at Post-Test 5 (134.00 ± 7.42 mmHg), representing an absolute reduction of 32.10 mmHg or approximately 19.33% from baseline. At Post-Test 6, SBP increased to 143.65 ± 16.97 mmHg but remained 22.45 mmHg below baseline. Thus, the overall pattern was characterized by a substantial early decline, progressive reduction through the middle intervention period, and subsequent fluctuation rather than a strictly linear downward trajectory.

In contrast, the diastolic response was considerably smaller and more stable. Mean DBP decreased from 88.00 ± 6.16 mmHg at baseline to 82.20 ± 7.59 mmHg at Post-Test 1, followed by values ranging from 83.00 to 85.80 mmHg during the subsequent measurements. The difference between the magnitude of SBP and DBP responses is clinically and physiologically relevant. SBP is influenced substantially by cardiac output, arterial compliance, and systemic vascular resistance, whereas DBP is more closely related to arteriolar resistance and the maintenance of vascular tone during diastole [22-23]. The greater change in SBP observed in this study may therefore be compatible with a transient reduction in peripheral vascular resistance following thermal exposure. However, because vascular resistance, cardiac output, endothelial function, and autonomic activity were not directly measured, this explanation should be regarded as a plausible physiological interpretation rather than a demonstrated mechanism.

The temporal pattern observed in Table 3 is also consistent with the potential hemodynamic effects of warm-water immersion. Thermal stimulation can increase peripheral blood flow and promote vasodilation, thereby potentially reducing vascular resistance and blood pressure. Previous reports on warm-water foot-bath therapy have documented reductions in blood pressure among individuals with hypertension, including interventions using warm water alone or in combination with

salt and other complementary components [26-27]. A narrative review similarly concluded that warm-water foot bathing may be considered a complementary intervention for blood-pressure management, while emphasizing that temperature and exposure duration require appropriate control because excessive thermal exposure may produce undesirable hemodynamic changes [12]. More broadly, recent systematic evidence on passive heat therapy suggests that repeated thermal exposure can produce modest reductions in resting blood pressure, although the magnitude and consistency of the response vary across intervention protocols and populations.

An important finding in Table 3 is that the reduction was not progressively maintained across all six measurements. The increase in SBP at Post-Test 4 and particularly at Post-Test 6 indicates that the response to repeated intervention was not strictly cumulative. Several explanations are possible, including normal day-to-day blood-pressure variability, differences in pre-measurement physiological conditions, circadian influences, or changes in autonomic and vascular regulation. Nevertheless, these explanations cannot be confirmed because variables such as daily stress, sodium intake, physical exertion, sleep, and medication timing were not quantitatively assessed in the present study [3-5]. Therefore, the observed fluctuations should not be interpreted as evidence of physiological adaptation or baroreceptor resetting without direct measurement.

The reduction in the variability of SBP toward the middle of the intervention period is also noteworthy. The standard deviation decreased from 15.99 mmHg at baseline to 8.75 mmHg at Post-Test 3 and 7.42 mmHg at Post-Test 5, suggesting that individual SBP values became more concentrated around the group mean during these measurements. However, this pattern should be interpreted cautiously because a reduction in between-participant variability does not necessarily demonstrate physiological stabilization or treatment responsiveness. It may partly reflect regression toward the mean or differences in individual responses. The subsequent increase in SD to 16.97 mmHg at Post-Test 6 further supports the interpretation that the response remained heterogeneous across participants.

Taken together, Table 3 demonstrates a pronounced short-term reduction in systolic pressure accompanied by a smaller and less variable reduction in diastolic pressure. The lowest mean SBP occurred on Post-Test 5, whereas the subsequent increase on Post-Test 6 indicates that the observed response should be understood as a fluctuating short-term hemodynamic pattern rather than a continuously progressive treatment effect. This interpretation is supported by the subsequent paired analyses in Tables 4 and 5, which are necessary to determine whether the apparent reductions were statistically consistent within participants and to quantify their magnitude using confidence intervals and effect sizes.

Figure 2 provides a visual representation of the temporal pattern presented in Table 3 and strengthens the interpretation of the repeated blood pressure measurements across the six intervention days. Rather than showing a uniform linear decline, the figure demonstrates a marked reduction in systolic blood pressure followed by modest fluctuations, whereas diastolic blood pressure remains comparatively stable. This distinction is important because repeated blood pressure measurements may capture both the immediate hemodynamic response to thermal exposure and normal day-to-day physiological variability [5],[21], [23].

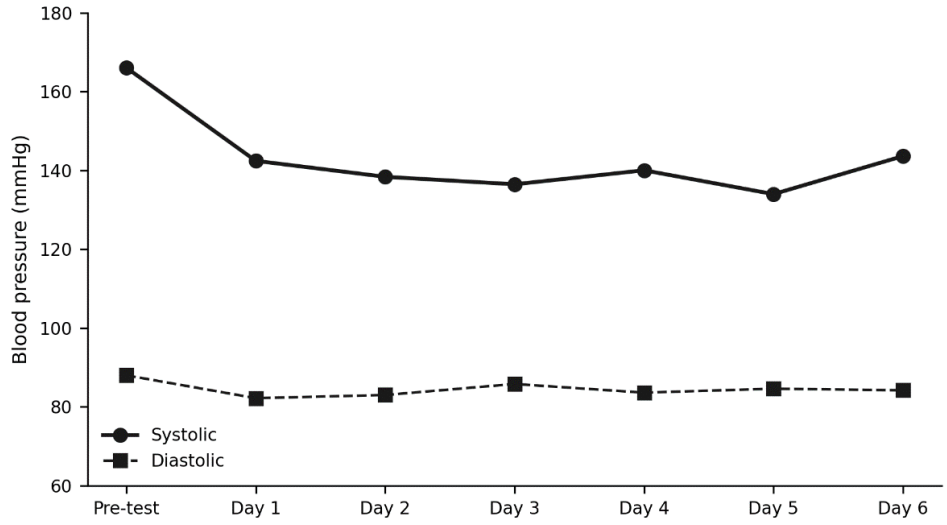


Figure 2. Temporal Pattern of Blood Pressure Changes

Figure 2 visually illustrates the temporal trajectory of blood pressure across the six intervention days. The most prominent feature is the separation between the systolic and diastolic trajectories. Systolic pressure shows a marked downward displacement from baseline, followed by a gradual flattening and intermittent fluctuations, whereas diastolic pressure demonstrates a smaller initial decline and subsequently remains relatively stable. Thus, the figure provides a visual representation of the differential response identified numerically in Table 3.

The shape of the systolic curve is particularly important because it does not represent a continuous linear decline. The progressive reduction during the early intervention period, the slight increase on Day 4, the lowest mean value on Day 5, and the subsequent increase on Day 6 suggest a dynamic short-term response rather than a uniform cumulative reduction. This pattern may reflect the interaction between the transient vascular effects of warm-water exposure and normal day-to-day blood-pressure variability [5], [21], [23]. However, because relevant daily determinants were not directly measured, the observed fluctuations should be regarded as descriptive temporal variation rather than evidence of physiological adaptation or loss of treatment effect.

The visual pattern therefore complements rather than duplicates the numerical analysis in Table 3. While Table 3 quantifies the magnitude and variability of blood-pressure changes, Figure 2 highlights their temporal configuration and the contrasting trajectories of systolic and diastolic pressure. This distinction is relevant to the subsequent statistical analysis because the apparent systolic reduction across repeated measurements requires confirmation through paired inferential testing and effect-size estimation, as presented in Tables 4 and 5.

3.4. Statistical Analysis of Systolic Blood Pressure Changes

Table 4 demonstrates a consistent reduction in systolic blood pressure across all six post-test measurements compared with baseline. The mean paired differences ranged from −22.95 to −31.60 mmHg, and all Wilcoxon signed-rank tests yielded $p < 0.001$. Importantly, all six comparisons remained statistically significant after Bonferroni adjustment ($\alpha = 0.0083$), indicating that the observed systolic reductions were not dependent on a single post-test measurement. The 95% CIs were entirely below zero at every time point, further supporting the consistency of the direction of change. Thus, when considered together, the p-values and confidence intervals provide convergent evidence of a within-subject reduction in systolic blood pressure throughout the six-day observation period.

Table 4. Paired Changes, 95% Confidence Intervals, Cohen's dz, and Wilcoxon Tests for Systolic Blood Pressure by Intervention Day (n = 20)

Day	Median Diff (mmHg)	Mean Diff ± SD (mmHg)	95% CI	Cohen's dz	Wilcoxon p	Sig. at α=0.0083
Post-Test 1	-22.50	-23.65 ± 14.62	-30.49, -16.81	1.62	< 0.001	Yes
Post-Test 2	-27.00	-27.70 ± 11.60	-33.13, -22.27	2.39	< 0.001	Yes
Post-Test 3	-30.00	-29.60 ± 17.16	-37.63, -21.57	1.72	< 0.001	Yes
Post-Test 4	-29.00	-26.05 ± 14.57	-32.87, -19.23	1.79	< 0.001	Yes
Post-Test 5	-34.00	-31.60 ± 14.63	-38.45, -24.75	2.16	< 0.001	Yes
Post-Test 6	-20.00	-22.95 ± 13.96	-29.48, -16.42	1.64	< 0.001	Yes

The magnitude of the paired differences also provides an important cross-analysis across the six intervention days. The smallest mean reduction was observed at Post-Test 6 (−22.95 ± 13.96 mmHg; 95% CI −29.48 to −16.42), whereas the largest absolute mean reduction occurred at Post-Test 5 (−31.60 ± 14.63 mmHg; 95% CI −38.45 to −24.75). This corresponds closely with the trajectory shown in Table 3 and Figure 2, where the lowest mean systolic blood pressure was observed at Post-Test 5. However, the largest standardized effect size did not occur at Post-Test 5. Cohen's *dz* reached its maximum at Post-Test 2 (*dz* = 2.39), compared with *dz* = 2.16 at Post-Test 5. This distinction is important because Cohen's *dz* incorporates the variability of the paired differences; therefore, a larger standardized effect does not necessarily coincide with the largest absolute mean reduction.

The pattern of Cohen's *dz* further indicates that the observed systolic changes were substantial relative to the within-participant variability. Effect sizes ranged from 1.62 at Post-Test 1 to 2.39 at Post-Test 2 and remained above 1.60 at all subsequent assessments. In conventional interpretation, these values represent large standardized within-subject effects. Nevertheless, the magnitude of *dz* should be interpreted in the context of the study design: because there was no parallel control group, a large within-subject effect cannot by itself establish that the warm-water foot-bath intervention was the sole cause of the reduction. Regression to the mean, repeated measurement effects, medication use, and other time-varying factors may also contribute to the observed change [21].

The relationship between the statistical findings and the temporal pattern is particularly noteworthy. Post-Test 1 already demonstrated a substantial reduction of −23.65 mmHg, suggesting that a measurable systolic response was present during the initial intervention period. The reduction became greater at Post-Test 2 and Post-Test 3, with mean differences of −27.70 and −29.60 mmHg, respectively. Although the mean reduction became slightly smaller at Post-Test 4 (−26.05 mmHg), it increased to its largest absolute value at Post-Test 5 (−31.60 mmHg). At Post-Test 6, the reduction decreased to −22.95 mmHg but remained statistically significant and clinically substantial in absolute terms. This pattern corresponds with the non-linear trajectory observed in Figure 2 and suggests that the response was maintained across repeated assessments but fluctuated in magnitude rather than following a strictly progressive decline.

The consistency of the systolic response is broadly comparable with previous reports of warm-water foot-bath interventions among individuals with hypertension. Studies using warm water alone or in combination with salt, lemongrass, or other complementary components have reported reductions in blood pressure following the intervention [29-32]. Previous work involving warm-water foot bathing has also suggested that thermal exposure may increase peripheral circulation and contribute to reductions in vascular resistance [23]. However, the present findings should not be interpreted as demonstrating superiority over these approaches because the studies differ in sample characteristics, intervention protocols, duration, and methodological design. The present study instead adds repeated within-subject observations showing that systolic reduction was detectable across six consecutive assessments.

A plausible physiological explanation for the greater systolic response involves thermal stimulation and peripheral vasodilation. Exposure of the feet to warm water may increase peripheral blood flow and alter vascular tone, potentially reducing systemic vascular resistance and thereby contributing to lower systolic pressure [12], [24], [23]. The endothelial system has an important role in regulating vascular tone, and changes in peripheral circulation may influence blood-pressure dynamics [24] .Nevertheless, endothelial function, peripheral vascular resistance, cardiac output, and autonomic activity were not directly measured in this study. Consequently, these mechanisms should be regarded as biologically plausible explanations supported by previous literature rather than mechanisms directly demonstrated by the present data.

Another important aspect is the relationship between the large systolic effect observed in Table 4 and the baseline characteristics reported in Table 1. Participants entered the study with heterogeneous hypertension severity, including Stage 1, Stage 2, and participants meeting the study's operational definition of hypertensive crisis. The relatively high baseline SBP of 166.10 ± 15.99 mmHg also provided considerable room for absolute reduction. However, because individual-level analyses linking baseline severity, age, sex, duration of hypertension, or occupation to the magnitude of ΔSBP were not performed, the present data cannot establish whether particular subgroups experienced greater benefit. The large overall *dz* values should therefore be interpreted as a group-level within-subject finding rather than evidence that the intervention is equally effective across all demographic or clinical subgroups [21].

The findings also need to be interpreted in relation to the distinction between statistical and clinical interpretation. The consistently negative mean differences, CIs that excluded zero, and large *dz* values demonstrate a substantial statistical within-subject change in SBP. However, statistical significance and effect size do not establish long-term blood-pressure control or reduction in cardiovascular events. The observation period was limited to six intervention days, and the increase in SBP at Post-Test 6 demonstrates that the response was not continuously progressive. Therefore, the results provide evidence of a short-term blood-pressure response associated with repeated warm-water foot-bath exposure, but they do not establish the persistence of the effect after the intervention period or its implications for long-term cardiovascular outcomes [2], [5], [21].

Table 4 provides stronger statistical evidence for the systolic pattern identified in Table 3 and Figure 2. Across all six post-test assessments, systolic blood pressure decreased significantly from baseline, with mean reductions ranging from 22.95 to 31.60 mmHg, 95% CIs consistently below zero, and large within-subject effect sizes (*dz* = 1.62–2.39). The largest absolute reduction occurred at Post-Test 5, whereas the largest standardized effect occurred at Post-Test 2, demonstrating that the magnitude of absolute change and standardized effect are related but not interchangeable measures. Taken together, these findings indicate a consistent short-term within-subject reduction in systolic blood pressure following repeated warm-water foot-bath exposure. Nevertheless, in the absence of a control group and with potential time-varying confounders not fully controlled, the findings should be interpreted as an association rather than definitive evidence of a causal treatment effect [21], [22], [26].

3.5. Statistical Analysis of Diastolic Blood Pressure Changes

Table 5 demonstrates a generally downward numerical trend in DBP relative to baseline, but the magnitude of change was considerably smaller and less consistent than that observed for SBP. The mean paired differences were −5.80 mmHg at Post-Test 1, −5.00 mmHg at Post-Test 2, −2.20 mmHg at Post-Test 3, −4.40 mmHg at Post-Test 4, −3.40 mmHg at Post-Test 5, and −3.30 mmHg at Post-Test 6. Thus, although the direction of change remained negative at every measurement point, the reduction did not progressively increase across the six intervention days. The largest numerical reduction occurred at Post-Test 1, followed by a smaller response at subsequent assessments. This pattern is consistent with Figure 2, which shows an initial decline in DBP followed by relative stabilization.

The inferential results provide an important qualification to this numerical pattern. At Post-Tests 1, 2, 4, and 5, the unadjusted Wilcoxon p-values were below 0.05 (p = 0.009, 0.012, 0.030, and 0.014, respectively), whereas Post-Tests 3 and 6 were not statistically significant (p = 0.128 and 0.141). However, none of the six comparisons remained statistically significant after the Bonferroni-adjusted threshold of α = 0.0083. Therefore, the data provide evidence of a modest directional reduction in DBP, but not sufficiently robust evidence to conclude that the reduction was statistically consistent across repeated measurements after accounting for multiple comparisons [21].

Table 5. Paired difference, 95% CI, and Cohen's dz for diastolic blood pressure relative to pre-test, by day

Day	Median Diff (mmHg)	Mean Diff ± SD (mmHg)	95% CI	Cohen's dz	Wilcoxon p	Sig. at α=0.0083
Post-Test 1	-1.50	-5.80 ± 9.17	-10.09, -1.51	0.63	0.009	No
Post-Test 2	-5.00	-5.00 ± 7.95	-8.72, -1.28	0.63	0.012	No
Post-Test 3	0.00	-2.20 ± 8.85	-6.34, 1.94	0.25	0.128	No
Post-Test 4	-3.50	-4.40 ± 8.99	-8.61, -0.19	0.49	0.030	No
Post-Test 5	0.00	-3.40 ± 6.35	-6.37, -0.43	0.54	0.014	No
Post-Test 6	0.00	-3.30 ± 11.81	-8.83, 2.23	0.28	0.141	No

The effect-size estimates reinforce this interpretation. Cohen's *dz* ranged from 0.25 to 0.63, with the largest values observed at Post-Tests 1 and 2 (*dz* = 0.63), while the smallest occurred at Post-Test 3 (*dz* = 0.25). Compared with the systolic effect sizes in Table 4 (*dz* = 1.62–2.39), the standardized diastolic effects were substantially smaller. This cross-table comparison is important because it demonstrates that the difference between SBP and DBP was not simply a consequence of statistical power; the magnitude of the standardized within-subject change was also consistently lower for DBP. Thus, the intervention-associated response appears to have been more pronounced for systolic pressure than for diastolic pressure.

The 95% confidence intervals require careful interpretation in relation to the multiple-testing procedure. At Post-Tests 1, 2, 4, and 5, the 95% CIs did not include zero, whereas the intervals for Post-Tests 3 and 6 included zero. However, these are conventional 95% CIs corresponding to an unadjusted α = 0.05 and should not be interpreted as contradicting the Bonferroni-adjusted hypothesis tests. Because six paired comparisons were performed, the more conservative adjusted threshold was used to control the family-wise error rate. Accordingly, the combination of the confidence intervals and adjusted p-values indicates that the evidence for a DBP reduction is weaker and less robust than the evidence observed for SBP [21].

The weaker DBP response may have a plausible physiological basis. Warm-water exposure can increase peripheral circulation and promote vascular relaxation, potentially influencing systemic hemodynamics [12], [24], [33]. However, DBP is influenced by factors including arteriolar vascular tone and the characteristics of diastolic blood flow, and its short-term response to thermal stimulation may therefore differ from that of SBP. Previous studies of warm-water foot-bath therapy have reported reductions in both SBP and DBP, but the magnitude of change has not necessarily been equivalent between the two parameters [30], [33]. The present findings are therefore compatible with previous observations while suggesting that, under the specific protocol and population examined, the more pronounced measurable response occurred in systolic pressure.

This interpretation is further supported by the temporal relationship between Tables 3–5. Table 3 showed that DBP decreased most prominently during the first post-test, from 88.00 to 82.20 mmHg, but subsequently fluctuated within a relatively narrow range of 83.00–85.80 mmHg. Table 5 then demonstrated that the corresponding paired effect sizes were modest, while Table 4 showed much larger and consistently significant systolic effects. Thus, the three sets of findings converge on the same

pattern: the intervention period was associated with a stronger and more consistent change in SBP, whereas the DBP response was smaller, more variable, and statistically less robust.

Several factors may have contributed to the weaker DBP response. Daily physiological variation, medication timing, physical activity, psychological stress, sleep, dietary sodium intake, and measurement conditions may influence diastolic pressure and were not quantitatively controlled in this study [3], [5], [21]. In addition, the study did not directly measure endothelial function, autonomic activity, peripheral vascular resistance, or cardiac hemodynamics. Therefore, the smaller DBP response should not be attributed to a specific physiological mechanism without direct measurement. Rather, the present results indicate that the observable short-term response to the intervention was predominantly systolic.

Table 5 indicates that warm-water foot-bath exposure was associated with a modest numerical reduction in diastolic blood pressure across all six post-test measurements, but the statistical evidence was substantially weaker than that observed for systolic pressure. Although several comparisons reached nominal significance at $p < 0.05$, none remained significant after Bonferroni correction, and Cohen's *dz* values were consistently smaller than those observed for SBP. The convergence of Tables 3–5 and Figure 2 therefore suggests a differential blood-pressure response characterized by a pronounced systolic reduction and a smaller, less statistically robust diastolic change. These findings support the interpretation of warm-water foot-bath therapy as a potentially complementary approach associated with short-term hemodynamic changes, while the absence of a control group and the limited observation period preclude definitive conclusions regarding causality or sustained diastolic blood-pressure control [2], [12], [21].

3.6. Study Limitations

This study has several limitations that should guide the interpretation of its findings. First, the one-group pretest-posttest design without a control group cannot rule out regression to the mean or the influence of routine care as explanations for the observed decline. Second, daily stress levels and dietary sodium intake plausible confounders of blood pressure fluctuation were not directly measured and are acknowledged here as a genuine limitation rather than incorporated as explanatory findings. Third, the six-day observation window does not establish whether the hypotensive effect persists after the intervention is discontinued. Future studies should therefore adopt a randomized controlled design, directly measure psychosocial and dietary confounders, and extend follow-up beyond the

4. Conclusion

Six consecutive days of thermal salt foot-bath hydrotherapy (40–42°C water containing 1.5% sodium chloride, applied for 15–20 minutes daily) produced a significant reduction in systolic blood pressure among hypertensive residents of Kubangsari Village, with a median decrease of 28.50 mmHg and a mean reduction of 32.10 mmHg (19.33%) by day five ($p < 0.001$), alongside a fall in the proportion of participants in hypertensive crisis from 25% to 0%. Diastolic blood pressure also declined, most markedly within the first 24 hours of treatment, but the change did not remain significant after Bonferroni correction, indicating that the intervention acted more consistently on systolic than diastolic pressure.

These findings support thermal salt hydrotherapy as a safe, low-cost, non-pharmacological complementary measure that can be practiced independently at home and integrated into community hypertension-management programs, without replacing pharmacological treatment for severe or crisis-level hypertension. Because the study used a single-group design without a control group and did not directly measure confounders such as daily stress or dietary sodium intake, future research should adopt a controlled design, verify the durability of the hypotensive effect after treatment ends, and examine physiological correlates such as cortisol or catecholamine levels to clarify the underlying mechanism more comprehensively.

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