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Original Article

Clinicopathological and biochemical study on Selenium toxicity in sheep

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ABSTRACT

Selenium is an essential trace element in living organisms as an integral part of seleno-enzymes, and it plays an important role in the maintenance of health, growth, and many biochemical-physiological functions of animals and humans. However, the excessive amount of selenium is toxic for so-called non-accumulator plants, animals and humans. The problem of selenium toxicity arises from the point that the safety margin is limited and the difference between the daily requires of selenium and the toxic doses is very narrow which leads to selenium toxicity as recorded in Qena governorate. Blood serum samples were collected from 20 morbid sheep (20/50), GOT, GPT were determined by spectrophotometer. Selenium and serum tocopherol were measured fluorometrically. Internal organs (lung, liver, kidney and intestine) biopsies were cultured in different culture media for detection of bacterial infection in morbid cases. Twenty cases from fifty sheep showed selenium toxicity by the ratio of 16%, their ages ranged between 3 to 6 months. All the toxicated animals exhibited higher selenium levels in the serum, in addition, the P. M. changes were identical. All the other similar microbiological cases were excluded. It was concluded that selenium toxicity in sheep can be diagnosed by clinical symptoms, post mortem examination (P.M.), and detection of liver enzymes activity.

Keywords: Selenium, toxicity, tocopherol, GOT, GPT, Sheep.

Introduction

Selenium (Se) is an essential nutritional element for the maintenance of biological activity, but excessive Se can be toxic to animals and humans. Selenium has an atomic number of 34, and an atomic weight of 78.96. Selenium toxicity was recorded for the first time in livestock in 1933 due to the consumption of plants of the genus Astragalus, Xylorrhiza, Oonopsis, and Stanleya in the United States [1].

Selenium poisoning can occur due to one of the following conditions of exposure. 1. Animals consumed plants which contained higher concentrations of selenium than normal from seleniferous soils. 2. Se poisoning from the environmental pollution due to the agricultural drain water, reclaimed soils from phosphate or ore mining, sewage sludge or fly ash. 3. Selenosis was caused accidentally due to the overdoses of Se supplements injection or via misformulation of feed mixes. The symptoms of toxicity may appear in the form of subacute, acute or chronic depending on the doses and duration of the exposure [2].

Generally, sheep and cattle are fed on suitable selenium intake, but not higher, the majority of selenium is excreted in the faeces. Previous studies in sheep, showed that most of selenium intake has been found to be in an inorganic form that seemed to be largely inaccessible to plants [3]. But with increasing the Se intake, urinary and volatile selenium excretion would accordingly increase. Experimentally, intravenous injection of high doses of selenium in sheep, about 60% was excreted via the urine, while more than 20% as volatile selenium, with nearly 5% was excreted in the faeces [3]. Blodgett and Bevill [4] reported that the biological half life of selenium in sheep treated with toxic doses of selenium was averagely 14.7 days.

The level of Se in the liver of toxic animals was found in some studies to be greater than 15,000 nmol/ kgwet matter basis (wmb) [5]. Hodges et al. [6] found that Se concentration in the liver

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was higher than 30,000 nmol/kg (wmb). Some investigators reported higher levels in toxic liver of sheep which reached higher than 190,000 nmol/kg (wmb) and in cattle more than 88,700 nmol/kg wmb [7]. Whereas, some researchers recorded a higher value (250,000 nmol/kg (dmb) or nearly 70,000 - 87,500 nmol/ kg (wmb) in cases of acute selenium toxicity [8]. Liver selenium levels can surpass 25,000 nmol/kg (wmb) within 6-12 hours of injection of 5 mg of sodium se1enate subcutaneously [9]. The LD_{50} dose rate for selenium in sheep was previously recorded by many investigators to be 0.455 mg/kg body weight, when sodium selenite was administered by a single intramuscular injection, and 1.9 mg/kg body weight when given orally [10].

Selenium toxicity has been recognized in localized areas in many countries. The range between the required amounts (about 0.2p.p.m) and the toxic levels (1-4p.p.m) was very narrow. Harmful selenium intake by animals arises either from the ingestion of silage plants with relatively high selenium concentration, or from the environmental pollutions, or overdoses in accidental conditions. "Alkali disease" and blind staggers are chronic and acute forms of selenium poisoning [11].

The main symptoms from selenium poisoning, in blind staggers were resulted in which the affected animal may circle or flounder or push against the obstructions. The appetite gets depraved and finally the animal becomes paralyzed, and will die from failure [12]. Mortality and/or morbidity rates among feedlot sheep usually occurred as a result of bacterial, viral and parasitic infections along with toxic agents which have been incriminated to produce severe damages [12]. Toxic level of selenium may be accumulated in certain plants including certain forage plants and grasses growing on high selenium soils. Increased level may occur accidentally in feeds, the diet should contain under p.p.m. Acute-blindness, depression, circling, head-pressing, colic, paralysis death, Chronic-loss of condition lameness, emaciation, hair loss from base of tail and hoof damage have been observed [13].

Selenium poisoning develops after the ingestion of plants that have accumulated higher concentrations of selenium, but it can occur with injection of over dosage of selenium supplement. In acute poisoning, symptoms related to the intoxication are emaciation, poor quality of hair and partial alopecia. The mechanism of the actions has not been completely understood, and it has been probably multifactorial [14].

Post mortem findings demonstrated hydrothorax, oedema in the interlobular septa, forth in the bronchi, trachea and nose, congested lungs, pallid liver with areas of necrosis, clotted blood and congestion in the intestine, oedema and gelatinous exudates of muscles and subcutaneous fascia.

Because of all the previous symptoms, P.M. findings had occurred and recorded for the first time in this area. The farm belonged to the previously mentioned company. Attentions were paid to exclude all the possible causes of such diseased condition. From the available literature [12, 15], some microorganisms such as Pasteurella, smuns were incriminated to cause signs (blind staggering) in feedlot cattle [16].

The aim of the present study was to clarify the cause of the illness in this farm since the animals under investigation were daughters and sons of imported valuable breeds. The disease was recorded for the first time in this area. The morbid animals did not respond to any course of treatment and contiguously died. Thus, the big target was to know the cause in order to minimize the possible risk of distribution of such disease among the remained living animals, and avoid the severe damages caused by the illness.

Material and Methods

A total of 20 blood samples were collected from 20 morbid sheep. Sera were separated and kept in a refrigerator at-20^{0}C till used for the further analysis.

The enzymes Glutamic Oxaloacetic Transaminase (GOT) and Glutamic Pyrvic Transaminase (GPT) were assayed in the sheep serum. The chemicals for this enzyme assay were obtained as test kits from G.F. Boehringer and Soehne Co. Mannheim, W. Germany. The general instruction for enzyme determinations, as given by the company, regarding preparation of solutions deproteiniztion, spectrophotometric measurements and calculations were followed.

Selenium (SE) content of the serum and serum tocopherol level were estimated following the method of Olson's fluorometric [17, 18]. The estimated values were compared with the normal value to assess their status both in the diseased and apparently healthy sheep.

For bacteriological isolation, in a total of 20 specimens from dead sheep, two samples from each (liver, lung and intestine) were collected under aseptic condition, and transferred in sterile container in ice box, and were directly inoculated onto Columbia CNA (Biomereux), defiberinated sheep blood agar (Difco), and MacConkey's agar plates (Difco), and incubated at 4^{0}C and 370C under 5-10% Co_2 for 24-48 hrs.

Trypticase soya broth (Difco) was inoculated and incubated in refrigerator at 4^{0}C for at least 4 weeks with periodical subculture onto Columbia CAN, blood agar and brucella agar media, and incubated in suitable environment. The plates were incubated at 4^{0}C and 37^{0}C under 5-10% Co_2 tension for 24-48 hrs. Suspected colonies were picked up for further identification according to Chandrasekharan and Yeap, [19]. Listeria as well as Pasteurella species were identified by Gram's stain, cellular morphology, catalase test, mortality test, haemolysis onto blood agar, indole test and other biochemical test according to Chandrasekharan and Yeap [19].

Results

Bacteriological finding:

Bacteriological examinations of the samples taken from lung, liver and intestine revealed the absence of Listeria, Haemophilus and Pasteurella microorganisms from the cultured specimens.

In addition, some microorganisms such as Saprophytic bacteria as proteusspcies, *E. coli* and *B. anthracoides* could be identified in the specimens from different internal organs.

Biochemical analysis:

Estimation of selenium level, teocopherol level and liver enzymes (SGOT and SGPT) in 52 serum samples subjected to biological analysis were recorded in the following table:

Table 1: Biological analysis of the samples

No. of serum samples obtained	Selenium level u/ml	Tocopherol level ug/100 ml	SGOT	SGPT
From apparently healthy sheep (Control)				
2	20±0.4	100.1±0.1	28.22±0.3	5.1±0.5
2	34.0±0.1	109.2±0.1	27.14±0.5	4.8±0.1
From dead sheep				
3	127.0±0.1	186.4	120.0±0.2	37.8±0.2
1	110.1±0.3	195.0	118.2±0.1	36.0±0.3
1	99.8	108.6	121.0	32.0
From sick sheep				
11	86.2±0.3	170±3.0	96.0±0.1	27.0±0.3
7	81.8±0.2	100.0	99.0±0.3	23.3±.04
8	79.2±0.1	260.0	95.0±0.2	21.0±0.01
5	76.4±0.1	173.0	87.3±0.2	19.1±0.01
4	65.2±0.2	182.0	89.2±0.1	15.3±0.1
3	61.6±0.1	99.6	69.0±0.1	13.0±0.1
1	57.8	273.1	35.0	6.4

Discussion and Conclusion

Selenium has been permitted to be added in the rations of dairy sheep at the concentration of 1 mg/kg ration [20].

Smith and Conard [21], reported that white muscle disease was documented as a result of selenium and vitamin E. deficiency in ruminants, affecting particularly the younger ages (1 to 4 weeks). The symptoms of Se and Vit. E deficiency (White muscle disease) are characterized by necrosis in the cardiac and skeletal muscles, and the clinical symptoms have been varied and ranged from stiffness and lameness to sudden death.

Selenium poisoning has been considered as one of the most dangerous diseases affecting ruminants. The occurrence of selenium poisoning in specific regions may be attributed to the higher level of selenium in the soil or it may arise from the abuse of selenium as food additive, or it may be arisen from the environmental pollutants due to the contamination of pasture by continuous deposition of industrial products or residues.

From the present study, looking carefully to the obtained results, one can conclude that the cause of sheep mortality and morbidity in Misr Aluminum farms was not due to the bacterial infections such as (listeria, pasteurella or haemophilus) as all of the bacterial cultures failed to identify such agents, but it required more investigations to exclude the bacterial causes for morbidity and mortality.

On the other hand, biochemical analysis revealed that the main cause of mortality and morbidity among sheep in this region was due to selenium poisoning. The presence of some sick animals showing high selenium level and illness symptoms supported the findings of some authors [16, 22], who found that there was a narrow safety margin between the toxic level and vital or normal levels.

From the obtained results, one can conclude that the level of tocopherol detected in control group in dead sheep or in sick sheep revealed no variation or played no role in inducing such toxicity because there was no sharp elevation or sudden drop varied greatly from normal levels which were recorded by Blood and Henderson [15] who mentioned that normal tocopherol level varied from 196 ± 64.9 ug/ml and also agreed with Thomas and Ronald [23] and Jiro [24]

Estimation of SGOT and SGPT levels in all the examined sheep revealed that both enzymes level appeared nearly similar to that of control group as shown in obtained results. This was in agreement with the findings of Schalm [25], who found that sharp increase in both SGOT and SGPT levels was an indicator of the toxic effect of selenium and pathogenic lesions occurring in alive and dead sheep and morbid mammals reflexing all the clinical symptoms appeared in sick sheep which were recorded in this farm. The findings were in agreement with the symptoms which were recorded previously due to selenium toxicity [12, 16].

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