

Successful Medical Management Of Urea Poisoning In A Cow - A Case Report

K.K. Ponnuswamy*, S. Sivaraman, and R. Ezakial Napolean

Veterinary Clinical Complex, Veterinary College and Research Institute, Salem - 636112, Tamil Nadu, India.

(Received : July, 2022 175/22 Accepted : July, 2022)

Abstract

An adult cross breed jersey cow was presented to the Veterinary Clinical Complex, Veterinary College and Research Institute, Namakkal, Tamil Nadu, India with history of accidental ingestion of urea. Clinical examination revealed twitching of ears, muscular fasciculation, bruxism, frothy salivation, moderate bloat, abdominal pain, frequent urination, rapid breathing, weakness, staggering gait, bellowing and tenesmus. Cow was successfully treated with intra ruminal administration of 5% acetic acid (5 litres) along with supportive for three days.

Key words : Cattle, Urea poisoning, Acetic acid

Urea is common non-protein nitrogen source for improving nutritional qualities of paddy straw for ruminants. Nitrogen from urea utilised by rumen microflora for synthesise protein (Smith and Sherman, 2009). Accidental ingestion of urea in cattle is characterised by excessive production of ammonia in the rumen which may produce gastrointestinal, hepatic, renal, circulatory and nervous disturbances (Radostits *et al.* 2006). The present report discusses the clinical signs, laboratory findings and successful medical management of urea poisoning in a cow.

Case History and Observations

A three year old cross breed jersey cow was presented to the Veterinary Clinical Complex, Veterinary College and Research Institute, Namakkal with the history of accidental ingestion of urea. On clinical examination, the animal had twitching of ears, fasciculation of

facial muscles, bruxism, ptyalism, moderate bloat, weakness, staggering gait, bellowing and tenesmus (Fig 1). All vital signs were normal except rapid breathing with expiratory dyspnea. Ammonia odour was noticed from the breath.

Haematology revealed haemoglobin (8.6 g/dl), total erythrocyte count ($4.8 \times 10^6/\mu\text{L}$), packed cell volume (26%), total leucocyte count ($6.8 \times 10^3/\mu\text{L}$), neutrophils (35%), lymphocytes (59%), monocytes (04%) and eosinophils (2%). The serum biochemical values are calcium (9.61 mg/dl), phosphorous (9.69 mg/dl), potassium (2.46 mEq/L), total protein (5.99 g/dl) and albumin (2.86 g/dl). Rumen fluid was blackish brown in colour, watery in nature, pH was 9.3 and on sedimentation activity test no flotation was noticed. Methylene blue reduction test was more than 20 min; titrable acidity was 8 units and only a few small protozoa were present. Depending upon the history, clinical and laboratory examination the case was diagnosed as urea poisoning.

Treatment and Discussion

Cow was treated with intra ruminal administration of 5 litres of 5 % acetic acid mixed with 20 litres of water using rumen pump along with supportive therapy with intravenous ringers lactate (5litre/day) and multivitamin injection for three days. By the second day, the animal showed improvement in the feed intake with reduction in salivation. Twitching of muscles and bruxism was not recorded and the rumen was resilient on palpation with normal rumen motility (Fig 2). By the third day cow was able to walk. No appreciable variation was recorded on haematological examination at the end of three days. Serum biochemical values recorded were

*Corresponding author : Email : drponnie@gmail.com



Fig 1 : Cow showing the salivation and tenesmus with elevation of tail.

calcium (4.2 mg/dl), phosphorous (4.7 mg/dl), potassium (5.9 mEq/L), total protein (7.3 g/dl) and albumin (3.03 g/dl).

In cases of urea toxicity, break down of urea into two ammonia units leads to dramatic increase in ammonia levels in rumen and blood (Mathew, 1989). Bartley *et al.*, (1976) reported, high levels of blood ammonia causes sunken eyes, dehydration, laboured respiration, muscle tremors at the face and head region which coincides with the present findings. Reduction in the serum calcium and phosphorus levels was observed in the present study and it is due to decreased absorptive capacity of intestine (Gupta *et al.*, 1995). Treatment of the urea toxicity was done in accordance with the Horner (1982).

Conclusion

The history, clinical symptomatology, haematology, serum biochemical findings and treatment of urea poisoning in a cow was reported.

Acknowledgement

The Authors were thankful to the Veterinary College and Research Institute, Namakkal



Fig 2 : Cow recovered from the urea poisoning.

Tamil Nadu Veterinary and Animal Sciences University for providing the facilities.

References

- Bartley, E.E., Davidovich, A.D., Barr, G.W., Griffel, G.W., Dayton, A.D., Deyoe, C.W., and Bechtel, R.M. (1976) Ammonia Toxicity in Cattle. Rumen and Blood Changes Associated with Toxicity and Treatment Methods. *J. Anim. Sciences*. **43**:835-841.
- Gupta, G.C., Pacauri, S.P., and Rajora, V.S. (1995) Studies on post-parturient anorexia syndrome in bovines. *Ind. J. Vet. Med.* **15**: 67-70.
- Horner, R. F. (1982) Suspected Ammonium Fertilizer Poisoning in Cattle. *Vet. Record*. **110**: 472-474.
- Mathew, I. G. (1989) Large Animal Neurology: a handbook for veterinary clinicians. pp 380, Philadelphia: Lea & Febiger.
- O. M., Blood, D. C., and Gay, G. C. (2006) A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses : Radostits Veterinary Medicine. 9thedn. WB Saunders, Bailliere and Tindall, London.
- Smith, M.C., Sherman, D.M. (2009). Goat medicine : 2nd Edition, Wiley-Blackwell Publishers, Iowa, USA. pp: 381