TRENDS IN AGRICULTURE SCIENCE
ISSN:2583-7850

Popular Article
Domain: Agriculture Science
Vol 5 Issue 4, April 2026, 458-461

Urea Poisoning in Dairy Animals

Nirbhay Bhawsar[1], Khushboo Choudhary [2]
[1]M.V.Sc. Scholar, [2]Ph.D. Scholar, Division of Extension Education
ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, Uttar Pradesh – 243122
*Corresponding author: drnirbhaybhawsar@gmail.com
doi.org/10.5281/TrendsinAgriculture.19998689

Abstract

Urea poisoning is an important metabolic disorder in dairy animals associated with the improper use of non-protein nitrogen (NPN) in ruminant feeding. In Indian agricultural practices, urea is widely used as a low-cost nitrogen supplement, increasing the risk of toxicity when not properly managed. Excess intake leads to rapid ammonia production in the rumen, exceeding microbial utilization and causing hyperammonemia. Mismanagement practices such as over-supplementation, sudden dietary changes and uneven mixing are major contributing factors. Clinically the condition shows rapid onset of neurological and digestive disturbances and may lead to sudden death especially due to delayed diagnosis and lack of awareness under field conditions. This article aims to provide a concise understanding of the causes, pathogenesis, clinical signs, diagnosis and treatment of urea toxicity in dairy animals.

Keywords- Urea toxicity, dairy animals, hyperammonemia, rumen metabolism, non-protein nitrogen (NPN).

Introduction

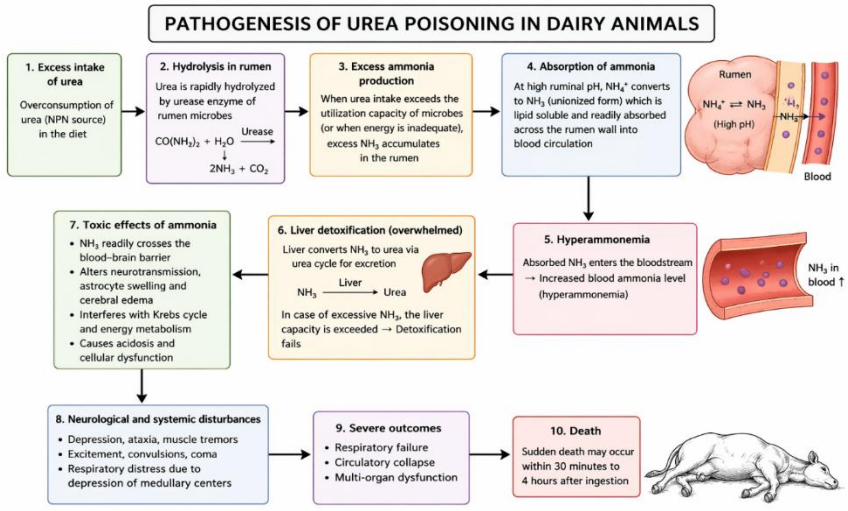
Urea poisoning is an important nutritional disorder in dairy animals caused by excessive intake of urea a widely used non-protein nitrogen (NPN) source in ruminant diets. In the rumen urea is rapidly hydrolyzed to ammonia, which is utilized by rumen microorganisms for microbial protein synthesis and subsequently contributes to the animal's protein requirements after digestion (Merck Veterinary Manual, 2023). However, when urea intake exceeds the capacity of rumen microbes to utilize ammonia, excess ammonia accumulates and is absorbed into the bloodstream leading to hyperammonemia. Although the liver converts ammonia back into urea for excretion this detoxification pathway can become overwhelmed, resulting in systemic toxicity (*Radostits et al.*, 2007). Clinically the condition develops rapidly, usually within 30 minutes to 4 hours after ingestion and is characterized by signs such as excessive salivation, muscle tremors, incoordination, bloat and respiratory distress which may progress to sudden death. Risk factors such as improper mixing of urea in feed, sudden dietary changes and low energy availability further increase the likelihood of poisoning (*Bartley et al.*, 1976).

Causes of Urea Poisoning – Urea poisoning in dairy animals primarily results from errors in feeding

management and improper utilization of non-protein nitrogen supplements. Excessive intake of urea is a major cause often observed in animals with aggressive or selective feeding behavior or when urea is supplied in higher-than-recommended amounts, leading to rapid accumulation of ammonia in the rumen (Department of Industry, Tourism and Trade, 2021). Another critical factor is the sudden introduction of urea into the diet without gradual adaptation, which limits the ability of rumen microorganisms to efficiently assimilate ammonia (*Radostits et al.*, 2007). Irregular or inconsistent consumption patterns further disturb rumen microbial balance and increase susceptibility to toxicity. The use of wet or liquid supplements containing urea can also elevate the risk as dissolved urea may promote higher and faster intake. Additionally, physical separation of urea from feed mixtures during storage or transportation can result in uneven distribution causing some animals to consume toxic concentrations unless the feed is properly remixed before offering (*Bartley et al.*, 1976).

Pathogenesis– The pathogenesis of urea poisoning primarily involves the rapid hydrolysis of urea into ammonia in the rumen by the enzyme urease produced by rumen microorganisms. Under normal conditions, ammonia is efficiently utilized by microbes for the synthesis of microbial protein in the presence of adequate fermentable carbohydrates. However, when excessive urea is ingested or when energy availability is limited, ammonia production exceeds microbial utilization, leading to its accumulation in the rumen. The elevated ruminal pH further facilitates the conversion of ammonium ions to free ammonia, which is readily absorbed across the rumen wall into the bloodstream. This results in hyperammonemia causing toxic effects on the central nervous system. Although the liver attempts to detoxify ammonia by converting it into urea, the capacity of this mechanism is quickly overwhelmed during acute exposure. High blood ammonia levels disrupt cellular metabolism interfere with the Krebs cycle and impair energy production in vital tissues, particularly the brain, leading to neurological disturbances, respiratory distress and ultimately death (*Radostits et al.*, 2007; *Bartley et al.*, 1976).

clinical signs – Urea poisoning in dairy animals is characterized by a rapid onset of clinical signs mainly affecting the nervous and digestive systems. Early signs include twitching of the ears and facial muscles, teeth grinding and excessive frothy salivation due to increased ruminal activity. As the condition progresses, animals may develop ruminal bloat, abdominal pain, frequent urination and rapid laboured breathing due to elevated ammonia levels in the bloodstream. Neurological manifestations such as weakness and incoordination



(staggering) are commonly observed often followed by excitability, violent struggling and bellowing. In advanced stages, animals may collapse and exhibit terminal convulsions or spasms before death. It is frequently observed that affected animals are found dead near the source of urea-containing supplements and multiple animals may be involved due to common exposure indicating a management-related cause (Radostits *et al.*, 2007; Department of Industry, Tourism and Trade, 2021).

Laboratory Diagnosis– Laboratory confirmation of urea poisoning primarily depends on the estimation of ammonia levels, although its diagnostic utility is limited by practical challenges. Blood ammonia can be measured in live affected animals but it becomes unreliable after death due to rapid protein degradation, which artificially elevates ammonia concentration (Radostits *et al.*, 2007). Proper handling of samples is essential, blood should be collected in lithium heparin or EDTA tubes, immediately chilled and plasma separated within 30 minutes of collection. The plasma may be stored at 4°C for a short duration or frozen until analysis. Because of these strict requirements, blood ammonia estimation is often impractical in field conditions. For better interpretation samples from both affected and apparently healthy animals should be collected and processed similarly to exclude artefacts related to storage (*Smith and Sherman*, 2009). Post-mortem estimation of ammonia in rumen fluid can provide supportive evidence if samples are promptly frozen. Among diagnostic samples aqueous humour collected from the eye of a recently dead animal is considered the most reliable, as it remains relatively stable and is less influenced by post-mortem changes making it valuable for confirming urea toxicity.

Treatment-

Immediate dilution and cooling of rumen contents - Early administration of large volumes of cold water orally helps dilute ruminal ammonia concentration and lowers rumen temperature, thereby reducing urease activity and slowing further ammonia production (*Radostits et al.*, 2007).

Oral administration of weak acids (antidote therapy) - Acetic acid in the form of vinegar (approximately 4 L in adult cattle) is commonly used to acidify the rumen, converting toxic ammonia (NH_3) into less absorbable ammonium ions (NH_4^+); treatment is most effective when given early and may be repeated at short intervals (*Bartley et al.*, 1976).

Relief of ruminal bloat - Passing a stomach tube helps to release accumulated gases and also facilitates

the administration of fluids, reducing pressure and improving respiration (Smith and Sherman, 2009).

Fluid therapy - Intravenous fluids are administered to dilute circulating toxins, correct dehydration and support metabolic balance during hyperammonemia (Merck Veterinary Manual, 2023).

Rumenotomy in severe cases - In valuable animals or advanced toxicity, surgical removal of rumen contents may be performed to eliminate the source of ammonia production (*Radostits et al.*, 2007).

Use of supportive medications - Antihistamines, corticosteroids, and broad-spectrum antibiotics may be given to control secondary complications such as inflammation, shock or infections (Smith and Sherman, 2009).

Diuretics for systemic support - Diuretics may be used to enhance renal excretion of toxins and help reduce edema associated with systemic toxicity.

Rumen stimulants and probiotics - Administration of stomachics and live yeast preparations supports restoration of normal rumen microflora and improves feed intake and digestion during recovery (Merck Veterinary Manual, 2023).

Conclusion

Urea poisoning in dairy animals remains an important yet preventable nutritional disorder that can lead to severe economic losses due to sudden morbidity and mortality. The condition primarily results from imbalanced feeding practices and lack of proper adaptation to urea supplementation. Early recognition of clinical signs and prompt therapeutic intervention are critical for improving survival outcomes. However, due to the rapid progression of toxicity, prevention through proper ration formulation, controlled urea supplementation and adequate energy supply is far more effective than treatment. Adoption of scientific feeding management and awareness among livestock owners can significantly reduce the incidence of urea poisoning and ensure sustainable dairy production.

References-

Allison, M. J. (1970). Nitrogen metabolism of ruminal microorganisms. *Journal of Animal Science, 30*(6), 1049–1057.

Bartley, E. E., Davidovich, A., Barr, G. W., Griffin, J. L., Dayton, A. D., Deyoe, C. W., & Bechtle, R. M. (1976). Ammonia toxicity in cattle. *Journal of Animal Science, 43*(4), 835–841.

Church, D. C. (1988). *The ruminant animal: Digestive physiology and nutrition*. Prentice Hall.

Department of Industry, Tourism and Trade. (2021). *Urea poisoning in cattle (Agnote K46)*. Northern Territory Government.

Hungate, R. E. (1966). *The rumen and its microbes*. Academic Press.

Leng, R. A. (1990). Factors affecting the utilization of 'poor-quality' forages by ruminants particularly under tropical conditions. *Nutrition Research Reviews, 3*(1), 277–303.

McDonald, P., Edwards, R. A., Greenhalgh, J. F. D., Morgan, C. A., Sinclair, L. A., & Wilkinson, R. G. (2011). *Animal nutrition* (7th ed.). Pearson.

Merck Veterinary Manual. (2023). Nonprotein nitrogen poisoning in animals. Retrieved from https://www.merckvetmanual.com

Radostits, O. M., Gay, C. C., Hinchcliff, K. W., & Constable, P. D. (2007). *Veterinary medicine: A textbook of the diseases of cattle, sheep, pigs, goats and horses* (10th ed.). Saunders Elsevier.

Smith, B. P., & Sherman, D. M. (2009). *Goat medicine* (2nd ed.). Wiley-Blackwell.

Van Soest, P. J. (1994). *Nutritional ecology of the ruminant* (2nd ed.). Cornell University Press.