Effect of acute selenium toxicity in broiler birds

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Abstract— **The present study was carried out to analyze acute selenium toxicity under experimental conditions. Examination of organs did not reveal changes except congestion and hemorrhages in the lungs, tubular degeneration in kidneys and occasional hemorrhages in the brain. From the results of the present studies, it is reasonable to infer that the selenium concentrations employed in both the groups proved toxic to the chicks**.

Keywords— **Acute, Selenium toxicity, Chicks**

I. INTRODUCTION

Selenium function is closely associated with Vitamin E. Both have antioxidant property and protect the biological membranes from oxidative degeneration. Glutathione peroxidase is the enzyme which has selenium as its constituent with 4 gm Se atoms per mole. Out of the total body selenium, 40 per cent is in the enzyme glutathione peroxidase as observed in rats. Both glutathione peroxidase and vitamin E prevent the formation of lipid hydroperoxides which damage the cellular membrane and disturbed the structural integrity of the cells. Selenium and vitamin E are mutually replaceable to some extent but up to a particular limit.

The present study was conducted on 300 day-old apparently healthy broiler chicks of either sex procured from a commercial hatchery in Kolkata, India. All the chicks were from the same hatch and also from the same breeding stock. The birds were maintained under standard feeding and management conditions.

II. MATERIALS AND METHODS

A total 120 broiler chicks at the age of two weeks were randomly divided into three Groups *viz*., A, B and C consisted of 40 birds in each group. All the birds in group A were given sodium selenite @ 30 ppm in drinking water. The chicks in Group B were also supplied with sodium selenite in drinking water but the concentration of sodium selenite was 15 ppm instead of 30 ppm. The birds of group C were given plain (without sodium selenite) drinking water throughout the experimental period.

III. RESULTS AND DISCUSSION

A. Determination of acute LD_{50} of sodium selenite (Na_2SeO_3)

Acute LD_{50} of Na_2SeO_3 was determined by Karber's methods as described by Ghosh *et al.* [1]. The results of oral ALD_{50} of sodium selenite of broiler chickens are represented in Table 1. The oral LD_{50} of selenium in sodium selenite in broiler chickens is 9.590838 mg per kg body weight, contained in that compound. The oral ALD_{50} of sodium selenite was determined by Karber's method and was found to be 9.59038 mg/kg body weight.

B. Estimation of selenium level in liver

For the estimation of selenium concentration in liver tissue, portions of liver from each bird were collected separately in dry and sterilized glass vials and were kept in deep freeze (- 20^{o}C) till further use. Weighed samples (1-2 gm) of livers from each case were subjected to slow digestion as per the method of Olson *et al*. [2]. The results of estimation of selenium levels (in mg) in liver of birds from different groups at various intervals of the experiment are represented in Table 2.

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C. Statistical analysis

All the estimated data were analyzed by analysis of variance and the significant result were shown by difference in superscripts with respective means [3].

TABLE I

Oral acute LD_{50} (ALD_{50}) of sodium selenite in broiler chickens

Group	Dose (mg/kg)	No. of birds	Dose difference (a)	No. of Deaths	Mean mortality (b)	a×b
A	1	20	5	0	0	0
B	5	20	5	0	0	0
C	10	20	5	2	1.5	7.5
D	15	20	5	5	3	15
E	20	20	5	8	5.5	27.5
F	25	20	5	15	8	40
G	30	20	5	20	18	90

Total = 180

LD_{50} = 30 – 180/20 = 21 mg/kg

The chemical formula of sodium selenite is Na_2SeO_3.

Therefore, the molecular weight of sodium selenite = (2×22.98+78.96+3×15.99) = 172.89

Thus, mg of sodium selenite contains 78.98 mg selenium.

So, 21 mg of sodium selenite contains

$$\frac{78.96\times 21}{172.89} = 9.590838 \text{ mg selenium.}$$

TABLE 2

Average values of selenium levels (mg) indifferent groups of broiler chicken at different time intervals

Days after selenium administration	Selenium levels (mg) in liver		
	Group A	Group B	Group C
21	1.30^{a} ±0.20	0.80^{ab} ± 0.05	0.30^{b} ± 0.03
28	2.60^{a} ± 0.66	2.18^{a} ± 0.23	0.43^{b} ± 25.65
35	2.90^{a} ± 0.41	2.26^{a} ± 0.14	0.56^{b} ± 0.08
42	NS	2.43^{a} ± 0.23	0.66^{b} ± 0.18

Mean within a column bearing different superscripts differ significantly (P<0.05). Values are presented as Mean ± SE. NS: Not significant.

Salyi *et al.* [4] induced acute experimental selenium poisoning in broiler chicks and concluded that oral LD_{50} of selenium in the form of sodium selenite was 9.7 mg/kg body weight, which were almost similar to the present determined values. The findings in the present study are in accordance with the observations of earlier workers [4], [5], [6] who have reported significant decline in body weight of selenium fed chickens. As revealed in the present study, the organic form of selenium is known to be accumulated in higher quantities and persist for longer periods in tissues as compared to inorganic toxicity [7]. Increasing trends in the liver concentration of selenium in chicks, hens and cockerels have been reported by other workers [5]. Khan *et al*. [5] proposed that accumulation of selenium was dose dependent which simulates with the observations of the present study. According to Cousins and Cairney [8] increased intake of selenium resulted in steady rise in tissue selenium concentrations until levels as high as 5-7 ppm in liver and kidneys.

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