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Effect of Coconut Water on Renal Parameters After Dichlorvos Toxicity in Albino Rats

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ABSTRACT

Background: Dichlorvos (2,2-dichlorovinyl dimethyl phosphate) is an organophosphate widely utilized insecticide in agricultural and domestic settings. Given its increasing misuse in suicides, there is a critical need to explore potential antidotes or protective agents against its toxic effects.
Objective: This study investigated the protective effects of tender coconut water on renal biochemical parameters in albino rats subjected to acute, sub-chronic, and chronic exposure to dichlorvos.
Materials and methods: Sixty-three male albino rats, weighing between 150-200g, were divided into three experimental phases: acute, sub-chronic, and chronic, with each phase consisting of three groups: a negative control (water only), a positive control (dichlorvos only), and a treatment group (dichlorvos and coconut water). The acute phase involved administration of 30 mg/kg dichlorvos with or without 1 ml of coconut water for 24 hours, while the sub-chronic and chronic phases involved lower doses (10 mg/kg) administered for 2 weeks and 1 month, respectively. Blood samples and kidney tissues were collected for biochemical analysis of electrolytes (sodium, potassium, chloride), urea, and creatinine.
Results: Dichlorvos significantly increased plasma creatinine (75.04±5.34 µmol/L) and urea (6.88±0.43 mmol/L) in the acute phase, with no significant changes observed in the treatment group. In the sub-chronic phase, creatinine levels were significantly higher in the positive control group compared to the treatment group.
Conclusion: The findings from this study suggest that coconut water has a protective effect on renal function in albino rats exposed to dichlorvos toxicity. In comparison, dichlorvos induced significant renal impairment, particularly in the acute and chronic phases.

Keywords: *Coconut, Dichlorvos, Nephrotoxicity.*

INTRODUCTION

Coconut water, the liquid endosperm found in young green coconuts, has been recognized in tropical regions as a refreshing beverage and a potent health tonic. Historically termed "noelani" by Hawaiians, meaning "dew from the heavens," coconut water has long been consumed for its nutritional and medicinal benefits [1]. It is particularly valued for its rich composition of electrolytes, vitamins, minerals, antioxidants, amino acids, and enzymes. Among these, potassium—a critical nutrient—is abundant, and its electrolyte-rich nature makes coconut water an effective oral rehydration solution [2].

Dichlorvos (2,2-dichlorovinyl dimethyl phosphate) is an organophosphate widely utilized insecticide in agricultural and domestic settings [3]. Giving its increasing misuse in suicides, there is a critical need to explore potential antidotes or protective agents against its toxic effects. Despite its efficacy in pest control, dichlorvos has garnered concern due to its

toxic effects on non-target species, including humans [3]. The compound functions by inhibiting acetylcholinesterase, leading to the accumulation of acetylcholine and subsequent overstimulation of the nervous system. This mechanism, while lethal to pests, poses significant risks to mammals, with the kidneys being particularly vulnerable to dichlorvos-induced toxicity [4]. Coconut water has been postulated to have nephroprotective properties, potentially mitigating renal damage caused by toxic agents such as dichlorvos [5].

The use of dichlorvos as a pesticide in Nigeria is prevalent, particularly in rural areas where it is applied to preserve grains and other food items. Its accessibility has also led to its misuse in suicide attempts, resulting in acute toxicity that can lead to renal failure and death [6]. Despite the known toxic effects of dichlorvos on kidneys, limited studies have investigated protective agents that could counteract its harmful impact. Coconut water, with its rich electrolyte content and potential nephroprotective properties, presents a possible intervention to mitigate renal damage caused by dichlorvos exposure [6]. The nephrotoxic effects of dichlorvos, particularly when ingested or inhaled in significant quantities, necessitate the exploration of protective or therapeutic agents. Coconut water, known for its hydration and mineral replenishment capabilities, may offer a natural and accessible means of countering dichlorvos-induced renal damage. This study investigated the protective effects of tender coconut water on renal biochemical parameters in albino rats subjected to acute, sub-chronic, and chronic exposure to dichlorvos.

MATERIALS AND METHODS

Study Area and Population

Sixty-five (65) albino rats consisting of males and females that weighed 150-200g were selected for the study. The animals were obtained from the University of Port Harcourt College of Health Sciences. They were transported in well-ventilated wired cages to the animal house at the Department of Medical Laboratory Science, Port Harcourt, and Rivers State University. During this study, the rats were maintained with a 12-hour light/dark cycle and were provided access to solid poultry chow as feed and water from the tap.

Experimental Design

The study was divided into three phases: acute (24 hours), sub-chronic (2 weeks), and chronic (1 month). Each phase included three groups: a negative control group (water only), a positive control group (dichlorvos only), and a treatment group (dichlorvos with coconut water), totaling nine groups.

a) Acute Phase: Group 1 received water only, serving as the negative control. Group 2 received 30 mg/kg of dichlorvos, serving as the positive control. Group 3 received 30 mg/kg of dichlorvos and 1 ml of coconut water.
b) Sub-Chronic Phase: Group 4 received water only (negative control), Group 5 received 10 mg/kg of dichlorvos (positive control), and Group 6 received 10 mg/kg of dichlorvos with 1 ml of coconut water.
c) Chronic Phase: Group 7 received water only (negative control), Group 8 received 10 mg/kg of dichlorvos (positive control), and Group 9 received 10 mg/kg of dichlorvos with 1 ml of coconut water.

Sample Collection

Blood samples were collected from all groups at the end of each phase. Following venipuncture, 5 ml of blood was drawn from each rat. The samples were divided into two portions: 2 ml in EDTA tubes for G6PD screening and 3 ml in plain tubes for biochemical analysis. The samples were centrifuged at 3500 rpm for 5 minutes, and the plasma was separated for analysis.

Renal Function Tests

The renal function markers, including sodium, potassium, chloride, urea, and creatinine, were analyzed using a semi-automated chemistry analyzer (Map-lab). The assays were performed following standardized protocols:

a) Serum Urea: Measured using the urease-Berthelot enzymatic reaction, where urea is hydrolyzed into ammonia and carbon dioxide. The ammonia reacts with salicylate and NaClO, forming a green indophenol, which is measured spectrophotometrically at 580 nm.
b) Serum Creatinine: Determined using the Jaffe's Slot method, where creatinine reacts with picric acid in an alkaline medium to form a colored complex. The intensity of the color, measured at 510 nm, is directly proportional to the creatinine concentration.
c) Serum Potassium: Assessed using a colorimetric method where potassium ions react with sodium tetraphenylboron in an alkaline medium to form a turbid suspension. The turbidity, proportional to potassium concentration, is measured at 500 nm.
d) Serum Sodium: Analyzed using a colorimetric method based on the reaction of sodium with a selective chromogen to produce a chromophore, whose absorbance at 630 nm corresponds to the sodium concentration.
e) Serum Chloride: Measured using the mercurous thiocyanate method, where chloride ions react with mercurous thiocyanate to form mercury perchlorate and thiocyanate. The resulting red complex is quantified at 505 nm.

Statistical Analysis
Data were analyzed using Medcalc software version 22.023. Categorical variables were presented as frequencies and continuous variables as mean ± SD. Differences between groups were determined using One-way ANOVA, followed by Dunnett's Multiple Posttest to locate specific differences. *P* value <0.05 was considered statistically significant.

RESULTS
Renal Parameters in Acute Study (24 hours)
The acute study results are summarized in Table 1. Significant differences were observed in several renal parameters when comparing the groups.

Table 1: Mean±SD of renal parameters in acute study (24hrs)

	Na mmol/L	**K mmol/L**	**Cl mmol/L**	**Urea mmol/L**	**Creat µmol/L**
GP 1	154.20±2.77	5.76±1.03	103.00±1.87	4.20±0.94	53.06±10.85
GP 2	159.80±4.71	6.50±0.37	106.60±1.52	6.88±0.43	75.04±5.34
GP 3	145.80±4.66	5.08±0.16	85.28±4.69	4.61±0.82	52.68±8.77
P-value	0.001	0.014	0.438	<0.001	0.002
F-value	14.434	6.176	0.886	17.798	11.016
Posthoc					
G1 vs G2	NS	NS	NS	S	S
G1 vs G3	S	NS	NS	NS	NS
G2 vs G3	S	S	NS	S	S

Key:
G1 = group 1 (Negative Control)
G2 = group 2 (Positive Control)
G3 = group 3 (Treatment Group)
S = Significant
NS = Not significant

For the sodium renal test, Group 2 (positive control) showed a significant increase in sodium levels (159.80 ± 4.71 mmol/L) compared to Group 1 (negative control) with 154.20 ± 2.77 mmol/L ($p = 0.001$). A significant reduction in sodium levels was observed in Group 3 (treatment group, 145.80 ± 4.66 mmol/L) compared to Group 2 ($p = 0.001$). Whilst the potassium test showed a significant increase in Group 2 (6.50 ± 0.37 mmol/L) compared to Group 1 (5.76 ± 1.03 mmol/L, $p = 0.014$). Group 3 showed a significant reduction in potassium levels compared to Group 2 (5.08 ± 0.16 mmol/L, $p = 0.014$).

For Urea and Creatinine, significant elevations in urea (6.88 ± 0.43 mmol/L) and creatinine (75.04 ± 5.34 µmol/L) were noted in Group 2 compared to Group 1 (urea: 4.20 ± 0.94 mmol/L, creatinine: 53.06 ± 10.85 µmol/L, both $p < 0.001$). Group 3 exhibited significantly lower levels of both urea and creatinine compared to Group 2 (urea: 4.61 ± 0.82 mmol/L, creatinine: 52.68 ± 8.77 µmol/L, both $p < 0.001$ and $p = 0.002$, respectively).

Renal Parameters in Sub-Chronic Study (2 weeks)
The sub-chronic study results, presented in Table 2, revealed the following:
For the 2 weeks phase of the study, creatinine levels showed a significant increase in creatinine in Group 5 (positive control) compared to Group 4 (negative control) (82.86 ± 6.11 µmol/L vs. 53.06 ± 10.85 µmol/L, $p < 0.001$). The treatment group (Group 6) showed a decrease in creatinine levels (71.08 ± 5.10 µmol/L) compared to Group 5, although the reduction was not statistically significant ($p > 0.05$). Whilst other parameters such as sodium, potassium, chloride, and urea levels showed no significant differences among the groups ($p > 0.05$ for all comparisons), indicating that dichlorvos at the administered sub-chronic dose did not markedly alter these renal parameters.

Table 2. Mean±SD of renal parameters in sub-chronic study (2weeks)

	Na mmol/L	**K mmol/L**	**Cl mmol/L**	**Urea mmol/L**	**Creat µmol/L**
GP 4	154.20±2.77	5.76±1.03	103.00±1.87	4.20±0.94	53.06±10.85
GP 5	152.40±3.13	5.52±0.89	105.00±3.74	5.55±0.44	82.86±6.11
GP 6	153.20±2.39	4.30±0.66	105.40±2.07	4.84±0.93	71.08±5.10
P-value	0.604	0.056	0.353	0.064	<0.001
F-value	0.526	4.032	1.138	3.484	18.670
Posthoc					
G4 vs G5	NS	NS	NS	NS	S
G4 vs G6	NS	NS	NS	NS	S

G5 vs G6	NS	NS	NS	NS	NS

Key:
G4 = group 4 (Negative Control)
G5 = group 5 (Positive Control)
G6 = group 6 (Treatment Group)
S = Significant
NS = Not significant

Renal Parameters in Chronic Study (1 month)
Chronic study results, as shown in Table 3, indicated significant changes in renal function markers:
The potassium, urea, and creatinine levels in Group 8 (positive control) displayed significant increases in potassium (2.94 ± 0.50 mmol/L), urea (5.50 ± 0.41 mmol/L), and creatinine (76.60 ± 3.47 µmol/L) compared to Group 7 (negative control: potassium: 5.64 ± 1.13 mmol/L, urea: 3.90 ± 0.44 mmol/L, creatinine: 56.86 ± 13.16 µmol/L; $p < 0.001$ for all). The full effect of the treatment was observed in Group 9 (treatment group) were a significant reduction in potassium (3.18 ± 0.55 mmol/L), urea (2.82 ± 0.37 mmol/L), and creatinine (66.16 ± 2.38 µmol/L) was observed compared to Group 8 ($p < 0.001$ for all), indicating the potential nephroprotective effect of coconut water. No significant differences were observed in sodium and chloride levels across all groups in the chronic study phase ($p > 0.05$), suggesting that these electrolytes were less affected by the chronic exposure to dichlorvos or the intervention with coconut water.

Table 3. Mean±SD of renal parameters in chronic study (1 month)

	Na mmol/L	**K mmol/L**	**Cl mmol/L**	**Urea mmol/L**	**Creat µmol/L**
GP 7	153.00±1.87	5.64±1.13	105.20±5.26	3.90±0.44	56.86±13.16
GP 8	151.80±6.53	2.94±0.50	106.20±5.85	5.50±0.41	76.60±3.47
GP 9	154.40±3.21	3.18±0.55	113.20±5.72	2.82±0.37	66.16±2.38
P-value	0.648	<0.001	0.087	<0.001	0.001
F-value	0.450	18.325	3.013	54.715	7.664
Posthoc					
G7 vs G8	NS	S	NS	S	S
G7 vs G9	NS	S	NS	S	NS
G8 vs G9	NS	NS	NS	S	NS

Key:
G7 = group 7 (Negative Control)
G8 = group 8 (Positive Control)
G9 = group 9 (Treatment Group)
S = Significant
NS = Not significant

DISCUSSION
The present study evaluated the nephroprotective effects of coconut water on albino rats exposed to dichlorvos (DDVP), an organophosphate insecticide known for its neurotoxicity and potential to cause renal damage [7]. The findings across the acute, sub-chronic, and chronic phases provide insight into the renal impacts of dichlorvos and the potential mitigating effects of coconut water. In the acute phase, significant elevations in urea and creatinine levels in the positive control group (Group 2) compared to the negative control (Group 1) confirm the nephrotoxic effects of high-dose dichlorvos within 24 hours. These findings align with previous studies indicating that organophosphates can induce acute renal impairment by disrupting glomerular filtration and tubular function [7].

The treatment group (Group 3), which received both dichlorvos and coconut water, exhibited significantly lower levels of urea and creatinine compared to Group 2. This suggests that coconut water may reduce the acute nephrotoxic effects of dichlorvos, possibly through its electrolyte replenishment and antioxidant properties which scavenge free radicals, thereby reducing oxidative damage to renal tissues [8].

During the sub-chronic phase, a significant increase in creatinine was observed in the positive control group (Group 5), indicating ongoing renal stress due to sustained dichlorvos exposure [9]. The lack of significant differences in sodium, potassium, chloride, and urea levels between the control and treatment groups suggests that the lower dose of dichlorvos used in this phase may not have been sufficient to elicit the full spectrum of renal toxicity observed in acute poisoning. However, the reduced creatinine levels in the treatment group (Group 6) compared to Group 5, although not statistically significant, hint at the potential long-term nephroprotective effects of coconut water [10]. In the chronic phase, dichlorvos exposure resulted in significant increases in potassium, urea, and creatinine levels in the positive control group (Group 8),

reflecting chronic renal damage. The treatment group (Group 9), which received both dichlorvos and coconut water, showed marked reductions in these parameters compared to Group 8, indicating that coconut water may mitigate the cumulative renal damage induced by prolonged dichlorvos exposure. These findings are consistent with studies that highlight the beneficial effects of coconut water in preventing renal dysfunction through mechanisms such as crystal inhibition, reduced oxidative stress, and enhanced renal function [11].

The lack of significant changes in sodium and chloride levels across all phases suggests that these electrolytes are less susceptible to alterations from dichlorvos toxicity or are more effectively regulated by compensatory mechanisms in the kidneys.

CONCLUSION

The findings from this study suggest that coconut water has a protective effect on renal function in albino rats exposed to dichlorvos toxicity. While dichlorvos induced significant renal impairment, as evidenced by elevated urea, creatinine, and potassium levels, concurrent administration of coconut water attenuated these effects, particularly in the acute and chronic phases. These results underscore the potential of coconut water as a natural nephroprotective agent against organophosphate-induced renal damage.

Given the widespread use of dichlorvos and its known toxicity, particularly in rural and agricultural settings, it is recommended that further research be conducted to explore the molecular mechanisms underlying the protective effects of coconut water. Additionally, public health initiatives should raise awareness about the risks associated with dichlorvos exposure and promote the use of natural remedies like coconut water to mitigate these effects. Future studies could also investigate the efficacy of coconut water in other models of nephrotoxicity and its potential role in human health.

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