

LEAD TOXICOSIS IN CROSS BRED CALVES

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ABSTRACT

Lead in the form of its acetate was administered to calves till the symptoms of lead toxicosis appeared in all the calves. The mean blood Pb concentration increased from $28.21 \pm 2.69 \mu\text{g}/100$ of zero day to 33.52 ± 2.37 ; 41.31 ± 2.17 ; 40.57 ± 1.02 and $43.47 \pm 00 \mu\text{g}/100$ ml on 3rd; 6th; 9th and 12th day of the experimental period respectively. The serum enzymes transaminases and phosphatases increased with increase in blood Pb concentration. The average concentration of Pb in brain and bone was 7 and 19 $\mu\text{g}/\text{g}$ which were observed to be minimum and maximum as compared with the levels estimated in other body tissues indicating that bone acts as an organ of Pb deposition and detoxification. Haematological studies showed a decrease in the haemoglobin concentration but no marked change was observed in the packed cell volume and erythrocyte sedimentation rate. Postmortem report revealed congestive haemorrhagic liver and catarrhal enteritis in all the calves. The most suitable biological samples used to confirm Pb poisoning were blood, bone, kidney and liver.

INTRODUCTION

M CINTOSH¹ while reviewing lead poisoning in farm animals stated that probably only a small number of deaths from Pb poisoning are correctly reported, diagnosed or even suspected. Pb is said to be the most common cause of poisoning in farm animals²⁻⁵. Orr⁶ and Priester and Hayes⁷ even reported that bovine is the most susceptible species of animals. Outbreak of Pb poisoning in cattle was also reported⁸ from Pb mine waste. Experiments^{9, 10} revealed that Pb in any of its several forms ingested daily at the rate of 20 mg Pb/kg body weight (b.w.) or fed as a single bolus containing 220-440 mg Pb/kg b.w. is the lethal dosage for calves. Earlier investigators^{9, 11-16} suggested that 5-7 mg Pb/kg b.w./day is the minimum intake which will eventually cause poisoning in ruminants. However, most of these studies have been conducted in ruminants during their early postnatal stage (pre-ruminant and transitional stage of rumen development), the period during which they are more susceptible to Pb poisoning^{5, 17}. Hence an experiment was undertaken to study physiological and clinical aspects of Pb toxicosis in 6-9 month old calves when the rumen is both anatomically and physiologically functional.

MATERIALS AND METHODS

Four normal healthy crossbred calves, 6-9 months

of age and weighing 45-75 kg were used in the study. The calves were kept on maintenance ration of green fodder, wheat bhusa and concentrate with water *ad lib*. The Pb as lead acetate was administered orally at the rate of 10 mg/kg b.w./day stuffed in wheat dough till symptoms of lead toxicosis were noticed in calves. Blood samples were collected via jugular veinpuncture and cerebrospinal fluid (CSF) by lumbar puncture from the calves, before the start of the experiment and after every 24 hr of 12 days experimental period. Heparin was used as the anticoagulant in samples collected for Pb analysis and EDIA was used for determination of haemoglobin, packed cell volume (PCV) and erythrocyte sedimentation rate (ESR). Serum was separated and stored at -20°C for enzymatic studies. The various enzymes such as aspartate transaminase (SGOT), alanine transaminase (SGPT), alkaline phosphatase and acid phosphatase were determined by the method of Wooten¹⁸. After death, postmortem was conducted of all the calves and various tissues such as heart, liver, spleen, skeletal muscle, lungs, kidney, testes, brain, skin and bone were collected from two calves for the estimation of Pb content by atomic absorption spectroscopy.

RESULTS AND DISCUSSION

The average blood Pb concentration in the calves was observed to be $28.21 \pm 2.69 \mu\text{g}/100$ ml (table 1)

Table 1 Effect of lead administration on blood lead concentration, haematological study and serum enzymes in cross-bred cows calves

Days/ parameters	0	1	2	3	4	5	6	7	8	9	10	11	12
Blood lead µg/100 ml	28.21 ±2.69	29.86 ±3.24	33.69 ±3.60	33.52 ±2.37	33.86 ±4.73	38.04 ±1.88	41.31 ±2.17	41.84 ±2.82	42.38 ±2.42	40.57 ±1.02	42.02 ±2.04	43.47 ±0.20	43.47 ±0.00
SGOT (AST)	53.27 ±2.36	52.76 ±1.36	63.31 ±2.75	78.15 ±3.29	78.02 ±1.19	78.02 ±1.16	78.02 ±1.16	82.56 ±1.16	92.46 ±1.16	94.12 ±2.27	107.20 ±1.16	105.19 ±1.16	105.19 ±3.19
$r = 0.95$													
SGPT (ALT)	37.25 ±2.19	41.56 ±2.76	48.55 ±2.19	83.13 ±1.78	93.10 ±4.67	100.08 ±4.01	115.71 ±0.00	128.07 ±3.50	128.00 ±2.99	130.55 ±1.67	129.66 ±3.02	136.32 ±5.20	138.32 ±6.30
$r = 0.90$													
Alkaline phosphatase	7.28 ±1.17	8.18 ±3.22	8.61 ±2.19	8.79 ±1.05	9.88 ±1.07	10.45 ±3.20	11.77 ±4.13	12.06 ±1.22	13.76 ±2.12	13.17 ±2.25	14.13 ±2.19	15.28 ±0.05	15.25 ±1.19
$r = 0.97$													
Acid phosphatase	2.15 ±0.35	2.36 ±0.56	2.71 ±0.82	3.08 ±0.80	2.98 ±1.10	4.02 ±0.78	3.27 ±0.77	4.19 ±1.17	4.65 ±0.56	5.42 ±0.90	4.40 ±1.15	5.62 ±0.92	4.72 ±0.50
$r = 0.90$													
Haemo- globin g°	10.05 ±0.16	9.65 ±0.40	9.45 ±0.62	9.20 ±0.70	9.10 ±0.60	8.80 ±0.70	9.05 ±0.76	8.40 ±0.82	8.80 ±0.83	8.83 ±1.30	8.10 ±1.10	8.30 ±0.30	—
PCV °	27.00 ±1.22	25.00 ±2.12	26.00 ±2.44	23.25 ±1.47	23.50 ±2.06	24.00 ±2.73	25.75 ±2.27	25.66 ±2.62	24.00 ±3.00	25.66 ±4.18	23.33 ±2.86	22.00 ±3.00	—
ESR mm hr	7.00 ±1.73	7.00 ±1.73	8.75 ±1.92	10.25 ±3.34	13.50 ±0.86	9.33 ±3.29	8.75 ±3.26	8.33 ±0.94	9.66 ±3.09	—	—	—	—

Serum transaminase are expressed as µ mol/l of blood/hr.
 Serum phosphatase are expressed as KA unit/100 ml of blood/hr.
 The values are mean ± S.E.M.

which increased by 54% during the experimental period of 12 days. Maximum increase was during the first half of the experimental period as compared with that of second half. Normal Pb values for ruminants have been reported as 35 $\mu\text{g}/100\text{ ml}$ blood. George and Duncan¹⁹ observed the range from 5 to 22.6 $\mu\text{g}/100\text{ ml}$. Allcroft²⁰ and Buck *et al*²¹ reported that only a small proportion (1–2%) of the amount of a Pb compound ingested is absorbed from the gastrointestinal tract of calves; however, Zmudski *et al*⁹ observed the rapid rise (ten times) in blood Pb during the first 24 hr after a single dose of 5.0 mg/kg b.w. and the high tissue levels obtained in 5 to 7 days of Pb administration at the rate of 2.7, 5.0 and 20.0 mg Pb/kg b.w./day. Results of the present experiment revealed that as and when the concentration of Pb in blood reached around 43 $\mu\text{g}/100\text{ ml}$, the calves became depressed and unsteady, exhibited manical excitement, ataxia, anorexia, grinding of teeth, diarrhoea, colic and generalized seizures. Opisthotonus, extensive salivation, exaggerated limb movement, protrusion of tongue and gasping respiration were observed before death. Zmudski *et al*⁹ observed almost similar signs of intoxication regardless of dosage, however, the period of time between development of typical symptoms and death varied inversely with dosage of Pb. Seizures and death occurred within 48–72 hr in calves given 2.7 mg Pb/kg b.w., within 24–36 hr in calves given 5.0 mg Pb/kg b.w. and the calf given 20 mg Pb/kg b.w. died within 8 hr after the onset of symptoms.

In this study one of the calves attained toxic concentration of blood Pb on the 7th day of the administration and maintained that level during the next three days and died on the 10 day of the experimental period. Another calf exhibited toxic symptoms on the 12th day of the experiment and died on the next day. Thereafter Pb administration and blood examination were stopped. However, the 3rd and 4th calves developed toxic symptoms on the 13th and 14th day of the experiment and died after 24 hr and 48 hr respectively. Postmortem report revealed congestive haemorrhagic liver and catarrhal enteritis in all the calves. Hoffman *et al*¹⁴ reported that blood Pb value of 40 to 50 $\mu\text{g}/100\text{ ml}$ indicate Pb poisoning. Percentage increase of blood Pb in the present experiment was comparatively much lower even with higher dosage of Pb as compared with those of Hoffman *et al*¹⁴ in 5 day old lambs, of Bratton *et al*¹⁵ in 5–6 weeks old calves and of Zmudski *et al*⁹ in 9–12 weeks old calves. This difference can be attributed to the concept developed in other species²² and the study of Zmudski *et al*¹⁶ in 2–3 week old calves, that milk diet

play an important role in the rate of absorption and deposition of Pb. Whole milk constitute an essential component of the feeding schedule of calves upto 12 weeks of age.

Blood Pb and transaminases values were significantly higher ($P < 0.05$) from 3rd day and phosphatases from 5th day onward as compared with the corresponding values of zero day. Enzymic studies exhibited strong correlation with blood Pb concentration. With increasing concentration of blood Pb the level of serum enzymes (table 1) SGPT, SGOT, alkaline phosphatase and acid phosphatase also increased indicating that Pb has a toxic and damaging effect on body tissues with particular reference to hepatic tissue.

Haemoglobin concentration continued to decrease during the experimental period. This is supported by the observations that Pb intoxication interferes with heme synthesis^{23, 24}. Erythrocyte sedimentation rate and PCV fluctuated during the study but no significant differences were recorded. Similar haematological observations have been observed by other workers^{9, 15}.

Average Pb concentration ($\mu\text{g}/\text{g}$) of the tissues collected were: bone 19; liver 12; lung 11; spleen 11; kidney 11.5; heart 9.5; testes 9.5; skeletal muscle 9; skin 8.5 and brain 7.0. The Pb residues of bone, kidney and liver observed in this study were lower than that of Zmudski *et al*⁹ and Bratton *et al*¹⁵. On the other hand, results of liver and kidney tissues are compatible with other studies^{21, 25}. This could be attributed to the differences observed in the blood Pb values of the corresponding studies. Similar Pb residues in tissues observed in calves⁹ receiving 2.7 mg Pb/kg b.w./day and those of cattle^{12, 26} receiving extremely high levels of Pb indicate that age is one of the important factors in evaluating Pb toxicity. Efforts have been made to find out the relationship of Pb residues in various tissues with particular reference to bone, liver, kidney and brain. In this study, bone Pb was higher than all other tissues. This situation is reversed in acute non-cumulative exposures^{20, 21}. Zmudski *et al* observed that bone Pb was consistently higher than liver Pb. Kidney Pb was the same as bone Pb in calves dosed with 2.7 mg Pb/kg, higher than bone Pb in calves dosed with 5.0 mg Pb/kg, and lower than bone Pb in the calf dosed with 20 mg Pb/kg. In this study as well as in others^{9, 18} the minimum accumulation of Pb in brain tissue has been observed. The results of the present findings supported the report of Adrian *et al*²⁷ showing that the bone acts as a lead 'sink' and is an important lead detoxification mechanism of domestic animals. Relatively low Pb content (6.0–8.5 $\mu\text{g}/100\text{ ml}$) of cerebrospinal fluid (CSF) as compared with that of

blood during the experimental period indicate the possibility of blood brain barrier for Pb. Thus the mechanism of influx of blood Pb into CSF needs to be investigated before the calf model could be used to study Pb encephalopathy as proposed by Zmudski *et al*⁹.

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