25. Selenium Toxicity in the Dromedary Camels
Clinical Symptoms and Lesions

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Introduction

Selenium (Se) is a group VI element with chemical properties very similar to those of sulfur, it has been demonstrated as an essential element for ruminants. Selenium is required to prevent deficiency diseases such as white muscle disease, maintain growth rates of young animals and promote reproductive performance. Camel sensitivity to trace element imbalances has been reported (Faye and Bengoumi, 1994). Selenium is generally considered a highly toxic element and selenium toxicity may occur in camels through incorrect diet formulation or prolonged oral exposure to elevated dietary selenium (Se) in forage. The objectives of the current study were to determine effects of graded levels of soldium selenite intake on camel performance and to provide preliminary data on camel selenosis (clinical symptoms and lesions).

Materals and Methods

Twelve healthy young camels were obtained from local UAE breed, aged 2 years and were acclimated to experimental design for 15 days. During the acclimation period, camels were treated with a broad-spectrum antiparasitic compound. Animals were housed in groups of 4 and were fed with a similar basal diet composed of Rhodes grass (*Chloris Gayana*) with an average quantity of 3 kg DM and 2 kg of pelleted concentrate 10 % protein (Soya Bean Meal – Maize – Barley – Wheat bran – Molasses – Salt – Premix). Camels were provided water *ad libitum*. Oral individual doses of selenium : 8.16, and 16 mg per day were given as sodium selenite, corresponding respectively : 8 mg (i.e. 17.44 mg sodium selenite), 12mg (i.e. 26.16 mg sodium selenite) and 16 mg (i.e. 34.88mg sodium selenite). Selenium was given enrobed in dates every day at the same time for 90 days. Selenium supplementation was stopped at the time of apparition of chronic selenosisand camels returned to normal good health gradually. At day 45 one camel of each group was slaughtered and a second one at the end of the experiment (at day 90).

Urine and faecal samples were taken every month from each camel. A sample of 600 g was taken from each camel, dried for 48h at 65^{o}C, grinded and stored in dark and cool place until selenium analysis. Total 24 hours urine of each camel was also taken, using a special plastic bag placed on the vulva, weighed and a sample of 20 ml was taken and stored at –20^{O}C up to selenium analysis. Selenium content of the camel basal diet and water was also assessed at the beginning, the middle and at the end of the trial. Nutriments were dried, ground and stored in a dark cool place until analysis. Hair was taken before slaughtering from the neck and other part of the camel were taken at day 45 and 90 using a stainless steel knife. Before organs sampling, the weight of each whole carcass and each organ were recorded. Samples from lung, heart, liver, spleen, kidney, pancreas, suprarenal gland, shoulder and femoral muscle, anterior limb bones, posterior limb bones, brain, intercostals muscles, diaphragm muscle and urinary bladder were collected. Samples from the tissues were fixed in 10% neutral buffered formalin for microscopic evaluation; others samples were stored at -80°C until selenium analysis.

Selenium was determined in organs, hair, face, urine, diet and water by Inductively Coupled argon Plasma – Atomic Emission Spectrometer (ICP-AES), Varian vista MPX-CCD simultaneous, using 11 points of standard curve of Accu Trace™ Reference Standard solutions from Accustandard® – USA. Quality Control Standard.

Results

Selenium analysis in water showed no selenium. Selenium content in diet was 0.49 mg/kg in concentrate, 0.15 in Rhodes grass. The daily feed intake was 2 kg of concentrate and 3 kg of grass on

average. Thus, the selenium intake provided by the diet was 1.43 mg per day for camels during all the experiment, the mineral mixture providing 8, 12 and 16 mg of selenium per day. According to the treatment, the total quantity of selenium provided in the diet was 9.4 mg/day for camels in group 1, 13.4 mg in group 2 and 17.4 mg in group 3. So, the dietary Se concentration varied between 1.7 (group 1) and 3.5 ppm (group 3) DM.

Selenium concentration in urine and feces varied between 33.2 and 2230.5 ng/ml with a mean value 646.6 ± 610.9 ng/ml and between 193.5 and 13487.4 ng/g DM with an average mean 2346.02± 2653.9 ng/g DM respectively. The urinary Se concentration was higher at month 3 in group 2 receiving 12 mg. Se concentration increased significantly starting from month 2 for 3 groups up to the end of the experiment for groups 1 (8 mg Se) and 2 (12 mg), but decreased at month 3 in group 3 (16 mg Se) when Se supplementation was stopped.

Clinical symptoms of selenosis appeared at week 2 (see annex1). Camels had visible reduced feed intake and weight loss, reluctance to move and tachypnea following minimal exercise. Alopecia was seen in 3 groups, but more extended in group 3 with rough skin. In groups 2 and 3, theurinary excretion increased and dark watery diarrhea was also observed. Tears with pale mucous were showed as well as an evidence of impairment vision. Fissured pads appeared in all groups but more pronounced in group 2 and 3. Consequently, camels in group 1 and 2 developed a vesicular stomatitis. Camels stood with their head down and neck extended, taking short, rapid, shallow breaths. The recovery period ranged from 1 to 2 weeks. Severity of clinical signs of disease and time to recovery varied and were dose dependent.

At necropsy all animals from the 3 groups showed gross lesions, characterized with severe pulmonary lesions with accumulations of serosanguinous fluid and foam in the trachea, bronchi, and bronchioles. The heart of these animals was soft and pale, all abdominal muscles, diaphragm and intercostals muscles were pale. The liver was red and mildly swollen. Heart, liver and kidney were congested and necroses. while pancreas was atrophied. Brain edema was observed in all treatment.

The major histopathologic changes camels that manifested clinical signs of selenosis included kidney lesions showed congestion in blood capillaries of cortex and medulla, degenerative changes in lining epithelial cells of convoluted tubules . Lesions were extended to other tissues with severe vacuolar degeneration in epithelial lining in urinary bladder and sub capsular focal hemorrhagic areas in spleen. Edematous fluid was seen in between the muscular fibers and slight congestion of blood capillaries in heart, hepatic cells, congestion in central hepatic vein and hepatic sinusoids. In addition, focal areas of muscular hyalinization (non –inflammatory) and edema were observed in intercostals and diaphragm muscles**.** Activation in lymphoid follicle was seen in cervical anterior lymph node. Focal hemorrhagic areas and blackish green fine granules accumulation were observed in focal areas of spleen. Brain showed perivascular oedema in brain.

Discussion

Se deficiencies have been reported in United Arab Emirates, camels are often supplemented with commercial Se and vit E compound; however, no data on camel selenosis have been reported. In this current study the amount of Se intake from basal diet is 1.43 mg Se per day i.e. 0.28 mg/kg DM that was considered approximatively the requirements for dairy cattle (NRC 2001). However, according to the mean weight of the camel in our study (183 kg), the selenium supply with the basal diet was 0.78 mg/100 kg LW. That was lower than recommendations for beef cattle (1 mg/100 kg LW). Selenium is needed in small amounts. The minimum level of selenium in diet that causes chronic selenosis in most animal species is 4-5 mg/kg DM (US NAS/ NRC, 1976) and the minimum level needed to prevent deficiency is 0.02 – 0.05 mg/kg DM (US NAS/ NRC, 1971). Excess Se intake can lead to Se poisoning, but species susceptibilityselenium toxicosis is variable. (Tiwary *et al.* (2006) did not observe lamb mortality with an oral sodium selenite up to 4 mg/kg LW. For other authors, the oral median lethal dose (LD50) of sodium selenite has been reported to be 1.9 ±1.2 mg of Se/kg LW (Blodgett & Bevill, 1987). A daily intake of 0.25 mg/kg LW was considered as toxic for sheep and cattle (Muth & Binns, 1964). These levels listed previously are higher than our dietary levels in the present study (0.051 to 0.095 mg/kg LW), which seems to show a high sensitivity of camel species to Se toxicosis. A limit marrow is to be considered between selenium requirement and toxicity. In this study, lesions appeared with a selenium intake of approximatively 2.5 mg/kg DM, while typical lesions of chronic selenium toxicosis were observed on young cattle receiving more than 5 mg/kg DM for 120 days (O'Toole & Raisbeck, 1995). The clinical symptoms showed in this study were in accordance with previous signs observed in chronic poisoning in other species (Casteel *et al.*, 1985;

Tiwary *et al.,* 2006). The necrosis of camel pad was comparable to those occured in chronic selenosis in cattle (O'Toole & Raisbeck, 1995) and horse (Raisbeck *et al*., 1993).

Conclusion

The results of this study indicate that the camel is sensitive to excess Se intake and selenosis, occurs with high-level selenium intake. Young camels are very sensitive. Clinical toxicity symptoms were observed at a dose of 8 mg Se daily within 3 weeks under sodium selenite form. According to dietary Se supply and to mean weight of the animal from the group 1, selenosis appeared with 0.05 mg/kg LW Se supply only. Severe intoxication occurred with 16 mg Se supplementation, i.e, 0.10 mg/kg LW. Those values were 5 times less than for sheep and cattle. According to such results, it could be important to limit Se supplementation in camel at 0.01-0.02 mg/kg LW, i.e. approximatively 4-8 mg per day for adult animals or 0.5-1 ppm in the diet.

Although meeting dietary selenium requirements is an important nutritional requirement for camels, mineral supplementation may also enhance the nutritional quality of the camel product (milk and meat).

References

Blodgett, D.J. & Bevill, R.F. (1987) Acute selenium toxicosis in sheep. Veterinary and Human Toxicology, 29, 233–236

Casteel, S.W., Osweiler, G.D., Cook, W.O., Daniels, G., & Kadlec, R. (1985) Selenium toxicosis in swine. Journal of American Veterinary Medical Association, 186, 1084–1085

Faye, B. & Bengoumi, M. (1994) Trace elements status in camels. A review. Biological Trace Element Research, 41, 1-11

Muth, O.H & Binns, W. (1964) Selenium toxicity in domestic animals. Annals of New York Academy Sciences, 111, 583-590

NRC (National research Council) (2001) Selenium requirements. In Nutrient requirements of beef cattle, 7th Ed., pp. 67-68, National Academic Press, New-York, USA

O'Toole, T. & Raisbeck, M.F. (1995). Pathology of experimentally induced chronic selenosis (alkali disease) in yearling cattle, Journal of Veterinary diagnostic investigation, 7, 364-373

Raisbeck, M. F., Dahl, E. R., Sanchez, D. A., Belden, E. L. & O'Toole D. (1993) Naturally occurring selenosis in Wyoming. Journal of Veterinary diagnostic investigation, 5, 84-87

Tiwary, A.K., Stegelmeier, B.M., Panter K.E., James, L.F. & Hall J.O. (2006). Comparative toxicosis of selenium selenite and sélénométhionine in lambs, Journal of Veterinary diagnostic investigation, 18, 61-70

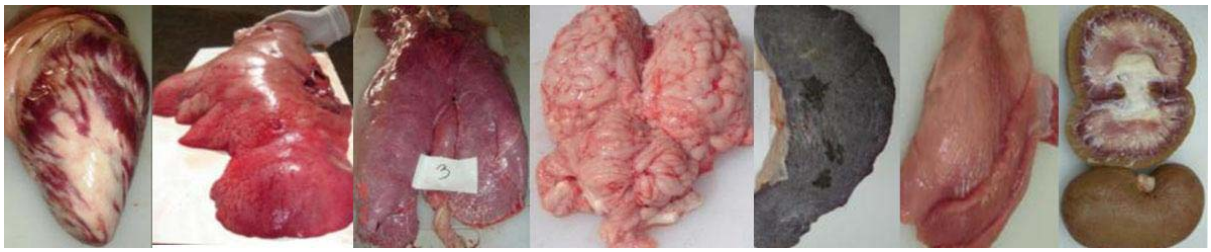
US NAS/NRC (1976) *Selenium,* Washington DC, National Academy of Science, National Research Council, Assembly of Life Sciences, Medical and Biological Effects of Environmental Pollutants, 203 pp.

US NAS/NRC (197l) *Selenium in nutrition,* Washington DC, National Academy of Science, National Research Council, Agricultural Board, Committee on Animal Nutrition, Subcommittee on Selenium, 79 pp.

Annexes 1 - Clinical symptoms

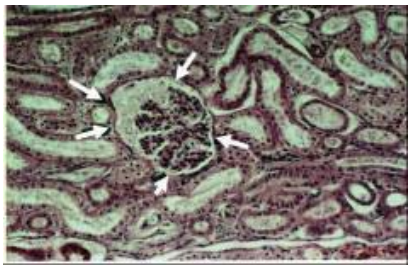
Alopecia | Pad leions | Sternal Position | Diarrhea | Hypertrophy of Cervial lymphnoid

Necropsy findings

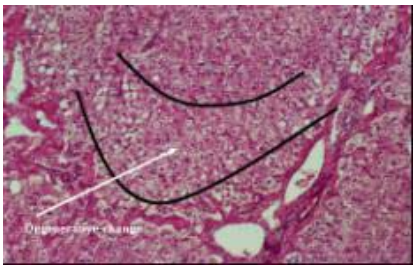


Heart Congestion, Necrosis, Soft Discoloration | Liver Congestion | Pulmunary Congestion | Brain Edema | Spleen Congestion | Kidney Necrosis | Muscle discoloration

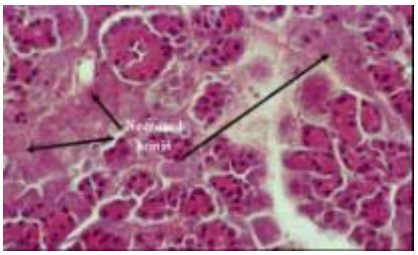
Histopathology findings

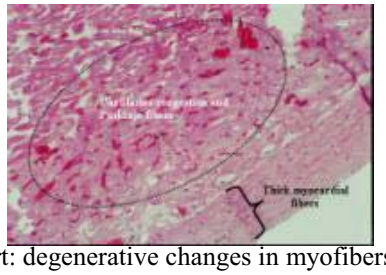


Kidney: degenerative changes in epithelial lining cells



Liver: sinusoids congestion, degenerative changes in periportal zone hepatic cells





Pancreas: necrotic areas and fibrosis Heart: degenerative changes in myofibers