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Original Article

Nephroprotective effect of *Costus afer* on lead induced kidney damage in albino rats

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Abstract: Background: Lead is a nephrotoxicant probably implicated in the rising incidence of chronic kidney injury in sub-Sahara Africa. With the prohibitive cost and unavailability of metal chelators, chronic kidney disease CKD prevention is very difficult hence the search for affordable alternative. *Costus afer* have been shown to be organo-protective. The present research investigated the nephroprotective effect of aqueous leaf extract of *Costus afer* on lead induced nephrotoxicity in male rats. Methods: Adult male rats were weight matched into five groups of five rats each. Groups 1 & 2 serve as normal and toxic control receiving deionized and leaded $(CH_3COO)_2Pb.\ 3H_2O$ water respectively. Groups 3, 4 and 5 were administered peroral 750, 1500 and 2250 mg/kg of aqueous leaf extract of *Costus afer* respectively while receiving Pb^{2+} water *ad libitum*. Hematological, antioxidant and histological parameters obtained from the result serve as scientific evidence in the study. Results: *Costus afer* treatment significantly reversed ($P < 0.05$) the decrease in the levels of gluthatione peroxidase (GSH-PX), superoxide dismutase (SOD), catalase (CAT), Glutathione-S-trasferase activity (GST) seen in the lead acetate only treated group. Similarly, the increased malondialdehyde (MDA) level in the lead acetate only treated group was significantly ($P < 0.05$) reduced in the *Costus afer* treated groups. There were significant ($P < 0.05$) decreases in serum serum level of sodium (146 ± 2.1 to 133 ± 6.0) and potassium (5.1 ± 0.4 to 4.4 ± 0.3) in lead acetate alone and treated group respectively. Also recorded was a significant ($P < 0.05$) decrease in serum levels of total protein and albumin (67 ± 7.9 to 47 ± 5.0 g/dl) and (45 ± 4.4 to 33 ± 5.5 g/dl) in lead acetate alone and *Costus afer* treated groups respectively. Conclusions: Aqueous leaf extract of *Costus afer* may be nephroprotective in albino rats.

Keywords: Multi-organ damage, lead acetate, antioxidant parameters, *Costus afer*

Introduction

Lead from sundry sources including crude oil drilling and refining activities especially in developing countries have been implicated in various forms of diseases [1]. Chronic lead exposure has been implicated in renal failure in experimental animals [2]. Clinical trials and epidemiological studies have demonstrated that lead poisoning can cause kidney damage. It is estimated that by 2030, more than 70% of patients with end-stage renal disease will be living in low-income countries like in sub-Saharan Africa [3]. In Nigeria the incidence of chronic kidney disease CKD ranges from 1.6-12.4 percent [4].

Now the widely adopted treatment for lead poisoning involves the use of chelating agents which form insoluble complex with lead and ultimately hasten the elimination of lead [5]. Although orally administered meso-2,3-dimercaptosuccinic acid (DMSA), a sulfhydryl-containing, water-soluble, metal chelator is non-toxic, like other chelators cannot remove metals from intracellular sites and may in fact cause redistribution of the toxic metals, essential metal loss, and renal dysfunction [6]. Also, metal chelators have some side effects. Succimer causes nausea, vomiting, diarrhea and skin rash; Penicillamine (abdominal pain, skin lesions, alopecia, stomatitis, glossitis, leucopenia, thrombocytopenia, enuresis); BAL (nausea, vomiting, sweating, high fever, hypertension, and tachycardia); DMPS causes transient increases in hepatic transaminase activity which however, resolves with discontinuation of drug; EDTA (renal toxicity, cardiac problems due to hypocalcemia) [7]. In addition to the adverse effects of these chelators, the prohibitive cost

and scarcity of these agents pose serious management challenge in resource poor countries in the developing nations.

There is a positive correlation between dietary supplementation with certain vegetables and the reduction of toxic effects of various toxicants and environmental agents, including heavy metals [8]. *Costus afer* a commonly consumed vegetable in Nigeria has been shown be to organoprotective [9]. Given the high cost, scarcity and wide range of adverse effects of chelators the classical antidotes of lead poisoning, continuous search for widely available "natural antidotes" that will ameliorate or reverse the deleterious effects of lead in developing nations has been research focus in our laboratory.

Objectives

The present study examined the efficacy leaf extract of *Costus afer* in mitigating lead-induced oxidative stress and injury in the kidney of male albino Wistar rats.

Materials and methods

Sample identification

Costus afer was identified and authenticated by Mr O.Ozioko A.O, International Center for Ethno Medicine and Drug Development (INTERCEDD), University of Nigeria Nsukka, Nigeria and the voucher Number is INTERCEDD/033.

Sample processing and extraction

The leaves were washed with clean water, shade dried in a well-ventilated place for 24 hrs. Two hundred and fifty grams of the leaves was weighed and macerated into 500 ml of deionized water placed in a stoppered container and allowed to stand for 48 hrs with constant agitation. After 48 hrs, the mixture was strained, the marc pressed, and the liquid filtered and stored in refrigerator at 4°C. The solution was discarded every three days and a fresh sample prepared and the process repeated till the end of the study.

Preparation of 2500 ppm leaded water

A 50 g lead acetate $(CH_3COO)_2Pb{\cdot}3H_2O$) was dissolved in 12 ml of 1 N HCL and made up to 20 L with deionized water. Ten grams of glucose was added to improve the taste [10].

Animal husbandry

Twenty male albino Wistar rats (*Rattus norvegicus*) weighing between 90-180 g were purchased from the Department of Experimental Pharmacology & Toxicology Animal house Abuja campus, Faculty of Pharmaceutical Science, University of Port-Harcourt, Rivers State. The rats were kept in polypropylene cages and maintained under standard conditions prescribed by the committee for the purpose of control and supervision on experiments on animals (CPCSEA). The experimental protocol was approved by the Institutional Animal Ethics Committee and the approval number UPH/PHARM/2017/033 assigned. They were weight matched into five groups of five animals each and allowed to acclimatize with for two weeks. They were housed in a standard cage and maintained in standard laboratory condition at ambient temperature (25 ± 2°C) with relative humidity (55-64%) and light and dark conditions (12/12 h). They were fed with Top Feeds (Flour Mills Lagos, Nigeria.) and leaded acetate $(CH_3COO)_2Pb{\cdot}3H_2O$) solution except for the normal control that received deionized water *ad libitum*. Animal ethics and proper handling method were strictly adhered to. The bedding of the cage was changed daily, and the cage was also washed and disinfected weekly.

Experimental design

Five groups of five male albino rats were used in the experiment. Each group was treated and fed as follows for four weeks:

Group 1: served as the normal control and received deionized water; Group 2: served as toxic control and received lead acetate solution; Group 3: lead acetate solution plus 750 mg/kg b.w. *Costus afer;* Group 4: lead acetate solution plus 1500 mg/kg b.w. *Costus afer;* Group 5: lead acetate solution plus 2250 mg/kg b.w. *Costus afer.*

The dose of the *Costus afer* extract used was based on the previous studies [11] while the method of Akram *et* al. [10] was adopted for the administration of the lead acetate solution. The body weights were monitored weekly, while the

fluid and feed intake of the rats in all the groups were monitored daily for 28 days.

Necropsy

On the 28th day, the rats were fasted overnight, weighed, and sacrificed under ether anesthesia on the 29th day. The blood samples were collected by cardiac puncture and kept at temperature of 4°C for 6 hours. The blood samples were then centrifuged at 3000 rpm for 15 minutes and stored properly for further analysis. The left kidney was stored in 10% formaldehyde and processed for histological examination whereas the right kidney was homogenized in ice-cold 0.1 M Tris HCl buffer (pH 7.4) to produce 10% homogenate. The homogenate was centrifuged at 5000 g at 4°C for 15 minutes. Supernatant was collected for antioxidant assay.

Biochemical analysis

Determination of kidney superoxide dismutase (SOD): SOD was estimated by the method that involved inhibition of superoxide-dependent reduction of tetrazolium dye, methyl thiazolyl tetrazolium (MTT) to its formazan [12]. The activity of superoxide dismutase was determined by the method of Misra and Fridovich [13].

Determination of kidney Malondialdehyde (MDA): Lipid peroxidation was determined by measuring the formation of thiobarbituric acid reactive substances (TBARS) according to the method of Ohkawa and Ohishi [14] and Balzasubramanian *et al*. [15]. The MDA level was calculated according to the method of Todorova *et al.* [16] and expressed as nmol of MDA/g of wet tissue using a molar extinction coefficient of the chromophore ($1.56*10^{-5}$/m/cm).

Estimation of kidney reduced glutathione (GSH) level

GSH was estimated based on a reaction of reduced glutathione with 5-5ditiobis-2-nitrobenzoic acid (DTNB).

Determination of kidney catalase activity

Catalase activity was determined according to *Clairborne* [17] with slight modifications.

Determination of kidney Glutathione-S-Trasferase Activity (GST): Glutathione-S-transferase (GST) activity was determined according to Habig *et al.* [18].

Determination of kidney Glutathione Peroxidase Activity: The activity of GSH-Px was assessed according to established methods of Rotruck *et al.* [19].

Renal biochemical parameters analysis

Total serum proteins [20, 21], serum Albumins [22], serum Globulin (calculated by subtracting the quantity of albumins from that of total proteins), serum creatinine [23] and serum urea [24, 25] were determined.

Hematological analysis

Five ml of blood was collected from the animals in each group in anticoagulant bottles for hematological analysis. The following hematological parameters were assayed RBC, TWBC, and differentials. Total White Blood Cell Counts (TWBC), Packed Cell Volume (PCV), Red blood cell (RBC) count, Lymphocytes% (L%) and Neutrophils% (N%) were determined using the Hemocytometer method [26]. Hemoglobin (Hb) concentration was determined by the Cyanmethemoglobin method [27].

Histology

For light microscopy examination, the formalin fixed tissues (kidney) were dehydrated through ascending grades of alcohol, cleared in three changes of xylene, and were embedded in paraffin. Serial sections, each of 4-micron thickness, were cut and stained with H and E as per standard protocol. Stained sections were morphologically evaluated, and the pictures of the slides were taken for comparison.

Ethical issues

The research followed the tenets of the Declaration of Helsinki. The protocol of this study is designed in accordance with the ethical principles of the International Committees for the Protection of Animal Rights Laboratory. This project was approved by Ethics Committee of the University of Port Harcourt, Rivers State, Nigeria.

Statistical analysis

The data were subjected to statistical analysis by applying one-way ANOVA using statistical

Table 1. Effect of the aqueous leaf extract of *Costus afer* on absolute and relative weights of kidney in lead acetate treated rats

Treatment Groups	Final Body-weight (weight gain/% weight gain)	Absolute weight (g)	Relative weight (%)
Water	99.9 ± 7.77 (27.10 ± 7.45/37.23)	1.57 ± 0.02	1.57
Pb Acetate	114.52 ± 11.3 (18.98 ± 6.43/19.87)	0.77 ± 0.01	0.67
Pb + 750 mk/kg CA	125.7 ± 10.2 (21.04 ± 6.65/22.46)	0.80 ± 0.01	0.64
Pb + 1500 mg/kg CA	130 ± 9.34 (20.8 ± 6.23/19.05)	0.80 ± 0.01	0.31
Pb + 2250 mg/kg CA	196.3 ± 11.6 (28.98 ± 12.39/17.22)	0.80 ± 0.01	0.41

Table 2. Effect of *Costus afer* leaf extract on the hematogical profile of lead acetate treated rat

Samples	Hb (g/dl)	PCV (%)	RBC (10^6 µL)	WBC (10^3 µL)	Platelets	N (%)	L (%)
Water							
Mean ± SD	13.43 ± 1.63	40.3 ± 4.9	5.7 ± 1.1	4.9 ± 0.3	216.7 ± 35.1	28.3 ± 7.6	71.7 ± 7.6
Max	15.30	46	7	5.2	250	35	80
Min	12.30	37	5	4.6	180	20	65
Pb Acetate alone							
Mean ± SD	11.7 ± 1.18*	34 ± 1.0*	4.4 ± 0.9*	9.9 ± 0.3*	216.7 ± 61.1	22.7 ± 2.1*	77.3 ± 2.1*
Max	13	35	5.8	10.2	270	25	79
Min	10.7	33	4.0	9.6	150	21	75
Pb Acetate + 750 mg/kg CA							
Mean ± SD	11.3 ± 0.35	35 ± 3.6**	4.7 ± 0.15	5.6 ± 0.4*	230 ± 55.7**	32.3 ± 2.5**	67.7 ± 2.5**
Max	11.7	39	4.8	6.0	280	35	70
Min	11.0	32	4.5	5.2	170	30	65
Pb Acetate + 1500 mg/kg CA							
Mean ± SD	12.5 ± 0.68	37.3 ± 2.1**	5.4 ± 0.6**	5.0 ± 0.5**	220 ± 36.1**	29.0 ± 3.6**	71. ± 3.6
Max	13	39	6	5.5	250	32	75
Min	11.7	35	4.8	4.5	180	25	68
Pb Acetate + 2250 mg/kg CA							
Mean ± SD	13.0 ± 1.18**	39 ± 3.6**	5.4 ± 0.9**	3.7 ± 0.3**	250 ± 43.5**	26.7 ± 7.6**	73.0 ± 7.6**
Max	14.3	43	6.5	4.0	280	35	80
Min	12	36	4.8	3.5	200	20	65

L = lymphocytes, N = neutrophils, PCV = packed cell volume, RBC = red blood cells, WBC = white blood cell. The data are expressed as mean ± S.D. *: Values differ significantly from control ($P < 0.05$). **: Values differ significantly from Pb only ($P < 0.05$).

package for social sciences (SPSS) version 12.0. Differences between means were tested using Duncan's multiple comparison tests and significance was set at $P < 0.05$.

Results

Effect on the body and organ weight

Lead acetate only or in combination with *Costus afer* did not cause any significant change in the body and organ weight of the rats. The percent gain in body weight ranged from 37.23% in the normal control (untreated) to 19.87% in lead acetate only treated group to 22.46, 19.05 and 17.22% in the *Costus afer* extract treated animals respectively. However, the body weight increases in all treated groups were not significantly different in all the groups (**Table 1**).

Effect of Costus afer on the heamatological parameters

Table 2 show the effect of *Costus afer* on the heamatological parameters. Treatment of rats with lead acetate caused significant ($P < 0.05$) decreased in PCV, Hb concentration and RBC count when compared with normal control. There was a significant ($P < 0.05$) dose dependent reversal of the effect of lead acetate administration following aqueous leaf extract of *Costus afer* administration. Lead acetate only

Table 3. Effect of *Costus afer* leaf extract on the renal parameters of lead acetate treated rat

Treatment Groups	UR (mg/dl)	CR (mg/dl)	TP (g/dl)	ALB (g/dl)	Na (U/L)	K (U/L)
Water						
Mean ± SD	1.0 ± 0.1	177 ± 2.1	55 ± 2.5	33 ± 0.6	106 ± 3.5	4.6 ± 0.6
Max	1.06	180	58	34	110	5.3
Min	0.92	176	53	33	103	4.2
Pb Alone						
Mean ± SD	1.1 ± 0.1	178 ± 3.0	67 ± 5.0**	45 ± 5.5	146 ± 2.1*	5.1 ± 0.4*
Max	1.21	181	70	49	148	5.4
Min	1.1	175	52	38	144	4.6
Pb + 750 mg/kg CA						
Mean ± SD	1.2 ± 0.04	177 ± 1.0	47 ± 9.2	34 ± 5.0	133 ± 6.0**	4.4 ± 0.3**
Max	1.26	178	70	39	139	4.7
Min	1.18	176	52	29	127	4.1
Pb + 1500 mg/kg CA						
Mean ± SD	1.2 ± 0.03	175 ± 1.5	58 ± 7.9**	33 ± 4.4**	142 ± 9.7**	4.6 ± 0.1**
Max	1.26	179	67	35	150	4.7
Min	1.2	176	52	27	131	4.5
Pb + 2250 mg/kg CA						
Mean ± SD	1.73 ± 0.6	177 ± 2.7	55 ± 3.2*	34 ± 5.7**	133 ±1.5**	4.1 ± 0.6**
Max	2.21	180	58	39	146	4.5
Min	1.12	175	52	28	117	3.5

The data are expressed as mean ± S.D. *: Values differ significantly from control ($P < 0.05$). **: Values differ significantly from Pb only ($P < 0.05$). CA = *Costus afer.*

treated group (group 2) had significant increase ($P < 0.05$) in total white blood count (WBC) when compared with the normal control group, while the total WBC in groups 3-5 animals were significantly ($P < 0.05$) decreased. There were no significant changes in platelets, lymphocyte and neutrophil percentages on the lead acetate treated group compared with normal controls.

Effect on the renal parameters

The effect of *Costus afer* on serum total proteins, Albumin, Urea, Creatinine, and serum electrolytes in lead acetate-treated male albino Wistar rats is shown on **Table 3**. There were significant ($P < 0.05$) decrease in serum levels of sodium (146 ± 2.1 to 133 ± 6.0) and potassium (5.1 ± 0.4 to 4.4 ± 0.3) in the lead acetate alone and lead acetate plus 750 mg/kg *Costus afer* treated groups. There were also significant ($P < 0.05$) decrease in serum levels of total protein (67 ± 7.9 to 47 ± 5.0 g/dl) and albumin (45 ± 4.4 to 33 ± 5.5 g/dl) in the in the lead acetate only and lead acetate plus 1500 mg/kg *Costus afer* treated groups respectively.

Effect on biochemical parameters

Table 4 shows the effect of *Costus afer* on kidney antioxidant and lipid peroxidation parameters of lead acetate-treated male albino Wistar rats. Lead acetate treatment significantly decreased GSH-PX, SOD, CAT, and GST when compared with the control group. There were significant differences ($P < 0.05$) between the levels of GSH-PX, SOD, CAT, and GST in the lead acetate only treated group and the lead acetate plus *Costus afer* treated groups. Similarly, MDA level was increased in the lead acetate only treated group. The increased MDA level in the lead acetate only treated group was significantly ($P < 0.05$) reduced in the lead acetate plus *Costus afer* treated groups.

Histopathology of the kidney

Kidney: **Figure 1** shows the photomicrograph of the kidney stained with H&E techniques of normal control group that received deionized water daily for 28 days (**Figure 1A**). There were normal dilated tubules and glomeruli at the periphery of the cortex. Lead acetate group daily for 28 days-undistorted interstitial space. The kid-

Table 4. Effect of aqueous extract of *Costus afer* on the anti-oxidant parameters in the kidney of lead acetate treated rats

Treatment Groups	GSH (µg GSH mg^{-1} Protein)	CAT (unit mg^{-1} protein)	MDA (µmol MDA mg^{-1} protein	GSH-Px (µg residual GSH remaining min mg^{-1} protein)	GST (units mg^{-1} protein)	SOD (unit mg^{-1} protein)
Water						
Mean ± SD	1.9 ± 0.1	2.1 ± 0.36	0.89 ± 0.25	0.19 ± 0.03	0.67 ± 0.02	0.09 ± 0.02
Max	2.0	2.5	1.12	0.21	0.69	0.1
Min	1.8	1.8	0.62	0.16	0.65	0.07
Pb alone						
Mean ± SD	1.77 ± 0.15	0.8 ± 0.11*	1.15 ± 0.08*	0.18 ± 0.01	0.69 ± 0.03	0.11 ± 0.06*
Max	1.9	0.91	1.24	0.19	0.72	0.17
Min	1.6	0.69	0.1	0.17	0.66	0.05
Pb + 750 mg/kg CA						
Mean ± SD	1.8 ± 0.1	2.1 ± 0.4**	0.92 ± 0.1**	0.2 ± 0.01	0.92 ± 0.15	0.11 ± 0.01
Max	1.9	2.5	1.02	0.21	1.07	0.12
Min	1.7	1.7	0.8	0.19	0.78	0.1
Pb + 1500 mg/kg CA						
Mean ± SD	1.8 ± 0.1	1.83 ± 0.7**	1.08 ± 0.1	0.19 ± 0.01	0.74 ± 0.07	0.15 ± 0.03
Max	1.9	2.5	1.16	0.19	0.8	0.17
Min	1.7	1.1	0.96	0.18	0.67	0.12
Pb + 2250 mg/kg CA						
Mean ± SD	2.03 ± 0.68**	2.77 ± 0.68**	0.97 ± 1.63**	0.21 ± 0.02	0.71 ± 0.04	0.15 ± 0.03
Max	2.8	2.8	1.15	0.23	0.75	0.18
Min	1.5	1.5	0.83	0.2	0.68	0.12

The data are expressed as mean ± S.D. *: Values differ significantly from control ($P < 0.05$). **: Values differ significantly from Pb alone ($P < 0.05$). CA = *Costus afer*.

ney of rats in Lead acetate treated group was not much affected Tissue sections from the kidneys of rats in extract treated groups (Lead acetate + 750 mg/kg CA; lead acetate + 1500 mg/kg CA and lead acetate + 2250 mg/kg CA daily for 28 days had varied areas of normal histology (**Figure 1C-E**).

Discussion

Chronic exposure to lead is known to cause nephrotoxic effects like interstitial nephritis, tubular damage, and at later stages glomerular damage leading to the chronic renal failure [28, 29]. Lead absorption into blood lead to lead accumulation in the erythrocytes [30]. Treatment of rats with lead acetate in this study caused significant reduction in PCV, Hb concentration and RBC count. The result is in agreement with Azoz and Raafat [31] and Ibrahim et al. [32] findings. On the other hand, total WBC was significantly increased. Administration of *Costus afer* significantly reversed these parameters. Lead directly affects the hematopoietic system through restraining the synthesis of Hb by inhibiting various key enzymes involved in the heme synthesis pathway, particularly the enzyme Aminolevulinic Acid Dehydratase (ALAD).

Association between lead exposure and nephrotoxicity is well-documented, even in a population with blood lead levels of 5 µg/dl [33]. Kidney damage is associated with distortions in various renal functions like albuminuria, reduced glomerular filtration rate, and decreased creatinine clearance in lead-exposed populations [34, 35]. There were significant changes in the levels of, ALB, CR, TP, K^+ and Na^+ following administration of lead acetate when compared with the normal control group that received only deionized water in this study. Sodium levels in the toxic groups showed a highly significant increase when compared with the control and treated. The increased level of blood urea and creatinine concentration in lead-acetate treated rats suggests the inability of the kidney to excrete these products causing their increase in blood and decrease their excretion in urine. The increase in uric acid concentrations may be due to degradation of purines or to an increase of uric acid levels by

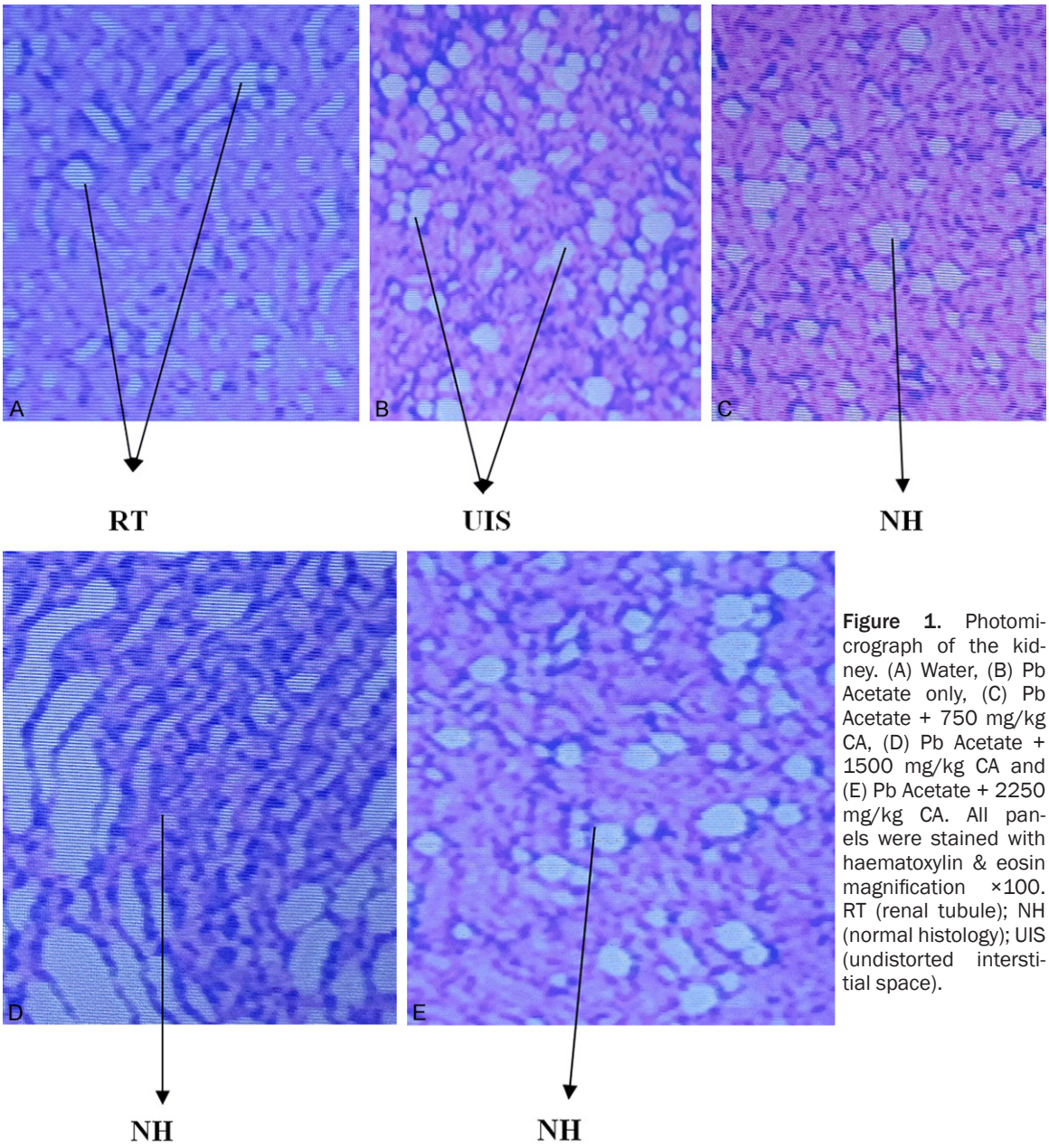

Figure 1. Photomicrograph of the kidney. (A) Water, (B) Pb Acetate only, (C) Pb Acetate + 750 mg/kg CA, (D) Pb Acetate + 1500 mg/kg CA and (E) Pb Acetate + 2250 mg/kg CA. All panels were stained with haematoxylin & eosin magnification ×100. RT (renal tubule); NH (normal histology); UIS (undistorted interstitial space).

either overproduction or inability of excretion as uric acid is the end product of the catabolism of tissue nucleic acid, i.e. purine and pyrimidine bases metabolism. Chronic exposure to lead resulted in electrolyte retention evidenced by elevation of sodium and potassium, since lead affects on renal tubular transport mechanisms. Another mechanism of increase sodium and potassium level is the decrease in functioning nephrons that trigger multiple adaptive processes in the hyper functioning remaining nephrons including augmented rates of electrolyte reabsorption [34, 35]. In this study, treatment of rats with lead acetate caused significant increase in the activity of serum bilirubin and urea, while the levels of albumin and total proteins were decreased. Similar results were reported by Azoz and Raafat [31], Ibrahim et al. [32], and Azab [35]. These parameters were significantly reversed by treatment with *C.afer*. Kidney may play an important role in the clearance of erythrocytes [36]. Erythrocytes infiltration has been observed in proximal tubules and tubular lumen of renal biopsies from patients with acute renal failure, acute glomerulo nephritis and hematuria [37].

Oxidative stress has been suggested to be the most convincing mechanism underlying lead-associated nephrotoxicity [38]. Pro-oxidant and anti-oxidant balance, along with decreased glutathione and increased lipid peroxidation, occur in the kidney following lead exposure in animal models [39, 40]. The levels of total glutathione, superoxide dismutase, gluthatione peroxidase were observed to be significantly reduced in lead acetate-treated rats. These reductions accompanying lead acetate treatment were significantly reversed by *Costus afer*, which also significantly reduced the value of the lead acetate-induced biomarker of lipid peroxidation, malondialdehyde. This result agrees with that reported by Azoz and Raafat [31]. Oxidative stress represents an imbalance between the production of free radicals and the biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage [42]. This has been reported as a major mechanism of lead induced toxicity [5] Lipid peroxidation, another indicator of oxidative stress occurs because of the action of reactive oxygen species ROS on lipid membranes.

Biochemical changes usually agree with distortions of the histoarchitecture of organs. In the present study with a rather short-term exposure of 4 weeks was not enough for obvious histopathological changes in the kidney. Histopathologically, kidney damage and renal impairment associated with lead poisoning is marked by proximal tubular nephropathy, glomerular sclerosis, and fibrosis in peritubular and interstitial lesions.

Conclusions

Taken together, *Costus afer* seems to have nephroprotective effect against lead induced damage in male albino rat model.

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Disclosure of conflict of interest

None.

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