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Evaluation of Oxidative Stress-related Biochemical Parameters in Experimentally Induced Hepatotoxicity by *Lantana camara* in Female Wistar Rats and its Amelioration with Neem (*Azadirachta indica*) Leaf Extract

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ABSTRACT

The experiment was conducted during August, 2024 to January, 2025 at College of Veterinary and Animal Science, Navania Vallabhnagar, Udaipur, Rajasthan, India to evaluate oxidative stress-related biochemical parameters in female Wistar rats subjected to *Lantana camara* induced hepatotoxicity, and to assess the hepatoprotective effects of *Azadirachta indica* (Neem) leaf extract against the induced toxicity. Five experimental groups were established. Group I served as the control, Group II received only Neem extract, and Group III was administered *Lantana camara* to induce hepatotoxicity. Group IV was pre-treated with Neem extract for 10 days prior to *Lantana camara* exposure, while Group V received Neem extract following *Lantana camara*–induced hepatotoxicity (post-treatment group). Biochemical markers, including lipid peroxidation (LPO), superoxide dismutase (SOD), glutathione reductase (GR), and catalase (CAT), were measured to assess oxidative stress and antioxidant defense mechanisms. The results showed that *Lantana camara* significantly increased oxidative stress, as evidenced by elevated LPO levels and decreased antioxidant enzyme activities. The study also revealed that *L. camara* induced significant hepatic damage mediated through oxidative stress, while neem leaf extract provided hepatoprotection by restoring antioxidant enzyme levels and preserving liver histoarchitecture.

KEYWORDS: Wistar rats, *Lantana camara*, oxidative stress, hepatotoxicity, neem

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1. INTRODUCTION

Hepatotoxicity caused by *Lantana camara* is a major veterinary concern, primarily due to the presence of lantadenes—compounds known to induce oxidative stress and impair liver function. This oxidative stress results in lipid peroxidation, enzyme dysfunction, and tissue degeneration. *Azadirachta indica* (Neem), a medicinal plant, is widely recognized for its hepatoprotective and antioxidant properties. This study aims to evaluate the biochemical changes associated with oxidative stress in *Lantana camara*–induced hepatotoxicity and to assess the protective effects of Neem leaf extract.

Lantana camara, an invasive plant species prevalent in India and several other regions, poses a significant threat to both plant and animal ecosystems. The red-flowered variety, *L. camara* var. aculeata, is particularly toxic to grazing animals. The plant spreads rapidly via cuttings, stumps, and seeds dispersed by birds and migrating livestock. Originally native to the Caribbean, Central America, and northern South America, *Lantana camara* has now invaded more than 60 countries. Early studies identified two principal toxic constituents-lantadenes A (LA) and B (LB); among them, LA is particularly toxic to sheep, while LB exhibits no evident toxicity (Sharma et al., 2007). The consumption of lantana leads to a decline in antioxidant defenses in animals, increasing their vulnerability to oxidative damage. Known for its hepatotoxic effects, *Lantana camara* disrupts hepatocellular function and triggers oxidative stress in grazing animals, ultimately resulting in liver injury (Sharma et al., 1982; Sharma et al., 2007).

Introduced to India in the 19th century, *Lantana camara* has proliferated extensively, especially in the Himalayan foothills (Sharma and Makkar, 1981). In Rajasthan-an agriculturally and livestock-dependent state-lantana infestations are widespread, particularly during droughts when grazing animals are more likely to ingest it (Singh, 2018). The Udaipur region, which receives an average annual rainfall of at least 900 mm, is especially prone to large-scale infestations (Ensbey, 2003). Climate change and global warming are expected to further facilitate the plant's spread due to increasingly favorable growth conditions (Zhang et al., 2014; Singh, 2018).

Oxidative stress is defined as an imbalance between the generation of reactive oxygen species (ROS) and the body's ability to neutralize them using antioxidant defenses. This imbalance causes damage to lipids, proteins, and DNA, playing a key role in cellular injury, inflammation, and organ dysfunction-especially liver toxicity (Yao et al., 2006). A critical indicator of oxidative damage is malondialdehyde (MDA), a lipid peroxidation byproduct that signifies cellular membrane disruption. Enzymatic antioxidants such as superoxide dismutase (SOD), glutathione reductase (GR), and catalase (CAT) are integral to the endogenous antioxidant system, helping to neutralize ROS and maintain redox homeostasis (Molina et al., 2003; Meister, 1984; Asghar et al., 2022). Upon ingestion, *Lantana camara* causes intrahepatic cholestasis and hepatocellular necrosis, which are accompanied by increased LPO and reduced antioxidant enzyme activity (Saini et al., 2007).

In contrast, *Azadirachta indica* (Neem) is rich in bioactive compounds like quercetin and azadirachtin and has demonstrated potent antioxidant and hepatoprotective properties (Chattopadhyay and Bandyopadhyay, 2005; Dkhil et al., 2012; Asghar et al., 2022). Neem has been shown to restore antioxidant enzyme activity, reduce oxidative stress, and ameliorate toxic liver injury (Rao et al., 2021; Singh et al., 2022). and protect liver function by modulating the redox balance and inhibiting free radical-mediated damage (Koul et al., 2007). Considering the oxidative damage induced by *Lantana camara* and the traditional hepatoprotective role of Neem (Jamshed et al., 2024), This study aims to evaluate the oxidative stress-related hepatotoxic effects of *L. camara* in female Wistar rats and the potential protective role of neem leaf extract. The study aimed to evaluate oxidative stress-related biochemical parameters in female Wistar rats following *Lantana camara* induced hepatotoxicity and to investigate the hepatoprotective effects of neem leaf extract against the resulting toxicity.

2. MATERIALS AND METHODS

The dose of *Lantana camara* leaf extract (450 mg/kg body weight) was selected based on one-tenth of its reported lethal dose (Rasool et al., 2024). *Azadirachta indica* (Neem) leaf extract was administered at a dose of 500 mg/kg body weight, as per previous literature. Both extracts were dissolved in distilled water and administered orally.

The thirty rats were randomly divided into five experimental groups to assess oxidative stress-related biochemical parameters:

- Group I (Control): Received distilled water for 28 days.
- Group II (Neem): Received Neem leaf extract for 28 days.
- Group III (*Lantana camara*): Received *Lantana camara* extract for 28 days to induce hepatotoxicity.
- Group IV (Pre-treatment): was Pre-treated with Neem extract for 10 days, followed by co-treatment with both Neem extract and *Lantana camara* for 28 days.
- Group V (Post-treatment): was Administered *Lantana camara* for 28 days to induce hepatotoxicity, followed by Neem extract for the subsequent 28 days.

This experimental design Table 1 was developed to evaluate the effects of Neem extract administered either before

Table 1: Experimental design

Group No.	Experimental groups	Duration of treatment
I.	Control (Received distilled water only)	28 days
II.	Neem leaf extract only	28 days
III.	*Lantana camara* leaf extract	28 days
IV.	Pre-treatment: Neem extract for 10 days, followed by co-treatment with *Lantana camara* and Neem for 28 days	38 days
V.	Post-treatment: *Lantana camara* for 28 days, followed by Neem extract for 28 days	28 days (Lantana)+28 days (Neem)

or after *Lantana camara*–induced toxicity, by measuring oxidative stress biomarkers across different treatment protocols.

2.2. Oxidative stress parameters analyzed

Lipid peroxidation (LPO) and the levels of antioxidant enzymes-superoxide dismutase (SOD), catalase (CAT), and glutathione reductase (GR)-were evaluated in liver tissue homogenates to assess oxidative stress.

2.3. Preparation of liver tissue homogenates

A 500 mg liver tissue sample was collected from each rat and placed in 5 ml of ice-cold phosphate-buffered saline (PBS, pH 7.4). The tissues were homogenized (10%) using a tissue homogenizer (MSW 346, IKA) under ice-cold conditions. The homogenates were then centrifuged at 3000 rpm for 10 minutes. The resulting supernatant was stored at -20 °C for subsequent oxidative stress analysis. The oxidative stress markers-LPO, SOD, CAT, and GR-were measured using standard spectrophotometric methods.

2.4. Lipid peroxidation (LPO)

Lipid peroxidation, an indicator of oxidative damage, was evaluated by measuring malondialdehyde (MDA) levels using the Thiobarbituric Acid Reactive Substances (TBARS) assay, as described by Fernanda et al. (2005).

2.5. Superoxide dismutase (SOD)

SOD activity was estimated following the method described by Madesh and Balasubramanian (1998).

2.6. Catalase (CAT)

Catalase activity was determined using the protocol established by Aebi (1984).

2.7. Glutathione reductase (GR)

GR activity was assessed using the method outlined by Goldberg and Spooner (1983). All oxidative stress marker analyses were performed using a UV–VIS double-beam spectrophotometer (Chino Scientific Instruments, Ajmer, India).

2.8. Clinical signs and mortality

All rats were observed daily for clinical signs, abnormal behaviour, and overall health status. Clinical observations and mortality were recorded throughout the duration of the experiment.

2.9. Body weight

The body weight of each rat was recorded weekly. Initial body weights were documented prior to dosing and used for dose-volume calculation and monitoring of weight changes during the study.

3.0. Histopathological examination

Liver tissues were fixed in 10% neutral-buffered formalin, embedded in paraffin, sectioned at 5 µm thickness, and stained with hematoxylin and eosin (H&E). Microscopic features assessed included hepatocellular necrosis, fatty degeneration, and inflammatory cell infiltration.

3. RESULTS AND DISCUSSION

3.1. Oxidative stress-related biochemical parameters

Liver tissue homogenates were prepared to evaluate oxidative stress markers; the mean values of LPO, SOD, GR, and catalase across groups were shown in Table 2. Similar biochemical profiling was adopted by Jamshed et al. (2024), who reported improved antioxidant enzyme activities with neem treatment in erythromycin induced hepatotoxicity in rats. Similarly, Sabra et al. (2025) demonstrated that

Table 2: Mean values of mean values of LPO, SOD, GR and catalase of rats of different experimental groups

Group	LPO (Mean±SE)	SOD (Mean±SE)	GR (Mean±SE)	Catalase (Mean±SE)
I. (Control)	45.84^{c}±0.99	10.57^{b}±0.77	15.88^{b}±2.80	8.84^{b}±0.33
II. (Neem @ 500 mg kg^{-1} b.wt.)	30.50^{b}±0.93	10.71^{b}±0.84	16.61^{b}±1.86	9.11^{b}±0.81
III. (*Lantana camara* @ 450 mg kg^{-1} b.wt.)	60.38^{d}±1.27	4.49^{a}±0.60	4.84^{a}±0.36	3.32^{a}±0.72
IV. (Pre-treatment)	49.30^{c}±0.82	14.90^{b}±2.79	23.78^{b}±2.62	10.14^{b}±0.84
V. (Post-treatment)	22.47^{a}±0.94	12.81^{b}±1.03	18.97^{b}±2.92	9.40^{b}±0.30

Note: Values are expressed as Mean±Standard error (SE); Values in the same column with different superscripts (a, b, c, d) are significantly different (p<0.05); **: Highly significant (p<0.01)

nanoparticle formulations of neem extract significantly restored oxidative enzyme levels (SOD, MDA, GPx) in CCl_4 induced liver injury models. The administration of *Lantana camara* induced significant oxidative stress in female Wistar rats, as evidenced by elevated lipid peroxidation (LPO) levels and a marked reduction in the activity of key antioxidant enzymes such as superoxide dismutase (SOD), glutathione reductase (GR), and catalase. These findings are in agreement with earlier studies demonstrating oxidative hepatic damage associated with *L. camara* toxicity (Sharma et al., 1982; Saini et al., 2007). Treatment with *Azadirachta indica* (neem) leaf extract, both as a pre-treatment and post-treatment strategy, significantly ameliorated oxidative stress parameters. Notably, pre-treatment provided more effective protection, likely due to the priming of endogenous antioxidant defense mechanisms prior to the onset of oxidative injury. In contrast, post-treatment facilitated biochemical recovery but was relatively less efficient in preventing initial damage.

3.2. Lipid peroxidation (LPO)

LPO levels significantly decreased in Group II compared to control. In contrast, Group III exhibited a significant increase. Groups IV and V showed notable reductions, with Group V demonstrating the lowest LPO, indicating stronger post treatment effects. These observations were consistent with Jamshed et al. (2024), who observed decreased MDA/LPO levels following neem administration in erythromycin intoxicated rats Sabra et al. (2025) also reported that neem nanoparticles significantly reduced malondialdehyde compared to conventional forms in oxidative liver injury.

3.3. Superoxide dismutase (SOD)

SOD activity significantly decreased in Group III vs. control. Groups IV and V significantly restored SOD, with Group IV slightly higher than V (not statistically significant).

Comparable findings were reported by Sabra et al. (2025), where neem nanoparticles markedly elevated SOD activity in CCl_4-induced hepatotoxicity Jamshed et al. (2024) similarly found recovery of antioxidant enzyme levels, including SOD, after neem treatment.

3.4. Glutathione reductase (GR)

Group III exhibited a highly significant reduction in GR. Treatment with neem in IV and V significantly restored GR, with Group IV marginally higher (not significant).

This trend aligned with the cisplatin neem study of Dkhil et al. (2013), cited in earlier reviews), where neem leaves extract significantly increased GR activity in rats. Sabra et al. (2025) also documented GR recovery with neem nanoparticles in CCl_4 models

3.5. Catalase

Catalase activity significantly declined in Group III. Neem pre and post treatment significantly improved CAT activity, with Group IV slightly higher than V (not significant). Studies such as Jamshed et al. (2024) and Sabra et al. (2025) similarly reported catalase improvements in neem treated hepatotoxicity models.

The enzymes SOD, GR, and catalase serve essential roles in cellular defense against reactive oxygen species (ROS), maintaining redox balance and preventing oxidative damage. The restoration of these enzymes in neem-treated groups reflects improved oxidative homeostasis and attenuation of lipid peroxidation. This confirms neem's dual role as both a preventive and therapeutic agent against oxidative hepatotoxicity induced by *L. camara.*

3.6. Clinical signs

Daily monitoring revealed that Groups I, II, and IV showed no clinical signs nor mortality. Group III exhibited reduced feed/water intake after day 14 with asthenia, lameness, epistaxis, alopecia, and diarrhea. Group V's intake was slightly decreased; forelimb swelling was noted, but other signs were absent as shown in Table 3. These systemic symptoms mirrored lantana toxicity reports in guinea pigs, goats, and rats in earlier studies, but recent reviews reaffirm clinical signs such as anorexia, lameness, and diarrhea in lantana exposed animals. Neem pre treatment prevented

Table 3: Clinical signs across experimental groups

Clinical Signs	Group I (Control)	Group II (*Azadirachta indica*)	Group III (*Lantana camara*)	Group IV (Pre-treatment)	Group V (Post-treatment)
Feed and water intake	Normal	Normal	Decreased after day 14	Normal	Slightly decreased
Asthenia	Absent	Absent	Present	Absent	Absent
Lameness	Absent	Absent	Present	Absent	Absent, but swelling on forelimb
Epistaxis	Absent	Absent	Present	Absent	Absent
Alopecia	Absent	Absent	Slightly present	Absent	Absent
Diarrhoea	Absent	Absent	Present	Absent	Absent

most disturbances, aligning with observations in neem hepatoprotection Jamshed et al., 2024.The present study also documented a range of clinical signs in *L. camara*-exposed rats, including reduced feed and water intake, asthenia, lameness, epistaxis, diarrhea, and mild alopecia. These observations are consistent with classical symptoms of lantana poisoning reported across species. Anorexia has been frequently observed in ruminants (Dhillon and Paul, 1971), rats (Pass et al., 1979), guinea pigs (Sharma et al., 1982), and goats (Obwolo et al., 1990). Neuromuscular symptoms such as hind limb weakness have been reported in cattle (Seawright and Allen, 1972), while reduced feed intake has been described in sheep (McSweeney and Pass, 1983). Similarly, gastrointestinal disturbances including diarrhoea and dehydration have been noted in goats (Obwolo et al., 1990), which correlate with the enteric symptoms observed in our study. Hepatic damage resulting from *L. camara* exposure underline systemic symptoms such as weight loss and bleeding. Epistaxis observed in affected rats reflect underlying coagulopathy secondary to liver dysfunction, as also noted by Chakrabarti (2006). Dermatological findings like dermatitis and alopecia, similar to those seen in our rats, have been reported in goats (Mathew et al., 2013) and calves (Srikanth and Kumar, 2013), further substantiating the toxic profile of *L. camara*. Rats treated with neem extract showed notably milder clinical symptoms compared to the untreated *L. camara* group, highlighting neem's protective role in mitigating systemic toxicity. The attenuation of clinical signs suggests that neem not only reduces biochemical and histological damage but also improves overall physiological resilience against lantana toxicity.

3.7. Body weight

No significant changes in daily body weight were observed across the experimental groups shown in Table 4. but weekly averages revealed differences: Group IV had significant weight gain vs. V by day 7 and 21; Group III showed significant weight loss by day 28 vs. all other groups; Groups II and IV displayed significant gains vs. III; Groups III and V had overall weight reduction vs. control. Body weight declines in lantana models have been documented historically, and similar weight preservation with neem

Table 4: Mean body weight (g) of rats in all experimental groups

Groups	0 days (Mean±SE	7 days (Mean±SE)	14 days (Mean±SE)	21 days (Mean±SE)	28 days (Mean±SE)	35 days (Mean±SE)	38 days (Mean±SE)
I. (Control)	139.33±6.47	150^b±5.77	161.33±6.14	171.50^c±5.56	179^a±5.81	–	–
II. (Neem 500 mg/kg b.wt.)	129.00±2.58	139^ab±2.02	151.50±2.78	163.33^bc±3.95	177.33^a±6.12	–	–
III. (*Lantana camara* 450 mg/kg b.wt.)	134.00±2.34	142.50^ab±2.36	146.66±1.67	144.83^a±1.89	140.17^b±5.02	–	–
IV. (Pre-treatment)	133.00±1.06	144.5^ab±1.30	159.83±1.07	168.50^bc±1.33	177.50^a±2.73	183.5±3.04	194.33±1.98
V. (Post-treatment)	129.50±2.94	134^a±3.38	147.66±3.69	156^ab±4.24	166^a±4.44	–	–

Note: Values are expressed as Mean±Standard error (SE); Values in the same column with different superscripts (a, b, c, d) are significantly different (p<0.05); **: Highly significant (p<0.01)

treatment has been reported in recent studies Jamshed et al., 2024; Sabra et al., 2025. A progressive decline in body weight was also recorded in *L. camara*-treated rats, likely resulting from impaired nutrient absorption and poor feed conversion efficiency. Similar findings of weight loss due to lantana-induced toxicity have been reported in various species, including mice, rabbits, guinea pigs, and rats (Pour et al., 2011; Khafaji and Zubaedi, 2013; Soren et al., 2019; Kumar et al., 2018; Ali et al., 2023). However, Pingale et al. (2020) reported no significant weight loss with a 14-day exposure to *L. camara* leaf powder, suggesting that variations in dosage, preparation, or experimental conditions can influence toxicity outcomes.

3.8. Histopathological findings

Liver sections from *L. camara*-treated rats showed moderate to severe hepatocellular necrosis, fatty degeneration, and lymphocytic infiltration. Vacuolar degeneration and cytoplasmic eosinophilia were also prominent Sharma and Makkar, 1981. In contrast, neem-treated rats showed preserved liver architecture with only mild cellular changes. The combined treatment group showed reduced congestion, inflammation and necrosis compared to the *L. camara*-only group, highlighting the protective effect of neem (Figure 1) also reported by, Hossain and Rahman, 2013. *Azadirachta indica* has been extensively

documented for its antioxidant efficacy in experimental models of hepatic injury. By enhancing endogenous antioxidant enzyme activities and scavenging ROS, neem offers cytoprotection to hepatocytes (Chattopadhyay and Bandyopadhyay, 2005; Dkhil et al., 2012, 2013). Our results align with these findings, demonstrating that neem administration reduced malondialdehyde (MDA) levels while increasing GSH, SOD, and catalase activities in the liver. Additionally, histopathological improvements, such as reduced hepatocellular necrosis and inflammation, further support neem's hepatoprotective potential.

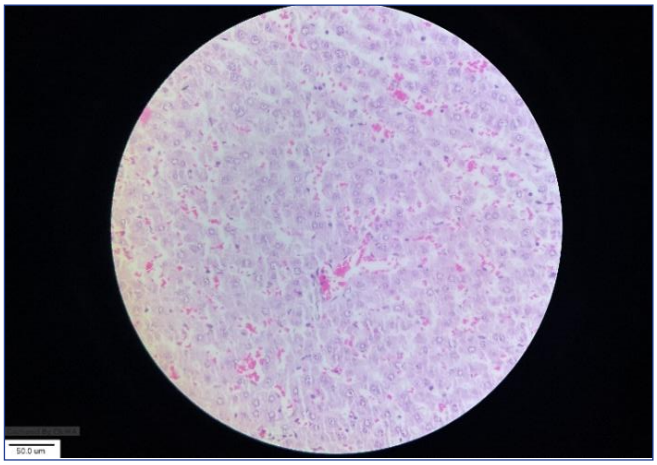

Figure 1: Photomicrograph of liver (V group) showings mild congestion, H&E Staining, 100x

4. CONCLUSION

Neem (*Azadirachta indica*) extract demonstrated showed significant hepatoprotective effects against *Lantana camara*–induced hepatotoxicity by reducing oxidative stress and restoring the activities of key antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione reductase (GR). Treatment with Neem extract decreased lipid peroxidation levels and preserve liver tissue integrity, thereby mitigating hepatic damage caused by the toxic effects of *Lantana camara*. These findings highlighted Neem's potential as a natural antioxidant and hepatoprotective agent.

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