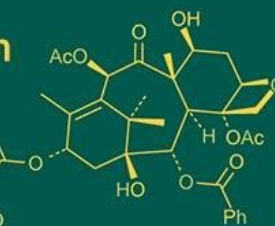
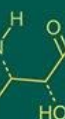
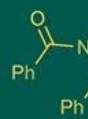


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2025): 5.29
IJABR 2025; 9(9): 508-511
www.biochemjournal.com
Received: 21-07-2025
Accepted: 25-08-2025

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Therapeutic management of atrazine poisoning associated with nervous form of ketosis in a crossbred cow

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DOI: <https://www.doi.org/10.33545/26174693.2025.v9.i9g.5682>

Abstract

Atrazine is a triazine herbicide widely used in agriculture, and accidental ingestion by cattle may lead to toxicosis characterized by gastrointestinal, hepatic, renal and neurological dysfunctions. Nervous form of ketosis, is a metabolic disorder of high yielding dairy cattle in early lactation and it is characterized by abnormal behaviour with certain degree of neurological signs. This report describes a unique case of atrazine poisoning associated with nervous form of ketosis in a crossbred cow, its clinical presentation, diagnosis and successful therapeutic management. Prompt diagnosis and an integrated approach involving detoxification, hepatoprotective agents, energy supplementation and supportive care resulted in complete recovery.

Keywords: Atrazine poisoning, nervous ketosis, crossbred cow, therapeutic management, case report

Introduction

Atrazine, a selective triazine herbicide which is commonly used in crop cultivation has a potential hazard for accidental ingestion by cattle make it a toxicological concern [1]. Clinical signs of atrazine poisoning include anorexia, ruminal atony, depression, hepatotoxicity, nephrotoxicity and nervous manifestations [2].

Ketosis is a frequent metabolic disorder in high-producing dairy cows, especially during early lactation, when energy demand exceeds dietary intake. The nervous form of ketosis, though less common, presents with dramatic behavioural changes such as excessive licking, circling, head pressing, aggression and bellowing [3, 4].

Ketosis in this cow occurred as a sequelae to atrazine poisoning, thereby complicating the clinical picture and posing significant diagnostic and therapeutic challenges. This case report documents the progression from atrazine intoxication to nervous ketosis in a crossbred cow and outlines the clinical findings and successful management approach.

Case history

A five-year-old crossbred Jersey cow in its second lactation was presented to the Veterinary Clinical Complex, Veterinary College and Research Institute, Salem, with a history of accidental ingestion of atrazine-mixed water intended for field use. The cow had been administered activated charcoal by the owner prior to arrival at the clinic. At the first presentation, the animal exhibited acute toxicosis, characterized by elevated rectal temperature (39.3 °C), slightly congested mucous membranes, hypersalivation, dilated pupils, muscle tremors, twitching and bradycardia (Fig. 1, Fig. 2). These clinical manifestations reflected systemic involvement, particularly cholinergic overstimulation and neuromuscular disturbances caused by the toxic insult. The animal appeared reluctant to move and depressed, with intermittent straining during defecation were observed. The animal was treated as per guidelines and sent to farm.

On the second following day, the cow was showing abnormal clinical signs such as vigorous licking of its own skin and inanimate objects, excessive salivation, head pressing against solid surfaces, circling movements and marked restlessness. On Clinical examination the animal was dull, depressed and was reluctant to move.

The rectal temperature was 38.7 °C, while the respiratory rate (24 breaths per minute) and pulse rate (70 beats per minute) remained within normal physiological limits. The mucous membranes were pink and moist, indicating adequate perfusion. Ruminal movements were severely reduced, with only one weak contraction per minute. Rectal examination revealed scanty, dry faeces coated with mucus, which indicated reduced gut motility and dehydration.



Fig. 1. Atrazine-poisoned cow exhibiting systemic signs of intoxication muscle tremors and hypersalivation.



Fig. 2. Dilated pupils observed during clinical examination of the affected cow.

Diagnosis and Treatment

On the first day of presentation, when the cow showed acute signs of atrazine intoxication such as hypersalivation, dilated pupils, muscle tremors and bradycardia, immediate therapeutic measures were undertaken. The animal was administered atropine sulphate intravenously at 0.1 mg/kg body weight to alleviate cholinergic effects along with intramuscular chlorpheniramine maleate at 0.2 mg/kg as an antihistaminic to reduce hypersensitivity reactions. Approximately five litres of intravenous fluids comprising normal saline and Ringer's lactate were infused to correct dehydration, maintain renal perfusion and hasten toxin clearance. About 6-7 litres of rumen contents were extracted using a rumen pump to reduce toxic load and rumen protectants, including activated charcoal with an osmotic laxative, were given orally to adsorb residual atrazine and promote elimination (Fig. 3, Fig. 4). Supportive therapy was continued at the field level, with the owner advised to maintain oral rumen protectants and fluid therapy on the following day.



Fig. 3. Evacuation of rumen contents through a suction apparatus

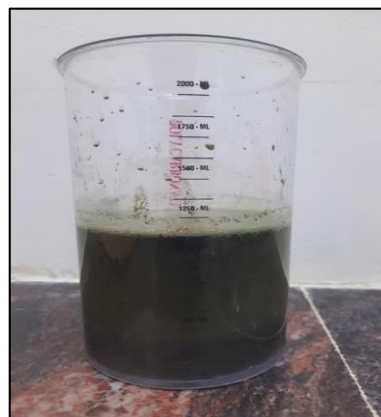


Fig 4: Rumen contents removed using a rumen suction pump.



Fig 5: Cow showing nervous form of ketosis with abnormal licking and circling

By the second following day, the animal developed abnormal behavior (Fig. 5) and neurological signs consistent with the nervous form of ketosis, including excessive licking, head pressing, circling, and restlessness, along with biochemical evidence of hypoglycemia and elevated ketone bodies. Despite these behavioural and gastrointestinal abnormalities, no significant changes were detected in haematological analysis, and blood smear examination ruled out haemoprotozoan infections. The presence of ketone bodies were confirmed using Keto-Diastix® (Bayer)-urine test strip revealed a strong positive reaction (Fig. 6, 7), indicated by a distinct deep purple colour which was around 160 mg/dl, on the reagent pad signifying a high concentration of ketone bodies in the urine.

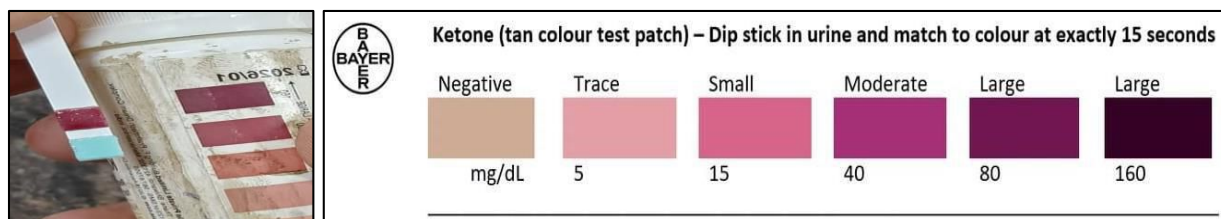


Fig 6, 7: Keto-Diastix® urine strip test.

Therapeutic management at this stage focused on correcting the negative energy balance and restoring glucose homeostasis. The cow received 1000 ml of 25% dextrose intravenously for two consecutive days, together with gel ketonil (Neospark) at 200 ml twice daily for three days as a sustained glucose precursor. A single intramuscular injection of prednisolone acetate (MSD animal health) at 0.5 mg/kg was administered intramuscularly to stimulate gluconeogenesis and parenteral injection of B-complex vitamins with liver extract were administered. Rumen transfaunation advised for 3 days. The animal showed gradual clinical improvement in feeding (Fig. 8).



Fig 8: Complete clinical recovery following therapeutic management.

Discussion

Atrazine, a widely used triazine herbicide, has been reported to adversely affect rumen microbial ecology when ingested accidentally by ruminants [6, 7]. In the present case, a crossbred Jersey cow developed acute toxicosis following ingestion of atrazine-mixed water, with systemic signs including hypersalivation, muscle tremors, bradycardia and dilated pupils. These manifestations were consistent with cholinergic overstimulation and systemic toxic effects of atrazine, which were managed successfully with atropine, rumen decontamination and fluid therapy.

Beyond its systemic toxicity, atrazine exerts a profound effect on rumen microbial populations. Rumen protozoa, which play a pivotal role in carbohydrate fermentation and fiber digestion, are highly sensitive to agrochemicals such as herbicides. Studies have shown that atrazine disrupts protozoal activity, reducing their numbers and impairing their fermentative capacity. Since protozoa contribute significantly to the breakdown of complex polysaccharides and the production of volatile fatty acids (VFAs), particularly acetate, propionate, and butyrate, their suppression results in a marked reduction in ruminal fermentation efficiency.

Among the VFAs, propionate is the principal gluconeogenic precursor in ruminants [8], serving as the primary substrate for hepatic glucose synthesis. In this case, atrazine-induced

suppression of protozoa and ruminal dysfunction (as evidenced by sluggish motility and reduced faecal output) likely led to diminished VFA production, particularly propionate [6]. This disruption would have contributed to decreased gluconeogenic flux in the liver, compounding the already stressed metabolic state of the high-yielding lactating cow.

The combined effect of reduced feed intake due to ruminal stasis and impaired VFA production led to a significant negative energy balance. With inadequate glucose supply from propionate and reduced hepatic gluconeogenesis (further impaired by atrazine-induced hepatocellular damage and oxidative stress), the animal developed hypoglycemia. To compensate, adipose tissue lipolysis was initiated, releasing large amounts of non-esterified fatty acids (NEFA) into circulation. Under normal conditions, NEFA are oxidized in the liver or esterified; however, hepatic impairment and reduced oxaloacetate availability (due to limited propionate) prevented complete oxidation. This led to the diversion of acetyl-CoA into ketogenesis, resulting in marked ketonemia [9, 10].

Clinically, the cow presented with abnormal behaviour such as excessive licking, circling, head pressing, and restlessness, consistent with the nervous form of ketosis [11]. The strong purple reaction on the Keto-Diastix® test confirmed a high concentration of urinary ketone bodies, while serum biochemistry revealed hypoglycemia and normal protein parameters, aligning with both toxic and metabolic derangements.

Therapeutic intervention was directed at both the toxic and metabolic components. Atrazine toxicosis was managed through rumen evacuation, activated charcoal, osmotic laxatives, atropine and supportive fluid therapy, which likely reduced the systemic herbicide load. Nervous ketosis was treated with intravenous dextrose, oral ketonil gel and prednisolone acetate to stimulate gluconeogenesis and improve appetite. Silymarin and B-complex vitamins provided hepatic and metabolic support.

This case demonstrates the intricate link between rumen microbial disruption by atrazine, reduced VFA production, impaired gluconeogenesis and the subsequent development of ketosis. The nervous form of ketosis, although rare, is particularly striking due to its neurological manifestations, which may mimic other toxic or infectious encephalopathies. Therefore, veterinarians should consider toxico-metabolic interactions when confronted with such complex presentations. Rapid cow-side tests like Keto-Diastix remain invaluable in confirming ketosis in field conditions.

The outcome highlights the importance of early recognition, detoxification and appropriate therapy. It also emphasizes the broader significance of herbicide safety in preventing inadvertent livestock exposure, as disturbances in rumen ecology can cascade into severe metabolic disorders like

ketosis, with potential economic consequences in terms of production losses and treatment costs.

Conclusion

This case illustrates the rare but clinically significant progression of atrazine intoxication to nervous ketosis in a crossbred Jersey cow. Atrazine not only produced acute systemic signs of toxicosis but also disrupted rumen protozoal activity, reducing volatile fatty acid production particularly propionate, the primary gluconeogenic precursor in ruminants. The resulting impairment of gluconeogenesis, coupled with negative energy balance and hepatic dysfunction, precipitated hypoglycemia and severe ketonemia, culminating in the nervous form of ketosis.

Successful management required a multifaceted therapeutic approach, including early detoxification with atropine, rumen evacuation, activated charcoal and fluid therapy, followed by targeted correction of hypoglycemia and ketosis with dextrose, propylene glycol (ketonil gel) and prednisolone, along with B Complex vitamins. Complete clinical recovery within a week emphasized the importance of timely intervention.

This case highlights the need for veterinarians to consider toxic-metabolic interactions when diagnosing complex neurological presentations in dairy cattle. Furthermore, it underscores the necessity of herbicide use awareness and safe handling practices to prevent accidental livestock exposure and associated production losses.

References

1. Gupta RC. Veterinary Toxicology: Basic and Clinical Principles. San Diego: Academic Press; 2012. p. 1438.
2. Hayes WJ, Laws ER. Handbook of Pesticide Toxicology. San Diego: Academic Press; 1991. p. 1576.
3. McArt JAA, Nydam DV, Oetzel GR. Epidemiology of subclinical ketosis in early lactation dairy cattle. *J Dairy Sci.* 2012;95(9):5056-5066.
4. Enjalbert F, Nicot MC, Bayourthe C, Moncoulon R. Ketone bodies in milk and blood of dairy cows: Relationship between concentrations and detection of subclinical ketosis. *J Dairy Sci.* 2001;84(3):583-589.
5. Radostits OM, Gay CC, Hinchcliff KW, Constable PD. Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats. 10th ed. Philadelphia: Saunders; 2007. p. 2156.
6. Hussain MI, Reigosa MJ. Herbicide atrazine affects soil and microbial dynamics: implications for livestock feeding. *Ecotoxicol Environ Saf.* 2011;74(8):2070-2077.
7. Chaudhry AS, Ali H. Rumen protozoa and their role in rumen fermentation. *J Appl Anim Res.* 2012;40(1):1-9.
8. Bergman EN. Energy contributions of volatile fatty acids from the gastrointestinal tract in various species. *Physiol Rev.* 1990;70(2):567-590.
9. Raboisson D, Mounié M, Maigné E. Diseases, reproductive performance, and changes in milk production associated with subclinical ketosis in dairy cows: A meta-analysis and review. *J Dairy Sci.* 2014;97(12):7547-7563.
10. Duffield TF. Subclinical ketosis in lactating dairy cattle. *Vet Clin North Am Food Anim Pract.* 2000;16(2):231-253.
11. Andrews AH. Nervous form of ketosis in cattle. *Vet Rec.* 1982;110(5):95-97.