Research Article

Toxicological Profile of Aluminium Sulphate and Effect on Protein Profile in Albino Rat

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Abstract: World is full of xenobiotic substances such as heavy metal, pesticide and other pollutic agents. Heavy metal is a metallic element of relatively high density greater than $4gm/cm^3$ or 5 times more greater than water and is toxic even at low concentration. Heavy metals occur as a natural constituent of earth crust, can be emitted into environment by both natural and anthropogenic causes. Major cause of emission is the mining operations. Predetermined doses of aluminium sulphate in acute (1d) and subacute (7, 14, 21, 28ds) treatments revealed significant increase in albumin and globulin. The results are encouraging and highlight the toxic profile of aluminum sulphate and its effect on protein profile in albino rat.

Keywords: Toxicological Profile, Protein Profile, Aluminium Sulphate, Albino Rat.

1. Introduction

Pollutants are any heavy substances in environment which causes objects to enable effects, impairing the welfare of environment reducing quality of life and may eventually cause death (Duruibe *et. al.,* 2007). The poisoning effects of heavy metals are due to their interference with the normal body biochemistry in the normal metabolic processes. When ingested, in the acid medium of the stomach, they are converted to their stable oxidation states (Zn^{2+}, Pb^{2+}, Cd^{2+}, As^{2+}, As^{3+}, Hg^{2+} and Ag^+) and combine with the body's biomolecules such as proteins and enzymes to form strong and stable chemical bonds. Although heavy metal poisoning could be clinically diagnosed and medically treated but the best option is to prevent heavy metal pollution and the subsequent human poisoning (Duruibe *et al.*, 2007).

Aluminium is the third most abundant element after O_2 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 aluminium is bauxite aluminium. Toxicity can be traced to deposition in bones and CNS which reduce renal function. Aluminium competes Calcium absorption reduce skeleton mineralization (Osteopenia) high doses of aluminium is associated with blood brain barrier. Use of aluminum as a vaccine adjuvant is found to be toxic,

Al creates dysfunctional cell that foul signaling system and CNS. Al^{3+} induces oxidative, genotoxic and interfacial water stress-A triple threat (Shaw *et al.*, 2014).

Aluminium is not as toxic as other heavy metals but there is evidence of some toxicity greater than 40 mg/day/kg of body mass. Humans are increasingly exposed to Al from food, medicinal, vaccines and cosmetics as well as industrial occupational exposure (Shaw *et al.,* 2014). Aluminium sulphate is a chemical compound with formula $Al_2(So_4)_3$, soluble in H_2O and mainly used as a flocculating agent. It is sometimes referred to as a type of alum (double sulphate salt). It is used in water purification and as a mordant in dying and printing textiles. Aluminium sulphate is sometimes used to reduce pH of garden soil and Aluminum potassium sulphate is the active ingredients in some antitranspirants and is usually found in baking powder. In the construction industry, it is used as waterproofing agent and accelerator in concrete.

Albino rat is selected for this study because of its easy rearing, quick acclimatization and easy handling at lab conditions and the most important is their similarity with humans because of mammals. Heavy metal found as a natural constituent of earth crust. It is become necessary to keep ourselves well known about the effect created by these heavy metals. Hence it is expected that the present investigation, hepatic functioning after

exposure of aluminium sulphate in Wistar rat will be useful to observe the ill effects of aluminium sulphate on liver and this study may also be useful in further researches related to other heavy metal intoxication.

2. Material and Methods

2.1 Experimental animal rearing

Randomly selected albino rats from 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.

2.2 Median lethal dose determination and sublethal doses

The albino rats were divided into 5 groups each consisting of 5 individuals. Standard solution of experimental test compound, aluminium sulphate was prepared by dissolving the powder into water. Different doses of aluminium sulphate were administered orally by gavage tube. The rat mortality was recorded for each dose after 14 days. The data were analyzed statistically by log-dose/ probit regression line method (Finney, 1971). 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.

2.3 Experimental protocol

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

2.4 Biochemical Analysis

Total protein was estimated by Biuret method described by Henry *et al.*, (1974). Proteins react with cupric ions of biuret in an alkaline medium to form a violet-blue coloured complex which appears as a result of the reaction between –CO and –NH_2 groups of protein cupric ions. The intensity of the coloured complex so developed is proportional to the total protein concentration in the sample. Albumin in serum binds with dyes bromocresol at pH-4.2 to form a green colour complex. The optical density is measured colorimetrically at 600nm (use red filter). The globulin was calculated by the deduction of amount of albumin from protein content. The A/G ratio was calculated by the dividing albumin content by globulin content.

2.5 Statistical Calculations

In the present investigation, the formulae were used for different statistical calculations after Fischer and Yates (1950) using statistical software.

3. Results and Discussion

Protein profile results (albumin, globulin, A/G ratio) are significant in correlation with dose and duration of aluminium sulphate (Fig. 1 and Table 1-3).
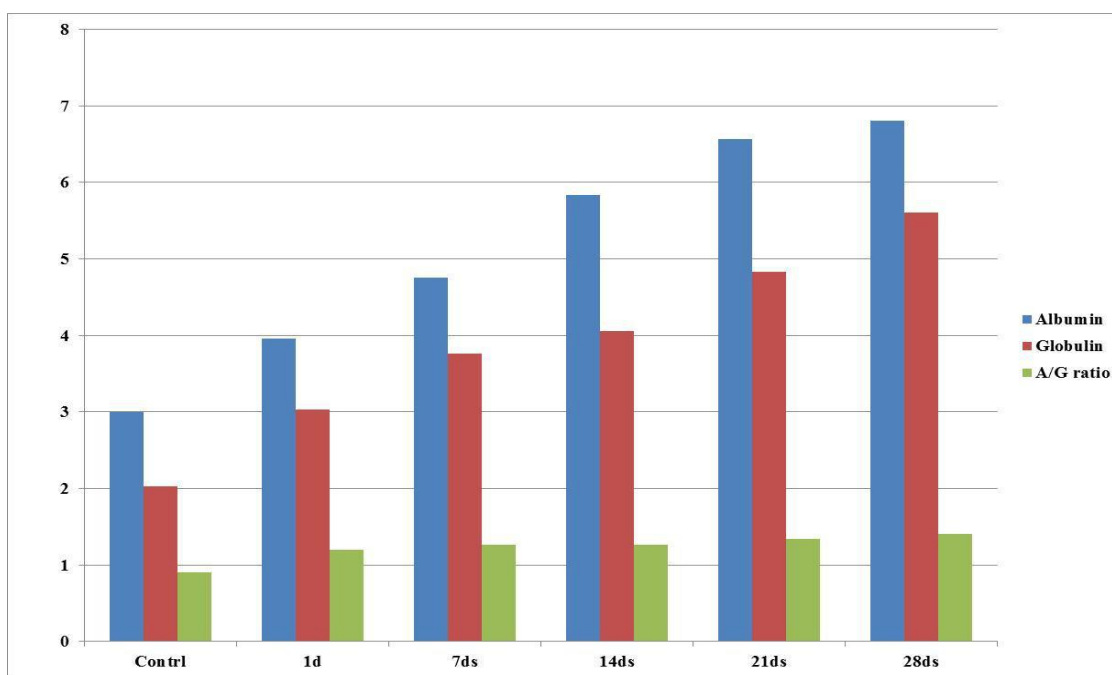

Fig. 1. Comparative chart of serum albumin, globulin and A/G ratio in albino rats following treatment of aluminium sulphate.

Table 1. Serum albumin (mg/dl) in albino rats following treatment of aluminium sulphate.

S. No.	Days of intoxication	No. of Rats	Serum albumin		Significance level
			Control Mean ± S.Em.	Treated Mean ± S.Em.	
1.	Acute (1d)	3	3.00±0.05	3.96±0.29	P < 0.05
2.	Subacute (7ds)	3	3.00±0.05	4.76±0.46	P < 0.01
3.	Subacute (14ds)	3	3.00±0.05	5.83±0.49	P < 0.001
4.	Subacute (21ds)	3	3.00±0.05	6.56±0.49	P < 0.001
5.	Subacute (28ds)	3	3.00±0.05	6.80±0.51	P < 0.001

Table 2. Serum globulin (mg/dl) in albino rats following treatment of aluminium sulphate.

S. No.	Days of intoxication	No. of Rats	Serum globulin		Significance level
			Control Mean ± S.Em.	Treated Mean ± S.Em.	
1.	Acute (1d)	3	2.03±0.12	3.03±0.24	P > 0.05
2.	Subacute (7ds)	3	2.03±0.12	3.76±0.43	P < 0.01
3.	Subacute (14ds)	3	2.03±0.12	4.06±0.44	P < 0.001
4.	Subacute (21ds)	3	2.03±0.12	4.83±0.49	P < 0.001
5.	Subacute (28ds)	3	2.03±0.12	5.60±0.40	P < 0.001

Table 3. Serum A/G ratio in albino rats following treatment of aluminium sulphate.

S. No.	Days of intoxication	No. of Rats	Serum A/G ratio		Significance level
			Control Mean ± S.Em.	Treated Mean ± S.Em.	
1.	Acute (1d)	3	0.90±0.05	1.20±0.05	P < 0.05
2.	Subacute (7ds)	3	0.90±0.05	1.26±0.04	P < 0.01
3.	Subacute (14ds)	3	0.90±0.05	1.26±0.08	P < 0.01
4.	Subacute (21ds)	3	0.90±0.05	1.34±0.06	P < 0.01
5.	Subacute (28ds)	3	0.90±0.05	1.41±0.08	P < 0.001

The exposures of workers engaged in the synthesis of the simple aluminum sulfate salt were reported. Exposures were poorly controlled as a result of start-up problems, and atmospheric exposure concentrations ranged from 0-53 mg/cu m as dust with 25-30% in the respirable range. The employees manifested nocturnal wheezing and breathlessness characteristic of occupational asthma. This reversible airway obstruction persisted despite 2-5 years of nonexposure in 11 of the 19 workers initially affected. The similarity of these cases of reversible obstructive airway disease or occupational asthma cases to those seen in aluminum smelters is noteworthy. However, the occupational asthma syndrome is not uncommon, as it has been reported consequent to other irritant metal salt exposures, such as from chromates, platinum, nickel, and vanadium (Bingham *et al*., 2001).

Subchronic or Prechronic Exposure/ Male Sprague Dawley rats were given 0.3% aluminum sulfate (as 3.7% aluminum sulfate octadecahydrate solution) in drinking water for thirty days. During the last three weeks of aluminum sulfate administration, treated animals consumed 18 + or - 0.8mL of aluminum sulfate solution/day/rat (2.0mmoles of aluminum/day/rat) and controls consumed /about/ 28mL of water/day/rat. Using a Lafayette passive avoidance device, an impairment of both consolidation and extinction of a passive avoidance task was seen in rats (n=4 to 10). General motor activity was measured in an open field apparatus and a Lafayette step-up avoidance device was used for active avoidance tasks. To assess working memory in an appetitive task paradigm, a standard, wooden open eight-arm maze was used. No impairment of performance was observed on an active avoidance task (n=6, controls=4), radial arm maze (n=10, controls=4) or open field activity measure. Biochemical analysis of other aluminum sulfate treated rats indicated a slight but significant increase in hippocampal muscarinic receptor number as determined by tritiated quinuclidinyl benzilate, binding. No changes were found in choline acetyltransferase activity, phosphoinositide hydrolysis, (3)H-quinuclidinyl benzilate QNB binding in the cortex or (3)H-pirenzepine PZ binding in the hippocampus or cortex (Connor *et al*., 1988).

Proteins are the important organic substances required in tissue building and repair. Protein profile contents (albumin, globulin) have been increased with increase in duration of exposure. This was possibly due to the direct effect of the toxic compound on protein metabolic demands following exposure to the toxic stress of aluminium sulphate. Proteins are the mainly involved in the architecture of the cell. During chronic period of stress, they are also a source of energy (Umminger, 1977).

The enhancement in serum protein profile (albumin, globulin and A/G ratio) after acute and subchronic intoxication of aluminium sulphate reveals the possibility of liver disorders. In these circumstances, hyperglobulinemia may be the result of

inflammatory reactions of the reticuloendothelial system. This usually occurs during acute and chronic hepatocellular disorders. In these disorders, defective enzyme system may promote degradation of protein in liver and other tissue and amino acids so formed may be converted to glucose through gluconeogenesis or by transamination into keto acids (Arora *et al*., 2014)

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