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A study of the haematological changes in lead intoxication in mice

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Abstract
In the present study lead induced histological alterations in mice studied. The male mice were divided into 3 groups and treated as follows: Group 1: healthy control and Group II with 0.4 and Group III with 1.2 mg/kg body weight of doses of lead acetate respectively in their daily supply of drinking water for twelve weeks. It was observed that there was a significant decline in the the total erythrocyte count (TEC) following exposure of lead acetate in group II and III in comparison with the control group. It was also observed marked decrease in the levels of haemoglobin (Hb) and packed cell volume (PCV). MCV, MCH, and MCHC were also significantly reduced in treated mice relative to the healthy ones.

Keywords: Arsenic, histopathology, rat

Introduction
Lead has been mined and used by mankind for 6,000 years, and the history of lead poisoning is nearly 2,500 years old. The older history of lead poisoning has been reviewed many times in articles, book chapters, and textbooks [1, 2]. Lead metal has toxic effect on living organisms and is often considered as contaminant and weak carcinogen [3]. Upon ingestion, absorption of lead influenced by many factors including dose, age and diet. After being absorbed, lead is distributed through the body and accumulates in the various vital organs.

Materials and Methods
The study was conducted on thirty male mice. The animals were kept in standard compartmented rectangular and well-ventilated cages. They maintained on standard healthy laboratory conditions at temperature of 18-24°C and twelve hours light and darkness. Animals were adapted to the new environment for fourteen days prior to study start. All mice had free access to drinking water and food, ad libitum, during the experimental period. They were fed with standard pellet diet. The animals were divided into three equal groups. Each group comprised of ten male and was marked as groups I, II and III. The first group represented the healthy control animals, while the second and third groups were given 0.4 and 1.2 mg/kg body weight of doses of lead acetate respectively in their daily supply of drinking water for twelve weeks.
For the haematological investigations, blood was collected from each mouse individually. The animals were fasted for twelve hours prior to blood collection. All animals were anesthetized by chloroform and blood samples were collected immediately from their heart using heart puncture technique with the aid of disposable sterile syringe and needle. Blood sample of each mouse was then transferred to a sterile capped tube containing anticoagulant EDTA for haematological estimation.
Blood cell counter automated haematology analyser was used to determine haematological indices including total erythrocyte count (TEC), total leukocyte count (TLC), Packed cell volume (PCV), Haemoglobin (Hb) concentration, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), and the amount of platelets.

Results and Discussion
Haematological values alteration in blood serum of the experimental animals after 12 weeks of treatment with lead.

Table 1

Heamatological Parameters	Group I (control) 0.0 mg/kg b. wt.	Group II 0.8 mg/kg b. wt.	Group IV 1.2 mg/kg b. wt.
TEC (× 10³ / µl)	6.22±0.54	5.64±0.76	5.01±0.93
TLC (× 10³ / µl)	7.10±0.08	8.12±0.24	8.90±0.12
PLT (× 10³ / µl)	205±3.87	358±5.32	379±4.01
HGB (g/dl)	15.2±1.28	12.6±1.49	9.24±1.04
MCV (fl)	62.6±2.03	56.04±2.73	55.94±1.08
MCH (pg)	19.11±1.02	15.03±1.32	11.72±1.37
MCHC (%)	32.00±1.46	24.74±2.39	20.10±2.03
PCV (%)	44.03±2.02	31.83±1.14	30.07±1.11

The results in the above table indicated significant reduction ($P<0.05$) in the total erythrocyte count (TEC) following exposure of lead acetate in group II and III in comparison with the control group. It was also observed marked decrease in the levels of haemoglobin (Hb g/dl) and packed cell volume (PCV). MCV, MCH, and MCHC were also significantly reduced in treated mice relative to the healthy ones. The reduction of TEC and decreased level of other haematological parameters indicated that alterations were observed in the groups where lead acetate was administrated [4, 5] and showed microcytic hypochromic anaemia [6, 7]. Similarly progressive decease of TEC count, PCV and in Hb (g/dl) was revealed.

These haematological changes might be attributed to the toxic effect of lead on cell metabolism, interaction with some reactions where calcium act as a secondary mediator, and inhibition of some enzymatic activities such as aminolevulinic acid dehydratase which play key role of heme biosynthesis [8], and other erythrocyte enzymes e.g. GA3PD and G6PD [9]. Continuous exposure to lead might adversely affect the heme biosynthesis in the body due to the inhibition of cytoplasmic and mitochondrial enzymes [10]. The depressing effects of lead acetate on the activity of the major enzymes in heme biosynthesis process might be refer to imperfection of iron metabolism [11, 12]. The inhibitory effect of lead acetate on conversion of coproporphyrinogen III to protoporphyrin IX resulting in shortening of erythrocyte life span and decrease the production of haemoglobin [8]. The reduction of haematological values might be attributed to binding of lead to red blood cells which increase membrane fragility and RBCs destruction [13]. Total leukocyte count (TLC) was also significantly increased in all groups which were administrated lead acetate relative to the control groups. It has been reported that lead induced inflammation which lead to increasing in white blood cells [5] which concur this study. Platelets count revealed considerable increase in intoxicated animals compared to the control. This may be due to thrombocytopenia after lead intoxication [13] followed by thrombocytosis [5, 13].

Conclusion

To conclude, treatment with lead acetate at low doses has harmful effects on experimental animals and induced haematological and biochemical alterations. We also concluded that toxic metal is to be avoided for its hazardous impact.

References

1. Cantaraw A, Trumper M. Lead poisoning. Clinical Toxicology. 1944; 6:419-429.
2. Ganguly S. Heavy Metal and Insecticide Toxicity in Animals and Poultry Birds. Agri-BioVet Press (an unit of Prashant Book Agency), Daryaganj, New Delhi, India, 2018. ISBN 978-93-84502-63-8
3. Nriagu JO. Lead and Lead Poisoning in Antiquity, Wiley, 1983.
4. Falke HE, Zwennis WCM. Toxicity of lead acetate to female rabbits after chronic subcutaneous administration: Biochemical and clinical effects. Arch. Toxicol. 1990; 64:522-529.
5. Yagminas AP, Franklin CA, Villeneuve DC, Gilman AP, Little PB, Valli VE. Subchronic oral toxicity of triethyl lead in the male weanling rat. Clinical, biochemical, hematological, and histopathological effects. Fundam. Appl. Toxicol. 1990; 15:580-596.
6. Mugahi MN, Heidari Z, Sagheb HM, Barbarestani M. Effects of chronic lead acetate intoxication on blood indices of male adult rat. DARU. 2003; 11:147-151.
7. Suradkar SG, Ghodasara DJ, Patel J, Jaiswal V, Prajapati KS. Haemato-biochemical alterations induced by lead acetate toxicity in wistar rats. Vet World. 2009; 2:429-431.
8. Klassen CD. Casarett and Doull's Toxicology: The basic science of poisons. 6th ed. McGraw-Hill Medical publishing division. 2001, 812-841.
9. Calderon-Salinas V, Hernandez-Luna C, Maldonado M, Saenz D. Mechanisms of the toxic effects of lead. I. Free lead in erythrocytes. J Expo Anal Environ Epidemiol. 1993; 1:153-64.
10. ATSDR (Agency for Toxic Substances and Disease Registry). Toxicological profile for lead, Update. Prepared by Clement International Corporation under contact no. 205-88-060 for ATSDR, U.S. Public Health Services, Atlanta, GA, 1993.
11. Chmielnika J, Zareba G, Nasiadek M. Combined effect of tin and lead on heme biosynthesis in rats. Ecotox. Environ. Safety. 1994; 29:165-173.
12. Rous P. The effect of heavy metals boundary contaminated soil on haematological and selected biochemical parameters in blood plasma of rabbits. Acta-Universitatis-Agriculturae-et-, 2000.
13. Sudakova AI, Shevchenko ZT, Nosova LI. Peripheral blood and bone marrow cell status of white rats with long-term lead exposure. Tsitol Genet. 1983; 17:3-7.