Clinical ketosis in lactating dairy cows and its therapeutic management

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Abstract

Ketosis is a state of hyperketonemia that runs in lactating dairy cows resulting in significant drop in milk yield. This condition arises due to heavy demand for glucose in lactation period resulting in hypoglycemia and acute energy crisis. In this study, 6 cases of lactating cows with clinical ketosis were treated. There was a sharp drop in milk yield and rapid loss of body condition. Urine and milk were positive for ketone bodies detected by Rothera's test. Blood glucose levels were markedly lower. The cows were treated with parenteral 50% dextrose solution to meet energy crisis. Propylene glycol @300gm for three days was orally fed as a gluconeogenic precursor. Cyanocobalamin was administered as a supportive therapy. Absolute clinical recovery was achieved in all cases.

Key Words: Ketosis; Glucose; Lactation; Propylene Glycol

Introduction

Ketosis is one of the most common metabolic disorders of dairy cows occuring during a period of negative energy balance that occurs almost in any high yielding cow at the beginning of lactation (Baird, 1982; Herdt, 2000). This leads to a period of reliance on alternative fuels and body stores to meet the needs of production (Ingvartsen, 2006). If animals are unable to adapt, due to management factors, concurrent disease, or many other factors, the body switches over to fat metabolism forming ketone bodies which are used as fuel for many tissues, sparing glucose for milk production (Ingvartsen, 2006). Blood levels of non-esterified fatty acids (NEFA) and beta hydroxybutyric acid (BHBA) are increased. This is exacerbated by the limited ability of ruminants to absorb glucose directly from the diet and thus these heavily rely on gluconeogenesis especially during early lactation (Herdt and Emery, 1992). Lactation demands exceed the gluconeogenic capacity of liver fuelled by insulin resistance and the pathologic hyperketonemia lands the animal into a clinical case of ketosis (Holtenius and Holtenius, 1996). Ketosis is categorized as clinical and subclinical depending on individual animal's ability to tolerate and process ketone bodies (Duffield, 2000; Herdt, 2000). Clinical ketosis manifests as increase in blood, urine or milk ketone bodies with visible clinical signs like rapid weight loss, inappetence, dry feces, dry hair coat and sharp drop in milk yield (Rodostitis, 2000).

Case History and Diagnosis

At Teaching Veterinary Clinical Complex, Faculty of Veterinary Science and Animal Husbandry, SKUAST-K, six cows, calved 15-30 days back, were presented during two months period with clinical signs pertaining to ketosis. Five cows were crossbred Jersey (jersey x local) and one cow was non-descript local. Four cows were in their forth parity and the rest two in third parity. Milk yield of cows was 8-10 litres/day earlier but now reduced to only 4-6 litres/day. History revealed that the cows were underfed and feeding frequency and energy intake were below the general recommendations. This was also suggested by the socio-economic conditions of the owners who were not able to feed the animals properly. The cows were in negative energy balance. On clinical examination, the cows had acetone smell in breath and fresh milk, dull, depressed and reduced rumen motility apart from obvious clinical signs stated earlier. For diagnostic confirmation, Rothera's test was carried out to detect ketone bodies. For this, urine and milk were collected and subjected to the test. 5ml blood was collected from jugular vein with sterile disposable syringes and transferred to screw cap vials. Serum was collected and assayed for glucose and calcium.

Treatment

Both urine and milk were positive for ketone bodies by Rothera's test (Table.1). On rectal examination, feces were scanty and dry. There was a significant decrease in milk yield of 40-50% in all 6 cases. Blood glucose levels were markedly decreased (21-29 mg/dl) and the cows were hypocalcemic (6.9-7.8 mg/dl) to some extent. On the basis of history, clinical signs and Rothera's test, the cases were diagnosed as clinical ketosis. Treatment was carried out as per standard protocols. Cows were administered intravenous 500 ml of 50% solution of glucose twice on first day. This was followed by an oral drench of propylene glycol @300 g once daily for four days. Single injections of triamcinolone acetonide (glucocorticoid- Inj. Vetalog 5ml intramuscularly) and Vitamin B_{12} (cyanocobalamin) were also administered. After four days of treatment, all cows showed marked improvement in clinical signs. There was resumption of feed intake with normal rumen motility and fecal output returned to normal consistency. Milk yield increased and returned to normal after 8-10 days post treatment in all cows. Urine and milk tested negative for ketone bodies by Rothera's test 3 and 5 days post treatment, respectively.

Table 1: Parameters in Cows with Clinical Ketosis

S. No	Milk Yield (L/day)		Blood Glucose	Blood Calcium	Rothera's Test	
	Before	Ketosis	(mg/dL)	(mg/dL)	Urine	Milk
1.	9	4	25	7.4	++++	+++
2.	8	6	29	7.8	++	+
3.	10	5	21	6.9	+++	++
4.	8	4	24	7.4	+++	++
5.	11	7	24	7.3	+++	++
6.	10	6	26	7.4	+++	+

Among many of the metabolic disorders, ketosis is important as it affects dairy cows leading to heavy economic impacts for milk losses and treatment costs. Not only this, the negative effects of ketosis can carry into lactation and include abomasal displacement, impaired reproductive

performance, sustained low milk output and even culling of the animals (Duffield *et al*., 2009; McArt *et al*., 2012b). Risk factors for ketosis include overcrowding, improper feed and water delivery, previous disease, body condition score, etc. (Dohoo and Martin, 1984; Overton *et al*., 2011). Urine ketone body concentrations are four times higher than blood concentrations (Schultz, 1971). We chose Rothera's test for diagnosis as qualitative tests on urine and milk are good indicators of clinical ketosis (Keneko, 1989). Low blood glucose was suggestive of hypoglycemia which is the fundamental cause of ketosis. Providing glucose, stimulating gluconeogenesis, and decreasing fat breakdown form the standard protocol for ketosis treatment (Herdt and Emery, 1992). Intravenous 50% dextrose solution was given as a prompt treatment measure as it increases the blood glucose levels immediately after administration (Sakai *et al*., 1996). Propylene glycol as an oral drench is given to prevent the recurrence of hypoglycemic condition. Oral propylene glycol has been shown to improve resolution of ketosis (McArt et al., 2011). Propylene glycol is either absorbed directly from rumen, or is converted to propionate both of which stimulate gluconeogenesis and insulin release (Studer *et al*., 1993). A dose of glucocorticoid was also included in therapy. It produces a state of hyperglycemia within 24 hrs due to repartitioning of glucose in the body (Herdt and Emery, 1992) which lasts for 4-6 days. A single dose injection of Cyanocobalamin (vitamin B_{12}) was administered. Cyanocobalamin has an established role in stimulating gluconeogenesis as it stimulates the activity of methylmalonyl-CoA mutase enzyme which increases the activity of Krebs cycle leading to more efficient energy production (Kennedy *et al*., 1990).

It is recommended that the cows be provided proper supplementation of energy dense diets before and after parturition to prevent the animals from slipping in a state of negative energy balance – ketosis.

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