

Successful Treatment of Clinical Ketosis in Dairy Cattle

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Abstract

Ketosis is a post-parturient metabolic disease encountered in high yielders due to imbalance in production and feeding leading to gluconeogenesis along with formation of abnormally high levels of ketone bodies. The disorder is particularly seen within the first few weeks after calving. It arises due to a negative energy balance when energy demands for milk production exceed dietary energy intake, leading to excessive mobilization of body fat reserves and resulting in elevated ketone body production. The present report describes a clinical case of ketosis in a 5-year-old HF crossbred cow in its second lactation, approximately 3–4 weeks post-calving, maintained at a private dairy farm in Rewa, Madhya Pradesh. The animal exhibited clinical signs, such as progressive anorexia, selective feeding behavior with a refusal to consume concentrate feed, a significant decline in milk yield (from 18–20 liters/day to 12–14 liters/day), reduced ruminal motility, and firm, dry, mucous-coated feces. Clinical examination and laboratory evaluation revealed a positive Rothera's test and hypoglycemia (blood glucose: 30 mg/dL), a definitive diagnosis of clinical ketosis was established. Therapeutic management included administration of intravenous 25% dextrose solution, anabolic steroids, and supportive vitamin-mineral supplementation, resulting in noticeable clinical improvement within two days and restoration of normal appetite and milk yield by the fifth day. This case emphasizes the importance of early diagnosis and a multimodal therapeutic approach in the effective management of ketosis. Furthermore, it highlights the need for proper transition cow nutrition and monitoring during the periparturient period to prevent such metabolic disorders. The findings underscore the economic and productivity implications of ketosis in dairy herds and the value of practical treatment protocols.

Keywords: Ketosis, gluconeogenesis, ketone bodies, HF crossbred cattle, Dairy Cattle

INTRODUCTION

Ketosis is a post-parturient metabolic disease encountered in high yielders due to imbalance in production and feeding. It is caused by abnormally high metabolism of carbohydrates to meet the animal's glucose requirement leading to defective metabolism, gluconeogenesis along with formation of abnormally high levels of ketone bodies. Ketosis is characterized by sudden weight loss, selective feeding of animals as evidenced by eating of roughage but refusal to eat concentrate, drastic reduction in milk yield. Ketosis, also known as acetonemia or ketonemia, is a common metabolic disorder affecting high-producing dairy cows, particularly during early lactation – generally within the first three to six weeks postpartum [1]. This period is marked by an increased energy demand for milk production that often exceeds the cow's dietary energy intake, resulting in a negative energy balance. To meet the body's glucose requirements, especially for lactose synthesis in milk, the cow begins mobilizing stored fat. This fat is broken down into non-esterified fatty acids (NEFAs), which are transported to the liver. When the liver's

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capacity to completely oxidize these fatty acids is overwhelmed, they are converted into ketone bodies, such as acetoacetate, β -hydroxybutyrate (BHBA), and acetone. Accumulation of these ketone bodies in the blood, urine, and milk is the hallmark of ketosis.

Ketosis can manifest in two forms: subclinical and clinical. Subclinical ketosis often goes unnoticed because it lacks obvious symptoms, yet it significantly reduces productivity and reproductive efficiency. Clinical ketosis, by contrast, presents with clear signs including reduced appetite, selective feeding behaviour (preferring roughage and rejecting concentrate), decreased milk production, dullness, and dry, firm feces. In more severe cases, cows may display neurological signs, such as abnormal licking, incoordination, or aggression [2].

The condition has a widespread impact on dairy herd economics. Losses stem from decreased milk yield, increased veterinary costs, delayed breeding, and culling. Certain cows are more susceptible to ketosis, especially those with high milk output, poor body condition management during the dry period, or those affected by concurrent health problems, such as retained placenta or mastitis.

Diagnosing ketosis relies on both clinical signs and laboratory testing. Common diagnostic tools include urine dipstick tests for ketones, blood glucose measurement, and specific tests for ketone bodies like the Rothera's test. Early diagnosis and intervention are essential for minimizing economic losses and preventing progression to more serious metabolic issues.

The present case report describes a classical occurrence of clinical ketosis in a high-yielding crossbred dairy cow during early lactation. It emphasizes the clinical presentation, diagnostic approach, effective therapeutic intervention, and recovery. The case also underscores the need for preventive nutritional management, especially during the transition period, to reduce the incidence of metabolic disorders like ketosis in dairy farms [3].

CASE HISTORY AND DIAGNOSIS

A 5 year old HF crossbred cattle during 3–4 weeks of calving in their 2nd lactation belonging to private dairy farm at Rewa, M.P., was attended with the history of gradual loss of appetite and body condition, refusal of eating concentrate but eating roughage and marked reduction in the milk yield from 18–20 liters to 12–14 liters/day.

Clinical examination of animals revealed normal temperature (101°F), respiratory rate 20–22 /min, heart rate 55–58 /min, conjunctival mucus membrane was normal. Superficial and pre-scapular lymph nodes were normal. Ruminant motility was 2–3 per 3 min. Faeces were firm, dry and mucous coated. The cow lost her body condition gradually and was depressed.

From the history of selective feeding and clinical examination, the case was diagnosed tentatively as ketosis. For laboratory confirmation, urine and blood samples were collected from affected cows. Urine sample was subjected to Rothera's test, which showed positive reaction (+++) for ketone bodies. The blood glucose level of cow was 30 gm/dl which also confirmed the diagnosis [4].

TREATMENT AND RESULT

The cow was treated with Inj. Dextrose 25% 1 liter (dextrose @ 0.5 g/kg b.wt.) for 2 consecutive days as replacement therapy, Inj. Isoflud – 5 ml I/M once, Inj. Pinkojet – 10 ml I/M daily for 4 days. This is a multivitamin and a liver tonic. Syr. Anabolyte @ 150 ml daily for 5 days Anabolyte functions supports metabolic recovery orally and Bolus Wokagest – 2 boli BD orally for 5 days. This formulation typically contains metabolic stimulants that aid in correcting metabolic imbalances. Animals showed improvement from the 2nd day onwards. By the 5th day of treatment, the cow regained its appetite and milk production showed marked improvement. Cooper (2014) and Biswal et al. (2016) [5, 6] reported that 27.2% of dairy cows are affected with clinical ketosis and age wise highest prevalence rate (40.8%) was found in the age group of 5.5–6.5 years. Cao (2017) [7] also reported higher levels of ketone bodies

in the urine of cows suffering from ketosis as confirmed by Rothera's test. Radostits et al. (2010) [8], in their studies, reported hypoglycaemia in cows with ketosis. Trebukhov and Elenschleger (2019) [9] also reported decreased glucose levels in HF cows affected with ketosis. Hypoglycaemia, ketonuria, reduction in milk yield, reduced appetite for concentrates and low levels of hepatic glycogen were reported in ketosis by Xu et al. (2014) [10]. Thus, the above treatments have markedly alleviated the metabolic abnormality caused due to heavy production and excessive loss of carbohydrates leading to low levels of blood sugar [11]. The affected cow was treated using a combination therapy aimed at correcting the negative energy balance, improving hepatic function, and enhancing appetite.

The therapeutic regimen included:

- *Inj. Dextrose 25% (1 liter, IV for 2 consecutive days)*: Intravenous dextrose was administered to provide an immediate source of glucose, addressing the hypoglycemia that is characteristic of clinical ketosis. Dextrose infusion is a widely practiced initial step in managing ketosis as it offers a quick and direct method of elevating blood glucose levels. While its effect is rapid, it is also transient, necessitating additional supportive therapy to maintain glucose homeostasis.
- *Inj. Isoflud (5 ml I/M, once)*: This preparation contains corticosteroids which promote gluconeogenesis by mobilizing protein and fat stores and enhancing liver enzyme activity. Corticosteroids such as dexamethasone or isoflupredone acetate are frequently used in the treatment of ketosis because they stimulate endogenous glucose production and suppress ketogenesis.
- *Inj. Pinkojet (10 ml I/M daily for 4 days)*: This is a multivitamin and a liver tonic containing B-complex vitamins and other cofactors essential for energy metabolism. Vitamin B12 and other components of the B-complex group play a crucial role in carbohydrate metabolism and appetite stimulation.
- *Syr. Anabolyte (150 ml orally daily for 5 days)*: Anabolyte functions supports metabolic recovery. Oral administration of such agents improves nutrient assimilation and overall body condition, helping the animal regain strength and milk production more effectively.
- *Bolus Wokagest (2 boli orally, twice daily for 5 days)*: This formulation typically contains metabolic stimulants that aid in correcting metabolic imbalances, potentially enhancing rumen activity and liver function.

Following the start of this therapeutic regimen, clinical improvement was noted from the second day of treatment. The cow began showing signs of increased appetite and responsiveness. By the fifth day, milk yield improved significantly and the animal's demeanor became more active and alert. This prompt response confirms the efficacy of the combined therapeutic approach in resolving the clinical ketosis.

Alternative therapeutic approaches often incorporate additional or substitute treatments.

- *Propylene Glycol*: Commonly used as a glucose precursor in ketosis management. It provides substrates for hepatic gluconeogenesis when administered orally. It is particularly effective in both subclinical and clinical cases and is often recommended for 3–5 days during treatment.
- *Niacin (Vitamin B3)*: Helps reduce lipolysis in adipose tissue and consequently decreases ketone production. It is often used in preventive strategies, particularly during the transition period.
- *Insulin*: In some protocols, insulin (especially long-acting formulations) is administered to enhance glucose uptake and suppress fat mobilization. It is more commonly reserved for severe or recurring cases.
- *Choline and Methionine Supplements*: These are lipotropic agents that aid in hepatic fat metabolism, reducing the risk of fatty liver which often accompanies or precedes ketosis.
- *Rumen-protected amino acids and trace minerals*: Supplementation with zinc, selenium, and manganese supports overall metabolic and immune function, contributing indirectly to faster recovery [12].

Supportive management practices, such as optimizing the cow's body condition score before calving, ensuring a smooth transition to lactation, and formulating energy-dense rations also play a critical role in preventing ketosis. Regular monitoring of high-risk cows during the periparturient period can lead to early detection and reduce the occurrence of clinical cases.

In this case, the integration of rapid glucose replenishment with supportive anabolic and hepatic therapy facilitated a swift recovery. The positive response underscores the importance of timely intervention and the use of a multimodal treatment strategy that addresses the underlying metabolic disturbances in ketosis. Furthermore, it highlights the need for farmer awareness and veterinary monitoring of cows during early lactation, particularly those with a history of metabolic disease or high milk yield [13].

CONCLUSIONS

Ketosis remains a significant metabolic disorder in high-producing dairy cows, especially during the early stages of lactation when energy demands peak. The present case report illustrates the typical clinical presentation of ketosis, with selective feeding behavior, and reduced milk yield. A positive Rothera's test for diagnosis allowed for timely intervention. The therapeutic approach combining intravenous glucose, corticosteroids, liver tonics, vitamins, and anabolic agents resulted in rapid clinical improvement and restoration of milk production. This case reinforces the importance of early recognition and a comprehensive treatment protocol to reverse the metabolic imbalance and prevent long-term productivity losses. It also emphasizes the necessity of preventive management strategies during the transition period, including optimized nutrition, metabolic monitoring, and cow comfort, to reduce the incidence of ketosis. Educating dairy farmers on the signs of ketosis and encouraging regular veterinary checks can further help in reducing the prevalence and economic burden of this condition. With prompt diagnosis and appropriate treatment, affected animals can recover fully, thereby safeguarding their health, fertility, and contribution to the dairy enterprise.

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