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RENOPROTECTIVE EFFECTS OF AQUEOUS AND DIETHYL ETHER *DIOSCOREA VILLOSA* LEAF AND ROOT-EXTRACTS ON RENAL BIOMARKERS AND ELECTROLYTES IN GENTAMICIN-INDUCED NEPHROTOXICITY

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***Abstract*—**Gentamicin-induced renal injury impairs filtration and electrolyte balance. This study aimed to evaluate aqueous and diethyl ether-leaf and root-extracts of *Dioscorea villosa* against gentamicin-associated renal dysfunction in rats. In the study, 114 rats were allocated to 19 groups (n = 6), including controls and four extracts administered at 200–800 mg/kg. Urea, creatinine, uric acid, sodium, potassium, chloride and bicarbonate were measured. Gentamicin increased in all seven indices compared with normal controls ($P < 0.05$). From the original graphs, urea increased from approximately 18 to 60 mg/dL; 800 mg/kg aqueous leaf, aqueous root, ether leaf and ether root treatments yielded approximately 30, 27, 21 and 16 mg/dL, respectively. Uric acid rose to approximately 6 mg/dL in untreated animals, versus approximately 1–3 mg/dL across the highest-dose extract groups. Potassium increased from approximately 4.6 to 9.8 mmol/L and decreased to approximately 7.0, 6.4, 6.1 and 5.1 mmol/L, respectively, at 800 mg/kg. Sodium, chloride and bicarbonate likewise showed dose-related reductions, as did creatinine. The strongest overall response was reported for ether root extract, approaching the silymarin control at 800 mg/kg. *Dioscorea villosa* extracts were associated with improved renal biochemical indices, particularly the ether root preparation. Findings of this study are indicative that the extracts may possess ability to alleviate renal damage and renal related disoders. This potential may be attributed to chemical constituents of the extracts and may be responsible for ethno-medicinal applications of the plant.

Index Terms*—Dioscorea villosa*, gentamicin, nephrotoxicity, renal biomarkers, serum electrolytes

I. INTRODUCTION

Gentamicin remains an important aminoglycoside antibiotic for treating severe bacterial infections, but its clinical usefulness is constrained by nephrotoxicity. The kidney is particularly susceptible because it receives substantial blood flow and actively concentrates filtered substances within tubular epithelial cells. Gentamicin accumulates in proximal tubular cells and can disrupt intracellular organelles, mitochondrial function and membrane integrity, eventually compromising renal function (Quiros *et al.*, 2011; Randjelovic *et al.*, 2017).

The pathogenesis of gentamicin-associated nephrotoxicity involves overlapping mechanisms rather than a single toxic event. Endocytic uptake into proximal tubular epithelial cells promotes intracellular accumulation, endoplasmic-reticulum stress, mitochondrial dysfunction and inflammatory signaling. Reactive oxygen species and lipid peroxidation contribute to cellular injury, while altered renal haemodynamics and tubular transport may exacerbate functional impairment (Quiros *et al.*, 2011; Randjelovic *et al.,* 2017). Experimental animal models therefore provide opportunities to investigate biochemical responses to gentamicin exposure, although observations from such models should not be interpreted as evidence of clinical efficacy without further validation.

Serum urea and creatinine are routinely used to evaluate impaired renal excretory function. Their concentrations can rise when renal clearance decreases, although both are influenced by factors beyond glomerular filtration. Uric acid provides additional information on purine metabolism and excretion, but it is not a kidney-specific injury marker. This study assessed all three analytes in gentamicin-exposed albino rats and compared untreated animals with groups receiving extracts from different plant parts and solvents.

Renal regulation of sodium, potassium, chloride and bicarbonate is essential for extracellular fluid balance, electrical excitability and acid–base homeostasis. Disturbances in these ions may accompany kidney injury through altered filtration, tubular reabsorption, secretion or water handling. Nevertheless, electrolyte concentrations are affected by hydration status, intake, sampling conditions and systemic physiology; a change in concentration does not, by itself, establish restoration of kidney structure. Joint assessment of these four electrolytes with conventional renal biomarkers offers a broader description of functional disturbances than any single endpoint.

Medicinal plants and their secondary metabolites have attracted attention as potential modifiers of oxidative and inflammatory injury. However, crude botanical extracts contain multiple constituents whose recovery depends on the plant tissue, extraction solvent and preparation conditions. Therefore, aqueous and organic-solvent

preparations should not be presumed chemically equivalent or biologically interchangeable. Experimental evidence concerning diosgenin, a steroidal sapogenin associated with some Dioscorea species, has suggested attenuation of gentamicin-related biochemical and histopathological injury in rats; importantly, findings for an isolated compound cannot establish the same activity for an uncharacterized whole-plant extract (Mishra *et al.*, 2021).

Dioscorea villosa, commonly referred to as wild yam, was investigated in the source project using leaf and root-extracts prepared with deionised water or diethyl ether. The research reported four dose levels and assessed renal function and electrolyte outcomes following gentamicin administration. Yet a meaningful comparison requires careful separation of the source's reported treatment responses from independently verified numerical data. In particular, statistical significance, dose-dependence and superiority among preparations should be supported by the underlying animal-level observations and appropriately reported comparisons.

The present article focuses on the combined renal biomarkers and electrolyte results of that experiment, rather than extrapolating to liver function or other unmeasured outcomes. Transparent presentation of group allocation, controls, sample size, assay procedures and statistical methods is essential for interpreting animal studies, consistent with the ARRIVE 2.0 reporting recommendations. Accordingly, this study aimed to evaluate the reported effects of aqueous and diethyl ether leaf and root extracts of D. villosa on serum urea, creatinine, uric acid, sodium, potassium, chloride and bicarbonate in gentamicin-induced nephrotoxicity in albino rats, and to compare responses across extract types and administered doses.

II. MATERIALS AND METHODS

A. Study Design and Experimental Animals

The study describes an *in vivo* comparative experiment with 114 adult male albino rats weighing 150–300 g. Animals were acclimatized for seven days and randomly assigned to 19 groups of six rats. Three groups served as normal, gentamicin-only injury and positive comparator controls. Kidney damage was induced in groups B to S by intraperitoneal injection of 100 mg/kg gentamicin for seven consecutive days. Group B negative control received normal saline, while group C positive control received 25 mg/kg body weight of sylimarin. 16 treatment groups received four extract preparations at four doses. Groups D, E, F and G received deionized water leaf-extract (DWL), groups H, I, J and K received deionized water root-extracts (DWR) and groups L, M, N and O received diethylether leaf-extracts (DEL), while groups P, Q, R and S received diethylether root-extracts (DER) at doses of 200, 400, 600 and 800 mg/kg body weight respectively via oral intubation for 21 consecutive days.

B. Extracts, Doses and Controls

Preparations were deionized-water leaf (DWL), deionized-water root (DWR), diethyl ether leaf (DEL) and diethyl ether root (DER). Each preparation was administered at 200, 400, 600 or 800 mg/kg body weight. The normal-saline group, a gentamicin-induced untreated group (100 mg/kg body weight) and a gentamicin-induced silymarin group (25 mg/kg body weight). Others include sixteen (16) treatment groups received four extract preparations at four doses as follows: Groups D, E, F and G received deionized water leaf-extract (DWL), groups H, I, J and K received deionized water root-extracts (DWR) and groups L, M, N and O received diethylether leaf-extracts (DEL), while groups P, Q, R and S received diethylether root-extracts (DER) at doses of 200, 400, 600 and 800 mg/kg body weight respectively via oral intubation for 21 consecutive days.

C. Serum Collection and Biochemical Determinations

Blood was collected by cardiac puncture. Samples intended for biochemical assays were collected in plain containers. Sodium was determined using a uranyl acetate/ferrocyanide colorimetric method described by Tietz *et al* (1976); potassium was measured by a sodium tetraphenylboron turbidimetric method of Tietz *et al* (1976) (500 nm). Chloride was determined using a mercuric thiocyanate/ferric-ion color reaction (480 nm), and bicarbonate by an enzymatic phosphoenolpyruvate carboxylase/malate dehydrogenase method (340 nm), described by Tietz *et al* (1976).

Serum creatinine was determined using an alkaline picrate kinetic assay (500 nm), described by Tietz *et al* (1976). Urea was assayed using urease and the Berthelot color reaction (600 nm), while uric acid was determined using a uricase–peroxidase color reaction (520 nm).

D. Statistical Analysis

Data generated were expressed as mean ± standard deviation (SD). Differences among treatment groups were analysed using one-way analysis of variance (ANOVA), followed by Duncan's multiple range test. Statistical significance was established at $p < 0.05$.

III. RESULTS

A. Serum Urea

Serum urea was higher in the gentamicin-only group than in the normal control, and the finding identifies this difference as statistically significant ($P < 0.05$). Fig. 1 shows the responses to deionized-water leaf (DWL), deionized-water root (DWR), diethyl ether leaf (DEL) and diethyl ether root (DER) extracts at 200, 400, 600 and 800 mg/kg body weight. All four preparations were associated with lower urea concentrations than the untreated gentamicin group, although the plotted responses varied across extract types and doses. The greatest reduction was reported with the diethyl ether root preparation at the highest administered dose. The silymarin-treated group, included as a reference

treatment, also showed a lower urea value than the injury control. Distinct superscript annotations on the original graph indicate the level of orderliness between-group differences designated as significant in the experimental analysis. The plotted values represent means with standard deviations for six rats per group. The overall pattern is a reduction in the gentamicin-associated elevation in blood urea following extract administration, with the most pronounced response among the higher-dose root treatments.

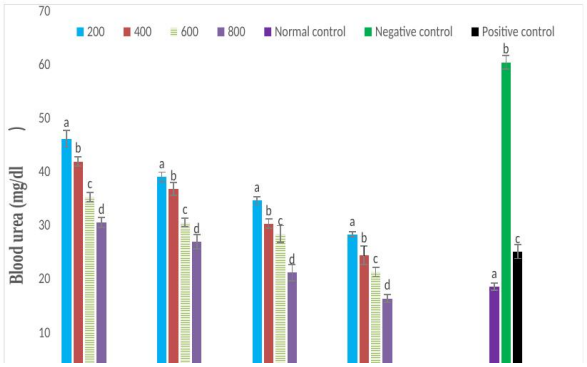


Fig. 1. Blood Urea level in gentamicin-induced damage in albino rats treated with leaf and root-extracts of *Dioscorea villosa*. Values are expressed as Mean ± Standard Deviation, n=6 rats per group. Values bearing different superscript differ significantly at (P < 0.05). DWL= Deionized water-leaf, DWR= Deionized water-root, DEL= Diethylether-leaf and Diethylether-root-extracts.

B. Serum Creatinine

Serum creatinine was elevated in rats receiving gentamicin without extract treatment compared with the normal control group (P < 0.05). The treatment comparisons in Fig. 2 include the four leaf and root preparations at each of the four administered doses, alongside the silymarin reference group. Extract-treated rats generally exhibited lower creatinine values than the gentamicin-only group. The study describes stronger responses at increasing extract doses and identifies the diethyl ether root preparation as the most effective overall treatment. This comparative pattern is visible across the plotted treatment groups, although individual values and pair-wise differences should be interpreted according to the superscript labels attached to the original bars. The control groups provide the baseline and untreated-injury reference points for interpreting the treatment response. Values generated are presented in as mean ± standard deviation for six animals in each group. Thus, Fig. 2 documents an attenuation of the gentamicin-associated rise in creatinine after administration of the investigated extracts, rather than evidence of complete recovery of renal function in every treated group.

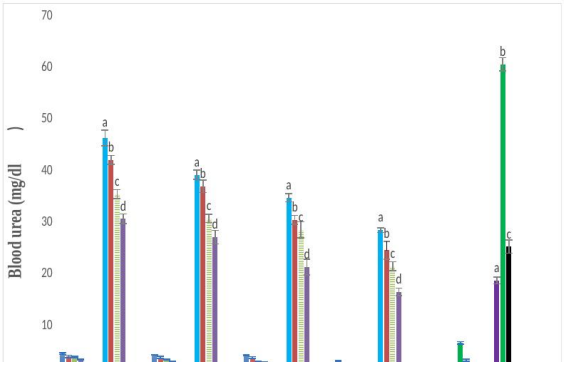


Fig. 2. Creatinine level in gentamicin-induced kidney damage in albino rats treated with leaf and root-extracts of *Dioscorea villosa*.Values generated are expressed as Mean ± Standard Deviation, n=6 rats per group. Values bearing different superscripts differ significantly at (P<0.05). DWL= Deionized water-leaf, DWR= Deionized water-root, DEL= Diethylether-leaf and Diethylether-root-extracts.

C. Serum Uric Acid

Gentamicin administration increased serum uric acid relative to the normal control group, with the original thesis reporting a significant difference (P < 0.05). Fig. 3 presents' uric acid concentrations for the untreated injury control, normal control, silymarin reference and the 16 extract-treatment groups. Treatment with deionized-water leaf, deionized-water root, diethyl ether leaf and diethyl ether root extracts was associated with reduced uric acid concentrations compared with gentamicin alone. The four dose levels, 200, 400, 600 and 800 mg/kg, permit assessment of the direction of the response within each extract preparation. The study describes greater reductions with increasing administered dose, while identifying diethyl ether root extract as the strongest preparation overall. Differences among the plotted groups are annotated using superscript letters, and the original figure reports means and standard deviations for six rats per group. Although the treatments reduced the elevated uric acid profile, the plotted group comparisons do not independently establish the mechanism responsible for the change. The result therefore indicates a treatment-associated shift in this renal biochemical index following gentamicin exposure.

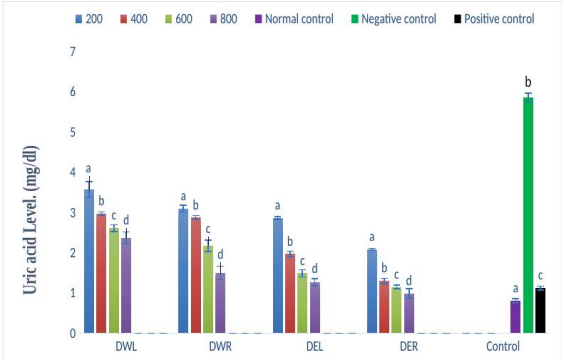


Fig. 3. Uric Acid level in Gentamicin-induced kidney damage in albino rats treated with leaf and root-extracts of *Dioscorea villosa*.Values are expressed as Mean ± Standard Deviation, n=6 rats per group. Values bearing different superscripts differ significantly at (P < 0.05). DWL=

Deionized water-leaf, DWR= Deionized water-root, DEL= Diethylether-leaf and Diethylether-root-extracts.

D. Serum Sodium

Serum sodium concentrations differed between the normal and gentamicin-only groups, with the thesis reporting an increase following gentamicin exposure ($P < 0.05$). Fig. 4 displays the sodium results for all control and extract-treated groups. Across the four preparations, administration of the leaf or root extracts was associated with lower sodium concentrations than those recorded in the untreated gentamicin group. The extract series includes 200, 400, 600 and 800 mg/kg body weight, enabling comparison of lower and higher doses within each preparation. The source describes a generally greater reduction with increasing dose and identifies the diethyl ether root treatment as having the strongest overall effect. Silymarin served as the reference treatment for the same outcome. The original bars include standard deviations and superscript annotations identifying statistically differentiated group means according to the thesis analysis, with six rats represented per group. The result shows that sodium was among the electrolyte indices altered by gentamicin and subsequently modified by extract administration. These findings describe the observed serum concentration pattern and do not, by themselves, establish a specific mechanism of sodium transport or fluid regulation.

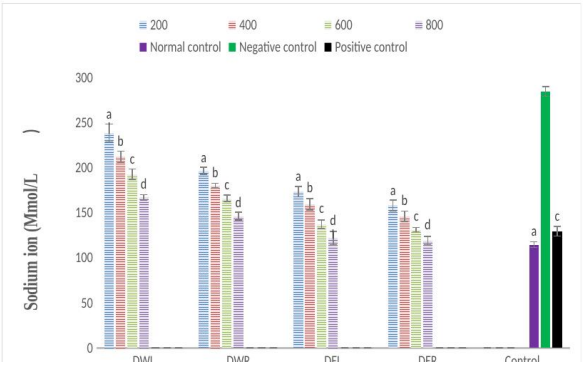


Fig. 4. Sodium ion (Na+) level in gentamicin-induced kidney damage in rats treated with leaf and root-extracts of *Dioscorea villosa*.Values are expressed as Mean ± Standard Deviation, n=6 rats per group. Values bearing different superscripts differ significantly at ($P < 0.05$). DWL= Deionized water-leaf, DWR= Deionized water-root, DEL= Diethylether-leaf and Diethylether-root-extracts.

E. Serum Potassium

The gentamicin-only group exhibited higher serum potassium than the normal control group, a difference reported as significant in the source study ($P < 0.05$). Fig. 5 compares the potassium values of the controls with those of rats receiving deionized-water leaf, deionized-water root, diethyl ether leaf or diethyl ether root extracts at 200–800 mg/kg. Potassium concentrations were lower in the extract-treated groups than in the untreated injury control, with the thesis describing an overall dose-related treatment response. The diethyl ether root extract at 800 mg/kg was identified as the strongest treatment response among the investigated preparations, while silymarin provided the reference comparison. The graph includes means, standard deviations and superscript letters for the statistical groupings, based on six rats in each treatment group. As potassium concentrations varied among preparations and doses, the original statistical labels should govern interpretation of any specific pairwise comparison. The primary finding is that extract administration was associated with attenuation of the gentamicin-associated increase in serum potassium. The direction and magnitude of these changes should be confirmed from the underlying measurements before numerical effect estimates are reported.

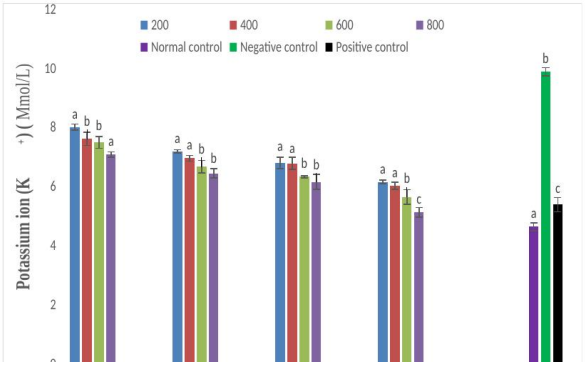


Fig. 5. Potassium ion (K+) level in gentamicin-induced kidney damage in albino rats treated with leaf and root-extracts of *Dioscorea villosa*.Values are expressed as Mean ± Standard Deviation, n=6 rats per group. Values bearing different superscripts differ significantly at ($P<0.05$). DWL= Deionized water-leaf, DWR= Deionized water-root, DEL= Diethylether-leaf and Diethylether-root-extracts.

F. Serum Chloride

Serum chloride was reported to be significantly higher in the untreated gentamicin group than in the normal control group ($P < 0.05$). Fig. 6 presents the chloride measurements for the three control conditions and the 16 groups treated with *Dioscorea villosa* extracts. Each of the four preparations was administered at 200, 400, 600 and 800 mg/kg body weight. Relative to gentamicin alone, the finding describes reductions in chloride after administration of the aqueous and diethyl ether leaf and root extracts. The magnitude of the plotted response varied by preparation and dose, with the diethyl ether root extract identified in the experiment as the strongest overall preparation. The silymarin group was included to provide a reference treatment response. These findings demonstrate a treatment-associated change in serum chloride within the experimental groups. They should not be interpreted as evidence of normalization of chloride balance without confirmation of the individual treatment means and their comparison with the normal control.

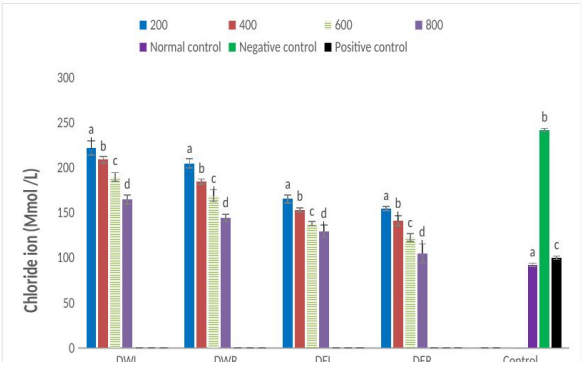


Fig. 6. Chloride ion (Cl-) level in gentamicin-induced kidney damage in albino rats treated with leaf and root-extracts of *Dioscorea villosa*. Values generated are expressed as Mean ± Standard Deviation, n=6 rats per group. Values bearing different superscripts differ significantly at ($P < 0.05$). DWL= Deionized water-leaf, DWR= Deionized water-root, DEL= Diethylether-leaf and Diethylether-root-extracts.

G. Serum Bicarbonate

Bicarbonate was also reported to increase in the gentamicin-only group relative to the normal control, with a significant difference indicated in the experimental analysis ($P < 0.05$). Fig. 7 compares serum bicarbonate across the normal control, gentamicin injury control, silymarin reference and the four extract preparations administered at 200, 400, 600 and 800 mg/kg. The findings indicate lower bicarbonate concentrations after treatment with the leaf and root extracts than in the untreated gentamicin group. Responses differed across the treatment series, and findings identify the diethyl ether root extract as the strongest preparation overall. The graph presents the treatment means with standard deviations for six animals per group, together with superscript letters indicating statistical groupings. Unlike the other electrolytes, bicarbonate requires particular care when interpreting the biological direction of change because it is related to systemic acid–base status. Accordingly, the result is presented here as the reported concentration change without labeling a lower bicarbonate value as necessarily beneficial. Confirmation against the raw measurements and accompanying acid–base information is needed before making a stronger physiological interpretation.

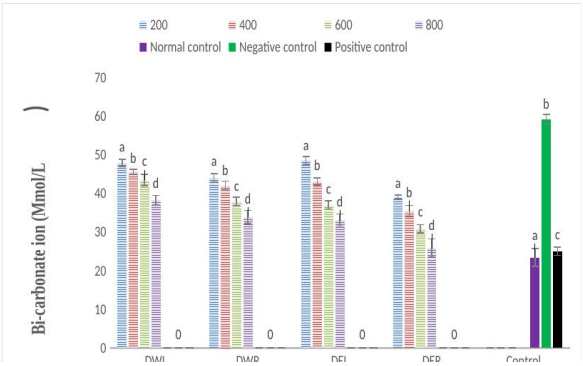


Fig. 7. Bi-carbonate ion ($HCO3^-$) level in Gentamicin-induced Kidney Damage in rats treated with leaf and root-extracts of *Dioscorea villosa*.Values are expressed as Mean ± Standard Deviation, n=6 rats per group. Values bearing different superscripts differ significantly at ($P<0.05$). DWL= Deionized water-leaf, DWR= Deionized water-root, DEL= Diethylether-leaf and Diethylether-root-extracts.

IV. DISCUSSION

The gentamicin-only group showed increased serum urea, creatinine and uric acid relative to the normal control, consistent with impaired renal excretory function. Gentamicin accumulates in proximal tubular epithelial cells and produces cellular injury through oxidative stress, mitochondrial dysfunction, inflammation and apoptosis (Quiros *et al.*, 2011; Randjelovic *et al.*, 2017). The concurrent changes in the three nitrogenous indices strengthen the interpretation of renal dysfunction, although these conventional serum markers alone cannot establish its anatomical severity or mechanism.

Serum urea increased markedly following gentamicin administration and decreased across extract-treated groups, particularly at higher doses. Urea accumulates when renal elimination is compromised, but its concentration is also affected by hydration, protein metabolism and catabolic state. Accordingly, the decline after treatment supports an improvement in the measured biochemical profile rather than proving complete restoration of glomerular filtration. The apparent dose response and the pronounced effect of the diethyl ether root preparation warrant confirmation using the original group means, standard deviations and individual-animal observations (Ali *et al*., 2011).

Serum creatinine followed a similar direction: elevation after gentamicin exposure and lower concentrations following extract treatment. This result is particularly relevant because creatinine is routinely used to assess renal filtration; however, it is an imperfect and delayed marker of acute kidney injury, and interpretation depends on baseline measurements and urine output (Kellum and Lameire, 2013). The agreement between creatinine and urea therefore provides convergent evidence of altered renal function, but a claim of normalized filtration would require measured creatinine clearance or other functional data.

Uric acid was also higher in gentamicin-exposed animals and declined with extract administration. As a product of purine metabolism eliminated in part through the kidneys, uric acid may change with renal excretion and systemic metabolism. Its reduction is compatible with the broader biochemical response, but it should not be treated as an independent demonstration of antioxidant activity or renal tissue repair. In particular, rat urate metabolism differs from that of humans, limiting direct clinical extrapolation.

The reported serum sodium pattern showed an increase after gentamicin exposure and lower values in extract-treated groups. Sodium homeostasis reflects the combined effects of renal solute handling, water balance and hormonal regulation. Gentamicin-induced tubular damage can disturb water and electrolyte transport (Randjelovic *et al*., 2017). Nevertheless, a high sodium concentration cannot be attributed solely to impaired filtration without fluid-intake, urine-volume and urinary-electrolyte measurements. A reduction toward the normal-control range is more

appropriately described as attenuation of the observed disturbance than as proof of a specific sodium-transport mechanism.

Serum potassium was substantially elevated in the gentamicin-only group and declined after treatment, with the strongest apparent response at the highest diethyl ether root dose. Potassium retention can accompany reduced renal excretion and may have important electrophysiological consequences. However, gentamicin may also produce renal tubular electrolyte wasting, and the direction of potassium change varies with the model and severity of injury. Indeed, a published diosgenin–gentamicin experiment reported decreased, rather than increased, serum potassium in the gentamicin group (Mishra *et al*., 2021). Thus, the potassium findings should be discussed as specific to the present experiment, with assay quality and specimen haemolysis checked before inferring generalized correction of potassium homeostasis.

Serum chloride increased in the gentamicin-only group and decreased after extract administration. Chloride is closely linked to sodium and extracellular fluid balance, but its concentration also depends on acid–base status and changes in other anions. The parallel sodium and chloride responses may indicate a shared disturbance of fluid–electrolyte regulation; the available serum measurements do not, however, identify whether altered intake, water loss, tubular transport or acid–base changes predominated. Interpretation would be strengthened by paired urine chemistry and contemporaneous serum sodium, chloride and bicarbonate measurements.

Bicarbonate also increased in the gentamicin-only group and fell following extract administration. This finding deserves particular caution: acute kidney injury frequently produces metabolic acidosis with reduced bicarbonate, whereas an elevated value may reflect alkalosis, compensation for respiratory disturbance, volume contraction or analytical variation. The simultaneous increases reported for sodium, potassium, chloride and bicarbonate therefore do not establish a single coherent acid–base mechanism. Blood pH, carbon dioxide tension, anion gap and specimen-handling information are needed before concluding that the extracts restored acid–base equilibrium. The present evidence supports a treatment-associated shift in the measured bicarbonate concentration, not a confirmed correction of an acid–base disorder.

Across the seven endpoints, the findings describe a stronger overall response for diethyl ether root extract than for the other preparations, particularly at 800 mg/kg. Variation between extracts could reflect differences in solvent selectivity and the distribution of bioactive constituents between leaves and roots. A related experimental study reported that diosgenin attenuated gentamicin-associated renal injury and altered inflammatory and oxidative-stress indices in rats (Mishra *et al*., 2021). However, the current experiment established that diosgenin was present at an active concentration and was responsible for the observed responses. Chemical profiling and mechanism-directed experiments would be needed to test that explanation. Likewise, the description of the highest-dose root-extract as comparable to silymarin should not be interpreted as statistical equivalence without a prespecified equivalence analysis. The relevant diosgenin study is cited by Mishra *et al.* (2021).

The principal limitations are the absence of accessible animal-level data for independent verification of group means and statistical comparisons, the reliance on conventional serum endpoints, and the lack of paired urine chemistry and blood-gas measurements. Despite these constraints, the coordinated reduction in urea, creatinine and uric acid provides the clearest evidence of a potentially renoprotective biochemical response. The electrolyte findings are useful complementary observations but require more cautious physiological interpretation.

V. CONCLUSION

Aqueous and diethyl ether extracts of *Dioscorea villosa* attenuated the reported gentamicin-associated increases in serum urea, creatinine and uric acid, with the strongest overall response observed for diethyl ether root-extract at 800 mg/kg. Sodium, potassium, chloride and bicarbonate also changed following treatment, although their physiological significance requires verification. These findings support further investigation of the root extract using validated raw data, renal functional measurements and mechanistic assays.

ETHICAL APPROVAL

Ethical approval for this study was obtained from the Research Ethics Committee, Department of Biochemistry, Ebonyi State University, Abakaliki, Nigeria (Approval No. EBSU/BCH/ET/21/009), subject to confirmation that this approval covers the present animal experiment.

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CONFLICTS OF INTEREST STATEMENT

The author declares no conflict of interest.

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