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Case Report

Accidental urea poisoning in cattle

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ABSTRACT

This paper presents a case report on urea poisoning in cattle that occurred due to accidental feeding of fertilizer urea to five cattle at Nesikha village, Mewang *gewog* (block), under Thimphu *Dzongkhag* (district) where all five animals died. Due to lack of facilities and also the kits in the field, detailed lab tests could not be conducted except post-mortem examination and histopathology. The diagnosis was made mainly based on the history of the owner. In the animal husbandry practices in Bhutan, urea is used as feed supplement along with the paddy straw after treatment with the certain formulation. Further, the urea is also used in the form of fertilizer for dressing the pasture. In addition, fertilizer urea is also used commonly in agricultural activities including horticulture hence, there is every chance of such accidental poisoning in animals. This case report is aimed at creating awareness to the animal health workers and also guide to attempt treatment in such poisonings in animals.

INTRODUCTION

Urea is used as a source of non-protein nitrogen (NPN) in many feed supplements especially for cattle. In ruminants, nitrogen from urea is released in the rumen as ammonia and is used by rumen microflora for synthesis of protein which then becomes available to the animal through the normal processes of digestion and absorption. Urea should not provide more than 3% of the concentrate ration, or 1% of the total feed intake, and no more than one third of the total nitrogen intake should be NPN. If more quantities of urea are consumed by the animals, the rumen microbes metabolise and ammonia is absorbed from the rumen into the blood. The ammonia is then converted back to urea in the liver, and is then excreted by the kidneys. When excess ammonia and urea circulate in the blood, it causes poisoning, which can occur rapidly from a few minutes to four hours after consumption. Poisoning is common in ruminants when they have accidental access to urea or are fed large amounts of urea; when they are not adapted to it. In addition, poisoning is caused when feeds are improperly mixed with urea or high urea concentration is present in low energy, low protein, and high roughage diets (Ortolani et al. 2000).

ACCIDENTAL POISONING

A case of sudden deaths of five milking cows at Bjemina Nesikha Village, Mewang *gewog* (block), under Thimphu *Dzongkhag* was reported in the evening of 31st May 2014. On visiting the site, it was found that the herd belonged to a private individual Mr. Pema and the history revealed of accidental feeding of fertilizer urea. Urea solution (approximately 1kg dissolved in 10 l water) was prepared and used for spray on apple trees for controlling fungal infection. The left-over solution (about 5 l) was kept in the bucket. The mother of the owner mistook it as water and added about 2 l of this left-over solution to the feed and fed to the cattle at about 5 PM on 31 May 2014. After about an hour, the animals started showing signs of giddiness, circling, regurgitation of feed, restlessness, sweating, muscular tremor, weakness, coma and finally died. It was learnt that contaminated feed was fed to all the five milking animals and all the animals died. Post-mortem examinations revealed fully bloated carcass, congestion of lungs, highly icteric liver, sloughing off of ruminal walls revealing haemorrhagic surfaces, haemorrhages in the small intestine and softening and haemorrhagic kidneys.

On microscope examination, intravascular clotting of blood vessel and degeneration of hepatocytes were observed in the liver. In the lungs, sloughing off of bronchial epithelium with exudates were observed. Congestion and myocarditis were observed in the heart. Haemorrhages and necrosis of tubular cells were observed in the kidneys. Other laboratory analysis like estimation of ammonia in ruminal fluid and blood were not conducted due to lack of reagents and also since proteins in the blood break down rapidly after death and produce ammonia and hence cannot be of any value.

DISCUSSION

The most common source of urea poisoning in cattle and sheep is known to be common via ingestion of toxic levels in concentrate feed, due to an error in urea addition or due to uneven mixing. However, in this case it is accidental feeding of dissolved fertilizer urea. Similar accidental poisoning of goats and cattle with the fertilizer, urea has been reported in Nigeria (Obasaju et al. 1980) where all the fifteen goats and five cattle died. Similarly, Caldow and Wain (1991) reported the deaths of seventeen out of twenty-nine suckler cows due to

contamination of drinking water with urea fertilizer. Three cattle have also died due to Water hauled in tanks previously contaminated with a nitrogen-based fertilizer (Villar et al. 2003). Death of seventeen cattle were also reported in a military dairy fam at Chittagong, Bangladesh due to consumption of molasses contaminated with fertilizer urea (Shaikat et al. 2012).

Main signs of poisoning are known to be due to alkaline-corrosive effects of the high NH_3 levels in the digestive tract and other tissues and the neuro-toxic effect on the brain. The clinical signs mainly observed were of nervous signs like giddiness, circling; similar observations were also reported by Antonelli et al. (2004); Srinivasan et al. (2008) and Shaikat et al. (2012). Animals decompose rapidly after death from urea poisoning and there will be no specific signs of poisoning. Post-mortem examination immediately after death can show evidence of bloat, generalised congestion of the carcase, excess fluid in the pericardial sac, pulmonary oedema with excess stable white foam in the large airways (Antonelli et al. (2004); and haemorrhages on the heart (epi-cardial and endocardial). The post-mortem findings of inflammatory changes in the rumen has also been described earlier (Srinivasan et al. 2008; Parkes et al. 2011; Shaikat et al. 2012). No characteristic lesion has been described on microscope examination by other earlier authors however, fatty degeneration and focal areas of necrosis were observed in the liver. In kidneys, haemorrhages and necrosis of tubular epithelium were also observed.

Proteins in the blood break down rapidly after death and produce ammonia, hence, testing blood from dead animals is of no value. Measurement of blood ammonia levels can be done in live, sick animals. However, in this case, animals died suddenly so no blood samples could be collected. Ammonia levels in rumen fluid can also be measured, but only fluid taken immediately after death can be of some value. The sample must be frozen immediately and kept frozen until tested. The pH of fresh rumen contents is a useful test that can be done in the field. An alkaline rumen (pH greater than 7.5-8) is suggestive of urea poisoning in conjunction to the history.

Prognosis in such case is very grave and often treatment is rarely effective. However, a stomach tube can be passed to relieve the bloat and to drench the animal with a large volume of cold water (about 45 l) for an adult cow is suggested, followed by 2-6 l of 5% acetic acid or vinegar. This dilutes rumen contents, reduces rumen temperature and increases rumen acidity, which all help to slow down the production of ammonia. Treatment may need to be repeated within 24 hours, as relapses can occur. Rumenotomy and removal of rumen contents is suggested for valuable animals.

In conclusion, as a preventive measure, urea should not be given to fasting or malnourished animals, or animals with damaged livers. It should be introduced gradually and increased to a safe level in case of ruminants. Fertilisers and feeds containing urea should be stored, used and disposed of properly. Mixing urea with molasses, fodder/sugar beet or good quality silage reduces the risk of poisoning and urea should be properly mixed with the feeds before feeding. Spillage should be cleaned up or dispersed as it arises. Pasture should be rested for an adequate time before grazing or cutting after Nitrogen application.

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