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Clinical Article

CASE REPORT ON ACCIDENTAL SODIUM CHLORIDE POISONING IN SHEEP

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SUMMARY

Nine adult sheep were presented with the history of accidental intake of water with high salt concentration. Based on the history and clinico-haemato-biochemical findings, the affected sheep were diagnosed to be a case of sodium chloride poisoning. The treatment regimen adopted includes fluid therapy without sodium, steroids, diuretics and vitamin B complex daily for five days along with limited water intake on hourly intervals.

Keywords: Sheep, Sodium chloride, Poisoning

The concentration of sodium chloride (NaCl) in the feed or drinking water which meets the physiological demands of the organism without causing toxicity is less than 0.5% (Siroka *et al.,* 2017). Direct salt poisoning in animals is caused by excessive intake of salt (Sodium chloride), whereas, a shortage of drinking water may leads to indirect salt poisoning, and mostly not by a combination of these two factors (Kahn, 2010; Gupta, 2012). Dissolved or mushy forms of salt may be more toxic than solid forms due to highly accessible for absorption (Kahn, 2010). Generally, sodium:potassium ratio in the natural feeds of omnivorous and herbivorous animals are approximately 1:1 and 1:10, respectively. So, a higher dose of salt can be tolerated by herbivorous animals because of less amount of sodium in their natural feed ration (Siroka *et al.,* 2017). Lactating animals are more susceptible to salt poisoning due to the loss of water in the milk. Young animals are more susceptible to salt poisoning because they have a low concentration of plasma proteins, and poultry because they have no taste buds in the beak cavity and lower concentration ability of their kidneys (Svobodova, 2008). In animals and birds, swine and poultry species are reported to be more commonly found affected with salt poisoning, whereas, it is less common in horses and cattle, and it has also been reported in sheep (Sandals, 1978; Radostits *et al.,* 2007; Duarte *et al.,* 2014). Though, the sheep probably belong to the less susceptible species to salt poisoning and their lethal dose (LD) is approximately 6 gm of NaCl/kg of body weight (Buronfosse, 2000; Kahn, 2010), the cases of accidental salt poisoning in this species may occasionally be reported to veterinary practitioners. The present report describe about the clinico-haemato-biochemical findings and therapeutic management of accidental sodium chloride poisoning in sheep.

The present report is about 9 adult sheep presented to the Medicine Section of Veterinary Clinical Complex, College of Veterinary and Animal Science, Rajasthan University of Veterinary and Animal Sciences (RAJUVAS), Bikaner (Rajasthan). Detailed anamnesis revealed that a total of 20 adult sheep accidentally intake water with high sodium chloride concentration (approx. 15 kg common salt/200 lit. of water). Owner further revealed that within 2 hours of intake, affected animals showed abnormal signs and symptoms including anorexia, somnolence, nystagmus, intense thirst, tympany, opisthotonus and absence of urination and defecation. Eleven animals succumbed to death on the evening of the same day and remaining 9 animals were presented for the treatment. Detailed clinical examination of these 9 affected animals revealed dehydration, dull and depressed condition, decreased reflexes, mydriasis and generalized muscle fasciculations (Fig. 1). Recording of vital parameters revealed tachycardia (94-110 beats/min.), respiratory rate (15-25/min.), rectal body temperature (100.7-102.5^{0}F) and normal to slightly congested mucosae. Complete blood count (CBC) values of these affected sheep were found almost in normal reference range and revealed Mean ± SE values of Hb, TEC, TLC, neutrophils, lymphocytes, monocytes and basophils as 7.9 ± 0.32 g/dl, 10.27 ± 0.39 x10^6/µl, 10.80 ± 1.31 x10^3/µl, 50 ± 2.56%, 44.22 ± 2.67%, 5 ± 0.55% and 1.44 ± 0.44%, respectively. Mean ± SE values of serum biochemistry findings revealed hypernatremia (164.77 ± 2.38 mmol/L) with normal values of potassium (4.36 ± 0.16 mmol/L), calcium (11.7 ± 0.15 mg/dL) and phosphorus (5.88 ± 0.25 mg/dL) contents.

Based on the history and clinico-haemato-

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Fig. 1. Clinical presentation of sheep affected with sodium chloride poisoning.

biochemical findings, the affected sheep were diagnosed to be suffered from sodium chloride poisoning and accordingly the treatment was instituted immediately. Each animal was treated by intravenous route with 200 ml of 10% dextrose solution, steroid (dexamethasone) @ 1 mg/kg and diuretic (furosemide) @ 2 mg/kg to reduce the likelihood of cerebral oedema in affected sheep. Vitamin B complex (Thiamine hydrochloride, Pyridoxine hydrochloride and Cyanocobalamin) was administered in standard recommended doses to each animal by intramuscular route to rejuvenate the body metabolism. Broad spectrum antibiotic (Ceftriaxone) @ 5 mg/kg was given intramuscularly to check any secondary bacterial infection during stress conditions in the affected animals. Additionally, water intake was limited @ 0.5% of body weight of affected sheep at hourly intervals until normal hydration is accomplished. Similar line of treatment was recommended for next 4 days also for therapeutic management of salt poisoning in the affected sheep.

The mechanism of the effect of sodium chloride poisoning is based on the ion and osmotic imbalance. Sodium and chloride ions are responsible for the osmotic balance in the body. They are almost completely absorbed in the digestive tract and are distributed into the whole body. Increased blood osmolality causes thirstiness, stimulates water uptake and because it affects the antidiuretic hormone, it causes water retention in the body. This compensation mechanism reduces the osmolality and is effective only when the animal has sufficient water at its disposal. When this compensation mechanism fails, water is drawn out of the cells (intracellular dehydration) by sodium ion concentration into the extracellular space and resulting in swelling. It also crosses the blood brain barrier and in the process, if it is acute, dehydration of neurons and swelling in the intercellular matter take place in the brain, as well as changes in blood circulation of the brain and haemorrhages. In severe cases, neurological symptoms can be observed (Gupta, 2012). Clinical symptoms of direct salt poisoning (excessive intake of sodium chloride) in animals may develop within 1-2 days as in the case of present report, and if the poisoning is caused by water deprivation (indirect poisoning), the symptoms may appear as late as after 4-7 days (Gupta, 2012). Intake of higher amount of salt causes irritation of the mucous membrane of the gastrointestinal tract, and if the irritation is not severe, the symptoms of the poisoning may appear in the form of loss of appetite, regurgitation and diarrhoea. In the cases of rapid intake of very high doses of salt, there will be serious gastroenteritis, electrolyte imbalance, dehydration of more than 10%, and may result in acute death in the affected animals. When the intake of salt is slower, the concentration of sodium in the blood and in the intercellular region gradually increases and other symptoms may develop (Siroka *et al.,* 2017). Clinical signs and symptoms in the form of opisthotonus and convulsions have been described in goats (Buronfosse, 2000), while somnolence, decreased reflexes, mydriasis, nystagmus, tachycardia, intense thirst, tympany, opisthotonus and generalized muscle fasciculations have been reported in sheep affected with salt poisoning (Scarratt *et al.,* 1985; Duarte *et al.,* 2014). Almost similar clinical findings have also been found in the present study conducted in sheep affected with accidental salt poisoning. Sodium chloride poisoning is also reported in various other animal species with varying nature and degree of clinical findings. In pigs, the clinical findings appear in the form of nervous symptoms such as compulsive walking, head pressing and circling. Acute salt poisoning in cattle may be manifested in the form of gastroenteritis, salivation, thirst, dehydration, weakness, ataxia, and convulsions. In some cases, blindness or aggressive behaviour is also reported. In laboratory examination, salt poisoning may be diagnosed if the concentration of sodium in the serum or plasma is high (hypernatraemia) (Svobodova *et al.,* 2008). During post mortem examination, sodium chloride poisoning may be conclusive if sodium concentrations of more than 2000 mg/kg is detected in the brain (Kahn, 2010) and the content of NaCl in the liver is found higher than 3000 mg/kg (Siroka *et al.,* 2017). Therapeutic management of salt poisoning in animals is based on the controlled hydration. The water uptake must be limited and gradual so as not to

cause or intensify brain oedema. Hypernatraemia should be treated preferably within 48 to 72 hours. If monitoring of sodium level in the serum/plasma is not possible, the water uptake should be restricted to 0.5% of live weight at 60 minutes intervals (orally or with a stomach probe) (Svobodova *et al.,* 2008). Intravenous fluid therapy along with correction of hypernatraemia is recommended for small animals. Yet, even when therapy is timely, the mortality may exceed 50% (Kahn, 2010). In the present case study, telephonic conversation with the owner revealed that in spite of every effort made, 3 more animals (out of a total of 9 sheep reported) succumbed to death on third day of treatment. The dead animals could not be necropsied due to reluctance of the owner. Remaining 6 animals recovered uneventfully after 5 days of treatment.

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