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Ethanolic *Allium sativum* Extract Attenuates Early Renal Dysfunction in High-Salt Diet-Induced Hypertensive Wistar Rats

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ABSTRACT

Hypertension remains a major public health concern and one of the leading causes of cardiovascular and renal complications worldwide. Prolonged hypertension can progressively impair kidney function. This study investigated the effects of ethanolic extract of *Allium sativum* (garlic) on kidney function markers in high salt diet-induced hypertensive Wistar rats. Hypertension was induced by feeding rats a high salt diet, after which they were treated with varying doses of *Allium sativum* extract, a standard antihypertensive drug, or left untreated. Serum levels of urea, creatinine, and uric acid were analyzed as indicators of renal function. The study aimed to evaluate the renoprotective potential of *Allium sativum* and its influence on biochemical indices of kidney function in hypertension. Findings from this work are expected to contribute to the understanding of the therapeutic benefits of garlic in the management of hypertension-related renal dysfunction.

1. INTRODUCTION

Hypertension refers to a chronic elevation of arterial blood pressure beyond accepted physiological thresholds, placing it among the most prevalent non-communicable diseases globally [1]. Its burden extends across multiple organ systems, with well-established associations between poorly controlled blood pressure and the onset of coronary artery disease, cerebrovascular events, cardiac failure, peripheral vascular disease, cognitive decline, and progressive renal deterioration [2].

Excessive dietary salt intake has been identified as a major environmental contributor to the development of hypertension, particularly in low- and middle-income countries where salt consumption frequently exceeds recommended limits [3,4]. Experimental evidence has shown that prolonged intake of a high-salt diet can induce hypertension and promote renal injury through mechanisms involving oxidative stress, endothelial dysfunction, inflammatory responses, and altered renal hemodynamics [5]. Although the blood pressure response to salt intake varies among individuals, a significant proportion of hypertensive subjects exhibit salt sensitivity, making dietary sodium a critical modifiable risk factor in hypertension management [6].

Hypertension and kidney disease are closely interrelated pathophysiological conditions. Sustained elevation of blood pressure can progressively impair renal structure and function, while declining renal function can further exacerbate hypertension through impaired sodium excretion and fluid retention [7]. The kidney plays a central role in long-term blood pressure regulation by maintaining fluid balance, electrolyte homeostasis, and hormonal control. In the early stages of hypertensive renal involvement, commonly used renal biomarkers such as serum urea and creatinine may remain within normal ranges, thereby delaying the detection of renal dysfunction [8,9]. In contrast, serum uric acid has been identified as an early biochemical marker associated with hypertension-related renal and vascular alterations [10].

Allium sativum (garlic) is a medicinal plant widely used in traditional medicine and has been reported to possess antihypertensive, antioxidant, anti-inflammatory, and endothelial-protective properties. These biological effects are largely attributed to its organosulfur compounds, including allicin and related metabolites [11,12]. Several experimental studies have demonstrated that garlic administration can lower blood pressure and improve renal biochemical parameters in hypertensive animal models [13]. However, evidence regarding its effects on early renal biochemical changes during salt-induced hypertension remains limited.

Therefore, this study was designed to investigate the effects of ethanolic extract of *Allium sativum* on renal function markers in high-salt diet-induced hypertensive Wistar rats, with emphasis on early indicators of renal dysfunction.

2. MATERIALS AND METHODS

Ethical approval

All experimental procedures were conducted in accordance with the Guide for the Care and Use of Laboratory Animals and were reported in line with the ARRIVE guidelines. Ethical approval for this study was obtained from the Animal Ethics Committee, Faculty of Basic Medical Sciences (details omitted for double-blind reviewing).

Location of Study

The study was conducted in the animal house of the Department of Human Physiology, Faculty of Basic

Medical Sciences, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria.

Materials

Twenty-five (25) male Wistar rats, electronic weighing balance, oral cannula, 2 mL hypodermic syringe, plain blood sample tubes, latex medical examination gloves, fresh garlic bulbs (approximately 500 g), UV-VIS 752N Spectrophotometer (Shanghai Yoke Instrument Co., Ltd., China), Thermostat Oven (DHG-9023A, PEC Medical, USA), standard laboratory cages and feeding plates, amlodipine (10 mg tablets), distilled water, standard laboratory rat feed (Top-Pro), sodium chloride (NaCl), 95% ethanol, chloroform, and capillary tubes.

Plant Extraction Procedure

Fresh bulbs of *Allium sativum* (garlic) were purchased from a local market, cleaned, peeled, and sliced into smaller pieces. The sliced garlic was oven-dried and subsequently ground into a fine powder using a laboratory blender. Two hundred and fifty grams of the powdered sample were macerated in 1000 mL of 95% ethanol for 48 hours. The mixture was filtered using a clean cloth and Whatman No. 1 filter paper. The filtrate was concentrated using a rotary evaporator and further dried in a laboratory oven at 45 °C to obtain a gel-like extract, which was stored at 4 °C until use [14].

Experimental design and treatment

The rats were randomly assigned into five groups (n = 5 per group):
Group A: Normal control (standard diet and distilled water)
Group B: Hypertensive control (high-salt diet and distilled water)
Group C: Hypertensive + *Allium sativum* extract (150 mg/kg body weight)
Group D: Hypertensive + *Allium sativum* extract (300 mg/kg body weight)
Group E: Hypertensive + standard drug (amlodipine, 5 mg/kg body weight)
All treatments were administered orally once daily using an oral cannula for a duration of 14 days.

Experimental animals

Twenty-five (25) adult male Wistar rats with body weights ranging from 120 to 140 g were obtained from the Department of Human Physiology animal facility (details omitted for double-blind reviewing) and used throughout the experiment. All animals were maintained in well-ventilated standard cages under a controlled 12-hour light–dark photoperiod at an ambient temperature of 27 ± 2 °C. Unrestricted access to fresh water and standard laboratory chow (Top-Pro) was provided throughout the study. Prior to any experimental intervention, all rats underwent a two-week adaptation period to minimise handling-related stress and allow physiological stabilisation under laboratory housing conditions.

Induction of hypertension

Salt-induced hypertension was established using a well-documented dietary sodium loading protocol adapted from Gu et al. [5]. A high-salt diet was prepared by dissolving 8 g of sodium chloride (NaCl) in a small volume of distilled water and incorporating it homogeneously into 80 g of standard powdered rat chow (Top-Pro), yielding a diet containing approximately 9.1% NaCl by weight. This constituted roughly an eight-fold increase above the sodium content of the basal diet. Fresh high-salt feed was prepared daily to ensure uniform NaCl distribution and palatability throughout the loading phase. Rats in Groups B, C, D, and E received this diet exclusively as their sole feed source for five consecutive weeks, while Group A (normal control) received unmodified standard chow throughout the same period. Body weight was recorded weekly for all animals. At the conclusion of the five-week salt-loading phase, systolic blood pressure (SBP) was assessed in all rats using a non-invasive tail-cuff sphygmomanometer (NIBP system). Each animal was warmed at 37 °C for five minutes to facilitate tail vessel dilation, and a minimum of three consecutive readings were taken per rat; the mean value was recorded. Rats with a confirmed SBP above 140 mmHg were designated hypertensive and advanced to the 14-day treatment phase. Any salt-loaded animal that failed to achieve this threshold was excluded from the treatment phase to maintain model validity.

Sample collection

At the end of the treatment period, the experimental animals were euthanized under mild anesthesia using chloroform. Blood samples were collected via ocular puncture into plain sample tubes, allowed to clot for approximately 5 minutes, and centrifuged at 3000 rpm for 20 minutes. The resulting serum was carefully separated using a micropipette and stored for biochemical analysis [15].

Estimation of renal biomarkers

Serum creatinine levels were determined using the Jaffe alkaline picrate colorimetric method [16]. Serum urea concentration was estimated using the urease–Berthelot method [17]. Serum uric acid levels were determined using the uricase-peroxidase enzymatic colorimetric method as described by Fossati et al. [18]. All assays were performed using commercially available diagnostic kits and read on a UV-VIS 752N Spectrophotometer at the appropriate wavelengths.

Statistical analysis

All results are presented as Mean ± Standard Error of Mean (SEM). Differences among groups in renal biomarker concentrations were evaluated by one-way analysis of variance (ANOVA); where ANOVA indicated a statistically significant effect, pairwise group differences were further explored using the post hoc Least Significant Difference (LSD) test. For intra-group comparison of body weight before and after the experimental period, the paired Student's t-test was applied. A probability value of p ≤ 0.05 was adopted as the threshold for statistical significance throughout. All computations were carried out using IBM SPSS Statistics, version 23.0 (IBM Corporation, Armonk, NY, USA).

RESULTS

Effect of ethanolic *Allium sativum* extract on body weight

The effect of ethanolic extract of *Allium sativum* on body weight of high-salt diet-induced hypertensive Wistar rats is summarised in Table 1. Body weight increased significantly from baseline in every experimental group over the study period (p ≤ 0.05), confirming adequate nutritional status and general wellbeing across all cohorts. The hypertensive control (Group B) gained weight despite sustained salt loading. Animals receiving *Allium sativum* extract at both doses, and those given the reference antihypertensive drug, recorded greater absolute weight gains relative to the hypertensive control group. Tail-cuff SBP measurements taken before treatment commencement exceeded 140 mmHg in all salt-loaded animals, corroborating successful hypertension induction using the high-salt dietary model.

Effect of ethanolic *Allium sativum* extract on renal biomarkers

The effects of ethanolic *Allium sativum* extract on serum urea, creatinine, and uric acid concentrations in high-salt diet-induced hypertensive Wistar rats are detailed in Tables 2, 3, and 4, respectively. Serum urea values were broadly comparable across the normal control, hypertensive control, and all treatment groups, with no statistically significant inter-group differences identified (p > 0.05).

Serum creatinine concentrations were largely uniform across experimental groups. A slight yet statistically significant decline was noted exclusively in Group E (amlodipine-treated) relative to both the normal and hypertensive control groups (p ≤ 0.05), while values in the garlic-treated groups did not differ meaningfully from either control.

Serum uric acid was significantly higher in the hypertensive control group (Group B) relative to the normal control (Group A) (p ≤ 0.05), indicating early metabolic and renal disturbance attributable to salt loading. Administration of low-dose *Allium sativum* extract (Group C) and amlodipine (Group E) each produced a statistically significant attenuation of serum uric acid compared with the hypertensive control (p ≤ 0.05), whereas the reduction observed in the high-dose garlic group (Group D) did not reach significance.

Note: All data are presented as Mean ± SEM (n = 5). Inter-group differences were assessed by one-way ANOVA with post hoc LSD test; significance threshold p ≤ 0.05.

Table 1. Effect of ethanolic *Allium sativum* extract on body weight in high-salt diet-induced hypertensive Wistar rats

Groups	Initial weight (g) Mean ±SEM	Final weight (g) Mean ±SEM	Weight diff. (g)	p-value	t-value
Group A	130.00±3.79	219.00±17.50	89.00	0.008*	-4.97
Group B	132.00±3.22	210.33±2.60	78.33	0.001*	-18.937
Group C	129.00±5.20	225.33±14.50	96.33	0.003*	-6.256
Group D	134.33±2.03	204.33±1.45	70.00	0.001*	-28.062
Group E	138.67±2.03	241.33±4.41	102.67	0.001*	-21.154

Data represent mean ± SEM (n = 5). Within-group comparisons of initial vs. final body weight were analysed using the paired Student's t-test. *: significant increase in final vs. initial body weight (p ≤ 0.05).

Table 1 result reported an increase in body weight across all groups when compared to their initial values. The hypertensive control (Group B) showed a significant increase, but the low-dose (Group C), high-dose (Group D), and standard drug (Group E) groups demonstrated greater increases in final body weight compared to both the normal and hypertensive controls. The highest increase was recorded in the standard drug group (Group E), indicating a significant improvement in body weight gain relative to the hypertensive control.

Table 2. Effect of ethanolic *Allium sativum* extract on serum urea level in high-salt diet-induced hypertensive Wistar rat

Renal Biomarkers	Mean ±SEM	F-ratio	p-value (vs. Group A)	p-value (vs. Group B)
Urea(mg/dl)				
Group A	26.51±0.91	0.229		
Group B	26.64±0.98		0.923	
Group C	25.62±0.58		0.523	0.464
Group D	25.84±0.85		0.630	0.564
Group E	25.84±1.28		0.630	0.564

Data was analysed using ANOVA followed by post Hoc LSD comparison. *: significant when compared to Group A; #: significant when compared to Group B

Table 3. Effect of ethanolic *Allium sativum* extract on serum creatinine level in high-salt diet-induced hypertensive Wistar rats

Renal Biomarkers	Mean ±SEM	F-ratio	p-value (vs. Group A)	p-value (vs. Group B)
Creatinine(mg/dl)				
Group A	0.25±0.00	3.079		
Group B	0.25±0.00		0.420	
Group C	0.25±0.00		0.683	0.235
Group D	0.25±0.00		0.587	0.191
Group E	0.24±0.00*#		0.030	0.007

Note: Data are presented as Mean ± SEM (n = 5); group comparisons performed by one-way ANOVA with post hoc LSD test.
*p ≤ 0.05 vs. Group A (normal control).
#p ≤ 0.05 vs. Group B (hypertensive control).

Table 4. Effect of ethanolic *Allium sativum* extract on serum uric acid level in high-salt diet-induced hypertensive Wistar rats

Renal Biomarkers	Mean ±SEM	F-ratio	p-value (vs. Group A)	p-value (vs. Group B)
Uric acid(mg/dl)				
Group A	6.76±0.00	2.142		
Group B	6.79±0.00*		0.038	
Group C	6.76±0.01#		1.000	0.038
Group D	6.77±0.01		0.529	0.113
Group E	6.76±0.02#		1.000	0.038

Note: Data are presented as Mean ± SEM (n = 5); group comparisons performed by one-way ANOVA with post hoc LSD test.
*p ≤ 0.05 vs. Group A (normal control).

DISCUSSION

This study evaluated the effects of ethanolic extract of *Allium sativum* on renal biomarkers in high-salt diet-induced hypertensive Wistar rats. The findings provide insight into early renal biochemical changes associated with salt-induced hypertension and the potential renoprotective role of garlic during the early phase of disease development.

In the present study, serum urea levels did not differ significantly between the hypertensive control group and the normal control group. This observation suggests that the duration of salt loading employed may not have been sufficient to impair urea excretion markedly. Similar findings have been reported in experimental models where early salt-induced hypertension did not produce significant alterations in serum urea levels, indicating preserved renal clearance during initial disease stages [14].

Serum creatinine levels also remained largely unchanged across the experimental groups, with a slight but significant reduction observed only in the standard drug-treated group. Creatinine is considered a relatively insensitive marker of early renal dysfunction and typically increases only after substantial loss of glomerular filtration capacity [15]. Previous studies have shown that creatinine elevation is more commonly associated with prolonged hypertension and advanced renal damage, which supports the findings of the present study [16].

In contrast, serum uric acid levels were significantly elevated in the hypertensive control group, indicating early renal and metabolic disturbances associated with salt-induced hypertension. Elevated uric acid has been linked to endothelial dysfunction, oxidative stress, and impaired renal microcirculation in hypertensive states [17]. This observation agrees with previous reports identifying uric acid as an early biochemical marker of hypertension-related renal involvement [19].

Treatment with low-dose *Allium sativum* extract resulted in a significant reduction in serum uric acid levels compared with the hypertensive control group. This finding is consistent with earlier studies demonstrating the antioxidant and endothelial-protective properties of garlic, which contribute to improved renal handling of metabolic waste products [20]. Adeleke et al. also reported improvements in renal biochemical parameters following garlic administration in hypertensive rats, although longer treatment duration was employed in their study [21].

The non-significant reduction in uric acid observed in the high-dose garlic-treated group suggests a possible dose-dependent but non-linear response. Similar observations have been reported in previous studies, where moderate doses of garlic exerted more pronounced biological effects than higher doses, potentially due to differences in bioavailability and metabolic processing of active compounds [22].

Overall, the findings indicate that while short-term salt-induced hypertension may not significantly affect conventional renal biomarkers such as urea and creatinine, early biochemical changes reflected by elevated uric acid levels can occur. The ability of *Allium sativum* extract to attenuate uric acid elevation highlights its potential renoprotective role during the early stages of hypertension.

Limitations of the Study

Several limitations of the present study should be acknowledged. First, the sample size of five animals per group is relatively small, which limits statistical power and the generalisability of results. Second, only male Wistar rats were used; sex differences in caffeine metabolism, salt sensitivity, and renal response to garlic supplementation cannot be excluded and warrant investigation in future studies. Third, blood pressure was measured at the end of the salt-loading phase to confirm hypertension induction, but serial blood pressure monitoring throughout the treatment period was not performed; this would have enabled direct correlation of blood pressure changes with renal biochemical outcomes. Fourth, histopathological examination of renal tissue was not conducted, which would have provided structural evidence of garlic's renoprotective effects. Fifth, the 14-day treatment period is relatively short, and the effects of prolonged garlic administration on renal function markers remain to be determined. Finally, the present study measured serum renal biomarkers only; assessment of urinary biomarkers and glomerular filtration rate would provide a more comprehensive evaluation of renal function. Future studies should address these limitations to better define the therapeutic scope of *Allium sativum* in hypertension-related renal disease.

CONCLUSION

This study investigated the effects of ethanolic *Allium sativum* extract on renal biomarkers in high-salt diet-induced hypertensive Wistar rats. The findings demonstrate that short-term exposure to a high-salt diet induces early renal biochemical alterations without causing marked changes in conventional renal markers such as serum urea and creatinine, suggesting the presence of early-stage hypertensive renal involvement rather than established renal damage.

The significant elevation of serum uric acid observed in hypertensive control rats indicates that uric acid may serve as a sensitive early marker of renal and metabolic disturbance associated with salt-induced hypertension. Treatment with low-dose *Allium sativum* extract significantly attenuated this elevation, suggesting a protective effect on early renal biochemical changes. The standard antihypertensive drug produced a comparable effect, further supporting the relevance of uric acid as an indicator of early therapeutic response.

Overall, the results of this study suggest that ethanolic *Allium sativum* extract exhibits partial renoprotective effects during the early stages of salt-induced hypertension, particularly through modulation of serum uric acid levels. These findings support the potential use of *Allium sativum* as an adjunct in the management of early hypertension-related renal dysfunction. However, longer treatment duration and additional renal assessments are recommended to further elucidate its effects on established renal injury.

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