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Antioxidant Effects of Vitamin C on Some Hematological Parameters of Male Wistar Rats Exposed to Lead Acetate

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

The study investigated the effects of vitamin C on platelet parameters, white blood cell count and white blood cells differentials in Wistar rats exposed to lead acetate. A total of twenty-four male Wistar rats weighing between 160g and 200g were utilized. The experimental animals were divided into four groups of six rats each (n=6). Group 1 served as the control group (received normal feed and water), group II received 10mg/kg body weight of lead acetate, group III received 100mg/kg body weight of Vitamin C, and group IV received 10mg/kg body weight of lead acetate followed by 100mg/kg body weight of Vitamin C. Lead and Vitamin C, along with normal feed, were administered for four weeks. Blood samples were collected via jugular puncture and stored in EDTA bottles for analysis to determine the blood profile of the rats. The results showed significant

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increase in Platelet Count (PLT) in group III and a significant decrease in Mean Platelet Volume (MPV) in group IV (Pb + vitamin C). The Mean Platelet Width (MPW) showed decrease in groups 2, 3, and 4 compared to the control group, although this decrease was not statistically significant. The study also noted an elevated level of white blood cells (WBC) in response to the antioxidant treatment, indicating a potential positive impact on immune function. In conclusion, this study demonstrates the therapeutic effect of Vitamin C against the toxic effects of lead on platelet parameters and white blood cell count and differentials.

Keywords: Blood cell count; platelet; thrombocytopenia; human health.

1. INTRODUCTION

Lead is a toxic metal that can have detrimental effects on various aspects of human health, including the parameters of platelets in the blood. Platelets are vital components of the blood responsible for clotting and preventing excessive bleeding. Lead exposure has been shown to negatively impact platelet parameters, leading to potential health risks. One of the primary effects of lead on platelets is a decrease in platelet count, a condition known as thrombocytopenia. Studies have demonstrated that lead exposure can reduce the number of platelets in the blood, increasing the risk of bleeding and bruising [1]. Thrombocytopenia can impair the body's ability to form blood clots, leading to prolonged bleeding and difficulty in stopping bleeding after an injury. In addition to decreasing platelet count, lead exposure can also impair the function of platelets. Research has shown that lead can interfere with the normal function of platelets, making them less effective at forming blood clots [2]. This impaired platelet function can further exacerbate the risk of bleeding and complications related to inadequate clotting.

Furthermore, lead exposure has been associated with changes in the morphology of platelets. Studies have reported alterations in the shape and structure of platelets in individuals exposed to lead, which can affect their ability to function properly [3]. These morphological changes may contribute to the overall dysfunction of platelets and their role in maintaining hemostasis. Moreover, the effects of lead on platelet parameters can have broader implications for cardiovascular health. Lead exposure has been linked to an increased risk of cardiovascular diseases, such as heart attacks and strokes [4]. Platelets play a crucial role in the formation of blood clots that can lead to these conditions, and any disruption in platelet parameters due to lead exposure can exacerbate this risk.

Lead can also be deleterious to white blood cells (WBCs) count and differentials. White blood cells are a crucial component of the immune system responsible for fighting off infections and foreign invaders. Lead exposure has been shown to negatively impact WBC count and differentials, leading to potential health risks [5].

One of the primary effects of lead on white blood cells is a decrease in the total WBC count, a condition known as leukopenia [6]. Studies have demonstrated that lead exposure can reduce the number of white blood cells in the blood, compromising the body's ability to mount an effective immune response [3]. Leukopenia can increase the susceptibility to infections and impair the body's ability to fight off pathogens. Furthermore, lead exposure can also alter the differential count of white blood cells, affecting the proportions of different types of WBCs in the blood Research has shown that lead can disrupt the balance between various types of white blood cells, such as neutrophils, lymphocytes, monocytes, eosinophils, and basophils [2]. These alterations in WBC differentials can impact the immune response and increase the risk of inflammatory conditions. In addition to decreasing the total WBC count and altering WBC differentials, lead exposure can impair the function of white blood cells. Studies have reported that lead can interfere with the normal function of WBCs, reducing their ability to effectively respond to infections and other immune challenges [1]. This impaired immune function can compromise the body's ability to defend against pathogens and increase the risk of illness. Moreover, the effects of lead on white blood cells can have broader implications for overall health and well-being. Disruption of the immune system due to lead exposure can increase the risk of infections, autoimmune diseases, and other inflammatory conditions [7]. Chronic exposure to lead has been associated with a range of health problems, including respiratory issues, cardiovascular diseases, and neurological disorders [8]. Lead exposure can

have significant negative effects on platelets parameters, white blood cells count and differentials, leading to leukopenia, alterations in WBC proportions, and impaired immune function. These effects can increase the susceptibility to infections, compromise the immune response, and contribute to various health complications. It is essential to minimize exposure to lead through environmental and occupational measures to protect the immune system and overall health.

Vitamin C, also known as ascorbic acid, is a powerful antioxidant that plays a crucial role in protecting the body from oxidative stress and damage caused by free radicals. Free radicals are unstable molecules that can cause harm to cells and tissues, leading to various health issues, including toxicity [9]. As an antioxidant, vitamin C helps neutralize free radicals by donating electrons to stabilize them, thus preventing them from causing damage to cells. This protective effect can help reduce the risk of toxicity in the body, as oxidative stress is a common factor in the development of toxicity from environmental toxins, medications, and other harmful substances [10]. In addition to its antioxidant properties, vitamin C also has the ability to regenerate other antioxidants, such as vitamin E, glutathione, and coenzyme Q10, further enhancing its protective effects against toxicity. By working synergistically with other antioxidants, vitamin C can help maintain the body's defense mechanisms and reduce the risk of toxic overload [11].

Research has shown that vitamin C supplementation have the potentials to prevent toxicity from heavy metals, such as lead and mercury, by reducing their harmful effects on the body. It has also been shown to protect against oxidative damage caused by exposure to pesticides, pollutants, and other toxins [12]. Overall, vitamin C's potent antioxidant properties make it a valuable nutrient in preventing toxicity and promoting overall health. Incorporating vitamin C-rich foods, such as citrus fruits, berries, and leafy greens, into your diet can help support body's natural detoxification processes and reduce the risk of toxic overload. Additionally, vitamin C supplementation may be beneficial for individuals at risk of toxicity or those exposed to high levels of environmental toxins. Based on this, the current study was aimed at investigating the effects of oral administration of vitamin c on platelet parameters, white blood cells count and differentials of male wistar rats exposed to lead acetate

2. MATERIALS AND METHODS

In this study, the effects of lead acetate and Vitamin C administration on rats were investigated through a series of experiments. The rats were divided into four groups and administered different doses of lead acetate and Vitamin C over a period of 28 days. The administration was done using an oral gavage cannula, with precise measurements of the solutions given to each group. Adult male wistar rats were divided in to four (4) groups of 6 rats. Group 1: Wistar rats served as control and were given water and feed, Group 2: Wistar rats were given 10mg/kg body weight of lead acetate only, Group 3: Wistar rats were given 100mg/kg body weight of Vitamin C only, Group 4: Wistar rats were given 10mg/kg body weight of lead acetate followed by 100mg/kg body weight of Vitamin C. The administration process involved holding the rats and carefully placing the cannula in their mouths to avoid injury. This procedure was repeated daily for 28 days. After the administration period, the rats were sacrificed in a preparatory room within the animal house. Blood samples were collected from each rat by exposing them to di-ethyl ether anesthesia and performing a jugular puncture. The blood samples were then placed in EDTA bottles and centrifuged to separate the plasma for further analysis. The platelet count was determined using a dilution method with ammonium oxalate reagent. The blood samples were diluted, and platelets were counted microscopically using a neubaurer counting chamber. The platelet count per liter of blood was calculated based on the number of platelets observed. Automated Hematology Analyzer was used for determining WBC count and differentials. Automated hematology analyzers use flow cytometry technology to count and differentiate various types of white blood cells based on their size, shape, and granularity.

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) version 23.0. One-way analysis of variances (ANOVA) followed by post-Hoc multiple comparison tests were conducted to determine statistical significance, with a significance level set at $P<0.05$. The data were expressed as mean ± standard error of mean (SEM).

3. RESULTS

The data on Fig. 1 represents the Effect of Vitamin C administration on platelet count level in Lead (Pb) exposed male Wistar rats. The

changes on the level platelet count indicates a significantly (P<0.05) decreased value in group 2 which was treated with lead (Pb) only when compared to group 1, vitamin C only treated group 3 showed a significantly (p<0.05) increased value when compared to control group 1. Lastly, vitamin C + Pb treated group 4 showed an insignificantly increased value (p>0.05) when compared to control group 1. Group 3 treated with Vitamin C showed a significantly increased value (p<0.05) when compared to group 2 treated with lead acetate.

Table 1. Changes in platelet parameters prior to the administration of Lead acetate and Vitamin C for 28 days

Variables	Mean ± Std. Error	Mean difference (95% CI)	p-value
Mean platelets width (MPW) %			
Control	9.23 ± 0.30	(8.452; 10.015)	0.005[C]
Lead Acetate	8.50 ± 0.82	(8.290; 8.710)	
Vitamin C	7.90 ± 0.25	(7.246; 8.554)	
Lead Acetate and Vitamin C	8.70 ± 0.43	(7.591; 9.809)	
Mean Platelets Volume (MPV)fL			
Control	7.67 ± 0.15	(7.276; 8.058)	0.028[D]
Lead Acetate	7.167 ± 0.11	(6.879; 7.453)	
Vitamin C	6.98 ± 0.23	(6.399; 7.567)	
Lead Acetate and Vitamin C	5.20 ± 1.44	(1.506; 8.901)	
Plateletcrit (PCT)%			
Control	0.63 ± 0.41	(0.521; 0.732)	-
Lead Acetate	0.52 ± 0.80	(0.315; 0.728)	
Vitamin C	0.69 ± 0.89	(0.466; 0.926)	
Lead Acetate and Vitamin C	0.56 ± 0.04	(0.463; 0.647)	
Platelet Count [PLT] x 10^9			
Control	721.33 ± 83.55	(506.561; 936.106)	
Lead Acetate	621.50 ± 64.65	(455.302; 787.698)	
Vitamin C	910.83 ± 131.43	(572.99; 1248.675)	0.042[C]
Lead Acetate and Vitamin C	753.67 ± 84.90	(535.422; 971.912)	

*Values are presented in mean ± sem, n= 6. * means values are statistically significant (p≤0.05) when compared to the control*

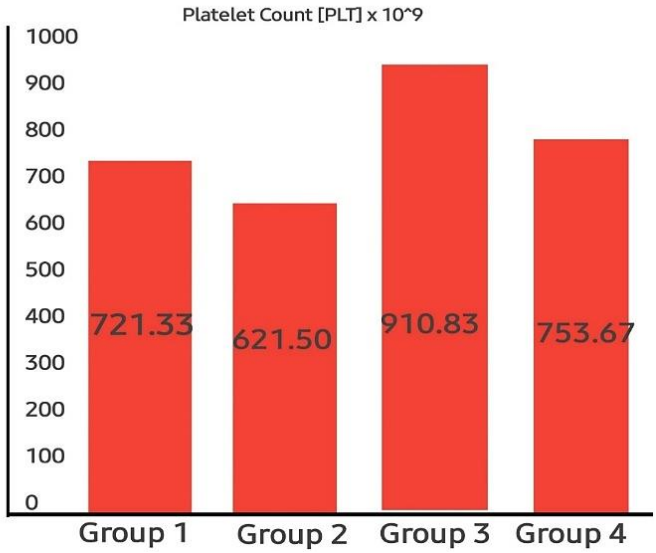

Fig. 1. The Effect of Oral administration of Vitamin C on Platelet parameters of Male Wistar Rats exposed to Lead Acetate (PbA)

Key: *Group 1: Control**;** Group 2: PbA alone treated**;** Group 3: Vitamin C Only Treated**;** Group 4: Vitamin C + PbA Treated*

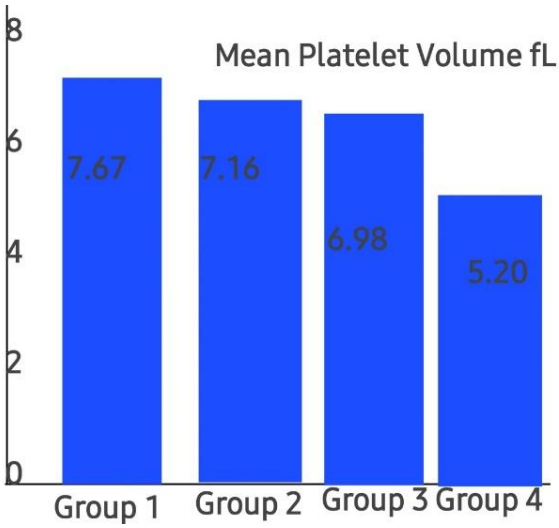

Fig. 2. Mean platelet volume (MPV) variation among Groups

Key: *Group 1: Control; Group 2: PbA alone treated; Group 3: Vitamin C Only Treated; Group 4: Vitamin C + PbA Treated*

The variation in the mean platelet volume (MPV) in the animal study following treatments with lead acetate and vitamin C. Only Group 4 which was treated with Lead acetate and Vitamin C showed a decrease. This decrease is statistically significant ($p<0.05$) when compared to Group 1 (control group/untreated group). Other groups (2 and 3) showed decreased values when compared with the control group (untreated group). This decrease was not statistically significant ($p>0.05$).

The data on Fig. 3 above shows Mean Platelet width (MPW) on the animal stud following treatments with lead acetate and vitamin C. Group 2 (PbA) showed an insignificantly decreased in value ($p>0.05$) when compared to group 1(untreated group). Group 3 (Vitamin C) showed a significant decrease ($p<0.05$) decreased value when compared to group 1. Group 4 treated with PbA + Vitamin C showed an insignificantly decreased value ($p>0.05$) when compared with group 1

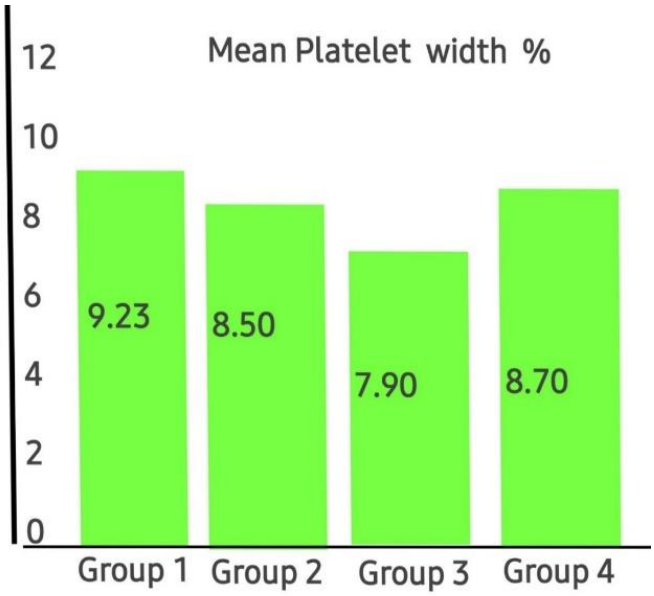

Fig. 3. Mean platelet width variation

Key: *Group 1: Control; Group 2: PbA alone treated; Group 3: Vitamin C Only Treated; Group 4: Vitamin C + PbA Treated*

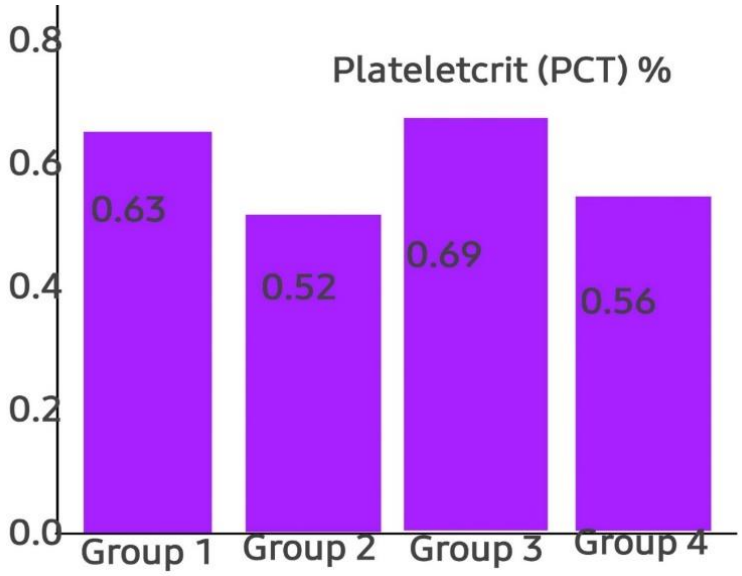

Fig. 4. Plateletcrit variation

Key: *Group 1: Control; Group 2: PbA alone treated; Group 3: Vitamin C Only Treated; Group 4: Vitamin C + PbA Treated*

Plateletcrit (PLT) on the animal study following treatments with lead acetate and vitamin C. Group 2 showed an insignificantly decreased value ($p>0.05$) when compared to the control group. Group 3 showed an insignificantly increased value ($p>0.05$). Group 4 showed an insignificantly increased value ($p>0.05$) when compared to the control group.

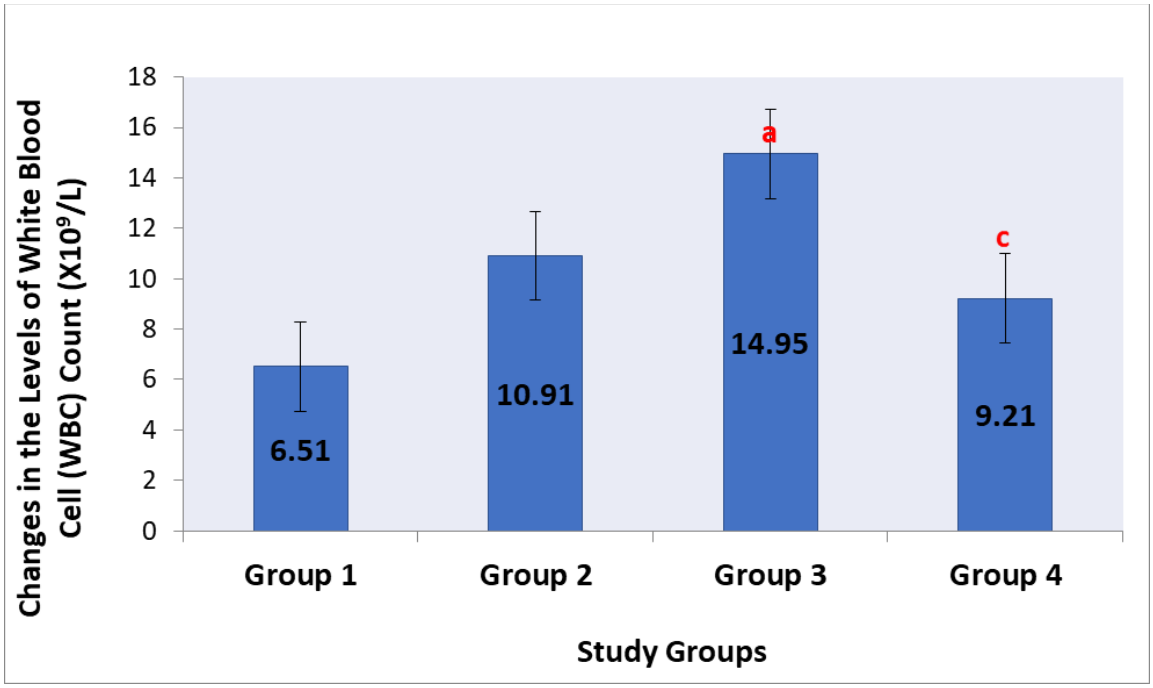

Fig. 5. Effects of oral administration of vitamin C on white blood cells (WBC) count in lead acetate (PbA) exposed male wistar rats

Values represent mean ± SEM, n=6; [a] Significant at $p<0.05$ when compared to Group 1; [b] Significant at $p<0.05$ when compared to group 2; [c] Significant at $p<0.05$ when compared to group 3. PbA= lead acetate.

Key: *Group 1: Control; Group 2: PbA alone treated; Group 3: Vitamin C Only Treated; Group 4: Vitamin C + PbA Treated*

Table 2. Effect of Oral administration of vitamin C on white blood Cells (WBC) differentials in lead acetate (PbA) exposed male wistar rats

Groups and Treatment	Neutrophils (%)	Lymphocytes (%)	Eosinophils (%)	Monocytes (%)
Group 1: Control	4.50 ± 1.12	91.00 ± 1.88	1.00 ± 0.00	3.50 ± 0.80
Group 2: PbA alone treated	4.50 ± 0.56	80.66 ± 9.62	1.00 ± 0.00	3.33 ± 1.23
Group 3: Vitamin C Only Treated	6.33 ± 1.05	81.33 ± 8.17	1.00 ± 0.00	3.16 ± 0.48
Group 4: Vitamin C + PbA Treated	3.83 ± 1.22	90.16 ± 3.83	1.00 ± 0.00	3.33 ± 1.20

Values represent mean ± SEM, n=6; [a] Significant at p<0.05 when compared to Group 1; [b] Significant at p<0.05 when compared to group 2; [c] Significant at p<0.05 when compared to group 3. PbA= lead acetate.

The data on Fig. 5. shows the result of the effect of oral administration of vitamin C on white blood cells (WBC) count in lead acetate (PbA) exposed male Wistar rats.

The result on the variation in the level of white blood cell (WBC) indicated a significantly (P<0.05) raised value in Group 3 rats (treated with Vitamin C alone) when compared to that of Group 1 (untreated control). The WBC levels of groups 2 and 4 were lower than that of Group 3 but only the value of Group 4 (treated with Vitamiin C + PbA) was statistically significant (P<0.05).

The data on Table 2 shows the result of the effect of oral administration of vitamin C on white blood cells (WBC) differentials in lead acetate (PbA) exposed male Wistar rats.

Obviously, the variations in the mean levels of neutrophils, lymphocytes, eosinophils and monocytes in all the treated groups were not statistically significant (P<0.05) when compared to that of group 1. However, it was noticed that Group 3 (treated with vitamin C alone) had the most raised level of neutrophils and that of group 4 (treated with Vitamiin C + PbA), the lowest. The variations in the levels of lymphocytes, eosinophils and monocytes did not show any uniform patterns across the all the treated groups.

4. DISCUSSION

Human health is negatively impacted by heavy metals, and industrialization, anthropogenic activities, and modern living have increased exposure to these metals [13]. Toxic metal contamination of air and water is a global environmental problem that affects hundreds of millions of people [13].

Some common sources of heavy metal contamination include but not limited to the following: eating food that contaminated with metals (fish); rinking water from older water supply systems, working at factories and place with increased exposure to heavy metals and even taking medications or supplements with high amounts of metallic elements [14,15].

Considering the foregoing, the present study evaluated the effect of oral administration of Vitamin C on platelets parameters and white blood cells (WBC) count in Lead Acetate (PbA) exposed male Wistar rats and some significant findings were made and discussed in the following paragraphs.

The lead acetate (10mg/kg body weight) only treated group (Group II) showed a significantly decreased value (p<0.05) when compared to the Control group (untreated group). This finding is consistent with previously published observations [16].

Exposure of the general population to lead mostly occurs through the ingestion of contaminated food and drinking water, as well as by the inhalation of particulate lead in ambient air [17]. Lead enters the bloodstream after it is absorbed. First, lead attaches to proteins in the blood and is carried to different tissues or organs in the body. The high amount of lead in the blood inhibits the secretion of thrombopoietin**.** Thrombopoietin is a key enzyme that triggers the megakaryocyte progenitors which leads to the synthesis of Platelets [18]. Hence, the inhibition of Thrombopoietin which leads to decline in activities of megakaryocytes progenitors affects the synthesis of Platelets (thrombocytes). Lead interferes with the production of platelet at several different steps [19].

According to studies [20], lead has a potential to induce oxidative stress and acts as a catalyst in the oxidative reactions of biological macromolecules. Hence, the toxicities associated with lead might be due to oxidative tissue

damage. Induction of Oxidative stress leads to reduction or decline in intracellular calcium level. Previous researches have established that intracellular calcium level modulate or enhanced proliferation of platelet count. This is consistent with the findings of previous studies [21].

Group 3 (Vitamin C) showed a significantly increased value ($p<0.05$) when compared to group 1(untreated group). The increase in platelet count in group III having 910.83 appeared to be significant ($p<0.05$) when compared to the control group that have a mean value of 721.33. Vitamin C contains Ascorbic acid, Sodium ascorbate, Magnesium stearate. Ascorbic acid is very necessary for thrombopoiesis because it facilitates iron absorption by forming a chelate with ferric iron at acid pH that remains soluble at the alkaline pH of the duodenum. The absorption of iron enhances the activities of intracellular calcium, intracellular calcium ions have a corresponding effect on Platelet count by increasing the number of platelets in the body [22].

Magnesium, another mineral contained in the vitamin C improves thrombopoiesis, reduces oxidative stress and plays an active role in improving the immune system [23].

Magnesium activates vitamin D needed for proliferation of burst-forming unit platelet progenitor cells. Magnesium also activates many enzymes in the body, is essential for the stability of cell function and cell repair and maintains the antioxidant status of cell. The sodium content helps vitamin C to be easily absorbed and stay longer in the body. It serves as an antioxidant that helps keep your cells from damage and keep them healthy [24].

Ascorbic acid (Vitamin C) has been studied extensively in modulating lead induced alterations. Ascorbic acid is known to have number of beneficial effects against lead exposure [25]. There has been considerable debate concerning the relationship between vitamin C nutritional status and heavy metal body burden in lead induced exposure. Early reports found that vitamin C might act as a possible chelator of lead, with similar potency to that of EDTA [26]. Most of the studies carried out to assess the effect of vitamin C on lead exposure used very high doses of vitamin C or administrate vitamin C concurrent with other vitamins such as vitamin E and vitamin D to reserve effect of lead. The effect of high dose vitamin C (1000mg/kg-2000mg/kg body weight) on lead levels has been clarified by studies but very little has been done to access the effect of vitamin C in low dose on lead induced alterations.

Group IV treated with PbA and Vitamin C showed an insignificantly increased value ($p>0.05$. This could be as a result of the effect of the antioxidant which mitigated the lead toxicities. Group II showed a significantly decreased value ($p<0.05$) when compared to group IV. In group II the rats were exposed to toxin (lead acetate). This exposure grossly caused toxicity to the rats and there was not antioxidant to ameliorate the effect of the lead acetate toxicity hence, decreased platelet count value (621.50). The Physiology of the antioxidant would be best understood when Platelet count value in group II (621.50) is compared to platelet count in group IV (753.67). The antioxidant was potent enough to counter the toxicity of the lead acetate. This finding is consistent with the findings of Mahdi [27] in a clinical study on lead-exposed worker.

From the result, Platelet count (PLT) in group IV appeared to be increased (753.67) when compared to group I which is the control group (721.33). This increase is statistically insignificant ($p>0.05$). The observation in group IV might be as a result of Vitamin C health benefits. The effect of vitamin C on lead levels has been seen in some studies show that ascorbic acid decreases intestinal absorption of lead [3]. Findings from this study indicated amelioration of platelet parameters after administrating lead acetate concurrent with vitamin C 100mg/kg body weight. However, from the research result, it is worth noting that the antioxidant (Vitamin C) was able to mitigate the lead toxicity hence, the increase in platelet count.

Mean Platelet Volume (MPV) was seen to be decreased (5.20) when compared to the Control group (7.67). This decrease is statistically significant. Abnormally low MPV values may correlate with thrombocytopenia when it is due to impaired production of megakaryocytes in the bone marrow, such as in aplastic anemia. It is an already established fact that lead acetate has an adverse effect on the human body, hence its administration may affect the platelets volume as seen from the study. A study carried out by Sharaf et al. [28] showed the administration with vitamin C concurrently with lead and cadmium metals prevented adverse effect of tested heavy

metals on hematological and biochemical parameters compared to vitamin C treated rats group and control untreated rats group. Vitamin C has potent antioxidant activity against lead toxicity. Hence the consumption of foods rich in vitamin C is often highly recommended to reduce the damage caused by the toxicity with lead. This is not the case with this present study since it was observed that there was further decrease in the mean values of the Vitamin C group to 6.98. The forth study group that received lead acetate and vitamin C had mean values of 5.20. This value was statistically significant ($p<0.50$).

In Fig. 3, it was observed that the control group had mean values of 9.23. Rats that received lead acetate showed a level of decrease in their mean values when compared to the control group, although this decrease was not statistically significant ($p>0.05$). The vitamin C group showed significant decrease ($p<0.05$) in their mean platelets width values when compared to the control group. While Vitamin C at normal physiological concentrations is good for human consumption, there is virtually no information on the impact of high concentrations of Vitamin C on PLT function. Vitamin C is a key modulator of platelet (PLT) function but its effect on platelet width is not known. The decrease in mean platelet values of vitamin C study group is in line with a study carried out by [29]. It was stated in the study that vitamin C may affect the values of PLT, MPV, PCT and RDW. The study group that received lead acetate and vitamin C had a mean value of 8.70. The result indicate that the control group had mean values of 0.63. The study groups recorded variations when compared to the control group. Lead acetate group showed a decrease in mean values (0.52). This decrease was statistically significant ($p>0.05$). However, the decrease could be as a result of the effects of lead acetate. Vitamin C group showed an increase in mean values (0.69), this increase was not statistically significant ($p>0.05$) when compared to group 1. Vitamin C has been known to improve platelet health. Also, Group 4 showed a further decrease (0.56) in the mean value when compared to the control group. This increase was not statistically significant ($p>0.05$). The current study found that the variation in the level of white blood cell (WBC), following oral administration of vitamin C on WBC in lead acetate (PbA) exposed male Wistar rats, indicated a significantly raised value in Group 3 rats (treated with Vitamin C alone) when compared to that of Group 1 (untreated control). It was also noticed that PbA only treated group had a lesser level of WBC than that of the Vitamin C only treated animals. In another related finding of the present study, the variations in the mean levels of neutrophils, lymphocytes, eosinophils and monocytes in all the treated groups were not statistically significant when compared to that of group 1. However, it was noticed that Group 3 (treated with vitamin C alone) had the most raised level of neutrophils and that of group 4 (treated with Vitamiin C + PbA), the lowest. Antioxidants have been shown to have a number of beneficial effects, protecting against RBC lipid peroxidation and increasing levels of reduced glutathione (GSH) while reducing levels of reactive oxygen species [30].

It has been said that Vitamin C has a major function of being an antioxidant as it boosts immunity through increasing white blood cells, in addition to supporting regeneration of vitamin E [31].

In fact, it has been established that the production of free radicals, which results in oxidative stress, damage to biological molecules like proteins, lipids, enzymes, and nucleic acids, damage to DNA, which is essential to carcinogenesis, and neurotoxicity are the main mechanisms of heavy metal toxicity. Thus, the above finding of this study reveals that treatment with Vitamin C (a known anti-oxidant) in PbA exposed mammals, helps boost the immune system via the modulating the WBC levels.

In the event of adverse alteration in the oxidants/antioxidants equilibrium system, perhaps due to heavy metal (like PbA) or other environment contamination, free radicals cause lipid peroxidation of cellular membranes, oxidation of proteins and DNA, chromosome structural alterations, genetic mutations, and/or modulation of cell growth, all of which promote carcinogenesis [32]. The finding of the present has yet revealed another important relationship between oxidant antioxidant system and the immune system. In fact, [33] succinctly puts it that the immune system is extremely vulnerable to oxidant and antioxidant balance, as uncontrolled free radical production can impair its function and defense mechanism.

5. CONCLUSION

The findings from this experimental study suggest that Vitamin C has the potential to improve platelet parameters and counteract the negative effects of heavy metal contamination,

particularly PbA. The study indicates that treatment with Vitamin C or the consumption of nutrients rich in this antioxidant may mitigate the impact of oxidative damage caused by environmental toxicants.

Given that oxidative damage is increasingly recognized as a contributing factor to various diseases such as cancer, diabetes, and neurodegeneration, it is crucial to promote the consumption of antioxidant-rich diets. By incorporating foods high in antioxidants into our daily meals, we can potentially protect our cells from damage and support overall cellular functions.

The study also observed a boosted level of white blood cells (WBC) in response to the antioxidant treatment, suggesting a potential positive impact on immune function. This underscores the importance of maintaining optimal antioxidant levels through a balanced diet to enhance cellular health and combat the effects of environmental toxins.

Therefore, it is recommended that individuals make conscious efforts to consume a balanced diet rich in antioxidants to support overall health and well-being. By incorporating antioxidant-rich foods such as fruits, vegetables, nuts, and seeds into daily meals, individuals can potentially reduce the risk of oxidative damage and promote cellular functions.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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