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Research Article

Evaluation of Ethanolic Extract of *Moringa oleifera* Root against Gentamicin-Induced Hepatotoxicity and Nephrotoxicity in Mice

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ABSTRACT

Background: Gentamicin is a potent aminoglycoside antibiotic whose therapeutic utility is constrained by drug-induced nephrotoxicity and hepatotoxicity driven by reactive oxygen species (ROS). **Objective:** To investigate the protective role of the ethanolic extract of *Moringa oleifera* root (MOE) against gentamicin-induced renal and hepatic dysfunction in mice. **Methods:** Female/male Swiss albino mice were allocated into five groups: Control, Gentamicin (100" mg/kg" , i.p.), MOE 300" mg/kg" + Gentamicin, MOE 600" mg/kg" + Gentamicin, and Silymarin (150" mg/kg") standard. Biochemical serum markers (creatinine, urea, BUN, uric acid, ALT, AST, ALP, total protein, bilirubin) and histopathological sections of renal and hepatic tissues were evaluated following eight days of treatment. **Results:** Gentamicin administration caused marked elevations in renal injury markers (serum creatinine: 3.65" mg/dL" ; serum urea: 178.4" mg/dL" ; BUN: 87.44" mg/dL") and liver enzyme dysregulation ($P < 0.001$). Co-treatment with MOE (300" and " 600" mg/kg") significantly ($P < 0.001$) restored parameter levels in a dose-dependent manner and attenuated structural necrosis, peritubular congestion, and inflammatory infiltration. **Conclusion:** *M. oleifera* root extract exhibits robust nephroprotective and hepatoprotective properties against aminoglycoside toxicity.

INTRODUCTION

Gentamicin (GM) remains a primary treatment option for severe Gram-negative infections, yet 10–20% of treated patients develop acute kidney injury (AKI). The mechanisms of GM toxicity involve drug accumulation in renal proximal tubular epithelial cells, lysosomal/mitochondrial

injury, and intense ROS generation. Similarly, bioactivation of therapeutics leads to direct liver cell death, organelle breakdown, and apoptosis.

The search for adjuvant therapies to mitigate drug-induced toxicities has highlighted natural antioxidants. *Moringa oleifera* Lam. root contains secondary metabolites with recognized

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antioxidant and membrane-stabilizing capabilities. This investigation evaluates the nephroprotective and hepatoprotective efficacy of an ethanolic extract of *M. oleifera* root (MOE) in a experimental mouse model.

2. MATERIALS AND METHODS

2.1. Plant Material and Extraction

M. oleifera roots were collected locally, authenticated by a botanist, shade-dried, and coarsely powdered. Coarse root powder (1 kg) was extracted with analytical-grade ethanol using a Soxhlet apparatus. The extract was concentrated under reduced pressure, yielding 4.8% w/w dry residue.

2.2. Preliminary Phytochemical Screening

Qualitative chemical tests were carried out according to standard procedures to verify the presence of carbohydrates, proteins, steroids, alkaloids, flavonoids, tannins, saponins, and resins.

2.3. Experimental Animals

Healthy Swiss albino mice (30--40 g) were acclimatized for one week under controlled conditions (12 h light/dark cycle, standard pellet diet, water *ad libitum*) with IAEC approval.

2.4. Experimental Design

Animals were partitioned into five experimental groups ($n = 6$ per group) over an 8-day regimen:

- **Group I (Control):** Distilled water orally (p.o.) for 8 days.
- **Group II (Gentamicin Toxicant):** Gentamicin 100 mg/kg/day intraperitoneally (i.p.) for 8 days.

- **Group III (MOE 300):** MOE 300 mg/kg/day p.o. + Gentamicin 100 mg/kg i.p. for 8 days.
- **Group IV (MOE 600):** MOE 600 mg/kg/day p.o. + Gentamicin 100 mg/kg i.p. for 8 days.
- **Group V (Standard):** Silymarin 150 mg/kg/day p.o. + Gentamicin 100 mg/kg i.p. for 8 days.

2.5. Biochemical and Histopathological Appraisals

On day 8, 24-hour urine collection was performed in metabolic cages. On day 9, animals were weighed and euthanized. Blood was collected for estimation of serum creatinine, urea, blood urea nitrogen (BUN), uric acid, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total protein, and bilirubin. Harvested renal and hepatic tissues were fixed in 10% formal saline, sectioned at 4–5 μm , stained with Hematoxylin and Eosin (H&E), and evaluated light-microscopically.

2.6. Statistical Analysis

Data are expressed as Mean $\pm$ Standard Deviation (S.D.). Differences between groups were analyzed using one-way ANOVA followed by Dunnett's / Tukey's post-hoc tests ($P < 0.05$ considered statistically significant).

3. RESULTS

3.1. Phytochemical Profile

Qualitative analysis confirmed the presence of resins, saponins, tannins, flavonoids, and alkaloids in the ethanolic root extract.



3.2. Renal Functional Markers and Body Weight

Gentamicin administration (Group II) produced marked acute kidney injury, reflected by significant increases in serum creatinine, serum

urea, urine creatinine, BUN, and uric acid ($P < 0.001$ vs. Control). Co-treatment with MOE (300 and 600 mg/kg) significantly ($P < 0.001$) prevented these elevation trends in a clear dose-dependent manner.

Parameter / Marker	Group I (Control)	Group II (Gentamicin 100 mg/kg)	Group III (MOE 300 mg/kg)	Group IV (MOE 600 mg/kg)	Group V (Standard)
Serum Creatinine (mg/dL)	0.65 ± 0.18	$3.65 \pm 0.68^{a***}$	$1.82 \pm 0.52^{b***}$	$1.06 \pm 0.43^{b***}$	0.78 ± 0.14
Serum Urea (mg/dL)	45.17 ± 0.17	$178.4 \pm 1.49^{a***}$	$108.4 \pm 1.23^{b**}$	$87.6 \pm 0.75^{b***}$	56.17 ± 0.32
Urine Creatinine (mg/dL)	99.76 ± 1.54	$289.56 \pm 3.06^{a***}$	$189.2 \pm 0.86^{b***}$	$113. \pm 0.97^{b***}$	102.6 ± 1.53
BUN (mg/dL)	18.81 ± 0.58	$87.44 \pm 1.92^{a***}$	$48.75 \pm 1.58^{b***}$	$38.16 \pm 0.63^{b***}$	23.31 ± 0.22
Uric Acid (mg/dL)	1.54 ± 0.14	$3.78 \pm 0.34^{a**}$	$2.98 \pm 0.24^{b*}$	$2.012 \pm 0.54^{b***}$	—
Body Weight Change (%)	$+3.93 \pm 0.28$	$-13.68 \pm 0.37^{a***}$	$-7.84 \pm 0.60^{b*}$	$-7.38 \pm 0.67^{b*}$	—

3.3. Hepatic Functional Markers

Gentamicin exposure caused hepatocyte membrane leakage, leading to elevated serum

transaminases, alkaline phosphatase, total bilirubin, and reduced total protein. Treatment with the extract restored hepatic integrity in a dose-dependent fashion.

Hepatic Parameter	Normal Control	Disease (Gentamicin)	Standard (Silymarin)	MOE Test-1 (300 mg/kg)	MOE Test-2 (600 mg/kg)
ALT (U/L)	60.17 ± 3.98	147.20 ± 3.40	82.50 ± 2.63	129.30 ± 2.67	102.80 ± 2.60
AST (U/L)	93.67 ± 4.43	172.20 ± 7.25	120.50 ± 4.55	155.20 ± 4.63	143.50 ± 5.97
ALP (U/L)	136.70 ± 4.36	315.80 ± 6.54	163.80 ± 3.98	272.00 ± 6.33	216.00 ± 8.62
Total Protein (g/dL)	6.15 ± 0.22	2.28 ± 0.21	5.27 ± 0.14	3.73 ± 0.29	4.93 ± 0.24
Total Bilirubin (mg/dL)	0.17 ± 0.03	0.68 ± 0.03	0.32 ± 0.03	0.53 ± 0.05	0.40 ± 0.06

3.4. Histopathological Evaluation

Kidney sections from Gentamicin-treated mice displayed marked pathological features including severe glomerular congestion (+++), peritubular congestion (+++), interstitial edema (++), tubular casts, mononuclear cell infiltration (+++), and proximal tubular epithelial cell necrosis.

Administration of MOE 600 mg/kg noticeably attenuated these histopathological changes, preserving tubular and glomerular structural integrity.



Histopathological Feature	Control	Gentamicin Toxicant	MOE 300 mg/kg	MOE 600 mg/kg	Standard
Glomerular Congestion	—	+++	++	+	+
Peritubular Congestion	—	+++	++	+	—
Blood Vessel Congestion	—	+++	++	++	—
Interstitial Edema	—	++	+	+	—
Inflammatory Cells	—	+	+	—	—
Mononuclear Infiltration	—	+++	++	+	+
Tubular Casts	—	+	+	—	—

Scoring: — (Absent), + (Mild), ++ (Moderate), +++ (Severe).

4. DISCUSSION

Drug-induced nephrotoxicity occurs primarily due to drug accumulation within renal cortical proximal tubules, initiating membrane breakdown, intracellular ROS accumulation, and tubular cell necrosis. Gentamicin administration triggers rapid declines in glomerular filtration, leading to retention of nitrogenous wastes (creatinine, urea, BUN, uric acid) and progressive emaciation/weight loss.

In the present study, co-administration of the ethanolic root extract of *M. oleifera* (MOE 300 and 600 mg/kg) produced significant, dose-dependent reductions in elevated serum and urinary markers of renal impairment while attenuating body weight loss. Furthermore, MOE administration attenuated elevated liver enzyme activity (ALT, AST, ALP) and total bilirubin, while restoring serum total protein levels.

These organ-protective benefits are likely mediated by the plant's secondary metabolites—namely flavonoids, phenolic acids, tannins, and alkaloids. These constituents act as free radical scavengers, inhibiting lipid peroxidation and cellular inflammation, thereby protecting renal tubular cells and hepatic parenchyma from drug-induced oxidative damage.

CONCLUSION

The ethanolic extract of *Moringa oleifera* root shows significant dose-dependent nephroprotective and hepatoprotective activities against gentamicin-induced toxicity. The extract preserves functional organ markers and histological architecture, likely through its antioxidant phytochemical constituents. *M. oleifera* root represents a potential therapeutic candidate for preventing drug-induced renal and hepatic injury.

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