The Pharma Innovation Journal 2023; SP-12(10): 1717-1719

www.ThePharmaJournal.com

The Pharma Innovation

ISSN (E): 2277-7695
ISSN (P): 2349-8242
NAAS Rating: 5.23
TPI 2023; SP-12(10): 1717-1719

www.thepharmajournal.com
Received: 07-07-2023
Accepted: 18-09-2023

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Therapeutic management of nervous form of ketosis in a dairy cow

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Abstract
A three-year-old crossbred Jersey cow in her second lactation was brought to Veterinary clinical complex, Tirunelveli with a history of parturition before one month ago, inappetence, refusal to eat concentrate feed, excessive salivation, and a significant drop in milk production over the past three days. Upon clinical examination, the cow had a woody appearance, salivation, licked herself and objects vigorously, was reluctant to move, and had pink, moist conjunctival mucous membrane. The vital signs, including temperature, heart rate, pulse rate, and respiratory rate, were all within the normal range. A blood sample was taken and analyzed for blood glucose and β-hydroxybutyrate (BHB) levels using a portable BHB Check Plus™ device. The results showed a low blood glucose level of 35 mg/dL and a high BHB level of 4.5 mmol/L. Whole blood, a blood smear, and serum samples were also evaluated. The animal was treated for three days with dose of 25% Dextrose and dexamethasone sodium phosphate injection along with supportive treatment. Clinical signs were completely subsided after three days of therapy. The cow had an uneventful recovery.

Keywords: Bovine, ketosis, beta hydroxy butyrate, dexamethasone, hypoglycemia

Introduction
Ketosis is a common metabolic disease among high-performance dairy cows which typically occurs during the first 6-8 weeks of lactation (Guliński, 2021) [6]. Ketosis is a multifactorial disorder of energy metabolism that leads to hypoglycemia, Ketonemia, Ketonuria and downer cow syndrome in adult cattle worldwide (Wang *et al.*, 2012) [23]. Throughout the gestation period, there is an increased demand for energy, primarily driven by fetal growth, lactogenesis, and early lactation. However, this increased energy requirement coincides with a limited feed intake, resulting in a negative energy balance for the animal (Jorritsma *et al.*, 2003) [7]. An excessive negative energy balance makes animals more susceptible to ketosis (Rukkwamsuk *et al.*, 1999) [15]. This condition results in significant economic losses and, in its less severe, subclinical form, is responsible for reduced milk production, a significant decrease in the solids-not-fat (SNF) content of milk, and the failure of affected animals to return to their normal production levels even after recovery (Andersson *et al.*, 1991; Lean *et al.*, 1994; Radostits *et al.*, 2000 [1, 10, 12]. It is characterized by an increased concentration of ketone bodies (including acetone, acetoacetate, and beta-hydroxybutyrate) in various bodily fluids (Duffield, 2023) [4]. Common clinical signs of ketosis are vague but encompass signs such as loss of appetite, reduced milk production, noticeable decline in the cow's body condition, firm and dry feces, and occasionally, neurological symptoms like walking in circles, straddling, leaning or pushing the head into the stanchion, apparent blindness, aimless wandering, vigorous licking of the skin and objects, depraved appetite, and chewing movements with salivation (Constable *et al.*, 2017; Duffield, 2023) [3, 4]. Ketosis can be diagnosed by measuring of ketone body concentrations using cow side tests of blood, milk, or urine. Among these options, measuring blood beta-hydroxybutyrate (BHB) levels using a cow side meter is considered the most precise on-farm test. Ketosis can be treated with administration of dextrose, corticosteroids, and a vitamin B-complex (Chahar *et al.*, 2018; Sandhya Morwal and Shantanu Kumar Kuldeep, 2019; Savita, 2020) [2, 17, 19].

History and clinical observations
A three years old female crossbred jersey cow in her second lactation, was presented to Veterinary clinical complex, Tirunelveli for treatment with the history of parturition before one month ago, inappetence, Refusal to eat concentrate feed, excessive salivation, and a significant drop in milk production over the past three days.

The clinical examination of a cow revealed that the animal had clinical signs of dullness, depression, woody appearance, reluctant to move, salivation, vigorous licking of skin and inanimate object (Fig 1). The cow had a rectal temperature of 38.7 ℃, a respiration rate of 20 breaths per minute, a pulse rate of 65 beats per minute, pink and moist mucous membranes and very sluggish ruminal movements at one per minute. Rectal examination revealed that dry, mucous-coated, and scanty feces. Vital signs, including temperature, respiration rate, and pulse rate, were found to be within the normal range, which was in concurrence with the findings of Sahinduran *et al.,* 2010 [16] and Tadesse *et al.,* 2012 [20]. In the current study, hematological analysis did not show any significant changes. Additionally, examination of blood smear did not reveal the presence of hemoprotozoan diseases which was in agreement with findings of Reddy *et al.,* 2014 [14]. A cow-side test conducted with the BHB Check Plus™ device (Fig.2) detected hypoglycemia with a glucose level of 35 mg/dL and an increased level of BHB, measuring 4.5 mmol/L (Radostits *et al.*, 2007) [13]. In the serum biochemical analysis, reduced level of glucose at 35 mg/dL was observed. However, normal values were noted for serum albumin at 2.5 g/dL and total protein at 6.7 g/dL. Conversely, there was an elevated level of blood urea nitrogen, measuring 136.16 mg/dL which was in accordance with findings of Reddy *et al.,* 2014 [14]. Based on the anamnesis, clinical observations, and laboratory diagnosis, the case was diagnosed as a nervous form of ketosis.

Treatment and Discussion

Treatment of this affected dairy cow include intravenous administration of 1000 ml of 25% dextrose and 15 ml Metaways®, 12 mg of dexamethasone sodium phosphate intramuscularly and 200ml of Boostasky® once a day for three days. Intravenous administration of glucose leads to transient hyperglycemia, an increase in insulin secretion, a decrease in glucagon secretion, and a reduction in the plasma concentration of nonesterified fatty acids (NEFAs). Hyperglycemia is observed within 24 hours of glucocorticoid administration and seems to arise from a repartitioning of glucose within the body rather than from an increase in gluconeogenesis (Constable *et al.,* 2017) [3]. The nervous symptoms are believed to be induced by the production of isopropyl alcohol, which is a breakdown product of acetoacetic acid in the rumen (Sato, 2009) [18]. However, it's worth noting that the need for glucose by nervous tissue to maintain normal function may also play a role in these cases (Radostits *et al.,* 2007) [13]. Cyanocobalamin, a type of vitamin B12, has been observed to decrease in cows during the period around calving (Kincaid and Socha, 2007) [9]. It has been hypothesized that the administration of vitamin B12 might enhance gluconeogenesis by boosting the activity of methylmalonyl-CoA mutase, an enzyme dependent on vitamin B12 in the tricarboxylic acid cycle (Kennedy *et al.,* 1990) [8]. Vitamin B12, when combined with folate, plays a crucial role in amino acid (AA) synthesis by facilitating the process of remethylation of homocysteine into methionine (Preynat *et al.,* 2009) [11]. Methionine is indispensable for protein synthesis and serves as a supplier of methyl groups, which are vital for numerous pathways involved in milk protein production (Gordon *et al.,*2017) [5]. The animal responded very well to the treatment and made a complete recovery within three days. This treatment in agreement with Upadhyay *et al.,* 2007, Tufani *et al.,* 2011, Savita *et al.,* 2020 [22, 21, 19].

Fig 1: Vigorous licking of Travis by nervous form of ketosis affected cow

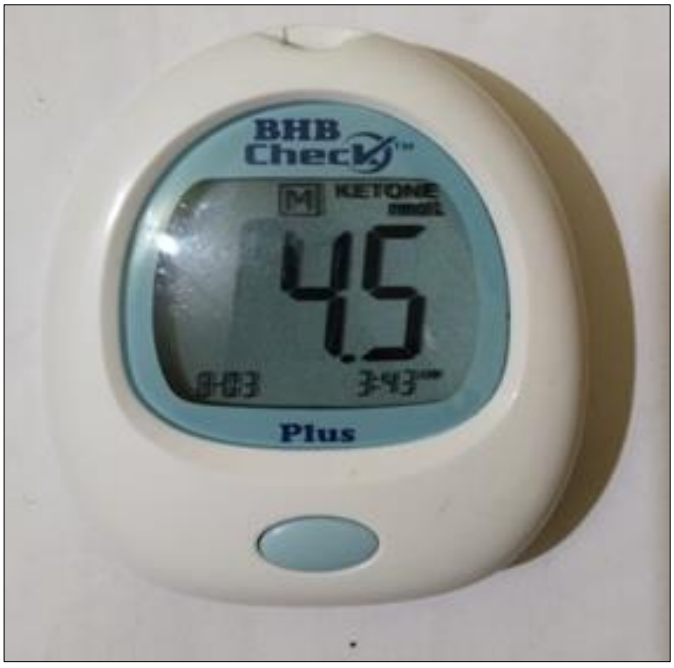

Fig 2: BHB Check plus™ meter showing a elevated level of β-hydroxybutyrate

Conclusion

Clinical ketosis cases in dairy animals can be confirmed in field conditions using portable devices. Nervous form of

ketotic dairy cow exhibited reduced appetite, declined to consume concentrated feed, had increased salivation, and experienced a notable decrease in milk production. Additionally, the cow had a woody appearance, reluctance to move, licked herself and objects vigorously. Timely identification is valuable for averting substantial economic losses for small-scale farmers due to clinical ketosis and ensuring the fast recovery of the affected animals.

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