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Cashew (*Anarcadium occidentale*) nut attenuates experimental model of nephrotoxicity induced by lead-mediated lipid peroxidation and impaired histoarchitecture in kidney tissue

Abstract

Background: Cashew *(Anarcadium occidentale)* nut possess anti-oxidative and anti-inflammatory properties which may be beneficial for nephrotoxicity caused by lead-mediated lipid peroxidation and impaired histoarchitecture of the kidney.

Methods: Serum urea and creatinine as well as kidney lipid peroxidation (MDA) and histoarchitecture was assayed on kidney tissues of 20 Wistar rats in order to ascertain the impact of cashew nut on nephrotoxicity induced by lead. The experimental animals were grouped into groups A, B, C and D; Group A served as control and received 1ml of distilled water. Experimental model of nephrotoxicity was induced on groups B, C and D with lead acetate 150mg/kg; group B was left untreated while groups C and D were treated with 500mg/kg and 1000mg/kg (P.O.) of cashew nut respectively for 14days.

Results: Lead acetate significantly increased serum urea, creatinine and kidney MDA at $P<0.05$ as well as impair histoarchitecture of the kidney tissue by causing great degree of distortion of glomerulus, thin macula densa, distorted thin segments of loop of Henle and infiltration of inflammatory cells on the tissues. Cashew nut showed beneficial input by averting the adverse impact of lead acetate via reducing serum urea, creatinine and kidney MDA significantly at $P<0.05$ as well as moderating the histoarchitecture of the kidney tissue back to normal architectural components with various cells of straight segments of loops of Henle and mild irregular contour of thin segments of loop of Henle.

Conclusion: Cashew nut attenuated nephrotoxicity caused by lead-mediated lipid peroxidation and impaired histoarchitecture of kidney tissues.

Keywords: *Anarcadium occidentale*, cashew nut, lead acetate, nephrotoxicity, kidney, lipid peroxidation

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Introduction

Nephrotoxicity refers to depletion in the structure and function of the kidney as a result of damage induced by chemicals, medications and diseased conditions leading to physiological alterations.[1] Lead (Pb), a known toxicant just like calcium carbide[2,3] and acrylamide[4,5] has been shown to induced toxicity on various organs like testes, stomach, liver including the kidney.[1,6] Lead induces organ damage via oxidative stress;[6,7] by increasing tissue lipid peroxidation (MDA) and decreasing tissues antioxidants such as catalase (CAT), glutathione (GSH) and superoxide dismutase,[6] although several studies are ongoing to avert toxicity induced by lead.

Cashew *(Anarcadium occidentale)* nut consumed as snack nut are used as food recipes, also refined into cashew cheese or cashew butter.[8] Cashew nut has been reported to have several nutritional benefits as well as medicinal impacts on various pathophysiological conditions.[9–11] Cashew nuts is said to be a medicinal plant with anti-oxidative and anti-inflammatory properties.[12] Studies has reported its therapeutic input in inhibiting oxidative stress and inflammations[13] as well as wound healing potentials.[14]

Despites the beneficial report of cashew nut, its input on lead-induced nephrotoxicity has not been fully elucidated. Considering the stated anti-oxidative and anti-inflammatory properties;[12–14] it was hypothesized that cashew nut may have beneficial impact on nephrotoxicity caused by lead-mediated lipid peroxidation and impaired histoarchitecture in kidney tissue. Hence, this study was designed to elucidate the impact of aqueous solution of cashew *(Anarcadium occidentale)* nut on nephrotoxicity caused by lead-mediated lipid peroxidation and impaired histoarchitecture in kidney tissue of experimental animal.

Materials and methods

Chemical: Laboratory graded lead acetate was obtained from the laboratory of Department of Biochemistry, Gregory University Uturu, Abia state, Nigeria.

Plant material: Roasted cashew nuts were purchased from a local (Achara) market at Uturu, Abia State, Nigeria. The nuts were cleaned of dirt and shells and blended with household blender into fine flour for easy administration to the rats. The blended flour was then stored in airtight containers and refrigerated for use during the experiment.

Animals and experimental grouping: This study was performed strictly in accordance to the principle of use and cares for laboratory animal (NIH, Publication, No 85-23). 20 wistar rats weighing 150-200g, procured from the animal house of the Faculty of Basic Medical Sciences, Gregory University Uturu, were used. The animals were housed in clean and comfortable cages under a dark/light cycle. The rats had access to standard growers chow and water ad libitum. The animals were allowed a period of 14 days for acclimatization. After

acclimatization, the rats were randomly distributed into 4 groups of 5 rats each. Group A: control and received 1ml of distilled water. Group B animals received 150mg/kg of lead acetate only. Group C rats received 150mg/kg of lead acetate + 500mg/kg of aqueous solution of cashew nuts. Group D rats received 150mg/kg of lead acetate + 1000mg/kg of aqueous solution of cashew nuts. Lead acetate dosage was done according to Nwosu et al.,[6] Cashew nut dosage was done according to Dias et al.[15] Cashew nut was dissolved in distilled water before administration. All administration was done orally for 14 days.

Animal euthanasia and sample collection: On day 15 after administration, the animals were euthanized using cervical dislocation. Blood was collected via cardiac puncture and put into a sample bottle and centrifuged at 3000rpm for 15 minutes. The serum gotten was used for urea and creatinine assay as described by Saka et al.[16]

Determination of Lipid peroxidation: The left kidney was excised out, cleared of fats, weighed and homogenized in cold phosphate buffer and centrifuged at 3000rpm. The supernatant was collected into plain sample bottle and stored at -20C. Malondialdehyde (MDA) activities were based on measurement of thiobarbituric acid malondialdehyde absorbance at 532nm.[17]

Histopathological studies: The right kidneys were excised out, weighed and immediately fixed in 10% formal saline for histological tests. After fixation, tissues were subjected to dehydration through graded alcohol, cleared with xylene and infiltrated with molten paraffin wax to harden. Tissues were then embedded into tissue blocks and later sectioned into 5µm sections with rotary microtome. The thin sections were floated in water bath, mounted on microscopic slides, dried in oven and stained with Harris hematoxylin and 1% eosin. Finally, microscopic examination was carried out using a photomicroscope and slides obtained.

Statistical analysis: Numerical data obtained were expressed as means ± standard error of mean (SEM). One way analysis of variance was estimated by using the Graph Pad Prism 5 to determine the difference among various treatments groups. Multiple comparisons among various treatments groups were determined by using Boneferroni post hoc comparison test. Values were considered significant at $p<0.05$.

Results

Cashew (*Anarcadium occidentale*) nut impact on urea and creatinine of experimental model of nephrotoxicity induced by lead-mediated lipid peroxidation and impaired histoarchitecture in kidney tissue: Serum urea level in group B (lead only) was significantly elevated when compared with control (group A) $P<0.05$. When compared with the control (group A), the cashew nut treated groups C and D showed a non-significant increase in serum urea levels. There was no significant decrease in group C, but there was significant decrease in serum urea of group D when compared to the lead only (group B) (Figure 1).

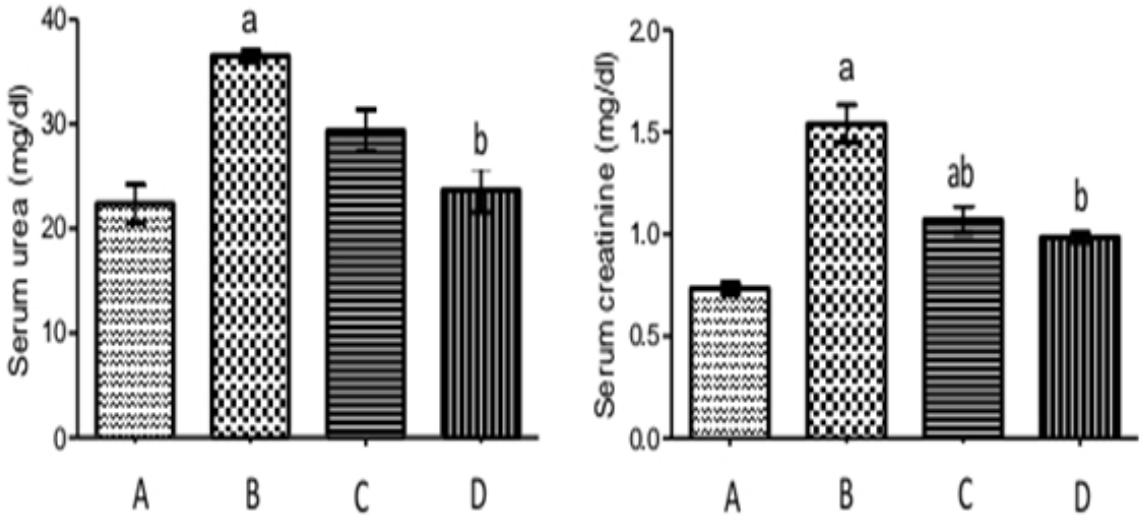

Figure 1 Serum urea and creatinine levels in kidney tissue of experimental groups. 'a' indicates statistically significant when compared with control (group A). 'b' indicates statistically significant when compared to lead only (group B) at $P<0.05$. Group C and D = 500mg/kg and 1000 mg/kg of cashew nut treated groups respectively.

Serum creatinine levels in groups B and C were significantly increased when compared with the control (group A) $P<0.05$. Group D showed a non-significant increase when compared with control (group A). Serum creatinine levels in the cashew nut treated groups C and D were significantly decreased when compared to the lead only (group B) at $P<0.05$ (Figure 1).

Cashew (Anarcadium occidentale) nut impact on kidney lipid peroxidation (MDA) of experimental model of nephrotoxicity induced by lead: Kidney MDA levels in the lead only (group B) and cashew nuts treated groups (C and D) were significantly higher when compared with the control group A $P<0.05$. Furthermore, there was significant decrease in MDA levels in a dose dependent manner in

cashew nuts treated groups (C and D) when compared to the lead only group B at P<0.05 (Figure 2).

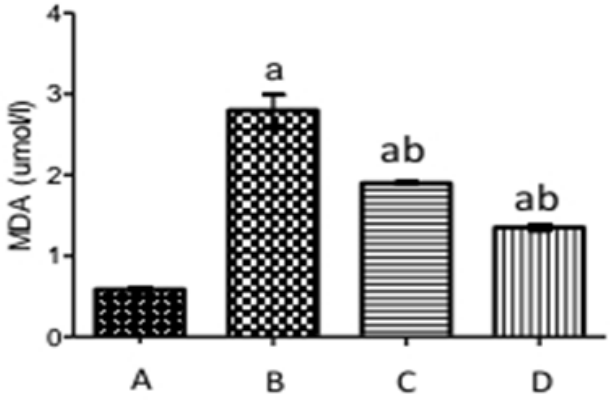

Figure 2 Lipid peroxidation (MDA) levels in kidney tissue of experimental groups. 'a' indicates statistically significant when compared with control (group A). 'b' indicates statistically significant when compared to lead only (group B) at P<0.05. Group C and D = 500mg/kg and 1000 mg/kg of cashew nut treated groups respectively.

Cashew (Anarcadium occidentale) nut impact on kidney histoarchitecture of experimental model of nephrotoxicity induced by lead: Figure 3A showed a normal architecture in the control (group A) with normal contour of the glomerulus, thick macula densa and ring shape of thin segments of loop of Henle. Lead exposure without treatment in group B caused a great degree of distortion of glomerulus, thin macula densa, distorted thin segments of loop of Henle and infiltration of inflammatory cells (Figure 3B). Kidney of group C treated with 500mg/kg of cashew nut after lead exposure, showed mild infiltration of inflammatory cells, irregular basal membrane separated from tubules and distortion in contour of collecting tubules (Figure 3C). Figure 3D showed normal architectural components with various cells of straight segments of loops of Henle and mild irregular contour of thin segments of loop of Henle.

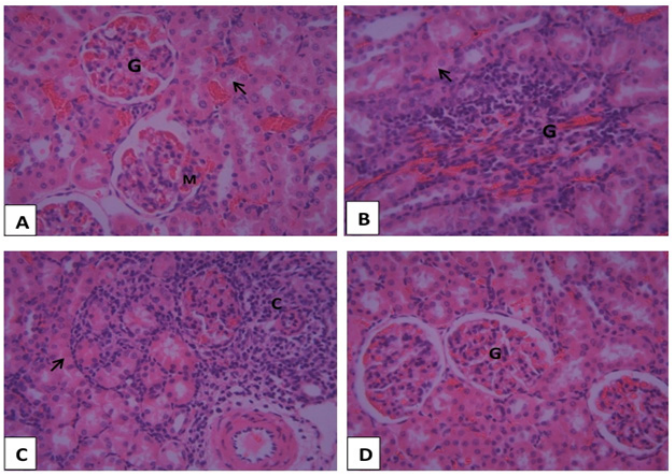

Figure 3 Photomicrographs of kidney in [a] control group; [b] lead only group; [c] lead + 500mg/kg cashew nut group; [d] lead + 1000mg/kg cashew nut group. G-glomerulus; M-macula densa; C-collecting tubules; arrow-loop of Henle (H&E x400).

Discussion

Cashew *(Anarcadium occidentale)* nut showed ameliorative input on the experimental nephrotoxicity induced by lead, as this study result showed that 1000mg/kg of cashew nut significantly decreased serum urea when compared to the impact exhibited by lead acetate (Figure 1) 500mg/kg and 1000mg/kg of cashew nut also showed significant dose-dependent decrease in serum creatinine levels when compared to the impact of lead acetate (Figure 1) suggesting an improvement in the function of the kidney that was impaired by lead. This is because serum urea and creatinine are biomarkers of kidney function test which is known to be decrease on proper functioning of the renal system.[18]

Lipid peroxidation (MDA) is a biomarker of oxidative stress.[4] MDA is increased in tissues undergoing oxidative stress.[5] Lead induced oxidative damage by increasing kidney MDA levels in this study (Figure 2) which is accordance to the report that lead induces tissue damage via increasing lipid peroxidation.[6] Findings from this study showed that 500mg/kg and 1000mg/kg of cashew nut attenuated lead mediated lipid peroxidation by significantly decreasing MDA levels when compared to the impact of lead acetate at P<0.05 (Figure 2) This ameliorative impact could be as result of the anti-oxidative properties of the cashew nut and this is in correspondence to studies 9-11 that stated its antioxidants and anti-inflammatory effects on various tissues via attenuated MDA activities.

The histopathology results corroborated other findings of this study as the group exposed to lead had severe histoarchitectural damage of the kidney tissues depicted by glomerular distortion, leukocyte infiltration and tubule separation. This is in line with lead induced kidney damage reported by other studies.[19–21] Treatment with 500mg/kg of cashew nut was able to provide mild ameliorative effect through reduction of inflammatory cell infiltration and tubular distortion. However, treatment with 1000mg/kg of cashew nuts restored the normal kidney histology. This may be as a result of the antioxidant and anti-inflammatory potentials of cashew nuts.[9–11]

Conclusion

Findings from this study shows that cashew *(Anarcadium occidentale)* nut has beneficial input on nephrotoxicity induced by lead-mediated lipid peroxidation and impaired histoarchitecture in kidney tissue by averting the impact of lead acetate on serum urea, creatinine, MDA and histoarchitecture of the kidney.[7]

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None

Conflict of interests

Authors declare no conflict of interests.

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