Original Research Article Toxicology

International Journal of Pharma and Bio Sciences

STUDIES ON ALUMINIUM SULPHATE INDUCED ALTERATIONS IN LIPID PROFILE IN ALBINO RAT

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ABSTRACT

This study evaluated the changes in lipid profile after aluminum induced toxicity in wistar rats. Pollutants are heavy metal substances in the environment which cause objects to enable effects, impairing the welfare of environment reducing the quality of life and may eventually cause death. Although heavy metal poisoning could be clinically diagnosed and medically treated, the best option is to prevent heavy metal pollution and the subsequent human poisoning. In the present study, an attempt has been made to evaluate the harmful effects of aluminum on blood lipid profiles to elucidate general health conditions in albino rats and results obtained can be applied to other mammals, including humans. The work has been done as per standard procedures and protocol in departmental laboratory. The results of present study clearly reflects significant alteration in lipid profile that can be considered as mark to show heavy metal toxicity. The outcome of the study showed that aluminum is capable to alter lipid profile parameters in mammals which can seriously affect the health and physiology of the affected animal. It is suggested to minimize the use of aluminum and whenever used, caution should be taken.

KEYWORDS: ***HDL, LDL, Triglyceride, Cholesterol, Heavy metal toxicity***

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Received on: 19-06-2019
Revised and Accepted on: 20-08-2019
DOI: http://dx.doi.org/10.22376/ijpbs.2019.10.3.b254-258

INTRODUCTION

Aluminum is a trivalent cation found in its ionic form in most kinds of animal and plant tissues and in natural waters . It is a ubiquitous element and the third most prevalent (abundant) element in the earth's crust, comprising approximately 8% of the earth's crust, exceeded only by oxygen (47%) and silicon (28%). The presence of this element has so heavily contaminated the environment that exposure to it is virtually inescapable. Natural processes account for most of the redistribution of aluminum in the environment as a result of the weathering of rocks and minerals in which it is present. Mobilization from natural sources can, however, also result from the deposition of acidic precipitation. Direct anthropogenic releases of aluminum compounds occur primarily to air and these are associated with industrial processes. Thus, the mining and processing of aluminum ores and the production of aluminum metal, alloys and compounds can lead to the release of aluminum compounds into the environment. The use of aluminum and its compounds in processing, packaging, and storage of food products, and as flocculants in the treatment of drinking water may contribute to its presence in drinking water and foodstuffs.[1] World is full of xenobiotic substances such as heavy metal, pesticide and other pollutic agents. Heavy metal are metallic element of relatively high density greater than 4 gm/cm3 or 5 times greater than water and are toxic even at low concentration[2]. Heavy metal occur as a natural constituents of earth crust, can be emitted into the environment by both natural and anthropogenic causes. Major cause of emission is the mining operations. Predetermined doses of aluminium sulphate in acute (1 d) and subacute (7, 14, 21, 28ds) treatments revealed significant increase in albumin and globulin. The results are encouraging and highlights the toxic profile of aluminum sulphate and its effect on protein profile in albino rat.[3] Aluminum is the third most abundant element after oxygen and silica in the earth crust, makes up about 8% mass of crust. Instead, it is found combined with 270 different minerals. The chief ore of aluminum is bauxite aluminum. Toxicity can be traced to deposition in bones and CNS which reduces renal function. Aluminum competes Calcium absorption reduces skeleton mineralization (Osteopenia). High doses of aluminum is associated with blood-brain barrier disruption. Use of aluminum as a vaccine adjuvant is found to be toxic, Aluminium creates a dysfunctional cell that foul signaling system and CNS. Al3+ induces oxidative, genotoxic and interfacial water stress-A triple threat [4]. Albino rat is selected for this study because of its easy rearing, quick acclimatization and easy handling at lab conditions . Hence it is expected that the present investigation, hepatic functioning after exposure of aluminum sulfate in Wistar rat will be useful to observe the ill effects of aluminum sulfate on the liver and this study may also be useful in future research related to other heavy metal intoxication.[5]

MATERIALS AND METHODS

Experimental animal rearing

Randomly selected albino rats from the inbred colony of almost equal size and weight (100±20g) at room temperature (27±0.5ºC) and relative humidity (55±3%) with 12 hours light/dark cycle were given standard laboratory pellet feed (Golden Feed, New Delhi) and water *ad libitum*. This is regarding renewal of registration/constitution of IAEC/Upation of the Dr. B. R. Ambedkar University, Agra, Uttar Pradesh (Reg.No.1608/CPCSEA).

Median lethal dose determination and sub-lethal doses

The albino rats were divided into 5 groups each consisting of 5 individuals. Standard solution of the experimental test compound, aluminum sulfate was prepared by dissolving the powder into water. Different doses of aluminum sulfate were administered orally by gavage tube. The rat mortality was recorded for each dose after 14 days. The data were analyzed statistically by the log-dose/ probit regression line method[4]. A regression line was drawn on the basis of two variables, log-dose and empirical probit on a simple graph paper used to determine the expected probit, necessary for LD_{50} determination. [7]

Experimental protocol

The rats were divided into 5 experimental groups, one acute (1d) and four sub-acute (7, 14, 21, 28 ds) groups consisting of 3 rats each. The controls were run simultaneously for both acute and subacute experimental groups. The sub lethal dose of aluminium sulphate for acute (1 d) treatment was 14.8 mg/kg b.wt., while for sub-acute (7, 14, 21, 28 ds) the doses were 2.11 mg/kg b.wt, 1.057 mg/kg b.wt., 0.704 mg/kg b.wt and 0.528 mg/kgb.wt respectively.

Biochemical Analysis

Lipid profile has been estimated using standard kit methods (Span diagnostics).

STATISTICAL CALCULATIONS

In the present investigation, the formulae were used for different statistical calculations after Fischer and Yates[8] using statistical software KpKy plot version 3.0.

RESULTS AND DISCUSSIONS

Lipid profile results (Cholesterol, triglycerides, HDL, LDL, VLDL) are significant in correlation with dose and duration of aluminium sulphate (Table 1-4, Fig. 1).

Table 1

Serum triglyceride (mg/dl) in albino rats following treatment of aluminium sulphate

S.No.	Days of intoxication	No. of Rats	Serum globulin		Significance level
			Control	Treated	
			Mean ± S.Em.	Mean ± S.Em.	
1.	Acute (1d)	5	81.00±2.08	91.33±4.05	P > 0.05
2.	Sub-acute (7 ds)	5	81.00±2.08	96.66±4.91	P < 0.05
3.	Sub-acute (14 ds)	5	81.00±2.08	118.0±4.35	P < 0.001
4.	Sub-acute (21 ds)	5	81.00±2.08	134.0±7.81	P < 0.001
5.	Sub-acute (28 ds)	5	81.00±2.08	152.33±8.68	P < 0.001

± - Mean±Standard Deviation

P>0.05- Non significant; P<0.05- Significant, P<- Highly significant; P<0.001)- Very highly significant

Table 2

Serum HDL (mg/dl) in albino rats following treatment of aluminium sulphate

S.No.	Days of intoxication	No. of Rats	Serum A/G ratio		Significance level
			Control	Treated	
			Mean ± S.Em.	Mean ± S.Em.	
1.	Acute (1d)	5	54.66±1.45	44.33±2.33	P < 0.05
2.	Sub-acute (7 ds)	5	54.66±1.45	39.66±1.20	P < 0.01
3.	Sub-acute (14 ds)	5	54.66±1.45	31.00±0.57	P < 0.001
4.	Sub-acute (21 ds)	5	54.66±1.45	22.00±1.15	P < 0.001
5.	Sub-acute (28 ds)	5	54.66±1.45	17.66±1.45	P < 0.001

± - Mean±Standard Deviation

P>0.05- Non significant; P<0.05- Significant, P<- Highly significant; P<0.001)- Very highly significant

Table 3

Serum LDL (mg/dl) in albino rats following treatment of aluminium sulphate

S.No.	Days of intoxication	No. of Rats	Serum A/G ratio		Significance level
			Control	Treated	
			Mean ± S.Em.	Mean ± S.Em.	
1.	Acute (1d)	5	49.33±0.88	56.33±2.33	P > 0.05
2.	Sub-acute (7 ds)	5	49.33±0.88	62.00±4.04	P < 0.05
3.	Sub-acute (14 ds)	5	49.33±0.88	67.66±504	P < 0.05
4.	Sub-acute (21 ds)	5	49.33±0.88	72.66±6.17	P < 0.05
5.	Sub-acute (28 ds)	5	49.33±0.88	77.00±7.00	P < 0.01

± - Mean±Standard Deviation

P>0.05- Non significant; P<0.05- Significant, P<- Highly significant; P<0.001)- Very highly significant

Table 4

Serum VLDL (mg/dl) in albino rats following treatment of aluminium sulphate

S.No.	Days of intoxication	No. of Rats	Serum A/G ratio		Significance level
			Control	Treated	
			Mean ± S.Em.	Mean ± S.Em.	
1.	Acute (1d)	5	13.66±0.88	18.33±1.45	P > 0.05
2.	Sub-acute (7 ds)	5	13.66±0.88	22.66±2.90	P < 0.05
3.	Sub-acute (14 ds)	5	13.66±0.88	25.66±2.90	P < 0.05
4.	Sub-acute (21 ds)	5	13.66±0.88	29.33±3.17	P < 0.01
5.	Sub-acute (28 ds)	5	13.66±0.88	33.00±4.72	P < 0.001

± - Mean±Standard Deviation

P>0.05- Non significant; P<0.05- Significant, P<- Highly significant; P<0.001)- Very highly significant

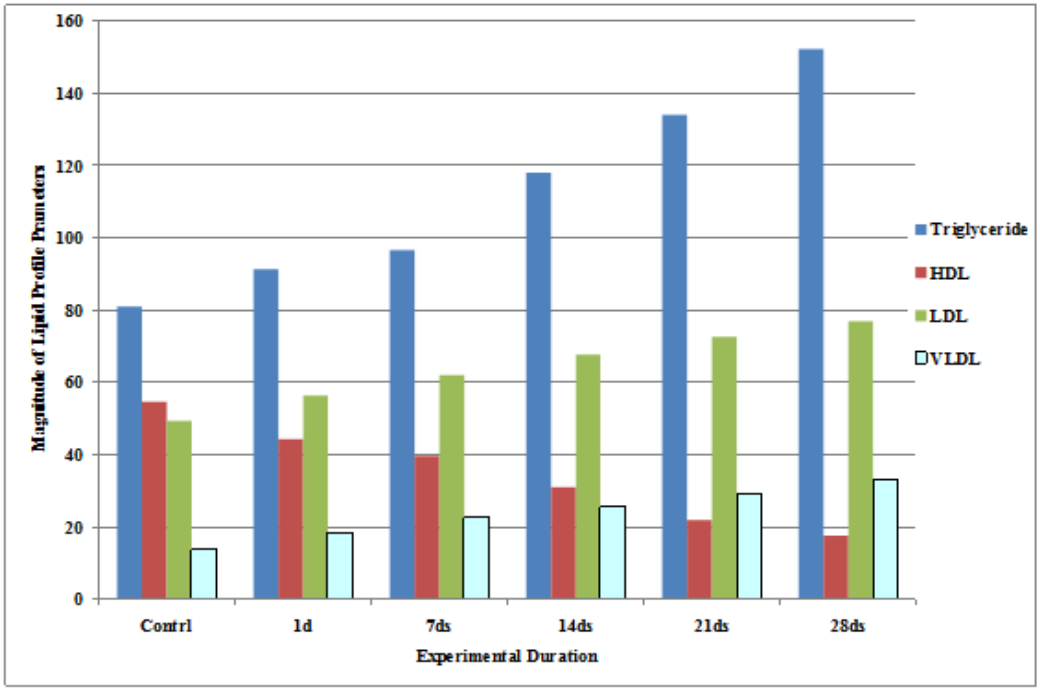

Figure 1
Comparative chart of lipid profile in albino rats following treatment of aluminium sulphate

The prevalence of cardiovascular disease (CVD) related deaths in the world are increasing and high blood pressure is one of the major contributors to these diseases. The clinical consequences of these conditions were severe and exert major research efforts to improve knowledge of its pathogenesis and thereby provide a more rationed approach to its prophylaxis and therapy[9]. From the result obtained, it was observed that there was a significant increase ($p<0.05$) in the levels of cholesterol, LDL and triglycerides in the serum of the aluminum treated rats compared to the control groups while the HDL was significantly reduced ($p<0.05$) in the aluminum treated group when compared to the control group. This may indicate aluminum toxicity. These findings agree with Salah *et al*.[10] who reported the effects of aluminum sulfate treated in deionized and tap water on lipid profile of Wistar rats. Their findings showed that there was an increase in lipid profile with an increasing dose of Aluminum sulfate. Similarly, Triglycerides and total cholesterol levels increase in response to aluminum toxicity observed in this study is consistent with increasing lipogenesis in the liver as observed by Thirunavukkarasu and Sakthisekaran[11]. This significant increase in lipid profile is an indication that inhalation exposure to aluminum sulfate may affect lipid metabolism. On one hand, lipid metabolism is affected once there is liver damage since the disturbance of cell membrane integrity is likely to cause some membrane lipids to be released into circulation; while on the other hand, it causes the tissue to compromise its effectiveness in regulating lipid metabolism. One of the main hypotheses of the mechanism of hepatocyte injury from aluminum sulphate metabolism is associated with oxidative stress and lipid peroxidation resulting from the imbalance between pro-oxidant and antioxidant chemical species[12]. Such an imbalance is associated with increased β-oxidation of fatty acids by mitochondria, peroxisomes, and cytochrome P450 2E1 (CYP2E1) pathways. These oxidative processes produce free electrons, H_2O_2, and reactive oxygen species (ROS) while depleting the potent antioxidants, glutathione[13]. The increased levels of free fatty acids present in the fatty liver provide a perpetuating and propagating mechanism for oxidative stress via lipid peroxidation, with secondary damage to cellular membranes and key organelles such as mitochondria. Lipid peroxidation usually leads to the formation of peroxyl radicals, which are central species in the peroxidation chain reaction. [14] These findings suggests that Aluminium which is ubiquitous in our environment and whose importance in our daily lives cannot be overemphasized causes toxicity when accumulated in various organs in the body and the blood cause different forms of damages to these organs. It can be concluded that aluminium toxicity increases the concentration of cholesterol and other lipid parameters in the body and hence result in cardiovascular disease. However the various antioxidants were able to ameliorate the effect of aluminium toxicity by reducing cholesterol, Triglycerides, LDL and increasing HDL. Therefore the cautious use of aluminum may be recommended for safe future of coming generation as like toxicity caused by heavy metals like aluminium[1]

CONCLUSION

This work highlights the threat to human beings from surrounding toxic material especially heavy metals used by man as utensils for cooking and for packing food materials. Man should be aware of heavy metals and avoid the usage of aluminium metal.

AUTHORS CONTRIBUTION STATEMENT

Dr. P.K. Singh conceptualized and gave guidelines with regard to this work. Mrs. Neelam Upadhyay collected data and conducted experiments in the laboratory. Mrs.

Reena Yadav help in analyzing these datas. All authors discussed the methodology and results and contributed to the final manuscript and gave necessary inputs towards the designing of the manuscript.

CONFLICT OF INTEREST

Conflict of interest declared none

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