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Acute Urea Poisoning in Buffaloes: Case Study

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Case Report

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INTRODUCTION

Urea is used not only as a fertilizer on crop and pasture fields but also as alternative non-protein nitrogen (NPN) source in ruminant. It is a cheap protein substance for ruminants. Urea poisoning is one of the common toxicities found in the ruminants especially cattle and buffaloes. Poisoning may occur when ruminants gain access to large quantities of urea accidently or fed in concentrate ration or urea-molasses treated dry roughages. Poisoning may also occur when animals are fed large amounts of urea, when they are not adapted to it or when feeds are improperly mixed or high urea concentration is present in low energy, low protein and high roughage diet [1]. Approximately 0.1 g of urea / kg b. wt. / day i.e., about 40 g urea per day for a 400 kg b. wt. cow) is considered sufficient. It is recommended that urea should be provided not more than 3 per cent of concentrate ration or 1 per cent of the total feed intake [2]. In cattle, 0.3 - 0.5 g / kg b. wt. / day (i.e., 120 - 200 g for a 400 kg b. wt. cow) is considered to be toxic and 1 - 1.5 g / kg b. wt. / day (i.e., 400 - 600 g for a 400 kg b. wt. cow) can be fatal [2,3].

In ruminants, after ingestion, urea undergoes hydrolysis by rumen microbial urease in to ammonia. Ammonia is then combined with carbohydrate derived keto-acids to synthesize protein or amino acids. This protein becomes available to the animal through the normal processes of digestion and absorption. However if more urea is consumed than the rumen microbes can metabolize, the excess ammonia is absorbed from the ruminal wall in to the blood and leads to hyperammonemia. The ammonia is then converted back to urea in the liver and is then excreted by the kidneys [2].

Several cases of urea poisoning have been reported by veterinarians and researchers in cattle, sheep, goat and horses. Present paper reports and study a case of urea poisoning in buffaloes in India.

CASE HISTORY AND OBSERVATION

A non-descript pregnant (approximately 5 months) buffalo, aged six years, was brought to the teaching hospital of the veterinary college, Vallabhnagar, Udaipur, Rajasthan in India with the history of tympany, frothy nasal discharge, respiratory distress and trembling. There was the death of another buffalo (non-descript, non-pregnant, aged 8 years, 3rd lactation) before treatment after showing similar symptoms. Detailed anamnesis revealed suspicion of accidental ingestion of urea fertilizer since the open bag of urea fertilizer and a concentrate mixture were kept in proximity and buffaloes showed the symptoms just after concentrate feeding during milking early in the morning. The clinical signs were observed after 45 min of feeding. Immediately, a field veterinarian was called upon but one buffalo had died and the other was referred to this hospital. The detailed physical and clinical examination was done. The clinical signs observed included restlessness, off feed, off water, suspension of rumination, subnormal rectal temperature (98.5°F), frothy nasal discharge, increased respiratory rate (60 per minute), dyspnoea, coughing, increased heart rate (95 per minute), arrhythmia, muscle tremors, tympany, atony of rumen, fluid filled rumen felt on percussion and palpation, severe abdominal pain, incoordination, shivering of muscles, brick red conjunctiva and pulmonary oedema.

Postmortem Lesions

Post-mortem of the buffalo died before treatment was performed. Post-mortem examination revealed congested liver and kidney, froathy bloat in rumen, marked ammonia smell when rumen was opened, oedema of lungs, white foam in airways i.e.,

trachea and bronchi, excess fluid in pericardial sac and haemorrhages in the heart.

Haemato-biochemical parameters

Examination of ruminal fluid revealed alkaline pH (8.7) with no live microflora present in the ruminal fluid. Haematological examination revealed normal blood profile (PCV – 40%, Hb - 13.8 g/dl, MCHC- 34.5 g/dl, WBC - 9.4 × 10^9/L, granulocytes - 4.1 × 10^9/L (44%) and L/M - 5.3 × 10^9/L (56%). Biochemical examination revealed significantly higher serum creatinine (3.9 mg/dl), blood urea nitrogen (47 mg/dl), ALT (105.3 µ/L), AST (335 µ/L) and serum glucose (121 mg/dl). The serum total bilirubin (0.1 mg/dl), serum amylase (51.3 µ/L), serum total protein (7.1 g/dl), serum albumin (2.8 g/dl) and serum alkaline phosphatase (61 µ/L) were found within normal range. Nothing abnormal was detected on examination of urine (Urobilinogen 0.1 mg/dl), protein negative, pH 6.5, Blood negative, specific gravity 1.015, ketone bodies negative and glucose negative).

Treatment

The case was treated with fluid therapy (Inj. 5% DNS and normal saline) @ 25 ml/kg b.wt. slow i.v. for 3 days with continuous monitoring because of pulmonary oedema, inj. isofluperidone 20 mg i.m. for 3 days, inj. Pheniramine maleate 5 ml i.v. for 3 days, inj. ceftiofur sodium 1 g i.m. for 5 days, inj. vitamin B1, B6, B12 @ 10 ml i.v. for 7 days, inj. Calcium borogluconate 450 ml half i.v. and half s.c. inj. Frusemide @ 10 ml i.m. for 3 days, 5% acetic acid @ 5 L orally, stomachic and live yeast extract orally for 7 days. The animal showed improvement on 3rd day after treatment and complete recovery on 7th day after treatment but there was abortion in the buffalo.

DISCUSSION

Accidental ingestion of large quantities of urea fertilizer causes poisoning in cattle and sheep. Ruminants are most sensitive to urea poisoning [4]. Dietary exposure of unacclimated ruminants to 0.3 - 0.5 g of urea per kg b. wt. may cause adverse effects. Doses of 1 - 1.5 g/kg are usually fatal [3]. Rumen and blood ammonia levels increases dramatically within 20 - 30 min of consumption [5]. Blood ammonia concentration generally causes the toxicity and clinical signs. The onset of clinical signs in the present study was after 45 min of ingestion. Similar finding was reported by [4]. The period from urea ingestion to onset of clinical sign is 20 - 60 min in cattle, 30 - 90 min in sheep and longer in horses [3]. Urea is rapidly hydrolysed upon entry in to the rumen resulting in peak rumen ammonia concentration with in the first hour of consumption. The clinical findings and post-mortem lesions observed in present study are in agreement with that of [2-4] in cattle except subnormal temperature. Kulkarni and Kulkarni reported subnormal body temperature in urea poisoning in a buffalo heifer that is in agreement with present study [6].

Symptoms of urea poisoning in animals are sunken eyes and loss of elasticity of skin due to dehydration, high temperature, labored respiration, muscle tremors or tetany and a fluid filled rumen with pH of 8-9 [7]. Mydriasis occurs in parasympatholytic status or a sympathetic stimulation [5]. The presence of signs such as mydriasis and tachycardia suggests that urea poisoning could elicit a sympathetic or parasympatholytic status Haliburtan and Morgan stated that during ammonia poisoning the parasympathomimetic status predominates and could be responsible for signs such as bradycardia and profuse salivation [8].

Post mortem findings of present investigation are in agreement with that of Shaikat et al. [4]. They also observed gastro-enteritis with haemorrhagic intestine.

In present study, pH of ruminal fluid was 8.7. Ruminal fluid pH became alkaline due to hydrolysis of urea to ammonia [2,4,6] Shaikat et al.; Kulkarni and Kulkarni; and Parkes et al., also reported alkaline rumen fluid pH. The PCV and serum concentration of ammonia, glucose, AST, ALT and BUN usually found increased in urea poisoning [3]. Increase in serum urea nitrogen and serum creatinine level was also reported by Haque and Dey; Kulkarni and Kulkarni [6,9]. Increased blood ammonia concentrations alter hepatic metabolism by increasing ureagenesis may also affect glucose metabolism in the liver and peripheral tissues [10].

Differential diagnosis include poisoning by nitrate or nitrite, cyanide, organophosphates and carbamate pesticides, raw soyabean overload, lead, chlorinated hydrocarbon pesticides and toxic gas, protein engorgement, grain engorgement and pulmonary adenomatosis. It is generally agreed that urea toxicity is equivalent to ammonia poisoning [11].

Ammonia toxicity prevents the release of carbon dioxide from the red blood cells while nitrites prevent the red blood cells from carrying oxygen to blood tissue.

In the present study, acetic acid orally was used as an antidote. Fluid therapy was administered to dilute the toxins in the blood. Antibiotic, antihistamine, and corticosteroid were given to prevent any secondary conditions. Diuretic was used to reduce oedema. Stomachic and live yeast extract were given to improve appetite and digestion. The antidote for a mature cow suffering from ammonia toxicity is oral administration of 4 L vinegar and this may need to be repeated every 20-30 min until the symptoms disappear [12,13]. Use of acetic acid much more than 90 min of the ingestion of urea do not appear to have much effect. Antihistamines, antibiotics and corticosteroids should be given to prevent any secondary conditions [14]. Cold water (45 L) may be administered. It lowers rumen temperature and dilutes reacting media which slows urease activity [2].

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