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Original Research Article

AMELIORATIVE POTENTIAL OF ETHANOLIC CRUDE EXTRACT *OF Khaya senegalensis* ROOT (KERE) AGAINST CADMIUM-INDUCED TESTICULAR TOXICITY IN MALE ALBINO RATS

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ABSTRACT

Khaya senegalensis (Meliaceae) is a plant used in traditional medicine for its therapeutic value against several ailments. Cadmium is a heavy metal with toxic effects on vital organs such as the testes. This study aims to evaluate the ameliorative potential of K. *senegalensis* root extract (KERE) against cadmium-induced testicular toxicity in rodents. Twenty-four male albino rats with weights between 180-250 g were randomly allotted into four groups of six rats thus: Group one rats received 1ml 0.9% normal saline orally for 14 days; group two rats received 600 mg/kg KERE; group three rats received 2 mg/kg $Cacl_2$ subcutaneously; group four rats received 600 mg/kg KERE + 2 mg/kg $Cacl_2$. LD_{50} of KERE was determined by the OECD 428 limit test. Animals were fasted and sacrificed on the 15th day under anaesthesia. Blood samples were collected using hypodermic needles into EDTA and plain bottles for haematological and biochemical evaluation, respectively. The testes of the rats were eviscerated and processed for semen analysis and histopathological assessment. Hormone analysis was determined using specific Kits. Haematological results revealed significant derangement of FBC parameters in Cd-treated rats compared to normal controls. SOD, MDA, CAT and GPx showed depletion in Cd-treated rats. Semen analysis revealed decreased sperm count, distorted sperm and poor morphology in animals treated with cadmium alone. KERE+Cd group revealed improved semen morphology, testosterone, FSH, and histology significantly against the Cd only group. Pathological examination revealed congestion, inflammation, and abnormal cells of the testes of the Cd group animals. Results from this study indicate KERE ameliorated the toxic effects of cadmium on the testes of treated rats.

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INTRODUCTION

The recent proliferation of human activity and industrialization has given rise to contamination of the environment by heavy metals. These metals, often bivalent with high molecular weight, exhibit toxic potentials in living systems of aquatic life, green plants, and animals, including humans. Several natural and human activities such as soil erosion, mining, blasting of rocks, Industrial wastes, agriculture, weathering, and urbanization have resulted in the increased contamination and accumulation of heavy metals in biological systems [1]. Metals

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such as cadmium, arsenic, lead, mercury, and chromium are classified as toxicants by the IAA, and some have been seen to be carcinogenic [2]. Cadmium is a toxic heavy metal that has no specific physiological function in plants or animals. It is abundant in soil and rocks and can pollute biological systems due to industrial activities such as the manufacturing of lithium batteries and mining [3].

In biological systems, Cadmium is known to bioaccumulate in the tissues and results in increased apoptosis in cells, especially in renal and reproductive tissues such as testes, ovaries, and prostate. The mechanism of action remains unclear, but some researchers have suggested that increased proliferation of reactive oxygen species (ROS) and the displacement of essential metals such as calcium and manganese by Cd. Moreover, Cd reacts with enzymes and proteins to form metalloproteins, which disrupt the enzymatic activities of the mitochondria of cells, resulting in cell death [4,5]. In addition, epidemiological data suggest a direct correlation between Cd toxicity and the increase of certain cancers of the nasopharynx, breast, prostate, pancreas, and kidney [6,7,8].

When heavy metals accumulate in tissues, they are often difficult to excrete. The use of ion chelators and dialysis remains the only form of treatment for heavy metal intoxication. However, chelators have some drawbacks, such as redistribution of some heavy metals across other tissues, nephrotoxicity and hepatoxicity [9]. As a result, the search for natural products such as medicinal plants exhibiting good antioxidant properties and the capability to mitigate the adverse effects caused by heavy metals such as cadmium is receiving increased attention.

Khaya sengalensis M. (red mahogany) is a plant that grows predominantly in the southern part of Nigeria and is used by traditional medicine healers in treating a plethora of ailments such as fever, arthritis, infections, and wounds [10]. The bark and leaves from the tree have been known to possess anti-inflammatory, antipyretic, antinociceptive, and wound-healing properties. However, there is flimsy information about its use in the amelioration of toxicants such as heavy metals.

This present study assesses the ameliorative potential of ethanolic root extract of *K. senegalensis* (KERE) in cadmium-treated rats.

MATERIALS AND METHODS

Cadmium chloride was procured from Sigma Aldrich (USA), and ethanol was procured from Biofem Pharma China. All other chemicals, reagents, and kits used in this research work were of analytical grade.

Collection of plant material

K. senegalensis roots were harvested in February 2024 from a medicinal garden in Dadin Kowa, Gombe state. The plant was identified by Dr. Tayo I. Famojuro, a Taxonomist in the Department of Pharmacognosy, Faculty of Pharmaceutical Sciences, Bingham University, Karu, Nigeria. A sample of the identified plant was kept in the University Herbarium; the voucher number assigned to the specimen was BHUH-938122.

Preparation of plant extract

K. senegalensis roots were sliced into small pieces and shade-dried at room temperature for three weeks. The dried plant material was then weighed and pulverized into powder using a milling machine. Extraction was carried out by macerating 800 g of root powder into 1.5 L 70% aqueous ethanol in a ratio of 1:4 (root powder: solvent) for 72 h. The resultant extract was filtered using a muslin cloth followed by a Whatman filter paper, and concentrated using a rotary evaporator. The dry extract was weighed and stored in an airtight container, labelled, and kept in a refrigerator at 4°C until required.

Ethical Approval

Ethical approval was sought and received from the Bingham University Ethics Review Committee with certificate number BHU/FPS/EAC/01/2025. All experiments in this research work followed the ethical principles for the care and use of laboratory animals [11].

Experimental Animals

Twenty-four male albino Wistar rats, whose ages were 3-4 months, obtained from the animal house of Bingham University, were used for this study. The animals were housed in clean propylene cages at an ambient room temperature (25±2°C) and allowed to acclimatize for 2 weeks. They were fed with standard rodent pellets (Ultima Feeds, Nigeria) and allowed access to water *ad libitum*.

Experimental Design

Twenty-four male albino rats of ages 3-4 months were used for this study. The rats were randomly divided into four groups of six rats each (n = 6). Group one rats were administered 1ml 0.9% normal saline orally for 14 days. Group two rats were administered 600 mg/kg body weight KERE extract solution orally for 14 days. Group three rats were administered 2 mg/kg body weight cadmium chloride subcutaneously for 14 days. Group four rats were administered 600 mg/kg body weight KERE extract orally and 2 mg/kg cadmium chloride subcutaneously for 14 days.

Determination of Median Lethal Dose (LD_{50})

Oral median lethal dose of KERE was determined using OECD Test Guide 420 [12]. Three rats weighing 150-200 g were randomly selected and administered a maximum dose of 2000 mg/kg KERE orally. The animals were observed for signs of toxicity over the next 48 hours. The body weight of animals was observed on the initial day, day 7, and day 14, and the weights were measured using an analytical weighing balance.

Sample collection

On the 14th day of study, rats were fasted overnight. The animals were anaesthetized on day 15 and sacrificed. Blood samples were collected through cardiac puncture using a

syringe and needle into plain sample bottles and ethylene diamine tetraacetic acid (EDTA) bottles for hematological and biochemical analysis, respectively.

Hematological Analysis
The auto blood analyzer machine (Siemens-ADVIA 2120, Germany) was used for the analysis of full blood count and differentials (PCV, Hb, WBC, PLT, MCH, MCV, MCHC).

Determination of reproductive hormones
Reproductive hormones (Testosterone, Luteinizing hormone, and follicle-stimulating hormone) were determined using specialized ELISA kits (USA) according to the Manufacturer's instructions. These hormones were determined using the sera from the treated animals.

Sperm Analysis
Sperm count and motility of the rats were assessed according to [13,14]. A cut of about 2.0 mm in thickness was performed on the epididymis. The piece obtained by cutting was incubated in a water bath in 5.0ml of TALP (Tyrode s-Albumin-Lactate-Pyruvate) at 37 °C for 10 min.

Preparation of testis homogenate
From each rat, 100 µg of right testis was homogenized in 50 mM HCl at pH 7.4 containing KCl (1.15%) for preparing a 20% (*w*/*v* 1/5) testicular tissue homogenate using Potter–a Potter-Elvehjem homogenizer. The homogenates were centrifuged via a cold centrifuge at 4°C for 10 minutes at 10,000 g. The testicular supernatants were used to assess levels/activities of oxidants/antioxidants, pro- and anti-inflammatory cytokines, and steroidogenic enzymes/proteins [14].

Determination of oxidant and antioxidant markers
The activities of testicular catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) and malondialdehyde (MDA) were determined according to the method described by [15,16,17,18]. According to the kit manufacturer's instructions, the supernatant was used to determine antioxidant and oxidant markers.

Histology of Testes
The testes of the rats were surgically removed and fixed in 10% buffered formalin for 24 hours. The process of dehydration was achieved by soaking in ethanol three times, clearing in xylene, and embedding in molten paraffin, 3µm of the paraffin mass was cut into a section using a microtome (Surgcare Microtome, Model 335A, USA). Cut sections were deparaffinized and stained with hematoxylin and eosin (H&E) for the observation of histopathological changes.

Statistical Analysis
Results were expressed as mean ± standard error of mean (SEM). Statistical comparisons were performed using one-way ANOVA, followed by Dunnett's post hoc test. Data was analyzed using Statistical Package for Social Sciences Software (SPSS, version 20.0). A p-value less than 0.05 ($p<0.05$) was considered statistically significant.

RESULTS

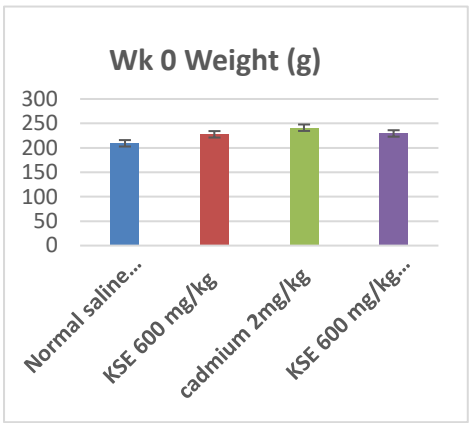
Oral Acute Toxicity
Table 1 shows the result of the acute toxicity test. From the study, the LD_{50} of KERE was estimated to be greater than 2000 mg/kg. Some signs of toxicity, such as excessive writhing, sedation, and tremors, were observed, but there was no mortality at a dose of 2000 mg/kg.

Effects of KERE on animal body weight
There was no significant difference in weight gain($p<0.05$) in the animals administered with KSE extract when compared with the control (Figures 1A-C). The cadmium group animals equally experienced an increase in body weight, however, at a lesser rate compared with the control group and the KERE group. All treated groups displayed a decline in weight from week two.

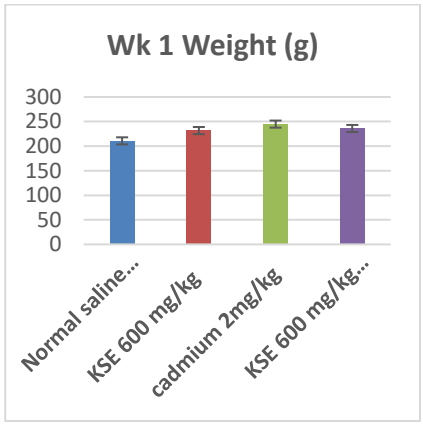
Effects of KERE on hematological parameters
There was a significant increase ($p<0.05$) in WBC count in animals treated with Cd alone compared to the normal control (Figures 2 A-F). WBC values for animals treated with KSE also showed a significant increase compared to the normal saline group. These values for WBC were higher for animals with Cadmium alone than those treated with Cd + KERE at 600 mg/kg. There was also a significant decrease($p<0.05$) in hemoglobin (Hb) values for animals treated with Cd alone compared with the control group and animals treated with KERE 600 mg/kg (Figure 2 A). Animals treated with Cd alone also showed a significant decrease in packed cell volume (PCV), hemoglobin concentration (Hb) and mean corpuscular volume (MCV), when compared to other treated groups and the control animals. There was no significant difference in PCV in animals treated with KERE 600 mg/kg and the control group. There was no significant difference in the platelet count ($p>0.05$) between KERE-administered rats and normal control (Figure 2D). However, there was significant decrease in platelet count in Cd administered rats compared with KERE alone and control animals. The KERE +Cd group showed improved values in platelet count compared with the Cd group alone. There was a significant difference ($p>0.05$) in Lymphocyte count in rats administered with 600 mg/kg KERE alone compared with normal control. There was also a significant decrease in lymphocyte count in the Cd group alone compared with other groups and the normal control.
MCH and MCV values were all increased significantly in KERE group animals alone compared to Cd animals only and normal control (Figure 2 F).

Table 1: Results of Acute Toxicity study using OECD Limit Test

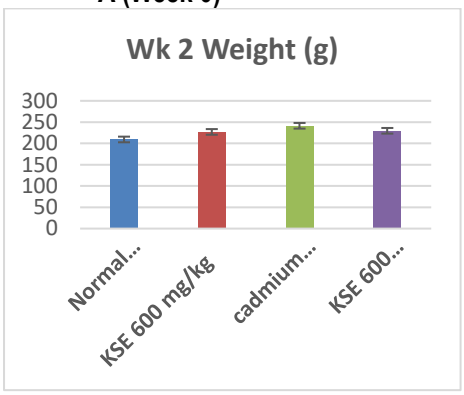
Parameter	Observation
Test Dose	2000 mg/kg KERE
Acute Toxic Signs	Tremors, sedation, writhing



A (Week 0)

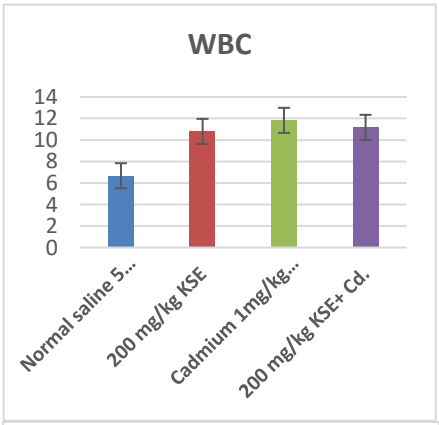


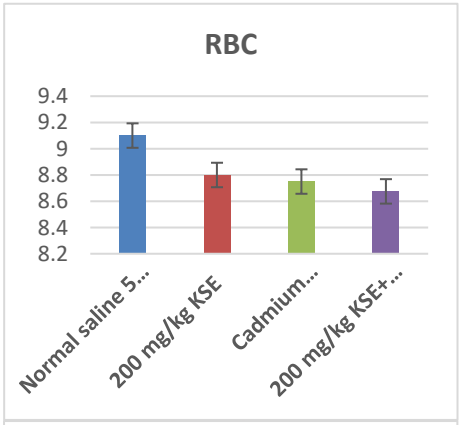
B (Week 1)

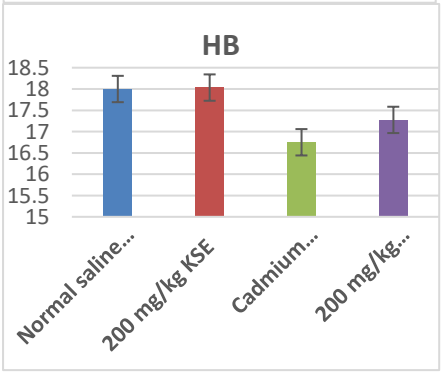


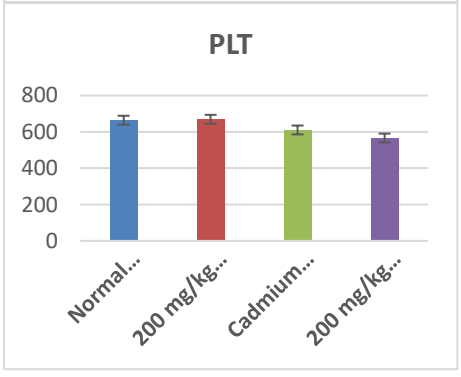
C (Week 2)

Figure 1 A-C: Weekly changes in animal body weight (g)

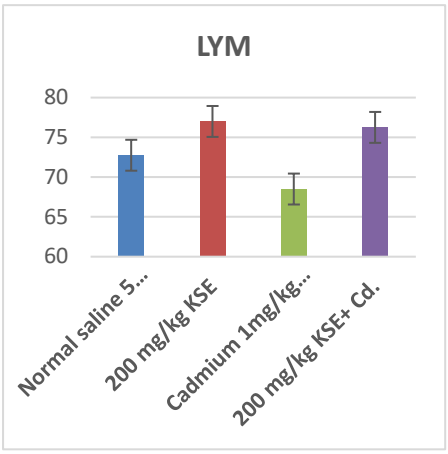




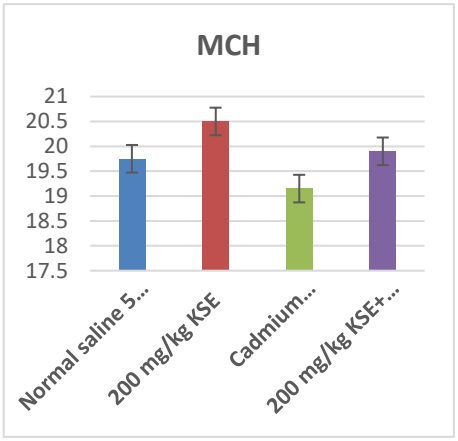




C (HB)



D (Platelets)



E (Lymphocytes)

F (MCH)

Figure 2: Effects of KERE on hematological parameters

Table 2: Sperm quality of animals supplemented with cadmium and *Khaya senegalensis* extract (n=5)

Parameters	Experimental test groups			
	Normal control	Cadmium only (3mg/kg)	KERE alone (600 mg/kg)	Cadmium + KERE
Sperm motility (% motile sperm)	72.24±2.45	[a]35.28±1.57	[a, b]79.13±2.04	61.79±1.87
Sperm count (x10^6 sperm/mL)	80.19±2.95	;p;'[b]31.06±5.29	82.41±1.87	[a]65.19±0.97
Sperm morphology (% normal cells)	85.56±5.35	48.36±3.48	[b]70.52±0.45	[a]64.24±1.42
Sperm viability (% viable sperm)	70.47±0.97	[b]45.49±3.04	[a, b]75.46±1.94	[a]62.33±4.27

Values expressed as mean± S.E.M with a, b as p<0.05 and p<0.001 respectively

Table 3: Effect of treatment on male sex hormone in rats

Groups	Testosterone (mg/ml)	Luteinizing hormone (mg/ml)	Follicle Stimulating Hormone (IU/L)
Normal control	5.25±3.21	3.42±0.97	3.2±2.95
Cadmium only (3mg/kg)	*0.26±1.89	*1.58±1.27	0.09±1.45
KERE (600 mg/kg)	*6.47±2.32	*2.36±2.07	4.21±0.74
Cadmium + KERE (600 mg/kg)	*3.80±4.74	*2.09±5.23	1.97±2.62

*Values are expressed as mean ± S.E.M Data compared with normal control, *p<0.05*

Table 4: Effects of treatment on Testicular antioxidant Enzymes

Groups	SOD (u/mg)	CAT (u/mg)	MDA (mmol/l)	GPx (u/mg)
Normal control	1.215±4.51	2.186±7.25	0.081±5.42	3.144±2.73
Cd 3mg/kg	*0.082±2.64	*1.059±2.97	*0.156±12.4	*1.048±8.95
KERE (600 mg/kg)	*1.261±5.27	1.014±15.26	*0.055±3.90	3.165±5.35
Cd+ KERE (600 mg/kg)	*0.425±8.32	*1.526±10.28	*0.102±3.8	1.922±4.34

*Values are expressed as mean ± S.E.M Data compared with normal control, *p<0.05*

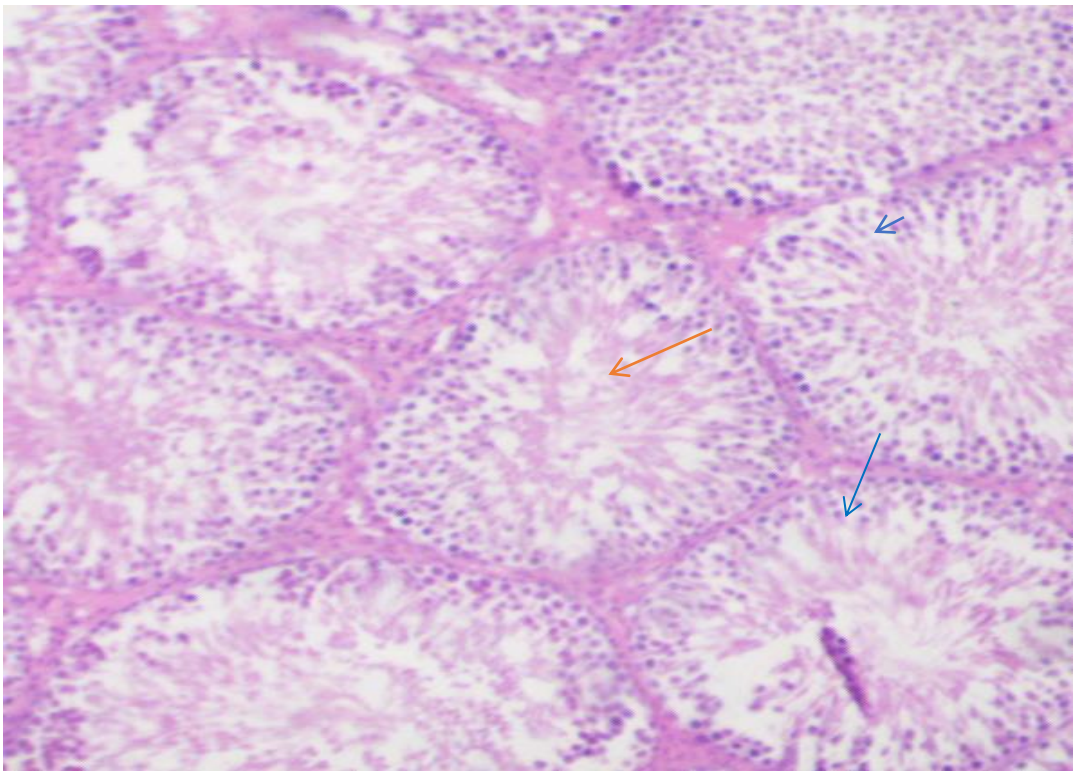


Figure 3 A: The testis of animals treated with 0.9% normal saline showed normal spermatocytes (blue arrow) and seminiferous tubule epithelial cells (red arrow). (H&E. X 400).

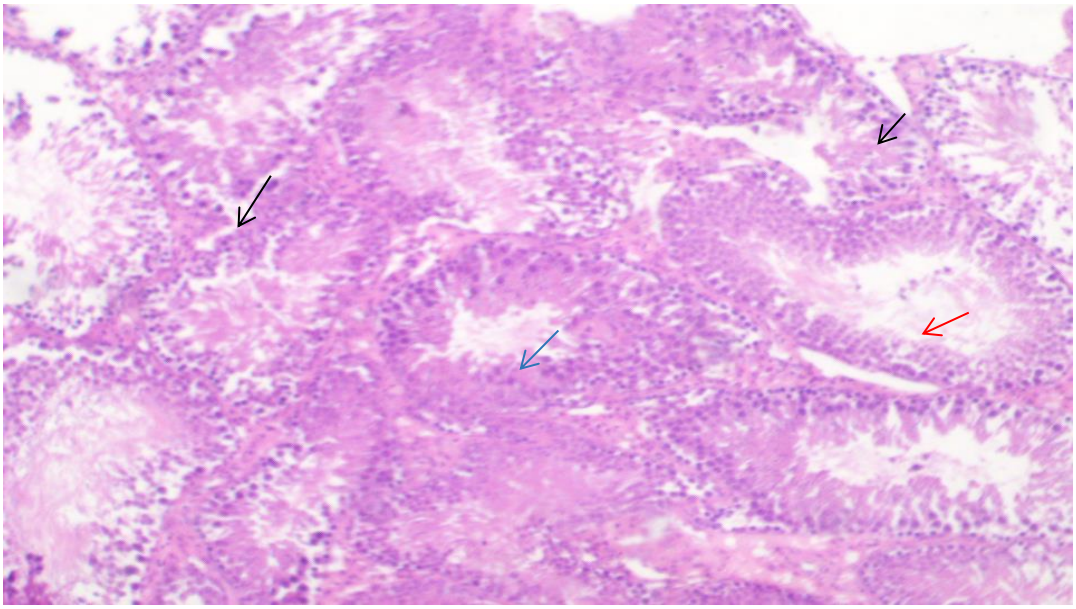


Figure 3 B: The testis of animals administered with 600 mg/kg extract showed well-preserved spermatids (black arrow) and seminiferous tubule epithelial (blue arrow) with a lumen (red arrow). (H&E. X 400).

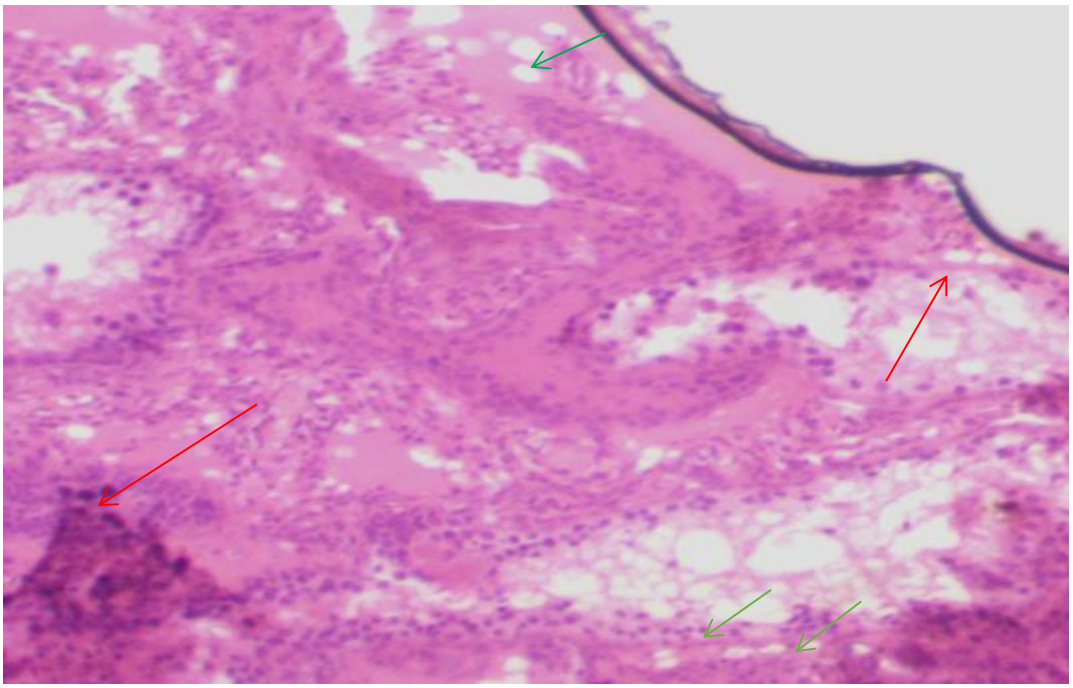


Figure 3 C: Testes of animals exposed to 3 mg/kg cadmium chloide exhibited reprotoxic effects by showing increased immature sperm cells (red arrow) and atrophy (degenerated) of the seminiferous tubules (green arrow). (H&E. X 400).

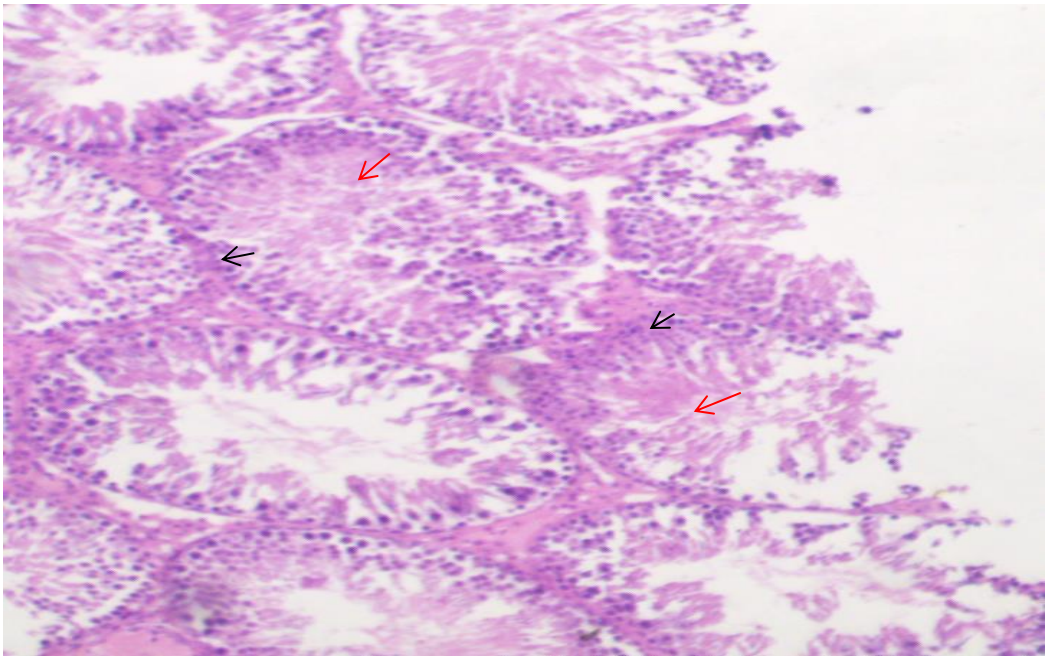


Figure 3 D: The testis of animals administered with 600 mg/kg KERE+ Cd showed healthy spermatids (black arrow) and vacuolated seminiferous tubule epithelial (red arrow). (H&E. X 400).

Sperm analysis

Table 2 summarizes sperm motility, count, morphology, and viability of rats treated with KERE and cadmium, or both. There was a significant increase in sperm count ($p<0.05$) between animals treated with 600 mg/kg KERE alone, when compared with normal control. This was also the case for sperm morphology and sperm viability. There was a significant decrease in sperm morphology and viability ($p<0.05$) between animals treated with Cd alone when compared with normal control and animals treated with Cd+600 mg KERE. Moreover, there was also a significant increase in all three parameters in animals treated with KERE compared with those treated with the Cd + KERE group.

Effects of KERE and Cadmium on male hormones of treated rats

Table 3 shows the results of the treated rats on the male hormone testosterone (T) and the luteinizing hormone (LH). There was a significant decrease ($p<0.05$) in testosterone and LH hormone of animals treated with Cd alone when compared

with normal control. There was also a significant ($p<0.05$) increase in testosterone, FSH, and LH hormones in animals treated with 600 mg/kg KERE alone when compared with normal control and Cd-treated rats. Table 3. also showed a significant increase in animals treated with 600 mg KERE+ Cd when compared with those treated with 3 mg/kg Cd alone.

Effects of KERE and Cadmium on Antioxidant Enzymes of treated rats

Table 4 summarizes the results of treated rats on antioxidant enzymes such as catalase (CAT), superoxide dismutase (SOD), Malondialdehyde (MDA) and Glutathione (GPx). There was significant depletion of all antioxidant enzymes in rats treated with 3 mg/kg Cd alone when compared with normal control. However, there was increased antioxidant enzymes in male rats treated with only KERE (600 mg/kg) compared with control. There was also significant ($p<0.05$) amelioration of antioxidant enzymes in rats treated with both the toxicant, Cd and KERE.

Histopathological Results

Histopathological assessment of the testes of treated animals is presented in Figures 3A-D.

DISCUSSION

The rising exposure of the human population to heavy metals has necessitated a need to search for more natural, eco-friendly molecules to be harnessed as chelators in ameliorating their toxic effects. Medicinal plants have shown great potential owing to their availability, reduced economic cost, and devoid of serious toxicity [19,20,21]. This present study was designed to assess the ameliorative potential of *Khaya senegalensis* ethanolic crude extract (KERE) on the testes of Cadmium-treated rats.

Hematological indices are used as good indicators of toxicity in vertebrates. Heavy metals such as Cadmium, arsenic, lead, and chromium have a potentially devastating impact on hematological parameters such as packed cell volume, hemoglobin concentration, and white cell count [23,24]. In this present study, there was a marked increase in WBC count in rats treated with 3 mg/kg Cd compared with the normal control group of animals (Figure1 A). This increase can be termed leukocytosis. Some researchers have found this to be a result of heavy metal toxicity [25, 26]. This proliferation of white blood cells was not seen in rats treated with 600 mg/kg KERE. However, there was also an increase in WBCs in the KERE+ Cd group, but this increase was not deemed to be significant. This result is similar to [28], who attribute an increase in WBC count as a physiologic response of the body to fighting infection or as a result of proliferation of inflammatory markers such as leukotrienes, tissue necrotic factor-alpha (tnf-α), interleukins-2,6, and prostaglandins. Platelets were also seen to be significantly raised in rats treated with Cd alone when compared with normal control rats. This proliferation in platelet count may be suggestive of the effects of Cd on the bone marrow of treated rats (Figure 1D). Platelets are responsible for the normal formation of clots and maintenance of internal homeostasis, but when increased significantly, they could pose a risk to clot blockage and embolism within the circulatory system [29, 30]. PCV, Hb concentration, and MCH for animals treated with cadmium alone decreased significantly when compared with animals treated with 600 mg/kg KERE (Figure1A-F), suggesting that Cd depressed the hematopoietic system while KERE had a stimulatory effect. This stimulatory effect may be a result of the abundance of phytochemicals and secondary plant metabolites such as flavonoids, vitamins, sterols, polyphenols, and carotenoids. These phytochemicals have been suggested by many researchers to stimulate red blood cell production and also reduce oxidative stress by the reduction of reactive oxygen species (ROS) in vivo [31, 32, 33]. Variations in the weight of animals are an important parameter and a good indicator for toxicity [34, 35]. Weight loss in organisms is a positive indicator of physiological derangement and can be caused by the generation of free radicals, a mechanism seen in most heavy metals such as lead, mercury, and arsenic [36, 37]. In this study, there was a significant increase in the weight of all treated animals in the first two weeks. This trend was also true for animals treated with 3 mg/kg Cd (Figure 2A-C). However, the rate of weight gain was less when compared with rats from the control and KERE groups. All animal groups showed a significant drop in mean body weight during the last week of the study. This insignificant drop may be a result of external factors the animals were exposed to, such as changes in temperature, feed ration, and humidity, and not necessarily as a result of administration of KERE nor exposure to cadmium. Although Cd has been established to interfere with certain physiologic functions that may affect the overall health of rodents, results from this study are not sufficient to ascertain if Cd drastically affected the overall weight gain of the animals, as there was a reduction in weight across all treated groups.

Cd has been established to affect spermatogenesis and is a known toxicant. The mechanism of action by which Cd affects sperm cells and overall quality is not clear, but past works by some researchers indicate increased production of ROS and activation of proinflammatory factors by binding to certain tissues and bioaccumulation within the cells. In this present study, there was a significant decline in sperm count, morphology, and quality in rats treated with 3mg/kg Cd compared with the normal control group (Table 2). However, this decline was attenuated in animals who received both Cd and 600 mg/kg KERE. This finding is in agreement with [38], who concluded that the toxic effects of heavy metals may be countered by medicinal plants. Phytochemical screening carried out on roots of KERE revealed rich presence of phytochemicals such as flavonoids, alkaloids, saponins, sterols, and tannins [39]. These bioactive compounds have been shown by research to be rich in antioxidants, which help reduce tissue peroxidation and promote overall health. In the present, the LD_{50} of an orally administered dose of KERE was determined to be greater than 2000 mg/kg (Table 1). This dose indicates that the plant is relatively nontoxic [40]. The finding is

at variance with [39], who estimated the median lethal dose of aqueous root extract of *K. senegalensis* to be 1280 mg/kg. [41] and [42] also reported zero toxicity following short-term oral administration of stem bark and leaf extracts of *K. senegalensis*. It should be noted, however, that the LD_{50} of plant extracts is not an absolute value, and these values may vary depending on the methods employed. Also, these disparities may be a result of the absence or presence of some secondary metabolites found in the plant from various parts of the world. This may be due to soil composition, climate, and other environmental factors.

Cadmium exposure leads to testicular oxidative stress by the upregulation of the generation of reactive oxidative species (ROS) and the down regulation of antioxidants, leading to increased oxidative stress and apoptosis [43, 44, 45]. In the present study, animals treated with $CaCl_2$ showed increased MDA production and depleted superoxide dismutase and catalase enzymes when compared with the control group (Table 4). Cadmium chloride has been observed to cause this action via two mechanisms; generation of ROS by accumulation of these species in testicular tissues and the suppression of the sulfhydryl groups and thiol groups by testicular SOD, GPx and CAT. It can replace the divalent bio-elements such as selenium, zinc, and magnesium that serve as an essential cofactor of antioxidant enzymes that causing a significant depletion of these antioxidant enzymes in testicular post-mitochondrial and mitochondrial fractions in rats exposed to cadmium chloride. Interestingly, animals treated with both cadmium and KERE showed a downregulation of MDA and increased SOD, GPx and CAT antioxidant enzymes when compared with animals treated with cadmium alone. This mitigation in MDA production was a result of the presence of antioxidants such as flavonoids present in KERE. The results are consistent with [46] who reported the downregulation of ROS generation in lead acetate testicular toxicity in rats using Kaempferol and Vitamin E, a known antioxidant. Moreover, Cadmium toxicity leads to testicular cellular damage, increased production of immature sperm cells, and disruption of spermatogenesis within the testes. This is as a result of increased lipid peroxidation and the displacement of Ca^{2+} from the cells, resulting in the complete arrest of spermatogenesis. The current histological analysis of rats treated with Cd only displayed cellular damage of the basement membrane, irregular thickening of interstitial and Sertoli cells, and increased production of immature cells (Figure 3C). These alterations were mitigated as shown in the histology micrographs of rats treated with 600 mg/kg KERE and Cd group, indicating testicular protection (Figure 3 B). The results are in agreement with [47] who displayed testicular protection in rats using Vitamin E. Similarly,[48] reported amelioration of testes of male rats treated with naringenin, a flavonoid found in grapefruit extract. In the current study, there was depletion in testosterone, the primary male hormone, in cadmium-treated animals. In addition, there was a depletion of FSH and LH hormones in the same group compared with control animals. Interestingly, there were increased hormonal values for rats treated with Cd and 600 mg/kg KERE, indicating a mitigation of toxic effects on the seminiferous tubules. These results are in agreement with the findings of [49] who reported the protective effects of quercetin in cadmium testicular toxicity in rodents. Quercetin, a secondary metabolite found in medicinal plants, increased testosterone, FSH, and LH levels in treated male rats.

CONCLUSION

From the results reported in this present study, KERE at 600 mg/kg shows good protective effects against the deleterious effects of $CaCl_2$-treated animals via its antioxidant mechanisms. Moreover, the toxic effects of Cd in the male reproductive system were seen in animals treated with Cd alone in terms of decreased sperm count, increased production of immature sperm cells, and this was confirmed in the histopathological results. In addition, KERE-treated rats exhibited improved sperm count, morphology, and quality, suggesting that it could possess aphrodisiac activity. Further research is required to ascertain the specific secondary metabolite responsible for this activity for possible isolation and purification.

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AUTHORS' CONTRIBUTION

This work was carried out in collaboration among all authors. The author UPI designed the research work, wrote the protocol, and the first draft of the manuscript. Authors DPK and AAD reviewed and vetted the first draft. TIF managed the literature searches, while author UPI effected corrections to the first draft. WOU performed the statistical analysis. DPK eviscerated the animal tissues from all the euthanized experimental rats. All authors made significant financial contributions as well as read and approved the final manuscript.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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None

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