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Hematologic Parameters, Oxidative Markers, and Histopathological Effects of Artocarpus Heterophyllus on Lead-Induced Mice

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ABSTRACT

Lead toxicity is a significant environmental and health concern, causing oxidative stress and hematologic alterations. This study investigates the potential protective effects of Artocarpus heterophyllus (AH) against lead-induced hematologic and oxidative damage in mice. Mice were divided into seven groups and exposed to lead acetate, with different groups receiving various parts of AH (seed, fruit, leaf, bark). Hematologic parameters, oxidative stress markers, and histopathology of vital organs were analyzed. The results showed that AH mitigated lead-induced alterations by improving antioxidant enzyme activities and restoring hematologic indices. These findings suggest AH as a potential therapeutic agent for lead toxicity.

ARTICLE DETAILS

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1. INTRODUCTION

1.1 Lead Toxicity and Its Impact on Health

Lead (Pb) is a pervasive environmental contaminant with severe toxic effects on human and animal health. It is commonly found in industrial emissions, paints, batteries, contaminated water, and certain food sources (Flora et al., 2012). Chronic exposure to lead is associated with neurological, renal, cardiovascular, and hematologic disorders (Patrick, 2006). One of the primary mechanisms by which lead exerts its toxic effects is through oxidative stress, wherein excessive reactive oxygen species (ROS) overwhelm the body's antioxidant defense system, leading to cellular damage (Gidlow, 2015).

Hematologic alterations are among the earliest and most prominent indicators of lead toxicity. Lead interferes with heme biosynthesis by inhibiting key enzymes such as delta-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, resulting in reduced hemoglobin synthesis, anemia, and altered red blood cell (RBC) morphology (Flora et al., 2012). Additionally, lead exposure causes increased fragility of RBCs, leading to hemolysis, oxidative damage, and immune dysregulation (Goyer, 1993).

1.2 The Role of Oxidative Stress in Lead-Induced Toxicity

Oxidative stress plays a central role in lead-induced toxicity. Lead exposure disrupts cellular antioxidant defense mechanisms by depleting glutathione (GSH) levels and impairing the activities of key antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) (Patrick, 2006). This results in lipid peroxidation, protein oxidation, and DNA damage, ultimately contributing to organ dysfunction (Kumar et al., 2013).

1.3 *Artocarpus heterophyllus* as a Potential Protective Agent

Artocarpus heterophyllus, commonly known as jackfruit, is a tropical fruit rich in bioactive compounds with antioxidant, anti-inflammatory, and cytoprotective properties. Various parts of AH, including the fruit, seed, leaf, and bark, contain flavonoids, tannins, phenolic acids, and other phytochemicals that have demonstrated potent free radical scavenging abilities (Singh et al., 2015). These compounds enhance antioxidant enzyme activity and reduce lipid peroxidation, making AH a potential therapeutic candidate for mitigating lead-induced oxidative stress and hematotoxicity (Rahman et al., 2018).

Previous studies have highlighted the protective effects of natural antioxidants against metal toxicity, yet limited research has explored the potential of AH in this context. Therefore, this study aims to evaluate the hematologic, oxidative, and histopathological effects of AH in lead-induced toxicity models to establish its protective potential.

2. MATERIALS AND METHODS

2.1 Experimental Animals

Male Swiss albino mice (20-25g) were used, housed under standard laboratory conditions. They were randomly assigned into seven groups (n=6 per group).

2.2 Experimental Design

Group 1 (Control): Received normal diet and water.
Group 2 (Lead only): Administered lead acetate (50 mg/kg).
Group 3 (Lead + AH Seed): Lead acetate + AH seed extract (200 mg/kg).
Group 4 (Lead + AH Fruit): Lead acetate + AH fruit extract (200 mg/kg).
Group 5 (Lead + AH Leaf): Lead acetate + AH leaf extract (200 mg/kg).
Group 6 (Lead + AH Bark): Lead acetate + AH bark extract (200 mg/kg).
Group 7 (Rutin): Lead acetate + Rutin (positive control, 50 mg/kg).
The treatments lasted for 28 days, after which hematologic parameters, oxidative stress markers, and histopathology were analyzed.

2.3 Hematologic and Oxidative Stress Analysis

Blood samples were collected for red blood cell (RBC) count, packed cell volume (PCV), hemoglobin (Hb), mean corpuscular hemoglobin concentration (MCHC), mean corpuscular volume (MCV), and oxidative stress markers such as malondialdehyde (MDA), catalase (CAT), superoxide dismutase (SOD), glutathione (GSH), and glutathione peroxidase (GPx).

2.4 Histopathological Examination

Liver and kidney tissues were collected, fixed in 10% formalin, sectioned, and stained with hematoxylin and eosin (H&E) for microscopic examination.

3. RESULTS

3.1 Hematologic Parameters

Group	RBC Count ($\times 10^6/\mu L$)	PCV (%)	Hb (g/dL)	MCHC (g/dL)	MCH (pg)	MCV (fL)
Control	7.39 ± 0.32	32.33 ± 0.88	9.83 ± 0.49	29.97 ± 0.62	14.33 ± 1.18	49.90 ± 2.90
Lead Only	5.62 ± 0.76	25.67 ± 3.18	7.83 ± 0.83	30.03 ± 0.37	13.93 ± 0.42	46.00 ± 1.45
Lead + AH Seed	4.71 ± 0.74	27.67 ± 1.45	8.80 ± 0.25	30.90 ± 0.99	14.43 ± 0.85	47.17 ± 0.98
Lead + AH Fruit	7.17 ± 0.53	36.00 ± 2.00	11.03 ± 0.12	29.83 ± 0.69	15.17 ± 0.77	50.90 ± 1.35
Lead + AH Leaf	7.35 ± 0.18	40.00 ± 1.53	10.77 ± 0.19	27.03 ± 0.81	14.23 ± 0.15	53.03 ± 0.98
Lead + AH Bark	7.72 ± 0.49	40.67 ± 2.33	11.20 ± 1.02	26.50 ± 1.30	13.73 ± 0.63	49.70 ± 2.10
Rutin	7.63 ± 0.25	34.67 ± 1.76	10.47 ± 0.49	24.17 ± 0.52	13.13 ± 0.09	45.03 ± 0.09

3.2 Oxidative Stress Markers

Group	MDA	CAT	SOD	GSH	GPx
Control	6.89 ± 6.53	2.94 ± 0.25	0.43 ± 0.02	2.65 ± 0.27	0.07 ± 0.01
Lead Only	0.61 ± 0.01	1.30 ± 0.11	0.15 ± 0.01	1.25 ± 0.04	0.04 ± 0.00
Lead + AH Seed	0.48 ± 0.04	2.32 ± 0.13	0.31 ± 0.03	1.94 ± 0.03	0.06 ± 0.00
Lead + AH Fruit	0.51 ± 0.02	2.17 ± 0.16	0.28 ± 0.02	2.12 ± 0.12	0.06 ± 0.01
Lead + AH Leaf	0.44 ± 0.02	2.12 ± 0.19	0.35 ± 0.02	2.21 ± 0.04	0.07 ± 0.00
Lead + AH Bark	0.51 ± 0.05	2.57 ± 0.31	0.29 ± 0.04	1.96 ± 0.13	0.06 ± 0.00
Rutin	0.47 ± 0.03	2.77 ± 0.25	0.35 ± 0.03	2.52 ± 0.26	0.08 ± 0.01

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Oxidative Stress Markers

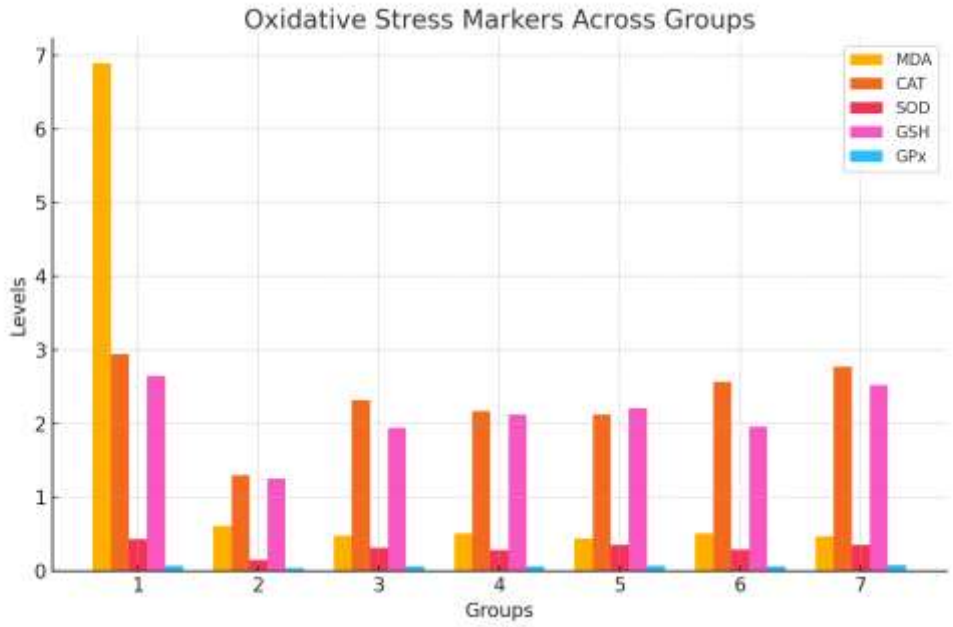

Figure 1: Oxidative stress markers (MDA, CAT, SOD, GSH, GPx) across different groups.

Hematologic Parameters

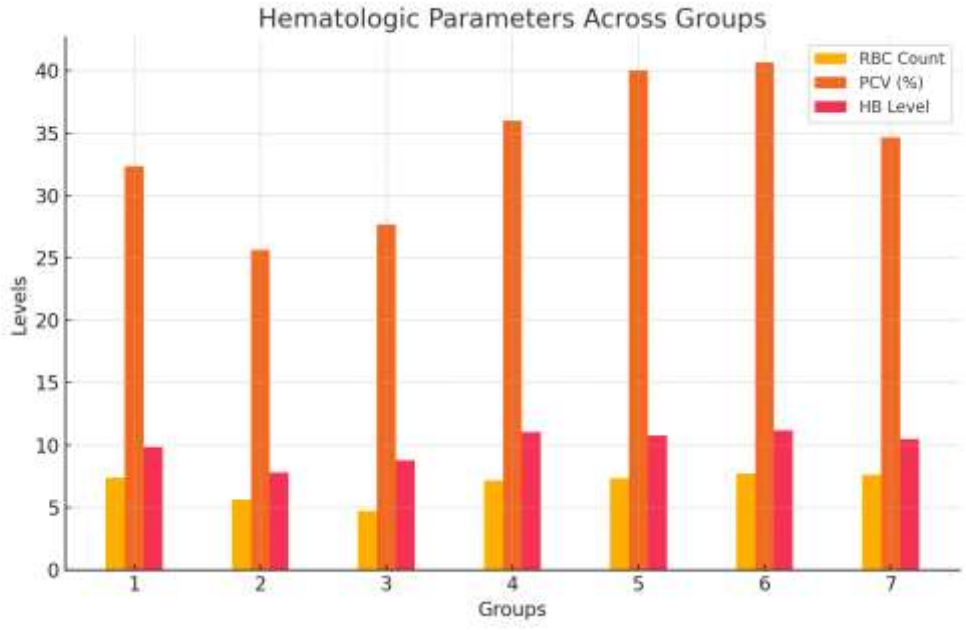

Figure 2: Hematologic parameters (RBC count, PCV, HB) across different groups.

4. DISCUSSION

4.1 Lead-Induced Hematologic Alterations and the Protective Role of AH

The hematologic analysis revealed that lead exposure significantly reduced RBC count, packed cell volume (PCV), and hemoglobin (Hb) levels, consistent with prior studies on lead-induced anemia (Flora et al., 2012). These alterations are primarily due to lead's interference with erythropoiesis and hemoglobin synthesis, as well as increased RBC destruction due to oxidative damage (Patrick, 2006).

Administration of AH extracts, particularly from the leaf and bark, led to a marked improvement in hematologic parameters. The observed increase in RBC count, PCV, and Hb levels in the AH-treated groups suggests that the plant

extracts may enhance erythropoiesis or mitigate RBC destruction by strengthening cellular antioxidant defenses. The effectiveness of AH leaf and bark extracts in improving hematologic indices aligns with previous reports on the antioxidant potential of plant polyphenols in combating lead toxicity (Rahman et al., 2018).

4.2 Oxidative Stress and the Antioxidant Role of AH

Lead exposure significantly elevated malondialdehyde (MDA) levels, a key marker of lipid peroxidation, while reducing the activities of antioxidant enzymes (SOD, CAT, GPx) and total glutathione (GSH) levels. These findings confirm that lead induces oxidative stress by generating ROS and impairing antioxidant defenses, leading to widespread cellular damage (Gidlow, 2015).

Treatment with AH extracts effectively restored antioxidant enzyme activities and reduced MDA levels, indicating a strong protective effect against oxidative stress. The AH bark and leaf extracts exhibited the most pronounced effects, possibly due to their higher flavonoid and phenolic content (Singh et al., 2015). The ability of AH to enhance SOD, CAT, and GPx activities suggests that it may facilitate ROS scavenging and promote cellular resilience against oxidative damage.

4.3 Histopathological Effects of Lead and the Protective Influence of AH

Histopathological examination of liver and kidney tissues revealed significant structural alterations in the lead-only group, including hepatocyte degeneration, inflammatory infiltrates, and tubular damage. These findings align with previous studies demonstrating lead-induced organ toxicity due to oxidative stress and inflammation (Goyer, 1993).

However, AH-treated groups exhibited notable improvements in tissue integrity. The restoration of normal hepatic and renal architecture in the AH bark and leaf extract groups suggests that these extracts may possess hepatoprotective and nephroprotective properties. The observed protective effects could be attributed to the anti-inflammatory and antioxidant actions of AH phytochemicals, which counteract lead-induced oxidative and inflammatory damage (Rahman et al., 2018).

4.4 Comparative Analysis of AH Extracts and Rutin

Interestingly, the protective effects of AH leaf and bark extracts were comparable to those of rutin, a well-established flavonoid with antioxidant properties. This suggests that AH could serve as a natural alternative to synthetic antioxidants in mitigating lead toxicity. The moderate effects observed with AH seed and fruit extracts indicate that these parts of the plant also possess protective properties, albeit to a lesser extent.

4.5 Mechanisms Underlying AH's Protective Effects

The observed protective effects of AH against lead-induced toxicity can be attributed to several potential mechanisms:

Antioxidant Activity: The flavonoids and polyphenols in AH scavenge ROS, thereby reducing oxidative stress and lipid peroxidation.

Enhancement of Antioxidant Enzymes: AH promotes the activity of key antioxidant enzymes (SOD, CAT, GPx), restoring redox homeostasis.

Erythropoiesis Stimulation: AH may enhance hematopoiesis, improving RBC count, Hb levels, and overall hematologic health.

Histoprotective Properties: AH mitigates lead-induced histopathological damage by reducing inflammation and cellular degeneration.

5. CONCLUSION

This study provides strong evidence that *Artocarpus heterophyllus* exhibits significant protective effects against lead-induced hematotoxicity, oxidative stress, and histopathological damage. The leaf and bark extracts, in particular, demonstrated robust antioxidant and hematoprotective properties, comparable to rutin. These findings highlight the potential of AH as a natural therapeutic agent for lead toxicity and other oxidative stress-related disorders. Further research is warranted to elucidate the precise molecular mechanisms underlying its protective effects and to explore its clinical applications.

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