RESEARCH PAPER

Evaluation of nephroprotective activity of Lepidium sativum in experimental animals

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ABSTRACT

Nephrotoxicity is a major clinical concern associated with the use of various therapeutic agents, particularly aminoglycoside antibiotics such as gentamicin. The present study aimed to evaluate the nephroprotective potential of the aqueous extract of Lepidium sativum seeds in gentamicin-induced nephrotoxicity in experimental animals. Nephrotoxicity was induced in Wistar rats by administering gentamicin (40 mg/kg, i.p.) for 14 days. The extract was administered orally at doses of 200 mg/kg and 400 mg/kg in both preventive and curative regimens. Renal function was assessed using biochemical markers including serum creatinine and blood urea nitrogen (BUN), along with oxidative stress parameters such as malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), and catalase.

Gentamicin administration resulted in significant renal impairment, evidenced by elevated BUN, serum creatinine, and MDA levels, along with decreased antioxidant enzyme levels. Treatment with Lepidium sativum significantly attenuated these changes, particularly at the dose of 400 mg/kg in the preventive group, which showed marked restoration of biochemical and antioxidant parameters. Histopathological evaluation further confirmed the protective effect by demonstrating reduced tubular damage and preservation of renal architecture.

The findings suggest that Lepidium sativum possesses significant nephroprotective activity, likely mediated through its antioxidant and cytoprotective properties. Thus, it may serve as a potential natural therapeutic agent for the prevention of drug-induced nephrotoxicity.

Keywords: Nephrotoxicity; Lepidium sativum; Gentamicin; Antioxidant; Renal protection; Oxidative stress.

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1. Introduction

The kidney plays a critical role in maintaining physiological homeostasis by regulating electrolyte

balance, fluid volume, and excretion of metabolic waste products. Due to its high blood flow, active transport mechanisms, and concentrating ability, the kidney is particularly vulnerable to toxic insults from drugs and environmental chemicals[1].

Nephrotoxicity is a major clinical problem characterized by functional and structural damage to renal tissues following exposure to xenobiotics such as antibiotics, anticancer agents, and non-steroidal anti-inflammatory drugs. It is estimated that a significant proportion of acute kidney injury cases are drug-induced, with aminoglycosides like gentamicin being among the most common causative agents[2]. Gentamicin-induced nephrotoxicity is primarily associated with accumulation of the drug in renal tubular cells, leading to oxidative stress, inflammation, and apoptosis[3].

The pathogenesis of nephrotoxicity involves excessive generation of reactive oxygen species (ROS), lipid peroxidation, mitochondrial dysfunction, and activation of inflammatory pathways. These events ultimately result in tubular necrosis, reduced glomerular filtration rate, and elevated levels of biochemical markers such as serum creatinine and blood urea nitrogen[4]. Oxidative stress has been recognized as a central mechanism in drug-induced renal injury, making antioxidants a promising therapeutic strategy[5].

In recent years, there has been growing interest in the use of medicinal plants for the prevention and treatment of nephrotoxicity due to their safety, affordability, and multitarget mechanisms. Herbal medicines are rich in bioactive compounds such as flavonoids, alkaloids, phenolics, and glycosides, which possess potent antioxidant and anti-inflammatory properties[6]. Several studies have demonstrated the nephroprotective effects of plant extracts through modulation of oxidative stress and restoration of endogenous antioxidant systems[7].

Among various medicinal plants, *Lepidium sativum* Linn. (family Brassicaceae), commonly known as garden cress, has been traditionally used for the treatment of various ailments including renal disorders, inflammation, and metabolic diseases. Phytochemical investigations have revealed that *Lepidium sativum* seeds contain flavonoids, alkaloids, proteins, and other bioactive constituents that contribute to its pharmacological activities[8].

Recent experimental studies have reported that *Lepidium sativum* exhibits significant antioxidant and nephroprotective effects in drug-induced renal injury models. The plant extract has been shown to reduce serum creatinine and urea levels, improve antioxidant enzyme status, and attenuate histopathological alterations in kidney tissues[9]. Furthermore, advanced studies indicate that *Lepidium sativum*, alone or in combination with antioxidants such as glutathione, can significantly reduce oxidative stress, inflammatory markers, and cellular apoptosis in gentamicin-induced acute renal failure models[10].

Despite these promising findings, there remains a need for systematic evaluation of the nephroprotective potential of *Lepidium sativum* using well-established experimental models. Therefore, the present study was undertaken to investigate the protective effect of aqueous extract of *Lepidium sativum* seeds against gentamicin-induced nephrotoxicity in experimental animals.

2. Materials and Methods

2.1 Plant Material and Authentication

Seeds of *Lepidium sativum* Linn. (family Brassicaceae) were procured from a local market and authenticated by a qualified taxonomist at a recognized research institute. A voucher specimen was deposited for future reference. The plant material was cleaned, shade dried, and coarsely powdered using a mechanical grinder[11].

2.2 Preparation of Extract

The powdered seeds were subjected to aqueous extraction by boiling in distilled water for 10 minutes, followed by cooling and filtration. The filtrate was centrifuged, and the supernatant was collected as the aqueous extract. The extract was freshly prepared prior to administration to ensure stability and efficacy[12].

2.3 Chemicals and Reagents

Gentamicin, thiobarbituric acid, reduced glutathione (GSH), epinephrine, and other analytical grade chemicals were procured from standard suppliers. All reagents used in the study were of analytical grade and prepared according to standard protocols[13].

2.4 Experimental Animals

Male Wistar albino rats weighing 180–250 g were used for the study. The animals were housed under standard laboratory conditions (temperature 25 ± 2°C, relative humidity 50–60%, 12 h light/dark cycle) with free access to standard pellet diet and water.

All experimental procedures were conducted in accordance with the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India, and were approved by the Institutional Animal Ethics Committee (IAEC)[14].

2.5 Acute Oral Toxicity Study

Acute oral toxicity study of the aqueous extract of *Lepidium sativum* was carried out as per OECD

guideline 423. The extract was administered orally at doses up to 2000 mg/kg body weight, and animals were observed for 24 hours for signs of toxicity and mortality. No mortality or adverse effects were observed, indicating the safety of the extract[15].

2.6 Experimental Design

Nephrotoxicity was induced using gentamicin in Wistar rats. Animals were randomly divided into six groups (n = 6):

Group	Treatment
I	Normal control (saline)
II	Gentamicin (40 mg/kg, i.p.)
III	Gentamicin + LS (200 mg/kg, p.o.)
IV	Gentamicin + LS (400 mg/kg, p.o., preventive)
V	Gentamicin + LS (400 mg/kg, p.o., curative)
VI	LS alone (400 mg/kg, p.o.)

Gentamicin was administered intraperitoneally for 14 days to induce nephrotoxicity. The extract was administered orally once daily[16].

2.7 Evaluation Parameters

2.7.1 Body Weight and Organ Weight

Body weight of animals was recorded before and after the experimental period. After sacrifice, kidneys were excised, weighed, and kidney weight/body weight ratio was calculated as an indicator of renal damage[17].

2.7.2 Biochemical Analysis

Blood samples were collected via retro-orbital puncture under light anesthesia. Serum was separated by centrifugation and analyzed for:

- Blood Urea Nitrogen (BUN)
- Serum Creatinine

These parameters were estimated using standard diagnostic kits as indicators of renal function[18].

2.7.3 Estimation of Lipid Peroxidation (MDA)

Lipid peroxidation in kidney tissue was estimated by measuring malondialdehyde (MDA) levels using thiobarbituric acid reactive substances (TBARS) method. Increased MDA levels indicate oxidative stress and cellular damage[19].

2.7.4 Estimation of Antioxidant Enzymes

Kidney homogenates were used for estimation of endogenous antioxidant enzymes:

- Reduced Glutathione (GSH)
- Superoxide Dismutase (SOD)
- Catalase

Standard spectrophotometric methods were employed for enzyme estimation, reflecting antioxidant defence status[20].

2.7.5 Histopathological Examination

Kidney tissues were fixed in 10% formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Slides were examined under a light microscope for structural changes such as tubular necrosis, interstitial inflammation, and glomerular damage[16].

2.8 Statistical Analysis

Data were expressed as mean ± SEM (n = 6). Statistical analysis was performed using one-way ANOVA followed by Bonferroni's multiple comparison test. A value of $p < 0.05$ was considered statistically significant.

3. Results

3.1 Phytochemical Screening

Preliminary phytochemical analysis of the aqueous extract of *Lepidium sativum* revealed the presence of various bioactive constituents including alkaloids, glycosides, flavonoids, proteins, and carbohydrates, which may contribute to its nephroprotective activity.

Table 3.1: Phytochemical Screening of *Lepidium sativum* Extract

Phytoconstituents	Result
Alkaloids	Present
Glycosides	Present
Flavonoids	Present
Carbohydrates	Present
Proteins	Present
Tannins	Absent
Steroids	Absent

3.2 Effect on Body Weight

Gentamicin-treated rats showed a significant decrease in body weight compared to the normal group. Treatment with *Lepidium sativum* (200 mg/kg and 400 mg/kg) significantly improved body weight.

Table 3.2: Effect on Body Weight

Group	Treatment	Body Weight Change (g)
I	Normal	18.32 ± 2.36
II	Gentamicin	-8.66 ± 2.45***
III	GM + LS (200 mg/kg)	-1.86 ± 2.32**
IV	GM + LS (400 mg/kg, P)	1.83 ± 2.87***
V	GM + LS (400 mg/kg, C)	-2.32 ± 3.56**
VI	LS (400 mg/kg)	17.17 ± 3.28

3.3 Kidney Weight/Body Weight Ratio

Gentamicin significantly increased kidney weight/body weight ratio, indicating renal damage. Treatment with *Lepidium sativum* reduced this ratio.

Table 3.3: Kidney Weight/Body Weight Ratio

Group	Treatment	Ratio (mg/g)

I	Normal	5.20 ± 0.16
II	Gentamicin	9.82 ± 0.42***
III	GM + LS (200 mg/kg)	8.48 ± 0.14**
IV	GM + LS (400 mg/kg, P)	5.90 ± 0.24***
V	GM + LS (400 mg/kg, C)	8.57 ± 0.13**
VI	LS (400 mg/kg)	5.41 ± 0.08

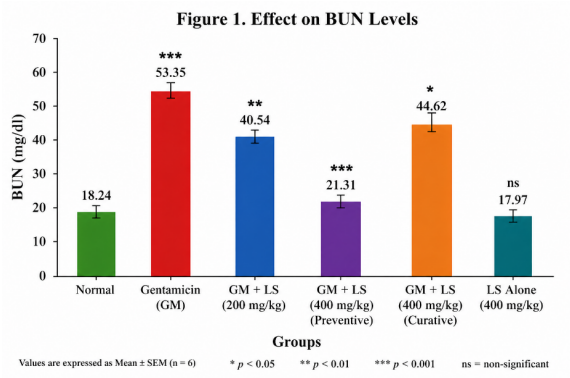
3.4 Biochemical Parameters

3.4.1 Blood Urea Nitrogen (BUN)

Gentamicin caused a significant increase in BUN levels, which was significantly reduced by *Lepidium sativum* treatment.

Table 3.4: Effect on Blood Urea Nitrogen

Group	BUN (mg/dl)
Normal	18.24 ± 1.03
Gentamicin	53.35 ± 1.82***
GM + LS (200 mg/kg)	40.54 ± 2.74**
GM + LS (400 mg/kg, P)	21.31 ± 1.90***
GM + LS (400 mg/kg, C)	44.62 ± 3.16*
LS alone	17.97 ± 1.36



3.4.2 Serum Creatinine

Gentamicin significantly elevated serum creatinine levels, which were reduced by *Lepidium sativum*, particularly in the preventive group.

Table 3.5: Effect on Serum Creatinine

Group	Creatinine (mg/dl)
Normal	0.62 ± 0.02
Gentamicin	1.51 ± 0.09***
GM + LS (200 mg/kg)	1.11 ± 0.08**
GM + LS (400 mg/kg, P)	0.80 ± 0.09***
GM + LS (400 mg/kg, C)	1.24 ± 0.08 (ns)
LS alone	0.61 ± 0.05

3.5 Oxidative Stress Parameters

3.5.1 Lipid Peroxidation (MDA)

MDA levels were significantly increased in the gentamicin group, indicating oxidative stress. Treatment with *Lepidium sativum* significantly reduced MDA levels.

Table 3.6: Effect on MDA Levels

Group	MDA (nmol/g tissue)
Normal	69.23 ± 6.34
Gentamicin	118.39 ± 6.16***
GM + LS (200 mg/kg)	83.11 ± 5.80**
GM + LS (400 mg/kg, P)	71.64 ± 9.57***
GM + LS (400 mg/kg, C)	87.08 ± 3.38*
LS alone	70.42 ± 7.63

3.5.2 Reduced Glutathione (GSH)

Gentamicin significantly decreased GSH levels, whereas *Lepidium sativum* treatment restored antioxidant levels.

Table 3.7: Effect on GSH Levels

Group	GSH (mg/g tissue)
Normal	172.64 ± 3.56
Gentamicin	35.17 ± 2.64***
GM + LS (200 mg/kg)	53.21 ± 3.21**
GM + LS (400 mg/kg, P)	72.46 ± 2.93***
GM + LS (400 mg/kg, C)	46.47 ± 3.01 (ns)
LS alone	161.37 ± 3.01

3.5.3 Superoxide Dismutase (SOD)

SOD levels were significantly decreased in the gentamicin group and improved with treatment.

Table 3.8: Effect on SOD Levels

Group	SOD (U/g tissue)
Normal	100.42 ± 1.38
Gentamicin	54.01 ± 1.98***
GM + LS (200 mg/kg)	67.65 ± 3.31**
GM + LS (400 mg/kg, P)	87.02 ± 3.35***
GM + LS (400 mg/kg, C)	63.60 ± 1.95*
LS alone	96.88 ± 2.56

3.5.4 Catalase Activity

Catalase activity decreased in the gentamicin group and was significantly restored by *Lepidium sativum*.

Table 3.9: Effect on Catalase Activity

Group	Catalase
Normal	1158 ± 8.05
Gentamicin	887.51 ± 5.80***
GM + LS (200 mg/kg)	927.87 ± 5.63*
GM + LS (400 mg/kg, P)	967.14 ± 5.63***
GM + LS (400 mg/kg, C)	919.33 ± 6.19 (ns)
LS alone	1124.84 ± 15.62

4. Discussion

Drug-induced nephrotoxicity remains a major limitation in clinical therapeutics, particularly with the use of aminoglycoside antibiotics such as gentamicin. Gentamicin is known to accumulate in renal proximal

tubular cells, leading to oxidative stress, inflammation, and subsequent tubular necrosis[21]. The present study was designed to evaluate the nephroprotective potential of *Lepidium sativum* against gentamicin-induced renal injury, and the findings clearly demonstrate its significant protective effects.

In the current investigation, administration of gentamicin resulted in a marked increase in serum creatinine and blood urea nitrogen (BUN), which are established biomarkers of renal dysfunction. This elevation indicates impaired glomerular filtration rate and renal damage[22]. Treatment with *Lepidium sativum*, particularly at the dose of 400 mg/kg in the preventive group, significantly reduced these biochemical parameters, suggesting restoration of renal function.

Gentamicin-induced nephrotoxicity is primarily mediated through the generation of reactive oxygen species (ROS), leading to oxidative stress and lipid peroxidation. Increased levels of malondialdehyde (MDA) observed in the present study confirm enhanced lipid peroxidation and membrane damage[23]. The significant reduction in MDA levels following treatment with *Lepidium sativum* indicates its potent antioxidant activity and ability to inhibit free radical-mediated damage.

The antioxidant defense system plays a crucial role in protecting renal tissues from oxidative injury. In this study, gentamicin administration significantly decreased the levels of endogenous antioxidants such as reduced glutathione (GSH), superoxide dismutase (SOD), and catalase. This depletion reflects an imbalance between oxidant and antioxidant systems[24]. Treatment with *Lepidium sativum* restored these antioxidant enzyme levels, thereby enhancing cellular defense mechanisms against oxidative stress.

The nephroprotective effect of *Lepidium sativum* can be attributed to its rich phytochemical composition, including flavonoids, alkaloids, and phenolic compounds. These bioactive constituents are known to possess strong antioxidant and free radical scavenging properties, which contribute to their protective effects against renal damage[25]. Flavonoids, in particular, are reported to stabilize cellular membranes, reduce oxidative stress, and inhibit inflammatory pathways involved in nephrotoxicity[26].

Histopathological findings further support the biochemical results. Gentamicin-treated rats showed severe tubular necrosis, interstitial inflammation, and glomerular damage, which are characteristic features of acute renal injury[27]. In contrast, animals treated with *Lepidium sativum* exhibited significant improvement in renal architecture with reduced tubular degeneration and inflammation, confirming its protective effect at the tissue level.

An important observation in the present study is that the preventive treatment was more effective than the curative approach. This suggests that *Lepidium sativum* may be more beneficial in preventing oxidative damage rather than reversing established renal injury. Similar findings have been reported in previous studies where early antioxidant intervention showed superior protection against nephrotoxicity[28].

Overall, the results of this study are consistent with earlier reports demonstrating the nephroprotective potential of plant-derived antioxidants. The ability of *Lepidium sativum* to reduce oxidative stress, restore antioxidant defenses, and improve renal function markers highlights its therapeutic potential in the management of drug-induced nephrotoxicity[29].

Furthermore, the observed effects may also involve modulation of inflammatory pathways and inhibition of apoptosis, although these mechanisms require further investigation at the molecular level[30-49].

5. Conclusion

The present study demonstrates that the aqueous extract of *Lepidium sativum* seeds possesses significant nephroprotective activity against gentamicin-induced renal toxicity in experimental animals. The extract effectively reduced elevated levels of serum creatinine and blood urea nitrogen, attenuated lipid peroxidation, and restored endogenous antioxidant defences, including glutathione, superoxide dismutase, and catalase. Histopathological findings further confirmed the protective effect on renal architecture.

Among the tested doses, 400 mg/kg (preventive regimen) exhibited superior nephroprotective efficacy, indicating its potential role in preventing oxidative renal damage. The observed effects are likely mediated through antioxidant and cytoprotective mechanisms.

Overall, *Lepidium sativum* may serve as a promising natural therapeutic agent for the prevention of drug-induced nephrotoxicity. Further studies are warranted to elucidate its molecular mechanisms and to validate its clinical applicability.

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