



E-ISSN: 2320-7078

P-ISSN: 2349-6800

JEZS 2017; 5(2): 1020-1023

© 2017 JEZS

Received: 13-01-2017

Accepted: 14-02-2017

Ritesh Jaiswal

College of Veterinary Science
and A.H. Anjora, Durg,
Chhattisgarh, India

SL Ali

College of Veterinary Science
and A.H. Anjora, Durg,
Chhattisgarh, India

S Roy

College of Veterinary Science
and A.H. Anjora, Durg,
Chhattisgarh, India

S Sakya

College of Veterinary Science
and A.H. Anjora, Durg,
Chhattisgarh, India

OP Dinani

College of Veterinary Science
and A.H. Anjora, Durg,
Chhattisgarh, India

Sudhir Kumar Jaiswal

Indian Veterinary Research
Institute, Izatnagar, Bareilly,
Uttar Pradesh, India

Correspondence

Sudhir Kumar Jaiswal

Indian Veterinary Research
Institute, Izatnagar, Bareilly,
Uttar Pradesh, India

Effect of induced lead intoxication on serological parameters without or with different antioxidants in broiler chicken

Ritesh Jaiswal, SL Ali, S Roy, S Sakya, OP Dinani and Sudhir Kumar Jaiswal

Abstract

In the present study effect of lead intoxication on serum biochemical of broiler chicken has been done. The birds were divided into six groups viz. A, B, C, D, E and F. Group-A served as control whereas group-B received lead acetate @ 200 mg/kg basal diet for 42 days simultaneously Ascorbic acid @ 200 mg/kg basal diet, Vitamin-E @ 100/mg/kg and Se @ 0.1 mg/kg basal diet, DL-methionine @ 100 mg/kg and methanolic extract of *Cissus quadrangularis* (CQE) @ 400 mg/kg basal diet were given to group B, C, D, E and F respectively. The biochemical profile of treated with various levels of antioxidants on day 21 and 42 were reported total protein (6.05±0.47 to 6.85±0.39 and 3.83±0.09 to 4.60±0.29), albumin (2.77±0.09 to 3.77±0.24 and 2.25±0.09 to 2.95±0.16), albumin: globulin ratio (0.74±0.04 to 1.89±0.49 and 1.08±0.13 to 2.06±0.25), Serum glutamic oxaloacetic transaminase (1.26±0.16 to 2.62±0.13 and 0.96±0.16 to 1.72±0.19), Serum glutamic pyruvate transaminase (14.07±1.08 to 17.28±0.90 and 8.88±0.25 to 14.87±0.49), blood urea nitrogen (6.07±0.75 to 11.22±0.45 and 2.98±0.21 to 4.60±0.17), creatinine (0.64±0.05 to 1.50±0.09 and 1.56±0.19 to 2.20±0.15) and alkaline phosphatase (3.41±0.25 to 4.40±0.32 and 2.50±0.57 to 4.00±0.78). All biochemical parameters in lead treated group were significantly different ($P<0.05$) from the normal range on day 21 and 42 as compared to various antioxidant treatments and control group. All the antioxidant treatments were found effective in bringing the biochemical parameters within the normal range. Methanolic extract of *Cissus quadrangularis* were reported to be most effective to ameliorate lead toxicity in broiler chicken.

Keywords: Lead, antioxidants, serological parameters, broiler

1. Introduction

Among all heavy metals lead is being an ubiquitous environmental pollutant, particularly widespread in industrial areas due its significant role in modern industry^[1]. However, both occupational and environmental exposure a serious problem in many developing and industrializing countries causing health problems in man and its contingent including animals^[2, 3]. Lead produces acute and chronic poisoning and induces a broad range of many undesired pathological effects in each and every organs and system of the body^[4] including physiological, biochemical, neurological^[5, 6] in animals. Lead produces acute and chronic poisoning and induces a broad range of many undesired pathological effects in each and every organs and system of the body including physiological, biochemical, neurological, immunological, renal, hepatic, behavioral, and especially haematological dysfunctions in animals^[4, 5, 6]. As ascorbic acid, α -tocopherol, DL-methionine, selenium and *Cissus quadrangularis* (herbal plant) have also been reported to modulate the oxidative effect of lead^[3, 9] Considering the above facts a study was conducted to study the effect of induced lead intoxication on serological parameters without or with different antioxidants in broiler chicken.

2. Materials and Methods

2.1 Housing and management

The present study was undertaken in the Department of Veterinary Medicine, College of Veterinary Science and A.H., Anjora, Durg (Chhattisgarh) during the year 2010 in the period of month of November-December. Broiler birds were maintained in deep litter system in open sided poultry house temperature ranging between 20-24 °C and relative humidity 44-48%.

The experiment was in accordance with animal welfare, and conducted under the protocols of Veterinary faculty, Anjora, Durg (Chhattisgarh) with the approval of Institutional Animal Ethics Committee (IAEC).

2.2 Experimental birds and feeding

For present study a total of 126 day old broiler chicks a of (Ven-Cobb strain) either sex along with broiler feed were procured from a well-organized private hatchery of Indian Broiler Group, Rajnandgaon, Chhattisgarh. The diets were formulated according to the broiler chicken requirements suggested by the National Research Council (NRC) guidelines [7]. All treatments were given to chicks on the basis of per kg basal diet daily for the period of 42 days. In this

small amounts of basal diet was first mixed with the respective amounts of lead acetate @ 200 mg/kg to induce lead toxicity and with treatment drugs viz. ascorbic acid @ 200 mg/kg, Vit-E @ 100 mg/kg and Se @ 0.1 mg/kg, DL-methionine @ 100 mg/kg and methanolic extract of *Cissus quadrangularis* powder @ 400 mg/kg as per treatment groups in a small batch. Then it was mixed with a larger amount of basal diet, until the total amount of respective diets were homogeneously mixed and was treated as per the experimental design.

2.3 Experimental design

Completely randomized design (CRD) has been used in this experiment as mentioned below in the table no.1.

Table 1: Experimental design

Groups	No. of birds	Treatment given on the basis of per kg basal diet
A	21	Basal diet
B	21	Lead acetate @ 200 mg/kg
C	21	Lead acetate @ 200 mg/kg + Ascorbic acid @ 200 mg/kg
D	21	Lead acetate @ 200mg/kg + Vit-E @ 100 mg/kg and Se @ 0.1 mg/kg
E	21	Lead acetate @ 200 mg/kg + DL-methionine @ 100 mg/kg
F	21	Lead acetate @ 200 mg/kg + Methanolic extract of <i>Cissus quadrangularis</i> (CQE) @ 400 mg/kg

2.4 Statistical analysis

To test the difference between different groups, analysis of variance with one way classification followed by Duncan's Multiple Range Test (DMRT) was applied as per the standard procedure outlined by [8].

3. Results and discussion

During the present study various biochemical parameters were analyzed by using semi-auto analyzer and Bayer's diagnostic kits. The results of various biochemical indicators of lead intoxication are summarized in the Table-2 for day 21 and Table-3 for day 42.

Table 2: Effect of lead and different treatments on biochemical indicators in broiler chickens on day 21

Biochemical parameters	Various treatment groups showing mean \pm SE values					
	A	B	C	D	E	F
Total protein (g/dl)	6.85 \pm 0.39 ^a	9.82 \pm 0.37 ^b	6.52 \pm 0.47 ^a	6.57 \pm 0.07 ^a	6.54 \pm 0.38 ^a	6.05 \pm 0.47 ^a
Albumin (g/dl)	3.48 \pm 0.36 ^{ab}	3.03 \pm 0.49 ^{ab}	3.77 \pm 0.24 ^b	2.77 \pm 0.09 ^a	2.81 \pm 0.08 ^a	3.29 \pm 0.24 ^{ab}
A :G ration	1.32 \pm 0.35 ^{abc}	0.49 \pm 0.12 ^a	1.89 \pm 0.49 ^c	0.74 \pm 0.04 ^{ab}	0.80 \pm 0.09 ^{ab}	1.57 \pm 0.41 ^{bc}
SGOT (U/L)	1.43 \pm 0.19 ^a	3.05 \pm 0.22 ^c	1.26 \pm 0.16 ^a	2.62 \pm 0.13 ^c	2.09 \pm 0.20 ^b	1.60 \pm 0.11 ^{ab}
SGPT (U/L)	14.07 \pm 1.08 ^a	17.52 \pm 0.94 ^b	17.17 \pm 0.59 ^b	17.28 \pm 0.90 ^b	16.80 \pm 0.85 ^{ab}	15.54 \pm 1.21 ^{ab}
Creatinine (mg/dl)	1.50 \pm 0.09 ^d	1.58 \pm 0.07 ^d	0.79 \pm 0.06 ^{ab}	1.04 \pm 0.16 ^{bc}	1.26 \pm 0.16 ^{cd}	0.64 \pm 0.05 ^a
BUN (mg/dl)	11.22 \pm 0.45 ^b	12.65 \pm 0.75 ^b	10.90 \pm 0.36 ^b	8.13 \pm 0.65 ^a	6.71 \pm 1.08 ^a	6.07 \pm 0.75 ^a
ALP (U/L)	4.24 \pm 0.37 ^a	7.63 \pm 0.55 ^b	4.35 \pm 0.23 ^a	4.40 \pm 0.32 ^a	4.30 \pm 0.31 ^a	3.41 \pm 0.25 ^a

^{ab} Mean values bearing different superscripts within rows differ significantly ($P < 0.05$)

Table 3: Effect of lead and different treatments on biochemical indicators in broiler chickens on day 42

Biochemical Parameters	Various treatment groups showing mean \pm SE values					
	A	B	C	D	E	F
Total protein (g/dl)	3.92 \pm 0.13 ^{ab}	5.72 \pm 0.33 ^d	4.48 \pm 0.05 ^{bc}	5.27 \pm 0.23 ^d	3.83 \pm 0.09 ^a	4.60 \pm 0.29 ^c
Albumin (g/dl)	2.25 \pm 0.09 ^a	3.40 \pm 0.07 ^d	2.95 \pm 0.16 ^c	2.66 \pm 0.08 ^{bc}	2.40 \pm 0.14 ^{ab}	2.61 \pm 0.16 ^{abc}
A :G ratio	1.43 \pm 0.18 ^{ab}	2.98 \pm 0.39 ^c	2.06 \pm 0.25 ^b	1.08 \pm 0.13 ^a	1.93 \pm 0.42 ^{ab}	1.37 \pm 0.14 ^{ab}
SGOT (U/L)	1.75 \pm 0.11 ^b	2.14 \pm 0.11 ^c	0.96 \pm 0.16 ^a	1.62 \pm 0.07 ^b	1.72 \pm 0.19 ^b	1.14 \pm 0.10 ^a
SGPT (U/L)	14.87 \pm 0.49 ^c	18.47 \pm 0.52 ^d	10.97 \pm 0.36 ^b	11.43 \pm 0.44 ^b	11.47 \pm 0.19 ^b	8.88 \pm 0.25 ^a
Creatinine (mg/dl)	1.78 \pm 0.23 ^{ab}	2.48 \pm 0.12 ^c	1.85 \pm 0.23 ^{ab}	2.04 \pm 0.11 ^{abc}	2.20 \pm 0.15 ^{bc}	1.56 \pm 0.19 ^a
BUN (mg/dl)	4.60 \pm 0.17 ^b	6.47 \pm 0.65 ^c	3.66 \pm 0.14 ^{ab}	3.78 \pm 0.16 ^{ab}	3.89 \pm 0.14 ^{ab}	2.98 \pm 0.21 ^a
ALP (U/L)	4.00 \pm 0.78 ^a	6.69 \pm 0.56 ^b	2.57 \pm 0.28 ^a	3.16 \pm 0.19 ^a	3.10 \pm 0.17 ^a	2.50 \pm 0.57 ^a

^{abcd} Mean values bearing different superscripts within rows differ significantly ($P < 0.05$)

3.1 Total protein

The values of total protein are presented in Table no.2. A significantly ($P < 0.05$) higher mean total protein level in plasma was recorded in group B (9.82 \pm 0.37 g/dl) as compared to control group A (6.85 \pm 0.39 g/dl) and other treatment group C (6.52 \pm 0.47 g/dl), D (6.57 \pm 0.07 g/dl), E (6.54 \pm 0.38 g/dl), and F (6.05 \pm 0.47 g/dl), but comparative changes among different treatment groups showed nonsignificant ($P > 0.05$) changes in group A, C, D, E, and F on day 21. On day 42

(Table-3) group B (5.72 \pm 0.33 g/dl) showed significantly ($P < 0.05$) higher total protein value than the group A (3.92 \pm 0.13 g/dl), C (4.48 \pm 0.05 g/dl), E (3.83 \pm 0.09 g/dl) and F (4.60 \pm 0.29 g/dl) and non-significantly ($P > 0.05$) from group D (5.27 \pm 0.23 g/dl). In present study lead treated group showed significant higher total plasma protein as compared to control and vitamin-C, vitamin-E & Se, DL-methionine and CQE treated group on day 21 and 42 except vitamin-E & Se treated group which showed non-significant ($P > 0.05$) decrease of

total plasma protein from lead treated group.

The present findings are not in conformity with the findings of [10] who reported supplementation of dietary lead @ 200 mg/kg diet alone or combination of lead @ 200 mg/kg diet plus ascorbic acid @ 100 mg/kg diet for period of 42 days of broilers did not show significant changes on serum total protein from the control group.

3.2 Albumin

This study showed that mean plasma albumin values of lead treated group B (3.03 ± 0.49 g/dl) were non-significantly ($P > 0.05$) comparable (Table-2) from group A (3.48 ± 0.36 g/dl), C (3.77 ± 0.24 g/dl), D (2.77 ± 0.09 g/dl), E (2.81 ± 0.08 g/dl) and F (3.29 ± 0.24 g/dl) on day 21. But comparison among different treatment groups showed significantly ($P < 0.05$) high plasma albumin level in group C from group A, D, E while others showed non-significant ($P > 0.05$) changes on day 21. At the end of study (Table-3) lead treated group B (3.40 ± 0.07 g/dl) showed significantly ($P < 0.05$) higher plasma albumin value from group-C (2.95 ± 0.16 g/dl), D (2.66 ± 0.08 g/dl), E (2.40 ± 0.14 g/dl), F (2.61 ± 0.16 g/dl) and control group-A (2.25 ± 0.09 g/dl). There seems to be no available reports to compare the findings of the present study with the others.

3.3 Albumin: Globulin ratio (A: G ratio)

Present study showed that mean A:G ratio of lead treated group B (0.49 ± 0.12) was significantly ($P < 0.05$) lower (Table-2) from group C (1.89 ± 0.49) and group-F (1.57 ± 0.41) but non-significantly ($P > 0.05$) from control group A (1.32 ± 0.35), D (0.74 ± 0.04) and E (0.80 ± 0.09) on day 21. On day 42 (Table-3) lead treated group B (2.98 ± 0.39) showed significant ($P < 0.05$) high A:G ratio from the group C (2.06 ± 0.25), D (1.08 ± 0.13), E (1.93 ± 0.42), F (1.37 ± 0.14) and control group-A (1.43 ± 0.18). In present study vitamin-E & Se showed lowest A:G ratio than the other treatment groups showed their better therapeutic efficacy followed by CQ extract > DL-methionine > vitamin-C against chronic lead intoxication in poultry on day 42.

Scarcely literature are available till date to define the A:G ratio in poultry due to lead toxicosis. However, it may be proposed that increase A:G ratio in lead treated group on day 42 might be due to suppressive effect of lead on immune system related organ like spleen and bursa of fabricus. These possible reasons and present findings corroborate with the findings of [10, 11] describing dose dependent lymphocytic depletion of lymphoid population in bursal follicle and spleen, decreased average bursal weight which confirms the immunosuppressive effect of lead in broilers.

3.4 Serum Aspartate Aminotransferase (SGOT/ AST)

The finding of present study showed that mean plasma SGOT value (Table-2) of lead treated group B (3.05 ± 0.22 U/L) was increased significantly ($P < 0.05$) from group C (1.26 ± 0.16 U/L), E (2.09 ± 0.20 U/L), F (1.60 ± 0.11 U/L) and control group A (1.43 ± 0.19 U/L) and non-significantly ($P > 0.05$) from group D (2.62 ± 0.13 U/L) on day 21. Observation of day 42 (Table-3) revealed significantly ($P < 0.05$) higher SGOT value in lead treated group B (2.14 ± 0.11 U/L) than the group C (0.96 ± 0.16 U/L), D (1.62 ± 0.07 U/L), E (1.72 ± 0.19 U/L), F (1.14 ± 0.10 U/L) and control group A (1.75 ± 0.11 U/L). In the present study highest therapeutic efficacy to reduce the plasma AST level against chronic lead intoxication was shown by Vitamin-C followed by CQ extract > Vitamin-E & Se > DL-methionine which was more or less comparable to

result of control group.

The present findings are in agreement with earlier workers who reported elevated serum AST level in the lead induced toxicity in birds following lead administration in birds [12, 13]. The enzyme AST / SGOT is widely distributed in tissues of all species of animal especially in muscle and liver and is located in the cytoplasm. In Plumbism the mean activities of AST increased significantly ($P < 0.05$) which might be due to liver damage or dysfunction.

3.5 Serum Alanine Aminotransferase (SGPT/ ALT)

Present study showed that mean plasma ALT values of lead treated group-B (17.52 ± 0.94 U/L) were non-significantly ($P > 0.05$) comparable (Table-2) from group C (17.17 ± 0.59 U/L), D (17.28 ± 0.90 U/L), E (16.80 ± 0.85 U/L), F (15.54 ± 1.21 U/L) and significantly ($P < 0.05$) higher from control group A (14.07 ± 1.08 U/L) on day 21. On day 42 lead treated group B (18.47 ± 0.52) showed significant ($P < 0.05$) high ALT level (Table-3) from the group C (10.97 ± 0.36 U/L), D (11.43 ± 0.44 U/L), E (11.47 ± 0.19 U/L), F (8.88 ± 0.25 U/L) and control group A (14.87 ± 0.49 U/L). In the present study highest therapeutic efficacy to reduce the plasma ALT level against chronic lead intoxication was shown by CQ extract followed by Vitamin-C > Vitamin-E & Se > DL-methionine which was comparable to result of normal group.

Increased plasma ALT level was recorded in the present study. There seems to appear no published report on effect of lead on the level of ALT in chickens to compare the present findings. However, elevated ALT activity was reported by [14] in rats and [15] in buffalo calves following lead administration. Restoration in the ALT values was recorded in the present study which might be due to the hepatoprotective effect and/or due to reducing the lead concentration in different tissues.

3.6 Creatinine

In present study the mean plasma creatinine of lead treated group B (1.58 ± 0.07 mg/dl) was significantly ($P < 0.05$) higher (Table-2) from group C (0.79 ± 0.06 mg/dl), D (1.04 ± 0.16 mg/dl) and F (0.64 ± 0.05 mg/dl) and non-significantly ($P > 0.05$) higher from group A (1.50 ± 0.09 mg/dl) and E (1.26 ± 0.16 mg/dl) on day 21. Comparison of values of day 42 revealed significant ($P < 0.05$) increased creatinine value (Table-3) in lead treated group B (2.48 ± 0.12 mg/dl) than the group C (1.85 ± 0.23 mg/dl), F (1.56 ± 0.19 mg/dl) and control group A (1.78 ± 0.23 mg/dl) while non-significant ($P > 0.05$) increased from group-D (2.04 ± 0.11 mg/dl) and E (2.20 ± 0.15 mg/dl). In the present study highest therapeutic efficacy to reduce the plasma creatinine level against chronic lead intoxication was shown by CQ extract followed by Vitamin-C > Vitamin-E & Se > DL-methionine which was comparable to value of control group on day 21 and 42.

Available literature showed increased creatinine level in various species following the lead administration. Elevated creatinine in lead induced toxicity was also observed in rats, buffaloes, broiler chickens and goats by [14, 15]. Earlier finding of higher creatinine level are in conformity with the findings of present study. It is proposed that accumulation of creatinine was a result of muscle metabolism of creatine and phosphocreatine and increased plasma level of creatinine indicates alterations in the glomerular filtration, which might be due to chronic renal damage.

3.7 Blood Urea Nitrogen (BUN)

The mean BUN level of lead treated group-B (12.65 ± 0.75

mg/dl) non-significantly ($P>0.05$) increased (Table-2) from group C (10.90±0.36 mg/dl) and control group A (11.22±0.45 mg/dl) and significantly ($P<0.05$) from group D (8.13±0.65 mg/dl), E (6.71±1.08 mg/dl) and F (6.07±0.75 mg/dl) on day 21. Observations of day 42 revealed that lead treated group B (6.47±0.65 mg/dl) had significant ($P<0.05$) higher BUN level (Table-3) from group C (3.66±0.14 mg/dl), D (3.78±0.16 mg/dl), E (3.89±0.14 mg/dl), F (2.98±0.21 mg/dl) and control group A (4.60±0.17 mg/dl).

^[15] Also reported significant increase in uric acid (14.80 mg/dl) as compared to control value (8 mg/dl) in broiler chicken exposed to lead for 40 days. Urea is the principal end product of protein catabolism and pathological conditions that reduce the glomerular filtration rate are the possible reasons of increased urea level in the serum.

3.8 Alkaline phosphatase (ALP)

The mean plasma ALP of lead treated group B (7.63±0.55 U/L) was significantly ($P<0.05$) higher (Table-2) from group C (4.35±0.23 U/L) and D (4.40±0.32 U/L), E (4.30±0.31 U/L), F (3.41±0.25 U/L) and control group A (4.24±0.37 U/L) on day 21. Observation of day 42 revealed significant ($P<0.05$) high ALP value (Table-3) in lead treated group B (6.69±0.56 U/L) than the group C (2.57±0.28 U/L), D (3.16±0.19 U/L), E (3.10±0.17 U/L), F (2.50±0.57 U/L) and control group A (4.00±0.78 U/L). In present study, highest therapeutic efficacy to reduce the ALP level in chronic lead intoxication was shown by CQ extract followed by Vitamin-C >DL-methionine >Vitamin-E & Se.

^[14, 15] also reported higher ALP values in rats and buffalo calves' respectively due to lead intoxication. The mean activities of ALP increased in lead treated group might be due to hepatic damage. Increasing serum enzyme activity is often the first change noticed in liver diseases and estimation of liver specific enzyme is the most sensitive and reliable test to detect hepatic damage.

4. Conclusion

Thus, it is concluded that the biochemical profile (total protein, albumin, A:G ration, AST, ALT, BUN, creatinine and alkaline phosphatase) deviated from the normal range in lead exposed birds. All the treatments including Ascorbic acid @ 200 mg/kg, Vitamin-E @ 100/mg/kg, Se @ 0.1 mg/kg, DL-methionine @ 100 mg/ kg and methanolic extract of *Cissus quadrangularis* (CQE) @ 400 mg/kg basal diet were found effective in bringing the biochemical parameters within the normal range. Methanolic extract of *Cissus quadrangularis* were reported to be most effective to ameliorate lead toxicity in broiler chicken.

5. Acknowledgement

IB Group, Rajnandgaon, Chhattisgarh for providing free of cost Ven-Cobb strain broiler chicks; Dean, Veterinary College Anjora, Durg (Chhattisgarh) for providing necessary facilities and Head, Department of Animal Nutrition for feed formulation.

6. References

1. Shalan MG, Mostafa MS, Hassouna MM, Hassab El-Nabi SE, Elrafaie A *et al.* Amelioration of lead toxicity on rat liver with vitamin C and silymarin supplements. *Toxicology*. 2005; 206:1-15.
2. McDowell LS. *Minerals in Animal and Human Nutrition*. Academic Press, Inc. California. 1992, 361-364.
3. Gurer H, Ercal N *et al.* Can antioxidants be beneficial in

the treatment of lead poisoning? *Free Radical Biology and Medicine*. 2000; 29:927-945.

4. Goyer RA. Lead toxicity from over subclinical to subtle health hazards. *Environmental Health and Safety*. 1990; 86:177-181.
5. Moreira EG, Vassilieff VS, Vassilieff I *et al.* Developmental lead exposure: behavioral alterations in the short and long term. *Neurotoxicology and Teratology*. 2001; 23:489-495.
6. Soltaninejad K, Kebriaeezadeh A, Minaiee B, Ostad SN, Hosseini R, Azizi E *et al.* Biochemical and ultrastructural evidences for toxicity of lead through free radicals in rat brain. *Human and Experimental Toxicology*. 2003; 22:417-423.
7. NRC. National Research Council. Washington DC. 1994.
8. Snedecor GW, Cochran WG. *Statistical Methods*, 8th Edn, 2, Iowa State Univ Press, Ames, IOWA. 1994, 2.
9. Erdogan Z, Erdogan S, Aksu T, Baytok E *et al.* The effect of dietary lead exposure and ascorbic acid on performance, lipid peroxidation status and biochemical parameters of broilers. *Turkish Journal of Veterinary and Animal Science*. 2005; 29:1053-1059.
10. Chauhan RS, Khurana SK, Mahipal SK *et al.* Pathology of chronic lead poisoning in chickens. *Indian Journal of Toxicology*. 1995; 2:56.
11. Youssef SAH, Sanousi AA, Afifi NA, Braway AL *et al.* Effect of subclinical toxicity on the immune response of chicken to New Castle Disease Virus Vaccine. *Research Veterinary Science*. 1996; 60:13-16.
12. Riggs SM, Puschner B, Tell LA *et al.* Management of an ingested lead foreign body in an Amazon parrot. *Veterinary and Human Toxicology*. 2002; 44:345-348.
13. Puschner B, Robert HP *et al.* Lead and Zinc intoxication in companion birds. *Compendium*. University of California, Davis. 2009; 31(1).
14. Mousa SA, Bashandy SA *et al.* Biophysical and biochemical changes in the blood of rats exposed to lead toxicity. *Romanian Journal of Biophysics*. 2008; 18(2):123-133.
15. Brar RS, Sandhu HS, Grewal GS *et al.* Biochemical alterations induced by repeated oral toxicity of lead in domestic fowl. *Indian Veterinary Journal*. 1997; 74:380-383.