Methanolic Leaf Extract of Dracaena trifasciata and Sub-Acute Renal Tubular Cell Death in Wistar Rats

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Abstract

Dracaena trifasciata (formerly *Sansevieria trifasciata*) is widely utilized in traditional medicine across West Africa and other tropical regions for managing various ailments. However, comprehensive safety evaluations regarding its systemic toxicity, particularly renal toxicity, remain insufficient. This study evaluated the quantitative phytochemical profile, acute oral toxicity, and sub-acute toxicological impact of the methanolic leaf extract of *Dracaena trifasciata* (MEDTL) on renal function and histoarchitecture in Wistar rats. Phytochemical screening was performed using standard qualitative and spectrophotometric quantitative protocols. For the acute toxicity study (LD_{50}), Lorke's method was employed using Wistar rats up to a dose of 5 000 mg/kg body weight. In the sub-acute study following OECD Guideline 407, 20 Wistar rats were divided into four groups (n = 5): Control (distilled water 10 mL/kg), 250 mg/kg, 500 mg/kg, and 1 000 mg/kg MEDTL administered orally once daily for 28 consecutive days. On day 29, blood samples were collected for renal biomarkers (creatinine, urea, Na^+, $\mathbf{K^+}$, Cl^-, HCO_3^-), and kidneys were processed for light microscopy using Hematoxylin & Eosin (H&E) and Periodic Acid-Schiff (PAS) staining. Acute toxicity testing (LD_{50}) demonstrated no mortality or overt signs of severe toxicity up to 5 000 mg/kg. In the sub-acute study, MEDTL induced significant dose-dependent renal impairment. Serum creatinine increased significantly ($p < 0.05$) from 88.33 (SD 4.41) µmol/L in controls to 118.33 (SD 3.76) µmol/L (250 mg/kg), 130.00 (SD 10.41) µmol/L (500 mg/kg), and 158.33 (SD 2.03) µmol/L (1 000 mg/kg). Serum urea was significantly elevated at 1 000 mg/kg (7.73 [SD 0.26] mg/dL vs. control 4.17 [SD 0.20] mg/dL; $p < 0.05$). Electrolyte alterations included significant hyponatremia at 250 mg/kg (121.67 [SD 2.03] \ mmol/L) and hyperbicarbonatemia at 1 000 mg/kg (27.67 [SD 1.45] mmol/L). Renal histopathology (H&E) revealed progressive cortical and medullary injury, characterized by glomerular hypertrophy, mesangial hyperplasia, tubular necrosis, epithelial sloughing, and hyaline cast formation. PAS staining demonstrated basement membrane thinning and loss of proximal tubular glycocalyx brush border integrity at higher doses. Caution is necessary during ethnopharmacological use

Introduction

Herbal medicines continue to serve as a primary healthcare resource for millions worldwide, particularly in developing regions where traditional practices are deeply rooted [4, 19]. *Dracaena trifasciata* (family Asparagaceae), formerly classified as *Sansevieria trifasciata*, is widely distributed across tropical regions including West Africa [3, 20]. Modern molecular phylogenetic analyses using plastid and nuclear DNA sequence data have definitively confirmed that *Sansevieria* constitutes a herbaceous clade nested within *Dracaena* [3]. Traditionally, decoctions and macerations of its leaves are used to manage inflammation, bacterial infections, respiratory illnesses, and hypertension [1, 2, 21].

Despite widespread therapeutic usage, the safety profiles of many herbal remedies are inadequately investigated, leading to potential organ toxicities following prolonged or high-dose administration [5, 12, 18]. The kidney is particularly vulnerable to xenobiotic-induced toxicity due to its high blood flow, metabolic capacity, and role in concentrating urinary solutes [15, 22]. Secondary metabolites such as

steroidal saponins, pregnane glycosides, and alkaloids—while possessing bioactivity—can exert cytotoxic effects on nephrons if accumulated in renal tissues over extended periods [23–25].

While previous preliminary screens have reported beneficial pharmacological activities, safety data regarding repeated exposure to *D. trifasciata* leaf extracts remain limited [17, 26]. Therefore, this study evaluated the quantitative phytochemical profile, acute safety threshold, and 28-day sub-acute nephrotoxic potential of the methanolic leaf extract of *D. trifasciata* in Wistar rats.

Methods

Plant Material Collection and Extraction

Fresh leaves of *Dracaena trifasciata* were collected from the Air Force area, Obio/Akpor Local Government Area, Port Harcourt, Rivers State, Nigeria. The plant material was identified and authenticated at the Herbarium of the Department of Plant Science and Biotechnology, University of Port Harcourt (Voucher Specimen No: UPH/P/567).

The leaves were washed thoroughly with distilled water, chopped, and air-dried to a constant weight. The dried leaves were ground into fine powder. Extraction was performed via maceration as described by Dewatisari et al. [20]: 500 g of powdered material was soaked in 70% methanol (1:3 w/v) for 3 x 24 hours with daily intermittent agitation [20]. The mixture was filtered through Whatman No. 1 filter paper and double-layered muslin cloth. The filtrate was concentrated at 45°C using a rotary evaporator and dried over a water bath. The resulting methanolic extract of *D. trifasciata* leaves (MEDTL) was stored in sealed glass containers wrapped in aluminum foil at 4°C until use.

Phytochemical Screening

Qualitative phytochemical analysis of MEDTL was conducted according to standard methods [6-9] to detect alkaloids, carbohydrates, flavonoids, saponins, anthraquinones, terpenoids, cardiac glycosides, phenols, fixed oils, phlobatannins, and cyanogenic glycosides.

Quantitative estimations were carried out as follows:

- **Total Phenolic Content:** Measured using the Folin-Ciocalteu reagent method at 765 nm and expressed as gallic acid equivalents (mg GAE/g) (ref. 27).
- **Total Flavonoid Content:** Assayed via the aluminum chloride colorimetric method at 420 nm and expressed as quercetin equivalents (mg QE/g) (ref. 27).
- **Total Alkaloid Content:** Determined gravimetrically using 20% acetic acid in ethanol and precipitation with ammonium hydroxide [6, 9].
- **Total Tannin Content:** Quantified spectrophotometrically using the ferric chloride-ferrocyanide reaction against a tannic acid standard curve [7].

- **Total Saponin Content:** Extracted using 20% ethanol, partition-purified with diethyl ether and n-butanol, evaporated, and weighed gravimetrically [9, 23].

Experimental Animals

Thirty-two (32) adult healthy Wistar rats (150--200 g) were obtained from the Animal House, Faculty of Basic Medical Sciences, University of Port Harcourt. The animals were housed in transparent polyacrylic cages lined with sterile wood shavings under standard environmental conditions (12-h light/dark cycle, room temperature). Rats had free access to standard rodent pelleted chow and drinking water *ad libitum*. All experimental procedures were approved by the Research Ethics Committee of the University of Port Harcourt (Ethical Reference: **UPH/CEREMAD/REC/MM111/131**).

Acute Toxicity Study (LD_{50})

The median lethal dose (LD_{50}) was evaluated using Lorke's method [10] and complementary fixed-dose screening approaches [13, 28].

- **Phase I:** Nine (9) rats were randomized into three groups (n = 3) and received single oral doses of MEDTL at 10, 100, and 1 000 mg/kg body weight, respectively [10].
- **Phase II:** Based on Phase I findings, three (3) additional rats were administered single oral doses of 1 600, 2 900, and 5 000 mg/kg body weight (n = 1 per dose) [10].

Animals were monitored continuously for the first 4 hours post-dosing, daily for 24 hours, and up to 14 days for clinical signs of toxicity, behavioral changes, feeding habits, and mortality [10, 29].

Sub-Acute Toxicity Study Design

Following OECD Guideline 407 for repeated dose 28-day oral toxicity testing in rodents (ref. 11), twenty (20) Wistar rats were divided randomly into four experimental groups (n = 5 per group) and treated orally via gavage once daily for 28 consecutive days [11, 12]:

- **Group I (Control):** Received distilled water (10 mL/kg/day).
- **Group II:** Received MEDTL (250 mg/kg/day).
- **Group III:** Received MEDTL (500 mg/kg/day).
- **Group IV:** Received MEDTL (1 000 mg/kg/day).

Blood and Organ Collection

On Day 29, following an overnight fast, rats were euthanized under anesthesia. Abdominal laparotomy was performed to expose internal organs. Blood samples were collected via cardiac puncture using 5

mL syringes. Blood for biochemical analysis was transferred into heparinized tubes and centrifuged at 3 000 rpm for 15 minutes at room temperature to obtain plasma/serum, which was stored at -80°C. The kidneys were harvested, weighed, and immediately fixed in 10% neutral buffered formalin for histological evaluation [16].

Renal Biochemical Assays

- **Urea:** Quantified using the Urease-Berthelot enzymatic method; absorbance was measured at 540 nm (ref. 14).
- **Creatinine:** Measured via Jaffe's alkaline picrate reaction at 520 nm (ref. 14, ref. 30).
- **Electrolytes (Na^+, K^+):** Quantified using flame photometry at 588 nm (Na^+) and 766 nm (K^+) (ref. 14).
- **Chloride (Cl^-) and Bicarbonate (HCO_3^-):** Determined via Mohr titration/colorimetric methods and back-titration according to standard protocols [14].

Histopathological Processing and Staining

Fixed renal tissues were processed following standard techniques: dehydrated through graded alcohols (75\%--100\%), cleared in xylene, and embedded in paraffin wax [16]. Microtome sections (5µm) were cut, mounted on albumin-coated glass slides, and stained using:

- **Hematoxylin and Eosin (H&E):** To assess general microarchitecture, cortical glomeruli, tubules, and medullary structures [16].
- **Periodic Acid-Schiff (PAS):** To evaluate basement membrane structural integrity, tubular brush border glycocalyx, and extracellular matrix changes [16].

Photomicrographs were acquired using an optical microscope fitted with a digital camera at x400 magnification.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics (Version 28.0; IBM Corp., Armonk, NY, USA). Results are expressed as Mean \pm Standard Error of Mean (SEM). Differences between groups were assessed using One-Way Analysis of Variance (ANOVA) followed by Dunnett's post-hoc test. Values of $p < 0.05$ were considered statistically significant.

Results

Acute Oral Toxicity (LD_{50})

Oral administration of MEDTL up to 5 000 mg/kg produced no mortality, severe clinical signs of toxicity, or obvious behavioral changes in rats over the 14-day observation period. The median oral lethal dose (LD_{50}) of MEDTL was therefore estimated to be greater than 5 000 mg/kg, classifying the extract as practically non-toxic upon acute oral exposure.

Sub-Acute Effects on Renal Function Markers

Administration of MEDTL for 28 days induced dose-dependent impairment of renal biochemical markers, as summarized in Table 1.

Table 1: Effects of 28-day oral administration of MEDTL on serum renal function markers in Wistar rats

Group	K^+ (mmol/L)	Na^+ (mmol/L)	Urea (mg/dL)	Creatinine (µmol/L)	Cl− (mmol/L)	HCO3− (mmol/L)
Control	4.47 (0.45)	142.67 (5.81)	4.17 (0.45)	88.33 (9.86)	75.00 (11.83)	23.00 (2.59)
250 mg/kg	3.50 (0.38)	121.67 (4.54)*	5.73 (0.34)	118.33 (8.41)*	57.00 (4.65)*	23.67 (3.24)
500 mg/kg	3.83 (0.54)	128.33 (5.37)*	6.30 (1.43)*	130.00 (23.28)*	63.00 (13.48)*	26.00 (3.42)
1 000 mg/kg	4.43 (0.51)	144.33 (9.17)	7.73 (0.58)*	158.33 (4.54)*	75.33 (18.81)	27.67 (3.24)*

*Data are expressed as **Mean (SD)** (n = 5 rats/group).*

**P < 0.05 compared with the control group (One-way ANOVA followed by Dunnett's post-hoc test).*

MEDTL: Methanolic extract of Dracaena trifasciata leaves.

- **Serum Creatinine:** Elevated significantly across all treated groups in a dose-dependent manner (118.33 [SD 8.41] µmol/l at 250 mg/kg, 130.00 [SD 23.28] µmol/l at 500 mg/kg, and 158.33 [SD 4.54] µmol/l at 1 000 mg/kg) compared with control (88.33 [SD 9.86] µmol/l; P < 0.05).
- **Serum Urea:** Showed a statistically significant increase in the 500 mg/kg group (6.30 [SD 1.43] mg/dl) and peaked at 1 000 mg/kg (7.73 [SD 0.58] mg/dl) compared with control (4.17 [SD 0.45] mg/dl; P < 0.05).
- **Electrolytes:** Sodium (Na+) levels decreased significantly at 250 mg/kg (121.67 [SD 4.54] mmol/l) and 500 mg/kg (128.33 [SD 5.37] mmol/l) compared with control (142.67 [SD 5.81] mmol/l; P < 0.05), before normalizing at 1 000 mg/kg (144.33 [SD 9.17] mmol/l). Chloride (Cl-) followed a

similar pattern. Bicarbonate (HCO_3-) was significantly elevated at 1 000 mg/kg (27.67 [SD 3.24] mmol/l) compared with control (23.00 [SD 2.59] mmol/l; P < 0.05).

Renal Histopathological Findings

H&E Staining: Cortex and Medulla (Figure 1 & Figure 2)

- **Control:** Showed normal renal cortical architecture with intact glomerular tufts, well-defined Bowman's spaces, and normal proximal/distal convoluted tubules. The medulla showed healthy collecting ducts and preserved interstitium.
- **250 mg/kg MEDTL:** Exhibited early alterations, including mild glomerular hypertrophy, mesangial cell hyperplasia, focal cortical tubular necrosis, medullary tubular ectasia, and mild epithelial sloughing.
- **500 mg/kg MEDTL:** Displayed increased tissue injury with occluded Bowman's spaces, extensive cortical tubular necrosis, tubular atrophy in the medulla, and widespread epithelial sloughing.
- **1 000 mg/kg MEDTL:** Demonstrated severe diffuse nephropathy. Cortical sections showed marked diffuse vascular congestion, extensive interstitial inflammatory cell infiltration, acute tubular necrosis (ATN), and collapse of glomerular capillaries. Medullary sections revealed severe pathology, including intraluminal hyaline cast formation, interstitial hemorrhage, and widespread tubular degeneration.

PAS Staining: Basement Membrane and Brush Border Integrity (Figures 3 & 4)

- **Control:** Demonstrated clear, intact basement membranes around glomerular capillaries and renal tubules, with a distinct, uniform apical glycocalyx brush border in proximal tubules.
- **250 mg/kg MEDTL:** Preserved relatively normal brush borders, but displayed localized thinning of cortical and medullary basement membranes.
- **500 & 1 000 mg/kg MEDTL:** Showed progressive loss of structural integrity. Both glomerular and tubular basement membranes were diffusely thinned and fragmented, accompanied by atypical, disrupted, or absent brush borders in proximal tubules.

Discussion

Although acute toxicity testing classified the methanolic leaf extract of *Dracaena trifasciata* as non-toxic (LD_{50} > 5 000 mg/kg), consistent with findings in similar plant toxicity screens [1, 10, 17, 32], sub-acute evaluation following OECD guidelines revealed significant dose-dependent nephrotoxicity over 28 days (ref. 11, ref. 12, ref. 18).

Elevations in serum creatinine and urea serve as primary clinical indicators of diminished glomerular filtration rate (GFR) and excretory dysfunction(ref. 14, ref. 15, ref. 30, ref. 31). Creatinine, a metabolic byproduct of muscle breakdown, is freely filtered by the glomerulus and not reabsorbed; thus, a progressive dose-dependent increase in serum creatinine directly reflects compromised glomerular

filtration capacity [14, 15]. Similarly, the marked elevation of serum urea in the higher dose groups corroborates impaired nitrogenous waste clearance [14, 22].

Electrolyte disruptions further demonstrate structural and functional damage to renal tubules [14, 15, 22]. The initial drop in sodium and chloride levels at 250 mg/kg and 500 mg/kg suggests impaired tubular reabsorptive mechanisms in the proximal convoluted tubule and loop of Henle [15, 25]. Furthermore, significant retention of bicarbonate at 1 000 mg/kg points to altered acid-base regulation, often secondary to impaired tubular secretion or systemic metabolic shifts caused by xenobiotic-induced renal insult [14, 31].

Histopathological evaluation provided clear anatomical evidence confirming the biochemical findings [12, 18]. Cortical H&E sections demonstrated dose-dependent glomerular hypertrophy, Bowman's space occlusion, acute tubular necrosis (ATN), and inflammatory cell infiltration [12, 18, 31]. These structural changes match features of xenobiotic-induced acute glomerulonephritis and tubular injury reported in systemic toxicity studies of herbal extracts [1, 12, 18, 33]. The medullary pathology—specifically tubular ectasia, epithelial sloughing, and hyaline cast formation—reflects protein leakage and severe disruption of urinary concentration mechanisms within the loop of Henle and collecting ducts [18, 22]. Hyaline casts form when filtered proteins precipitate with Tamm-Horsfall mucoprotein in concentrated tubular fluid, a hallmark of tubular damage and glomerular barrier breach [22].

PAS staining highlighted the sub-cellular targets of MEDTL toxicity. The progressive thinning of basement membranes and loss of the apical glycocalyx brush border in proximal tubules indicate damage to structural extracellular matrix proteins and loss of surface area for reabsorption. The proximal tubular brush border is essential for selective reabsorption of solutes, electrolytes, and low-molecular-weight proteins. Destruction of this brush border compromises tubular transport mechanics, exacerbating solute loss and secondary necrosis [15, 25, 34].

The nephrotoxic mechanisms of MEDTL are likely linked to high concentrations of secondary metabolites, particularly steroidal saponins (such as trifasciatosides A–J), pregnane glycosides, and homoisoflavonoids present in *D. trifasciata* leaves [23, 24, 35, 36]. While low levels of phenolics and flavonoids can exert antioxidant effects [19, 27], high cumulative doses of steroidal saponins can cause direct plasma membrane disruption, cellular cytotoxicity, oxidative stress, and mitochondrial dysfunction in renal tubular epithelial cells [25, 34].

While the methanolic leaf extract of *Dracaena trifasciata* exhibits a high oral safety threshold under acute single-dose conditions (LD_{50} > 5 000 mg/kg), repeated daily exposure over 28 days causes significant dose-dependent nephrotoxicity in Wistar rats. This toxicity is characterized by biochemical nitrogenous waste retention, electrolyte imbalance, glomerular damage, tubular necrosis, hyaline cast deposition, and structural breakdown of basement membranes and brush border glycocalyx. Consequently, prolonged or unmonitored use of high-dose *D. trifasciata* preparations in traditional medicine carries potential risks of renal impairment.

Declarations

Ethics Approval

Ethical approval for the care and use of laboratory animals was granted by the Research Ethics Committee of the University of Port Harcourt, Rivers State, Nigeria (Reference: **UPH/CEREMAD/REC/MM111/131**).

Consent for Publication

Not applicable.

Availability of Data and Materials

All data generated or analyzed during this study are included in this published article.

Competing Interests

The authors declare that they have no competing interests.

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Authors' contributions:

- **Davies, S.I:** Conceptualization, methodology, manuscript writing.
- **Nwachukwu D.C.:** Formal statistical analysis, critical review, and editing.
- **Kagbo H.D..:** Laboratory investigation, biochemical assays, histological processing.

All authors read and approved the final manuscript.

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Figures

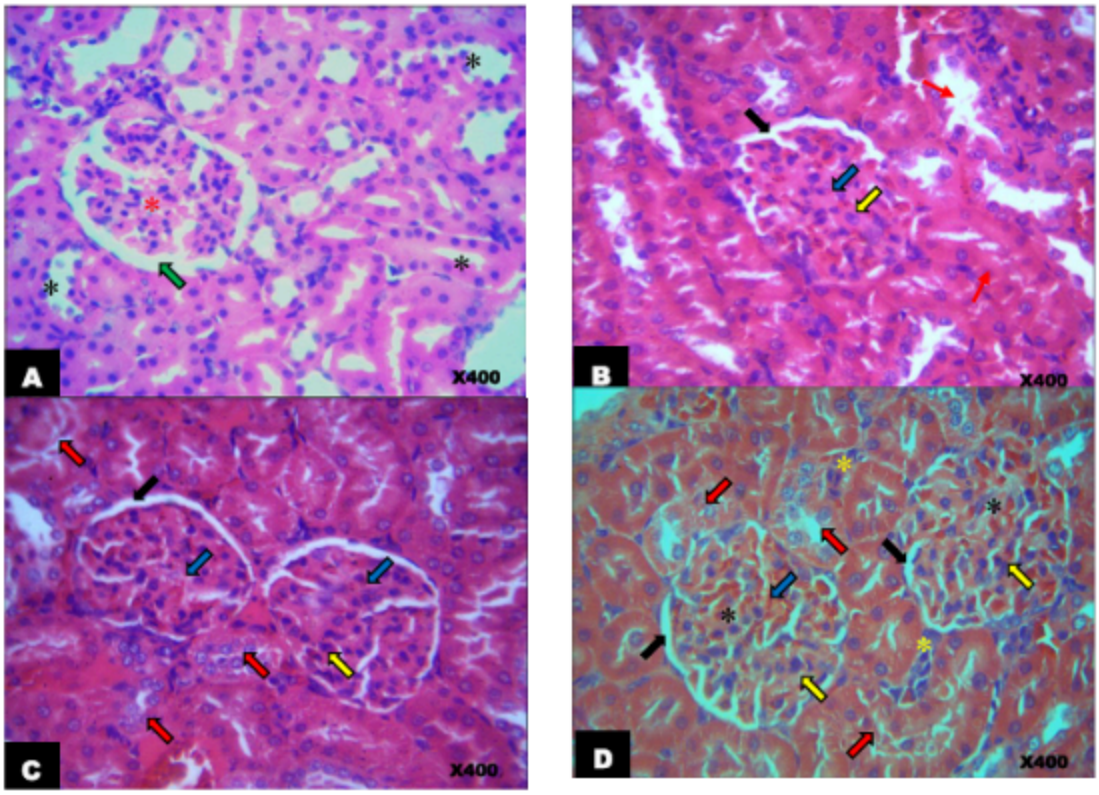

Figure 1

Histopathological alterations in renal cortical tissue following administration of *Dracaena trifasciata* extract (H&E, x400).

Photomicrograph sections of renal cortex across experimental groups:

(A) Control: Demonstrates normal renal corpuscular architecture with a intact glomerular tuft (**red asterisk**), urinary Bowman's space (**green arrow**), and surrounding normal renal tubules (**black asterisk**).

(B) *Dracaena trifasciata* (DT) 250 mg/kg b.w.: Displays glomerular tuft hypertrophy (**blue arrow**), mesangial cell hypertrophy (**yellow arrow**), Bowman's space occlusion (**black arrow**), and focal mild tubular epithelial necrosis (**thin red arrow**).

(C) DT 500 mg/kg b.w.: Shows marked glomerular hypertrophy (**blue arrow**), mesangial expansion (**yellow arrow**), Bowman's space occlusion (**black arrow**), and severe tubular cell necrosis (**thick red arrow**).

(D) DT 1 000 mg/kg b.w.: Demonstrates severe diffuse vascular congestion of glomerular capillaries (**black asterisk**), complete Bowman's space occlusion (**black arrow**), severe renal tubular cell necrosis (**thick red arrow**), and focal interstitial inflammatory cell infiltration (**yellow asterisk**).

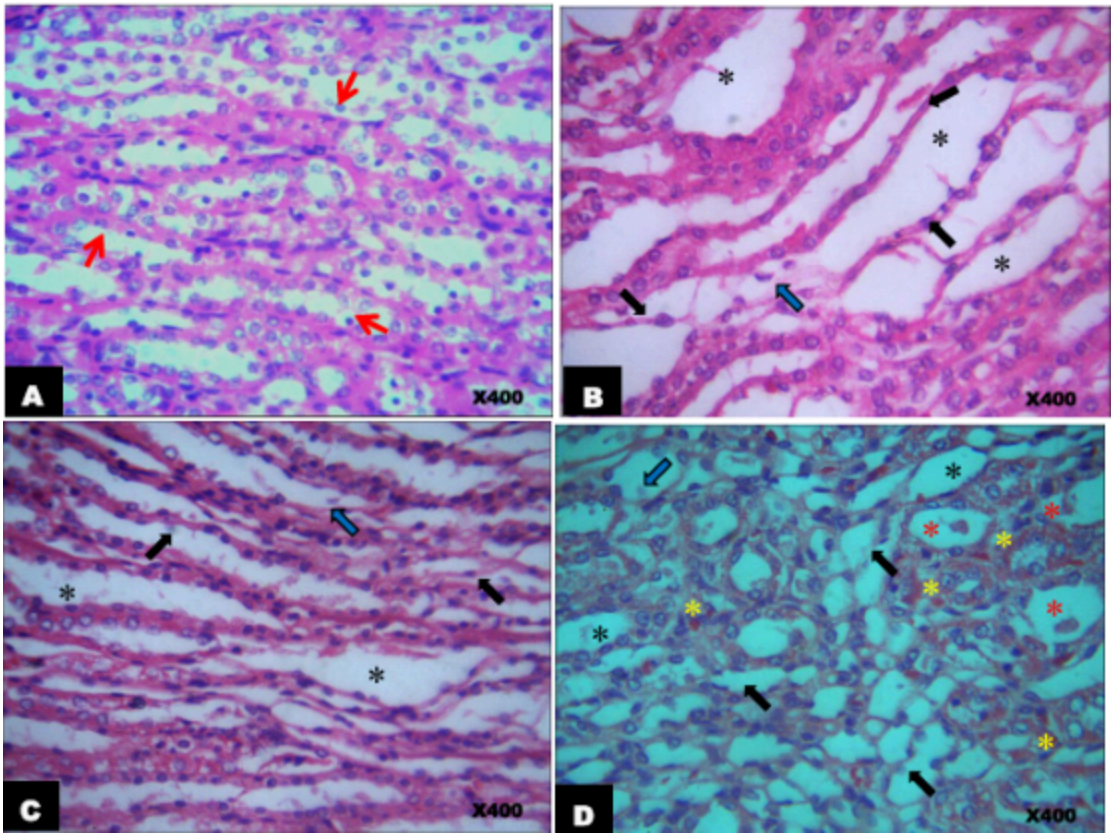

Figure 2

Histopathological changes in renal medullary tissue following administration of *Dracaena trifasciata* extract (H&E, x400).

Photomicrograph sections of renal medulla illustrating tubular injury and cast formation:

(A) Control: Shows normal medullary microarchitecture with intact collecting tubules and ducts (**red arrow**).

(B) DT 250 mg/kg b.w.: Demonstrates early degenerative changes including tubular dilatation/ectasia (**black asterisk**), luminal sloughing of necrotic epithelial cells (**blue arrow**), and focal tubular atrophy (**black arrow**).

(C) DT 500 mg/kg b.w.: Displays progressive tubular ectasia (**black asterisk**), desquamation of epithelial lining cells (**blue arrow**), and diffuse tubular atrophy (**black arrow**).

(D) DT 1 000 mg/kg b.w.: Shows severe tubular atrophy (**black arrow**), luminal obstruction by eosinophilic hyaline casts (**red asterisk**), tubular ectasia (**black asterisk**), and interstitial hemorrhage (**yellow asterisk**).

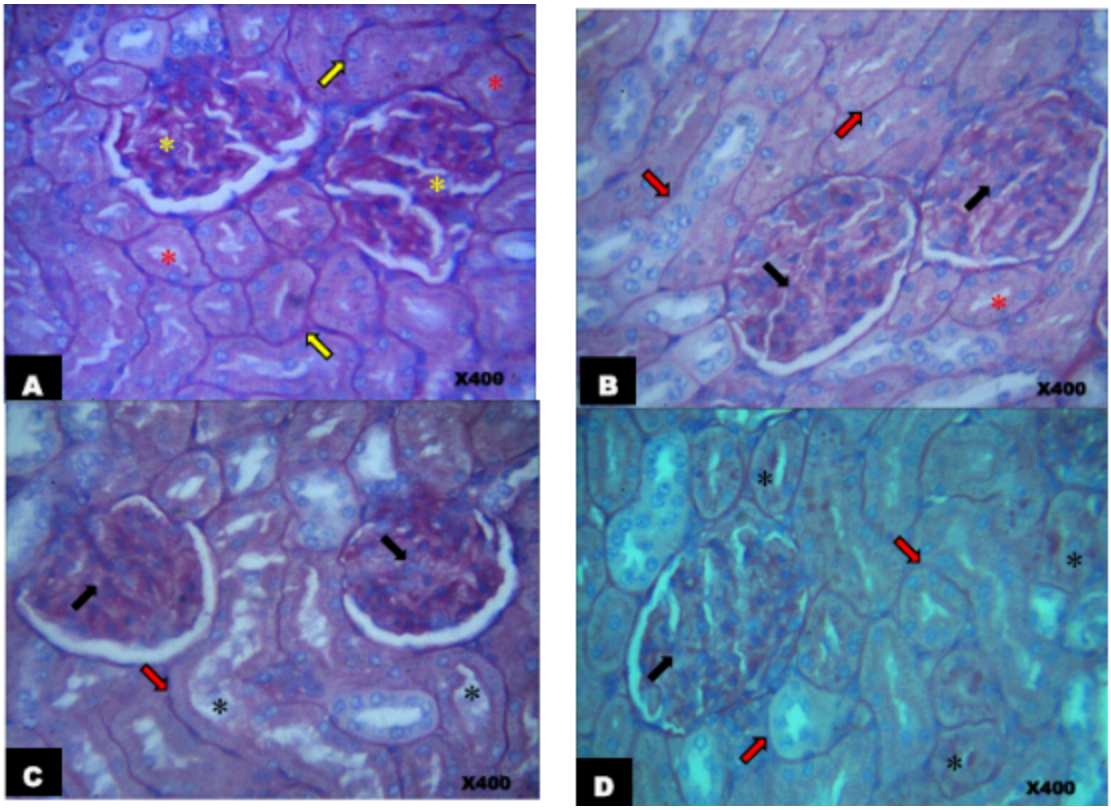

Figure 3

Histochemical evaluation of renal cortical basement membranes and apical brush borders (PAS, x400).

Photomicrograph sections of renal cortex stained with Periodic Acid-Schiff:

(A) Control: Demonstrates intact, intense PAS-positive basement membranes of the glomerular tuft (**yellow asterisk**), normal cortical tubular basement membranes (**yellow arrow**), and continuous glycocalyx brush border along proximal tubules (**red asterisk**).

(B) DT 250 mg/kg b.w.: Shows thinning of glomerular (**black arrow**) and tubular (**red arrow**) basement membranes, with preserved proximal tubular brush border integrity (**red asterisk**).

(C) DT 500 mg/kg b.w.: Displays attenuated basement membranes (**black arrow**, **red arrow**) accompanied by localized disruption and atypical loss of glycocalyx brush borders (**black asterisk**).

(D) DT 1 000 mg/kg b.w.: Demonstrates severe basement membrane fragmentation (**black arrow**, **red arrow**) and widespread loss/disruption of proximal tubular brush border glycocalyx (**black asterisk**).

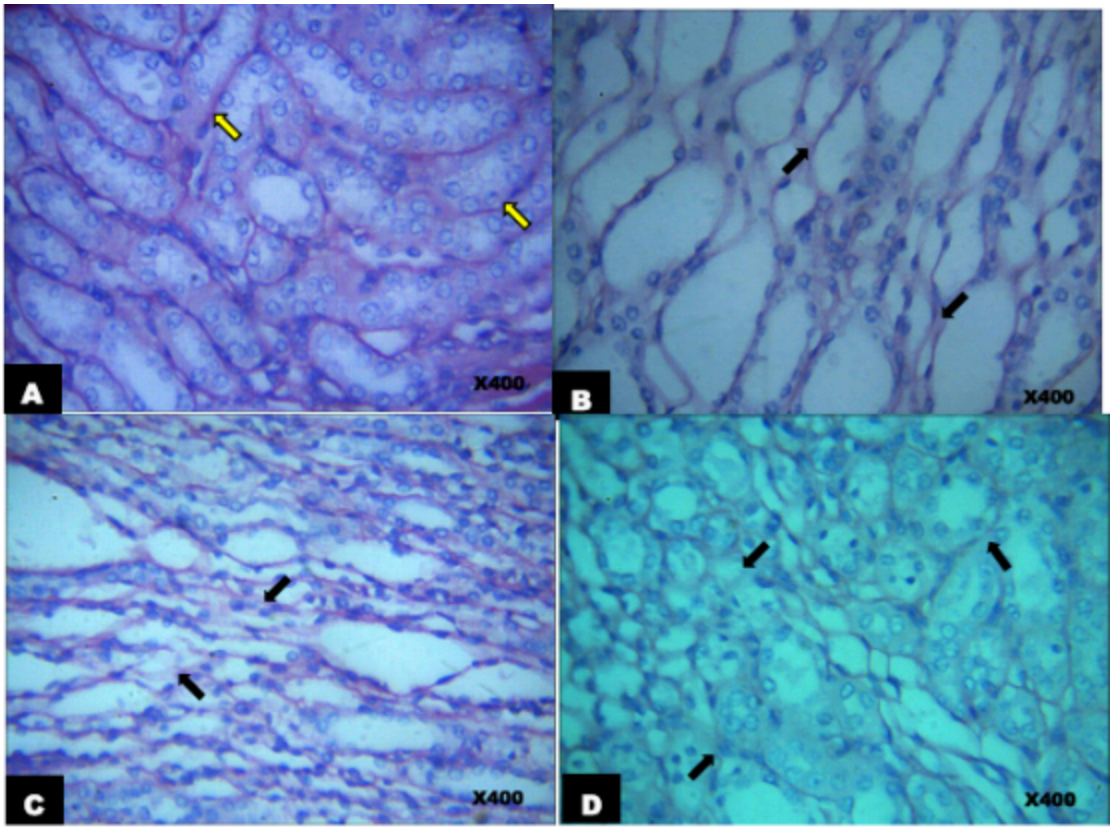

Figure 4

Histochemical changes in renal medullary basement membranes (PAS, x400).

Photomicrograph sections of renal medullary tubules stained with Periodic Acid-Schiff:

(A) Control: Shows normal, continuous PAS-positive basement membranes encapsulating medullary collecting tubules (**yellow arrow**).

(B) DT 250 mg/kg b.w.: Displays mild thinning and focal loss of intensity in medullary tubular basement membranes (**black arrow**).

(C) DT 500 mg/kg b.w.: Shows progressive thinning and attenuation of medullary tubular basement membranes (**black arrow**).

(D) DT 1 000 mg/kg b.w.: Demonstrates severe, diffuse basement membrane thinning and focal structural disruption surrounding medullary tubules (**black arrow**).