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Impact of Zinc acetate on Haematological parameters in Albino Rats

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Abstract

The hematological consequences of zinc poisoning in male albino rats (Rattus norvegicus) exposed to zinc acetate were studied. Group A (Healthy Control) was given sodium acetate (125 mg/kg), and Group B (Zinc-Exposed Group) was given zinc acetate (125 mg/kg) per orally for 60 days. Blood samples were drawn on days 0, 30, and 60 to be analyzed. The findings indicated remarkable differences in hematological values between the two groups. Group B had a lower red blood cell (RBC) count (6.32 ± 0.13 × 10^3/µl) than Group A (7.22 ± 0.13 × 10^3/µl), indicating compromised erythropoiesis or enhanced erythrocyte destruction. Hemoglobin (Hb) level was lower in Group B (11.85 ± 0.65 g/dl) than in Group A (12.66 ± 0.31 g/dl), indicating disturbed heme synthesis. Hematocrit percentage also fell remarkably in Group B (37.74 ± 1.15%) as opposed to Group A (41.24 ± 0.75%), consistent with the decreases found in RBC count and hemoglobin. Mean corpuscular volume (MCV) was increased in Group B (62.60 ± 2.27 μ^3) over Group A (55.12 ± 2.96 μ^3), indicating the existence of larger, perhaps immature red blood cells. Mean corpuscular hemoglobin (MCH) was lower in Group B (19.28 ± 0.69 pg) than in Group A (20.96 ± 0.26 pg), reflecting lower hemoglobin content per red blood cell. The results prove the deleterious effect of zinc toxicity on hematological values in rats and outline the possible health risks from extended zinc dissemination in the environment.

Keywords: Zinc toxicity, Hematological parameters, Erythropoiesis, Hemoglobin synthesis

Introduction

Industrialization and technological advancements in leading a country on the way to progress, can simultaneously put numerous stresses on individuals and nature. Man continued to explore new regions with an obstinate urge to surpass an irresistible urge for his welfare. The genius and unfulfilled greed for improvement enabled him to introduce newer goods in his life. But this boundless progress took a notorious toll on the natural systems. Present-day industrial and technological operations have been directly and indirectly accountable for emission of

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poisonous pollutants leading to a spectacular surge in concentration of numerous poisonous chemicals, especially heavy metals, in the environment that could prove hazardous to the well-being of man and animals. Growing cases of plumbism are being reported among children of urban dwellers on account of increased levels of zinc in the environment. However, owing to greater dissemination of zinc in the environment from industries, smelters and other stationary and mobile sources, concern regarding the toxicological importance of zinc has now moved towards the potential health risks in the general population of man and animals.

Zinc has pronounced toxic actions on the nervous system, circulatory system and kidney. Cutaneous and hepatic manifestations of metal toxicity from heavy metal contaminated ground water occur among the vast population of different region of India. Prolonged use like fowler's solution has been reported to induce hepatic lesions. Though the link between zinc toxicity and hepatic fibrosis has been strongly established, no clear mechanism of liver injury has been explained.

Material and Methods:

The research used male albino rats (Rattus norvegicus) obtained from a laboratory, which were exposed to a 60-day acclimatization period with baseline hematological assessment. Rats weighing 150–300 grams were marked with yellow stain patterns (back, head, tail, or combinations) for identification, with one left unmarked. They were segregated into two groups: Group A (Healthy Control) was administered sodium acetate (125 mg/kg), and Group B (Zinc-Exposed Group) was given zinc acetate (125 mg/kg), both oral administration by gavage for 60 days, as per Zbinden (1976). Blood samples were obtained through retro-orbital plexus puncture (Sorz and Buckner, 1964) on days 0, 30, and 60. The sample was divided into two aliquots; the first was placed in heparinized vials (pre-rinsed with hot nitric acid) at -20°C in sealed containers for analysis.

Results and Discussion:

The haematological indices of male albino rats from the study demonstrated clear-cut differences between Group A (Healthy Control, treated with sodium acetate) and Group B (Zinc-Exposed Group, treated with zinc acetate) following a 60-day experimental period.

Red Blood Cell (RBC) Count: Group A had an RBC count of $7.22 \pm 0.13 \times 10^3/\mu l$, which was higher than Group B's count of $6.32 \pm 0.13 \times 10^3/\mu l$ ($P < 0.05$). This decrease in RBC count in

Group B indicates that zinc acetate exposure can impair erythropoiesis or cause enhanced erythrocyte destruction, as evidenced by zinc-induced hematotoxicity.

Hemoglobin (Hb) Level: The level of hemoglobin in Group A was 12.66 ± 0.31 g/dl, as opposed to 11.85 ± 0.65 g/dl for Group B, with the variation being statistically significant ($P < 0.05$). The decreased Hb level in the zinc-exposed group suggests possible impairment of heme synthesis, a well-established consequence of zinc toxicity, which disrupts enzymes like delta-aminolevulinic acid dehydratase (ALAD).

Hematocrit Percentage: Hematocrit levels were 41.24 ± 0.75% in Group A and 37.74 ± 1.15% in Group B, the latter showing a significant fall ($P < 0.05$). This fall is consistent with the decreases seen in RBC count and hemoglobin, reinforcing the negative effect of zinc on total red blood cell mass and oxygen-carrying capacity.

Mean Corpuscular Volume (MCV): Group A had an MCV of 55.12 ± 2.96 μ^3, whereas the value in Group B was a statistically significant value of 62.60 ± 2.27 μ^3 ($P < 0.05$). An increased MCV in the exposed group indicates larger, potentially immature red blood cells, which could be indicative of compensatory erythropoiesis or faulty maturation process as a consequence of zinc poisoning.

Table 1: Effects of Zinc Acetate Administration on Haematological parameters of Albino Rat

Parameter	**Group A (Control)**	**Group B (Zinc Acetate Only)**	**Significance**
RBC Count (x 10^3/µl)	7.22 ± 0.13	6.32 ± 0.13	$P < 0.05$
Hb (g/dl)	12.66 ± 0.31	11.85 ± 0.65	$P < 0.05$
Haematocrit %	41.24 ± 0.75	37.74 ± 1.15	$P < 0.05$
MCV (µ3)	55.12± 2.96	62.60± 2.27	$P < 0.05$
MCH (pg)	20.96±0.26	19.28 ± 0.69	$P < 0.05$

Mean Corpuscular Hemoglobin (MCH): The MCH in Group A was 20.96 ± 0.26 pg, much greater than Group B's 19.28 ± 0.69 pg ($P < 0.05$). This reduction of MCH in the zinc-exposed group shows reduced hemoglobin content per red cell, which is consistent with the impaired hemoglobin synthesis associated with zinc exposure. In summary, the results show that zinc

acetate exposure (125 mg/kg) for 60 days profoundly impacted hematological indices in Group B versus the control Group A. The decreases in RBC, hemoglobin, hematocrit, and MCH, coupled with an elevation in MCV, reflect the erythropoietic and hemoglobin biosynthetic toxicity of zinc, with all of the differences attaining statistical significance ($P < 0.05$). These results highlight the hematotoxicity of zinc and its ability to interfere with red blood cell indices under the controlled experimental conditions.

The hematological indices analyzed in the present study demonstrate remarkable changes in the blood picture of male albino rats exposed to zinc (Group B) compared to the healthy control group (Group A), which was administered sodium acetate. The changes detected in red blood cell (RBC) count, hemoglobin content, hematocrit, and corresponding indices present unequivocal evidence of zinc-induced hematotoxicity, as has been found in earlier studies.

The RBC count in Group B ($6.32 \pm 0.13 \times 10^3/\mu l$) was considerably decreased compared to that of Group A ($7.22 \pm 0.13 \times 10^3/\mu l$) ($P < 0.05$), showing an extreme reduction by zinc exposure. Zinc toxicity has been generally reported to hamper erythropoiesis by inhibiting enzymes critical to heme biosynthesis, such as delta-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, zincing to abnormal hemoglobin synthesis and enhanced destruction of erythrocytes (Flora et al., 2012). Besides, oxidative stress caused by exposure to zinc plays a role in membrane lipid peroxidation, leading to enhanced hemolysis and a reduced lifespan of RBCs (Hossain & Ameen, 2018).

Hemoglobin level was reduced significantly in zinc-exposed subjects (11.85 ± 0.65 g/dl) as opposed to the controls (12.66 ± 0.31 g/dl) ($P < 0.05$). Zinc disrupts heme synthesis by inhibiting critical enzymes within the pathway, especially ALAD, and impeding iron loading into protoporphyrin IX, zincing to accumulation of zinc protoporphyrin and dysfunctional erythropoiesis (Patrick, 2006). As a result, impaired hemoglobin synthesis leads to compromised delivery of oxygen and anemia reported in zinc intoxication.

Group B hematocrit levels ($37.74 \pm 1.15\%$) were significantly lower compared to Group A ($41.24 \pm 0.75\%$) ($P < 0.05$), in line with the decrease in RBC count and hemoglobin level. Lower hematocrit levels reflect a decreased percentage of red blood cells in overall blood volume, which further emphasizes the harmful effects of zinc on erythropoietic function (Gidlow, 2015). Zinc-induced anemia has been described as normocytic and hypochromic in

the early stages but can become macrocytic anemia because of compensatory erythropoiesis through the release of larger, immature red blood cells (Hosseini et al., 2013).

Surprisingly, Group B had a significantly higher mean corpuscular volume (MCV) of 62.60 ± 2.27 μ^3 than Group A's 55.12 ± 2.96 μ^3 ($P < 0.05$). The high MCV in zinc-exposed rats indicates the occurrence of macrocytosis, which can be due to an elevated number of reticulocytes entering circulation as part of a compensatory mechanism against anemia (Mudipalli, 2007). Macrocytosis can also reflect disturbed erythropoiesis, wherein defective maturation of erythroid precursors takes place because of ineffective heme biosynthesis.

Group B had a decreased mean corpuscular hemoglobin (MCH) value of 19.28 ± 0.69 pg versus 20.96 ± 0.26 pg in Group A ($P < 0.05$). A decrease in MCH indicates that each erythrocyte in zinc-exposed rats carries less hemoglobin, further indicating impaired synthesis of heme and compromised oxygen-carrying capacity of the blood. This finding is consistent with earlier research that has shown zinc toxicity to be a cause of hypochromia because of the buildup of free protoporphyrins and poor iron incorporation (Khalaf et al., 2012).

The hematotoxicity seen in this study is due to various mechanisms. Zinc interferes with erythropoiesis by inhibiting ALAD and ferrochelatase, disrupting heme biosynthesis and decreasing the synthesis of functional hemoglobin (Flora et al., 2012). Oxidative stress induced by zinc also affects the erythrocyte membrane by zincing, leading to enhanced hemolysis and decreased RBC lifespan (Sandhir et al., 1994). In addition, zinc affects iron metabolism by enhancing iron sequestration within macrophages and hindering iron mobilization for erythropoiesis (Khan et al., 2008).

Results in this research concurred with those of existing work showing chronic exposure to zinc promotes anemia and changes the hematological parameters in animal models (Sharma et al., 2010; Hossain & Ameen, 2018). Research revealed the dose-dependent decrease in the level of RBC, concentration of hemoglobin, and hematocrit alongside compensation in MCV and distorted levels of MCH, mirroring the observations seen in this current study.
Overall, the important changes in the hematological values in zinc-exposed male albino rats reemphasize the harmful effect of zinc acetate on erythropoiesis, hemoglobin formation, and the overall integrity of the red blood cells. The declines in RBC, hemoglobin, hematocrit, and MCH with a rise in MCV indicate a robust case of zinc-caused hematotoxicity, wherein all the

differences reached statistical significance ($P < 0.05$). These results are in agreement with the known toxicological pattern of zinc and emphasize the requirement for more studies on possible interventions to counteract the harmful outcomes of zinc exposure.

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