

LEAD TOXICITY IN BULL

Mona S. Zaki⁽¹⁾; Nabila El Batrawy⁽²⁾ Olfat M. Fawzi⁽³⁾ and Nagwa S. Ataa⁽⁴⁾

⁽¹⁾ Dept. of Hydrobiology, N.R.C.

⁽²⁾ Dep. of microbiology, Animal Reproduction Research Inst.

⁽³⁾ Dept. of Biochemistry, N.R.C.

⁽⁴⁾ Department of microbiology, N.R.C

olfatdarwish@yahoo.com

Abstract: The problem of lead toxicity originated in a private farm in *El- Katta* "Giza governorate", due to ingestion of plant polluted with lead. About 8 out of 50 bull animals showed lead toxicity. The animal's age was 6 months. The animals suffered from depression, pressing head against objects, dilatation of eye pupils, total blindness (in 2 cases) with normal light reflex in both eyes, edema in briskets, enteritis with bloody diarrhea and pupil dilation. Also there were lacrimation, pale dirty mucous membrane and sunken eyes. Serum analysis from these animals revealed high lead concentration. In addition too, significant decrease in the levels of testosterone, LH, FSH. PCV, haemoglobin, R.B.C.s and total proteins were also decreased. Highly degeneration of kidney, and liver accompanied with elevation of AST, ALT, Urea, creatinine, cortisol, sodium, and potassium. Moreover, *Sterptococcus sp.*, *S. epidermidis* and *S. Aeruginosa* were isolated. We conclude that the cause of animals morbidity and mortality in this farm was not due to bacterial infections but due to lead toxicity and we can say that polluted environment, especially with lead, can cause severe harm to animal health, in addition to serious danger on human health, by eating food polluted with lead. [Report and Opinion. 2010;2(2):62-66]. (ISSN: 1553-9873).

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Introduction

Lead is a major environmental pollutant and its toxicity continues to be a major public health problems, and there is a growing consensus that lead cause toxic injury to human at a level of exposure that was considered to be safe only a decade ago. As the chronic exposure to lead, even at low levels, can result in slow progressive, in most of time, irreversible damage to haematopoeitic, nervous and renal systems [1]. Recent researches predicted that high levels of toxic metals in scalp hair, due to environmental pollution, in addition to deficiency in trace metals, play a role in the development of heart diseases [2].

In recent years, research efforts are directed towards quantification of the impact of lead exposure on human health, particularly from environment. The diagnosis based on blood lead levels doesn't always give an accurate estimate of total body burden of lead, so it's important to detect subcellular damage using reliable sensitive biomarkers [3]. Animals are very good indicator of the environmental pollution, as they inhabit the same space as humans and are exposed to the action of the same pollutants, for that reason, it's appropriate and advantageous to evaluate the negative impact of the polluted environment by heavy metals, and their influences load on human health by parallel evaluation of their load on animals [4, 5], so measuring of lead in blood animals [6], in cows and especially in lactating one, proved to be a good indicator of

environmental contamination and also for food contamination from polluted animal [7].

Materials & Methods:

Animals:

8 young bull of 6 months old were used in the present study. They were obtained from a private farm in *El-Katta* "Giza governorate". The animals suffered from depression, pressing head against objects, dilatation of eye pupils, total blindness (in 2 cases) with normal papillary light reflex in both eyes, edema in briskets, enteritis with bloody diarrhea and papillary dilation. Also there were lacrimation, pale dirty mucous membrane and sunken eyes.

Haematological studies:

Blood samples were collected from the jugular vein on EDTA as anticoagulant for determination of Hb, PVC, ESR, RBC's count, and WBC's count, according to [8].

Biochemical and Hormonal studies:

The activities of aspartic aminotransferase (AST) and alanine aminotransferase (ALT) as well as cholesterol, urea and creatinine levels were determined

according to the method of Varley *et al.*, [9] by using commercial kits (Bio Merieux, France).

Total serum protein was estimated according to Drupt [10]. Serum cortisol was analyzed by a Gamma counter using ^{125}I cortisol radioimmunoassay kit (Baxter Health Care Corporation USA) according to the method described by Pickering and Pottinger [11]. Potassium, Sodium and lead concentrations were determined by atomic absorption spectrophotometry.

Bacteriological studies:

Swabs from internal organs (liver, kidney, intestine and lungs) were collected under aseptic condition. The inoculated plates were incubated at 37°C for 24-48 h. The suspected colonies were picked and purified by further subculturing after which they were stained with Gram stain, for further biochemical identifications, the subjected isolates were classified according to Buchman *et al.*, [12] and Wilson & Miles [13].

Soil Forage:

The lead content of the soil and forage was measured by atomic absorption according to the

method of Rodrigues and Castellon [14].

Statistical analysis:

The obtained data were subjected to the student t-test according to Gad and Well [15].

RESULTS:

In the present study, haematological examination showed significant decrease in haemoglobin, PCV, and RBC's count and significant increase was found in ESR and W.B.C's count in all animals (Table 1).

Biochemical results detected significant increase in AST, ALT, urea, creatinine, cortisol, sodium, potassium and lead (Table 2). While, there was significant decrease in total protein level (Table 2). Serum LH, F.S.H, and testosterone hormones were significantly decreased (Table 3).

The lead content was found to be 293.72 p.p.m in the polluted soil and 164.3 p.p.m in forage (Table 4).

Bacteriological results revealed that the most predominant isolated micro-organisms was *Streptococcus*, *S. epidermis* and *S. Aeurogenosa* (Table 5).

Table (1): Effect of lead toxicity on some hematological parameters (Mean values \pm SE) in bull.

Parameters	Control (8)	Bull (8)
P.C.V	31 \pm 1.2	31 \pm 1.4
E.S.R	1.1 \pm 0.01	1.8 \pm 1.3*
R.B.C's count 10^6 /ml	6.33 \pm 0.28	4.4 \pm 1.24*
W.B.C's count 10^3 /ml	8.35 \pm 0.23	11.03 \pm 0.49*
HB g/dl	8.1 \pm 0.01	7.13 \pm 0.14*

*P < 0.01

Table (2): Effect of lead toxicity on some biochemical parameters (Mean values \pm SE) in bull.

Parameters	Control (8)	Bull (8)
AST U/l	131 \pm 1.23	174 \pm 2.45*
ALT U/l	31 \pm 1.46	62 \pm 1.62*
Total Protein g/ dl	10.4 \pm 1.68	8 \pm 0.27*
Urea mg/dl	2.90 \pm 0.62	3.09 \pm 0.82*
Creatinine mg/dl	0.98 \pm 0.03	1.7 \pm 0.02*
Cortisol ng/dl	0.6 \pm 0.01	1.67 \pm 0.23*

Na ⁺	M.E.Q	97±2.2	114±3.3*
K ⁺	M.E.Q	4.7±1.3	6.2±1.9*
Lead	p.p.m	0.87±0.12	1.68±0.10*

*P <0.01

Table (3): Effect of lead toxicity on L.H, F.S.H, and testosterone hormones (Mean values ± SE)

Parameters	L.H (mu/ml)	F.S.H (mu/ml)	Testosterone (ug/dl)
Control	3.14 ± 0.32	5.4 ± 0.32	5.1 ± 0.042
Affected Animal	2.1 ± 0.14*	4.1 ± 0.42*	3.8 ± 0.23*

*P <0.01

Table (4): Mean level of lead in soil and vegetables in the studied area

Samples	Polluted area concentration of lead in p.p.m	Control Area
Soil	293.72 ± 32.24 *	74.23 ± 9.12
Forage	164.3 ± 6.32 *	64.01 ± 6.23

*P <0.01

Table (5): Bacterial determined in internal organs liver and kidney in bull

Bacterial Isolated	Degree of presence
Streptococcus sp.	30 %
S. epidermidis	40 %
S. aeurogenosa	25 %

DISCUSSION

From our results, we noticed a decrease in the level of hemoglobin, PVC, and R.B.C's count, owing to the fact that lead intoxication causes a documented defect in haem synthesis. The results obtained agreed with several authors [16, 8] because lead pollution has an inhibitory effect on globin synthesis, inhibits iron to form haem and inhibits delta amino levulinic acid dehydratase in red cells.

Moreover, we can conclude that lead toxicity has dangerous effect on animals in the studied areas where the lead content of soil measured was about 293.73 p.p.m, and in contaminated pasture was 164.3 p.p.m, the lead poisoning in the studied bull may be due to grazing this contaminated pasture which may be

due to their contamination by industrial wastes [17]. Hob & Kirn [18] reported that, lead concentration in plants was 80-160 p.p.m and in soil was 100-300 p.p.m which was considered as toxic level. Referring to the FAO-WHO[19] recommendations, the acceptable daily lead intake is 0.05 p.p.m. and our finding agreed with that of Bryant and Rose [20]; Fayed and Abdallah [21]; Zaki et al., [22]

Bacterial microorganism e.g. *Streptococcus Sp*, *S. Epidermis* and *S. Aeuroginasa* were isolated from internal organs, similar finding were reported by Fingold & Martin [23] and Ducan & Prasse [24] who stated that bacterial microorganism are present in animals and birds suffering from high pollution with lead due to immunological suppression.

Both clinical signs and ocular changes, which were observed in the present work, might be attributed to the toxic effect of lead on the C.N.S as mentioned by Krameller-Froetcher, [25] and Schlerka [26], this toxic effect was characterized by severe cerebral disturbances leading to blindness in some cases which may be due to cerebrocortical oedema, or may be also due to associated optic neuritis and optic atrophy. We can conclude, that all the clinical symptoms reported in the studied bull might be due to acute lead intoxication, as mentioned by Ozmen and Mor [27].

The biochemical results detected in Table 2, showed significant increase in AST, ALT, while there was significant decrease in total protein level (Table 2). These findings agreed with those found by Swarap et al [28], as the elevation in transaminases activities and the decrease in total protein level may be attributed to the liver injury, so the exposure to lead in polluted environments alters serum biochemical parameters indicative of liver functions.

The biochemical results, detected in table 2, showed significant increase in urea and creatinine, which are indicative of abnormal kidney functions, agreed with those of Goswami and Gachhui [29].

The disturbed liver and kidney functions have been seen on the last stage of lead toxicity, this agreed with Zaki et al., [22].

The hormonal results, detected in Table 3, showed significant decrease in L.H., F.S.H. and testosterone, which are affected in the early stages of lead poisoning [30 and 31].

In conclusion lead toxicity cause atrophy of liver, kidney, gonads blindness, in addition to the locomotor disturbances. And the cause of animal morbidity and mortality in this farm was not due to bacterial infections but due to lead toxicity. We can also say that polluted environment, especially with lead, can cause severe harm to animal health, in addition to serious danger on human health, by eating food polluted with lead.

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REFERENCE

1. Silbergeld E.K. 1997: Preventing lead poisoning in children, *Ann Rev. Public Health*; 18:801-810.
2. Afridi H.I., T.G. Kazi, G.H. Kazi, M.K. Jamali., G.O. Shar, (2006): Essential trace & toxic element distribution in the scalp hair of Pakistani myocardial infarction patients, *Biol.Trace elem. Res.* 113(1):11-

- 34.
3. Endo G. , S. Honiglechi, I. Kiyota, (1990): Urinary NaG in lead exposed workers, *J Appl. Toxicol*, 10:235-38.
4. Korak M., E. Kralova, P. Sviatko, J. Bilek, A. Bugarsky, (2002): Study of the content of heavy metals related to environmental load in urban areas in Slovakia. *Bratsil Lek. Listy*; 103 (7-8): 231-7.
5. Rashed M.N. and M.E. Soltan, (2005): Animal hair as indicator for heavy metal pollution in urban & rural areas, *Env. Monit Assess*; 110 (1-3) : 41-53.
6. Zadnik T., 2004: Lead in top soil, hay, silage& blood of cows from farm near a former lead mine & current smelting plant before and after installation of filters. *Vet. Hum. Toxicol.* ; 46 (5): 287-90.
7. Swarp D. , R.C. Patral, R. Naresh, P. Kumar, P. Shekhar, (2005): Blood lead levels in lactating cows reared around polluted localities; transfer of lead into milk. *Sci Total Environ.*; 347 (1-3): 106-110.
8. Jain S.D. (1986): Evaluation of Haemogram in healthy and diseased sheep. *Res. Vet. Sci.* 33,21.
9. Varley H., A.H. Gwenbek and M. Bell, (1980): *Practical clinical chemistry vol. I General I top-'scommoner test 5^{SI} ed.* London, William medical books Ltd
10. Drupt, F., (1974): *Estimation of total protein, Pharm Biol*, 9, 77,
11. Pickering, A.D. and J. Pottinger, (1983): *Analysis of Hormone, Gen Comp Ender.* 49,232.
12. Buchman R.E, W. E Gibbuns and R.Y. Stajner (1975): *Bergeyes Manual of determinative Bacteriology*, Ed. Millions and Wilkins co Baltimore.
13. Wilson G.S. and A.A. Miles, (1975): *Topley and Wilsonies principles of Bacteriology and immunity 6th Ed*, Edwards and Annald, publishers LTD, London.
14. Rodrigues MF and Castellon (1982): Lead and cadmium levels in soil and plants near highways and their correlation with

- traffic density. *Environ. Pollute. (B)* 4:281-290.
15. Gad W. and G. Well (1976): *Statistical methods* 6th Ed the Iowa state Univ, Press Iowa U.S.A.
 16. Soliman, M.N. (1983): *Toxicity resulting from lead compounds in Veterinary practice*. Ph. D. Thesis, Cairo University Egypt.
 17. Lemos R.A., D. Driemeier, E.B. Guimacaes, I.S. Dutra, A.E. Mori and C.S. Barros, (2004): *Lead Poisoning in cattle grazing pasture contaminated by industrial Waste*, *Vet Hum Toxicol*, 46 (6) 326-8.
 18. Hob and Kirn (1988): *Elevated levels of a lead and other metals in roadside soil and grass and their use to monitor heavy metal depositions in Hong Kong*. *Environ. Pollut.*,49: 34-51.
 19. FAO/WHO 1983: 6th joint expert committee on food additives, evaluation of mercury, lead, cadmium and the food additives diethyl pyrocarbonate and acetyl gallate, WHO food additivitie Ser. No.4.
 20. Bryant SL and RW Rose, (1985): *Effect of cadmium on the reproductive organs of the male Ram "Australian Journal of Biological -sciences*, 28(3); 305-311-15
 21. Fayed A.H and E.B. Abdallah, (1997): *"Technique for studying the morphology of mammalian spermatozoa which are eosinophilic in a different live/dead stain*. *J. Report. Fertile*; 29; 443.
 22. Zaki M.S., A.M Haman and F.S Bayaumi and M.N Shalaby, (2001): *Some clinicopathological and microbial studies in lead toxicity on cows*, *Egypt comp path & clinic pathology* vol 14 29-35.
 23. Fingold S.M. 1982: *Diagnostic microbiology*, 6th ed., Mosby co. st. Lowis, Toronto, London.
 24. Ducan J. and K.W. Prasse (1986): *Vet laboratory medicine-clinical pathology*, 2nd ed. Iowastate Univ., press. Ames; Iowa U.S.A.
 25. Krametter-Frooestscher R., F. Tataruch, S. Hauser, M. Leschnick, A. Url and W. Baumgartner, (2007): *Toxic effects in herd of beef cattle, following exposure to ash residue contaminated by lead and mercury*. *Vet. J.*174 (1): 99-105.
 26. Scherleka G., F. Tataruch, R. Krametter-Frooestscher, A. Url, D. Kossler, S. Hogler and P. Schmidt, (2004): *Acute lead poisoning in cows due to feeding of lead contaminated ash residue*, *Berl Munch Tierarztl Wochenschr*, 117 (1-2):52-6.
 27. Ozmen O. and F. Mor (2004): *Acute lead intoxication in an old Battery Factory*, *Vet Hum.Toxicol.* 46 (5):255-6.
 28. Swarp D. , R. Naresh, V. P. Varshney, M. Balgangatharathilagar, P. Kumar, D. Nandi and R.C. Patral, (2007): *Changes in plasma hormone profile and liver functions in cows naturally exposed to lead and cadmium around diffirent industrial areas*, *Res. Vet. Sci.* 82 (1):16-21.
 29. Goswami K., R. Gachhui and A. Bandopadhyay, (2005): *Hepatorenal dysfunction in lead pollution*. *J. Environ. Sci. Eng.:* 47 (1)75-80.
 30. Gorbel F., M. Boujelbene, F. Makni Ayadi, F. Gucimazi, F. Croute J.P Soleilhcvoup and A.I. El-Feki, (2002): *Cytotoxic effects of lead on the endocrine & exocrine sexual function of pubescent male & female rats. Demonstration of apoptotic activity*, *C R Biol*; 325 (9): 927-40.
 31. Srivastava V., R.K. Dearth, K.J. Hiney, L.M. Ramisez, G. Bratton and W.L. Dees, (2004): *The effects of low level Pb. on steroidogenic acute regulatory protein in the pubertal ratovary*, *Toxicol Sci* 77(1): 35-40.