Bangladesh Journal of Veterinary and Animal Sciences
Journal homepage: www.bjvas.com
pISSN 2227-6416
eISSN 2709-2542

Case Report

Ketosis of an Early Lactating Crossbred Holstein-Friesian dairy cow: A Case Study

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A R T I C L E I N F O

Article history:

Received: 22/09/2020
Accepted: 30/12/2020

Keywords:

Metabolic disorder, retained placenta, ketosis, crossbred dairy cow

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A B S T R A C T

Ketosis is considered one of the economically important metabolic diseases in the dairy industry. This is a case study of 3 years old Holstein Friesian cow weighing 550 kg, presented with a primary complaint of inappetence and decreased milk production. The cow had a history of calving 3 weeks ago. Physical examination findings revealed severe dehydration, anorexia, rough hair coat, ruminal stasis. Moreover, the feces of the cow contained undigested materials. Clinical examination findings were temperature 101.5°F, decreased respiration, and decreased pulse rate. Metritis on rectal palpation was also found. Besides, routine examination of blood revealed lymphocytopenia. In biochemical examinations, we found Calcium 9 mg/dl, Magnesium 3.9 mg/dl, Blood urea nitrogen 36.6 mg/dl, Triglycerides 23.5 mg/dl, Total protein 84.2 mg/dl, Chloride 65.9 mmol/L, Sodium 106.3 mmol/L and a pH of ruminal fluid was 8.2. We may conclude that the high yielding crossbred dairy cow with the history of retained plancenta had been suffering from ruminal alkalosis, negative energy balance, and dehydration after parturition. These findings indicate secondary ketosis, which might be preventable by assessment of serum metabolic profiles around parturition and changes in the management practices accordingly.

To cite this paper: *A. Z. Tanzin, A. K. M. Saifuddin, J. Sultana and SKMA Islam, 2020. Ketosis of an Early Lactating Crossbred Holstein-Friesian dairy cow: A Case Study. Bangladesh Journal of Veterinary and Animal Sciences, 8(2):204-207. .*

1. INTRODUCTION

Peri-parturient period is the most important time when the energy demand is the highest in dairy cows. For example, a dairy cow producing 35L milk/day requires three times more energy for production than for body maintenance (Coppock et al., 1974). The energy requirements of a lactating cow are met through a combination of dietary intake and mobilization of body reserves. The high-yielding dairy cow could not maintain positive energy balance during early lactation and must mobilize body reserves (Coppock et al., 1974), which might affect reproductive performance. Physiological conditions associated with insufficient energy supply predispose dairy cows to various metabolic and microbial diseases such as milk fever, endometritis, ketosis, displaced abomasum, and retain placenta (Duffield 2000). Ketosis, in a dairy animal, may appear as a primary disease or in association with other pathological conditions (secondary ketosis). Economic losses are considerable due to decreased milk production, cost of treatment, and decreased market value of the animal and occasionally from death and disposal of animals. Cows affected with ketosis show a drop-in milk production; they refuse to eat good feed and often pick at poor hay or straw bedding (McSherry et al., 1960). Ruminations are often suppressed or suspended and constipation with hard, mucus-covered feces is frequent. Cows lose weight rapidly and have a typical acetone smell to the breath, urine, and milk. While some cases of ketosis occur in the prepartum period, most cases occur during the first month after calving

(McSherry et al., 1960). Nervous signs are seen in about 10% of cases of primary ketosis (Foster, 1988). Hypoglycemia and ketonemia are constant features in ketosis. Hudson (1928) reported that many cases of ketosis are associated with metritis and concluded that toxins from the uterus were responsible for the development of ketosis. Cows that freshen in the later part of the stable feeding period are frequently affected with subclinical ketosis. These cows have slightly lowered blood sugar levels, slightly elevated blood ketone levels, decreased milk production, and various degrees of hormonal stress. Any disease which then attacks these cows causes further stress and clinical cases of ketosis develop (Hudson, 1928). Furthermore, negative energy balance leads to hypoglycemia and ketonemia as the maintenance of blood glucose is critical in high yielding cows following parturition (during the first two weeks) (Remppis et. al. 2011). In Bangladesh, ketosis mostly occurs in secondary form. As a result, the cases of primary ketosis and nervous ketosis go unnoticed or not been diagnosed or differentiated properly. Hence the present case report aims to provide information about the successful diagnosis and management of a case of ketosis in a crossbreed (Holstein Friesian × Local). The following is an overview of the clinical syndrome of ketosis.

2. CASE STUDY

Presentation

A 3-years old Holstein Friesian crossbred was weighing 550 kg in the first lactation was presented with the complaint of inappetence and decreased milk production. It was fed on stall feeding condition.

History

The cow calved on 1st September 2018. After 10 days, the owner observed some pus-like material in the opening of the genital tract and called upon a veterinary field assistant (VFA) to check the uterus. On rectal palpation the VFA found some remnants of the placenta in the uterus and treated with oviprost® (prostaglandin) (Renata Ltd, Bangladesh) 2 ml i/mly and washed the uterus with Renamycin 100®(Oxytetracycline) (Renata Ltd, Bangladesh) using artificial insemination gun, administrated 250mg/50ml Amotrex (Metronidazole) (ACI Ltd, Bangladesh) i/vly and put a tablet of Amodis® (Metronidazole) (Square Pharma-ceuticals Ltd, Bangladesh) in uterus once daily for 3 days. Then the owner administrated Calphozyme, dextrose 10%, 1 liter, and calcium 200 ml, and dexamethasone. After that, the owner applied the liniment of mustard oil and common salt on the gum. Ten days afterwards, while the situation was not improving, he called a veterinarian, who examined the animal and recorded clinical findings. The day before the presentation the cow showed anorexia and slightly decreased milk production. As per the owner's observation, the milk yield decreased substantially from 21 liters to 10 liters and the cow had a very fast wasting.

Clinical Examination

The temperature was 101.5°F, presence of ruminal stasis, rough hair coat. On rectal palpation, the veterinarian diagnosed endometritis and cervicitis (Sheldon et al., 2004). The animal was defecated and urinated once within 7 days. Primary treatment was given with magnesium sulphate (100gm), vet saline® (half packet) orally, sodi bi carb (sodium bi carbonate 50ml), Cal-c-vet 200 ml (Calcium and Magnesium), and dextrose 10%, 2L was prescribed intravenously once only. Yougart (0.5 kg) was prescribed to increase rumen flora. Also, Lugol's iodine 20ml was applied to wash the uterus and continued for 3 days.

Laboratory Examination

Blood was drawn from the jugular vein with a 10 ml plastic syringe. Blood (3 ml) were immediately allotted into an Ethylene Diamine Tetra Acetic Acid containing tube for complete blood count and 4 ml blood was taken into a vacutainer without anticoagulant for serum preparation. Complete blood counts (Haemo-globin, packed cell volume, erythrocyte sedimentation rate, total erythrocyte count, total leukocyte count, and differential leukocyte count) were performed using haemato-analyzer (Celltacα, MEK-6550, Nihon-Kohden, Japan)®. The serum was obtained after clotting and centrifuged at 3000 rpm for 10 minutes, and serum was kept in the Eppendorf tube and stored at 4°C for further analysis. Rumen liquor was collected from the rumen by using a hypodermic needle (18G) and was taken in a 4 ml vacutainer tube. Then ruminal pH was determined using a benchtop pH meter (WTW®, inoLab, Germany). The microscopic examination of rumen fluid was done at 10X to observe the motility of microflora

following the procedure of coproscopic examination. Then the collected rumen fluid was centrifuged at 3000 rpm for 10 minutes to obtain clear rumen fluid for further biochemical analysis. Both serum and rumen biochemical tests including calcium, creatinine, sodium, potassium, chloride, triglycerides, total protein, glucose, magnesium, phosphorus, blood urea nitrogen(BUN), SGOT(AST) were performed using commercial kits through the biochemical analyzer (Humalyzer 3000®).

3. RESULTS

Physicochemical parameters of rumen fluid and cattle feed

Physical examination of the ruminal fluid showed greyish, alkaline pH (8.2) with no live microflora present in ruminal fluid. Biochemical examination of rumen fluid showed ruminal calcium (14.87 mg/dl), magnesium (20.6 mg/dl), phosphorus (43.7 mg/dl), creatinine (0 mg/dl), potassium (26.4 mmol/L), chloride (57.1 mmol/L), sodium (152 mmol/L). Biochemical examination of rumen fluid was done to know the status of ruminal environment that might influence the maintenance of rumen pH, which is very much important for normal microfloral activity. Proximate analysis of concentrated feed vs German grass revealed following parameters dry matter 93.15% vs 21.86%, moisture 6.85% vs 78.14%, crude protein 16% vs 8.75%, crude fiber 9.65% vs 41.25%, ash 5.75%-6.80%, ether extract 7.75% vs2.20%.

Haemato-biochemical parameters

Routine examination of blood showed PCV-41.6%, Hb-12.1 g/dl, ESR-0 mm 1st hour, TEC -8.8 $x10^6$/cumm, TLC-8.5 $x10^3$/cumm, DLC (Lymphocyte -31%, Neutrophil- 60%, Eosinophil-4%, Monocyte-5%, Basophil-0%), respectively. Serum biochemical examination revealed calcium (9 mg/dl), creatinine (0.8 mg/dl), sodium (106.3 mmol/L), potassium (3.9 mmol/L) chloride (65.9 mmol/L), triglycerides (23.5 mg/dl), total protein (84.2 gm/L), glucose (73.3 mg/dl), magnesium (3.9 mg/dl), phosphorus (6.3mg/dl), blood urea nitrogen (36 mg/dl), and SGOT (91.4U/L).

Treatment

After the primary treatment, the cow started to eat slightly. But milk production didn't increase. Therefore, oral administration via stomach tube of 0.5-liter rumen flora was done 3 times in 2-3 alternative days via stomach tube and dextrose saline was administered intravenously. Follow up treatment was given routinely for 20 days. The condition of the cow was improved slightly.

4. DISCUSSION

In the present study, the cow was found with retained placenta with metritis followed by ketosis. The negative energy balance is the precursor of ketosis. The same observation was recorded by other author (Hudson, 1928) who reported that many cases of ketosis are associated with metritis resulting from toxins produced in the uterus. A routine examination of blood revealed lymphocytopenia (31%) that might be due to renal insufficiency. Biochemical analysis revealed a higher triglyceride (23.5 mg/dl) level, which indicates negative energy balance. This finding corroborated with a previous study (Wotton, 1992) which reported that negative energy balance in early lactation due to increased utilization of glucose for milk lactose synthesis causes lower plasma concentrations of both glucose and insulin as compared with that in later stages of lactation (Hart et al., 1978). In the current study, the BUN level was found higher than the normal range that might be due to retention of urine for 7 days. Similarly, other authors (de Morais, 2017) stated that high levels of BUN may be the result of kidney disease or blockage of the normal flow of urine. There was also an elevated level of total protein (84.2 g/dl) due to severe dehydration which is coincided with the earlier report of de Morais, 2017. Again, there was hypochloremia (65.9 mmol/L) due to intestinal obstruction as there was no defecation for 7 days that leads to the failure of abomasal emptying and obstruction of the proximal part of the small intestine. A similar finding was also reported by de Morais 2017, that failure of abomasal emptying and obstruction of the proximal part of the small intestine will result in the sequestration of large quantities of chloride ion.

Hence the report also revealed that hyponatremia (106.3 mmol/L) is caused due to dietary low supplement of sodium. The glucose level was normal due to the administration of glucose parentally. The pH of rumen was 8.2 which indicates alkalosis and the color of the rumen fluid was grayish. Commonly, primary ketosis is due to primary energy deficiency in high milk

producers and heavy demand of energy in early lactation can cause persistent hypoglycemia with the resultant decrease in insulin production and subsequent lipid mobilization. The resultant hepatic lipidosis leads to an increase in hepatic ketogenesis, hypoglycemia, and hyperketonemia (Wotton, 1992). Furthermore, ketonemia and ketonuria are rather constant and a characteristic feature in ketosis (McSherry et al., 1960). The severity of clinical ketosis in cattle follows rather closely the degree of hypoglycemia (Repke, 1942). In some cases of ketosis, however, normal or even high blood sugar has been reported (Allocraft, 1941). Since typical cases of bovine ketosis are not produced by fasting, it has also been stated that lack of blood sugar per se is not the principal cause (McSherry et al., 1960).

After using dextrose saline glucose level got normal. Magnesium sulphate was given to subside deficiency of magnesium and Lugol'siodine was used as antiseptic to flush the uterus.

The limitations of the present study are only one case study and corresponding urine and milk test weren't done. The non-esterified free fatty acids and ketone bodies were not assessed due to the unavailability of test facilities. Replacement of rumen microflora was insufficient according to the recommended dose (4-5 liters of rumen flora). Although several limitations exist, the strength of the present study is that we assessed rumen fluid and blood to find out the biochemical changes. Finally, we may conclude that secondary ketosis from retained placenta might be recovered successfully with usual therapy of ketosis if done in time along with the replacement of an appropriate amount of ruminal fluid. Further studies are warranted with more sample size, assessment of ketone bodies in urine and feces as well.

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