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CLINICAL AND HAEMATOLOGICAL STUDIES ON EXPERIMENTALLY INDUCED SELENOSIS IN CROSSBRED COW CALVES

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ABSTRACT

The effect of sodium selenite induced subacute and chronic toxicity on clinical and haematological parameters was studied in cross-bred cow calves. Subacute and chronic selenosis was induced by oral administration of sodium selenite at dose rate of 2.5 mg/kg for 21 days and 0.25 mg/kg for 16 weeks, respectively. Toxic manifestations in subacute selenium toxicity included anorexia, salivation, redness of eyes, swelling of joints, wound formation in the pastern area, reluctance to move, diarrhea, stiffness of neck, labored breathing and subnormal body temperature and recumbeacy in terminal stages. In chronic selenosis, main symptoms observed were rough hair coat, alopecia, swelling of coronet, enlargement of the hooves, interdigital lesions and gangrene at the tip of tail. Both forms of selenosis significantly affected blood haemoglobin, packed cell volume, total erythrocyte count and total leukocyte count. However, mean corpuscular volume and mean corpuscular haemoglobin were significantly altered in chronic toxicity only. There was no significant effect of selenosis on erythrocyte sedimentation rate and mean corpuscular haemoglobin concentration.

Key words: Selenium, toxicity, subacute and chronic, haematology, cow calves.

INTRODUCTION

Selenium is a naturally occurring trace element and is nutritionally required in small amounts as a part of selenoproteins and selenoenzymes. However, selenium in twice the required concentration becomes toxic for animals. Selenium deficiency results in white muscle disease in cattle and sheep, hepatosis dietitica in pigs, exudative diathesis in chickens (Blood and Radostits, 1989) and Keshan's disease in human beings (Levander, 1987). On the other hand, elevated dietary selenium in forage and seleniferous plants is associated with alkali disease, blind staggers and Degnala disease (Toole and Raisbeck, 1995). Loss of hair and horn and hoof abnormalities, leading to cracks and detachment, tail necrosis and disturbed reproductive cycle are main toxicity symptoms observed in animals (Randhawa *et al*., 1992). Among different species, ruminants are more susceptible to selenosis than others (Allaway, 1973). So, the present project was undertaken to study the effect of subacute and chronic toxicity of sodium selenite on various clinical and haematological parameters in crossbred cow calves.

MATERIALS AND METHODS

Sixteen male crossbred cow calves (6-12 months of age), weighing between 70-120 kg, were randomly divided into four groups of four animals each. These animals were dewormed and acclimatized to uniform environmental conditions and were provided seasonal green fodder and water *ad libitum*. Groups I and III served as control for subacute and chronic toxicity studies, respectively. Animals of Group II were orally administered sodium selenite at the dose rate of 2.5 mg/kg body weight for 21 consecutive days. Group IV animals were drenched with sodium selenite at the dose rate of 0.25 mg/kg body weight daily for 16 weeks. Each animal was examined daily for general condition, hair coat, leg incoordination and virubility. Appetite of calves and consistency of faeces were also noted. After the treatment period was over, animals were observed for recovery. Post-mortem examination was performed on two animals that died during treatment period in subacute toxicity group. Blood samples were withdrawn via the jugular venipuncture and mixed with dipotassium salt of EDTA. Sampling was done on days 0, 6, 12, 15, 18 and 21 during treatment period and days 3, 7 and 10 during post-treatment period in subacute toxicity group, whereas, in case of chronic toxicity, sampling was done at weekly intervals for 16 weeks and for 3 subsequent weeks during the post-treatment period. Various haematological parameters viz haemoglobin concentration, packed cell volume (PCV), erythrocyte sedimentation rate (ESR), total erythrocyte count (TEC), total leukocyte count (TLC), mean-corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC) and mean corpuscular volume (MCV) were estimated by the

methods of Benjamin (1985). Significance of differences between contol and treatment groups of respective categories was determined with the help of student's t-test.

RESULTS AND DISCUSSION

The subacute and chronic toxicity of selenium was induced by daily oral administration of sodium selenite @ 2.5 mg/kg for 21 days and 0.25 mg/kg for 16 weeks, respectively. In subacute toxicity group, the symptoms started appearing from 6th day onwards (Table 1) and these included anorexia, redness of eyes, swelling of joints as well as base of ear and wound formation on pastern region. Two of the animals died within 18-21 days. In terminal stages, there was stiffness of neck (Plate 1), respiratory distress and subnormal body temperature. However, in case of experimentally induced chronic selenosis, typical toxic symptoms i.e. alopecia, cracks on hooves, elongation of hooves, interdigital lesions, ring formation on coronet region and gangrene at tip of tail (Plate 2) were observed after 12 weeks of exposure, but there was no mortality. After the discontinuation of sodium selenite treatment lesions subsided without any treatment in all the surviving animals probably because of very rapid elimination of selenium from the body.

Table 1: Chronology of appearance of symptoms in experimentally induced selenosis in crossbred cow calves

Symptoms	Subacute selenosis	Chronic selenosis
	Time (days)	
Slight redness of eyes, salivation	6-9	-
Wound formation on pastern region	9-12	-
Slight roughness of hair coat	12-15	30-60
Diarrhea	10-12	90-100
Swelling at joints and ear base	12-18	-
Reluctance to move/lameness	16-18	100-110
Stiffness of neck, labored breathing	18-21	-
Overgrowth of hooves	-	100-110
Gangrene at tail tip	-	110-120
Interdigital lesions	-	110-120

The blood haemoglobin content of animals experimentally induced with subacute selenium toxicosis showed a significant decrease from 12.6 ± 0.92 g/dl on 0 day to 8.99 ± 0.27 g/dl on 12th day and fell to a level of 7.76 ± 0.25 g/dl by the 18th day (Table 2). However, the blood haemoglobin concentration started increasing gradually after withdrawal of selenium treatment and returned to normal by 10th day of post-treatment period.

Plate 1: Stiffness of neck in crossbred cow calf with experimentally induced subacute selenium toxicity.

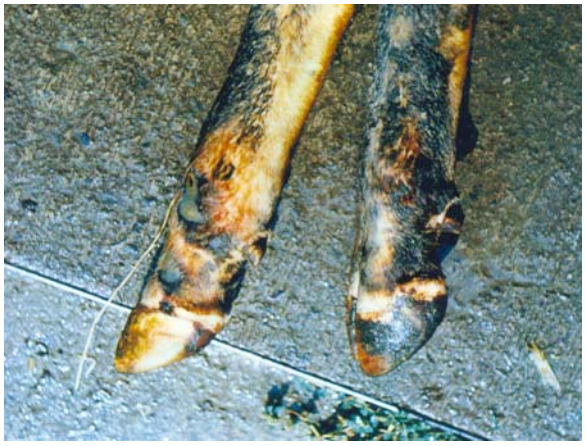

Plate 2: Ring formation in coronet region and wounds in the pastern area in a crossbred cow calf with experimentally induced subacute selenium toxicity.

In chronic selenosis, haemoglobin concentration decreased to almost half of pretreatment level by the 8th week (Table 3). Thereafter, the haemoglobin concentration was significantly lower even 3 weeks after the withdrawal of selenite treatment. The decline in the haemoglobin levels could be due to the inhibition of delta-amino levulinate dehydratase, a key enzyme required for the haemoglobin synthesis. Barbose *et al.* (1998) has reported that sodium selenite, an inorganic salt of selenium, inhibits the delta-amino levulinate dehydratase. These results are in tune with the findings of a decline in blood haemoglobin content during chronic selenosis in cattle (Mitra *et al.*, 1996) and buffaloes (Kumar *et al.*, 1989; Dhillon *et al.*, 1992a; Ghosh *et al.*, 1993). Randhawa *et al.* (1992) reported

Table 2: Effect of repeated oral administration of sodium selenite (2.5 mg/kg body weight) on haemoglobin concentration, packed cell volume and mean corpuscular haemoglobin in crossbred cow calves

	Treatment					Post- treatment			
Time (days)	**0**	**6**	**12**	**15**	**18**	**21**	**3**	**7**	**10**
Haemoglobin concentration (g/dl)									
Control	11.6 ± 0.66	12.4 ± 1.06	11.3 ± 0.57	11.6 ± 0.53	11.6 ± 0.48	11.9 ± 0.58	12.9 ± 0.26	11.4 ± 0.48	12.6 ± 0.72
Treated	12.6 ± 0.92	10.2 ± 0.35	8.99 ± 0.27**	7.94 ± 0.46**	7.76 ± 0.25**a	6.97b	8.43b	10.5b	11.5b
Packed cell volume (%age)									
Control	38.6 ± 0.95	38.2 ± 1.98	37.3 ± 1.76	37.5 ± 1.45	36.9 ± 1.46	39.2 ± 1.24	37.7 ± 1.42	37.4 ± 1.56	38.2 ± 1.39
Treated	39.7 ± 0.77	34.9 ± 0.88	29.2 ± 2.28*	27.3 ± 3.72*	26.1 ± 2.40**a	23.1b	28.2b	29.6b	32.0b
Mean corpuscular haemoglobin (pg/dl)									
Control	27.5 ± 2.05	30.1 ± 3.64	27.8 ± 2.40	28.0 ± 2.45	28.5 ± 2.59	29.2 ± 2.35	32.0 ± 1.63	28.0 ± 1.97	31.1 ± 3.11
Treated	31.5 ± 4.22	25.7 ± 1.68	23.2 ± 1.16	21.2 ± 1.57	19.2 ± 2.43a	18.2b	22.3b	27.5b	29.5b

*P < 0.05 ** P < 0.0

a: Mean ± SE of three animals b: Mean ± S.E of two animals

that animals affected with chronic selenosis had 35% less haemoglobin and suffered from macrocytic and hypochromic anaemia.

The trend of fall of PCV was in close resemblance with that of haemoglobin. In subacute toxicity studies, significant fall in PCV was recorded on 12th day (29.2 ± 2.28%), which further gradually declined to 26.1 ± 2.4% by the 18th day of selenium treatment (Table 2). The PCV values remained lower even in the post-treatment period. However, in chronic selenosis, PCV showed a significant decrease from 6th week to 16th week of treatment (Table 3). The PCV levels remained significantly lowered even three weeks after withdrawal of sodium selenite treatment. The findings of lowered PCV in chronic selenosis in crossbred cow calves correlates very well with the finding of lowered PCV in cattle (Mitra *et al.*, 1996), buffaloes (Kumar *et al.*, 1989; Dhillon *et al.*, 1992b; Ghosh *et al.*, 1993; Deore, 2000) and pigs (Baker *et al.*, 1989). However, on the contrary, Ellis *et al.* (1997) reported no effect of chronic selenosis on PCV in cattle. The decrease in Hb and PCV in our study may be due to selenium-induced haemolytic anemia (Halverson *et al.*, 1970), reduced synthesis of haemoglobin (Nagai, 1959) or depressed PCV (Goehring *et al.*, 1984).

The subacute toxicity of sodium selenite resulted in a significant decrease in TEC from 4.12 ± 0.11 x 10^6/mm^3 on 0 day to 3.88 ± 0.05 x 10^6/mm^3 on 12th day (Table 4). The counts were significantly lower at all the time intervals studied. The average value of TEC of all the animals experimentally induced with chronic selenosis on 0 day of treatment was 4.09 ± 0.06 x 10^6/mm^3 (Table 5). These showed a significant decrease by the second week of selenite treatment (4.03 ± 0.03 x 10^6/mm^3). Thereafter, TEC remained significantly lower during the treatment period. During the post-treatment period, TEC showed a gradual recovery.

TLC in case of subacute selenosis registered a significant decrease from 9320.0 ± 39.2 per mm^3 on 0 day to 8650.0 ± 165 per mm^3 by 12th day of treatment, which further decreased to 8333.3 ± 120 per mm^3 by the 18th day of treatment (Table 4). A gradual increase in TLC was observed during post-treatment period. However, in chronic toxicity group, TLC on 0 day was 9350-9600/mm^3, which showed a significant fall from the 2nd week (9200 ± 126.5 per mm^3) to the 16th week (8200 ± 60.4) of treatment (Table 5). During the post-treatment period, the TLC values started increasing. After three weeks of withdrawal of sodium selenite treatment TLC values were 8600 ± 126.5 per mm^3. This finding is, however, in contrast to finding of Dhillon *et al.*, (1992a), who observed no change in TLC values in cattle and buffaloes thriving on fodder raised on seleniferous soils. The decrease in the TLC could be due to the depressed neutrophil production (Hogan, 1986).

MCV and MCH showed no significant alterations in subacute toxicosis, whereas in chronic selenosis these recorded a significant decline from 6th and 8th weeks of treatment, respectively. During the post-treatment period, these erythrocytic indices returned to normal.

This finding is, however, in contradiction with the findings of Dhillon *et al.* (1992a), who reported an increase in MCV and decline in MCHC values, reflecting macrocytic and hypochromic anaemia in cows and buffaloes. There was no significant change in the values of ESR and MCHC in both subacute and chronic selenium toxicosis. Kumar *et al.* (1989) observed no change in MCHC value, which is suggestive of normochromic anaemia. Progressive microcytic, hypochromic anaemia with continuous decrease in haemoglobin values have been reported in cases of selenium toxicity in dogs (Moxan and Rhian, 1943), hamsters (Birt *et al.*, 1986), goats (Pathak, 1984) and guinea pig (Das, 1987).

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